

WEBINAR

The Neurobiology of Relapse and Models to Support Recovery



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Addiction Policy Forum



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addictionpolicy.org

Ned the Neuron visits The Art of Neuroscience at SfN



H Kathleen Childress Designs from the "Synapse Series"

Brain-Behavioral Vulnerabilities Laboratory

Principal Investigators



Childress



Kampman



Ayaz

Co-Investigators



Regier



Gawrysiak



Hager



Thase

Consultants



Carhart -
Harris



Nicholas

Support Team



Ivey
Project
Manager



Benedict-
Augustine
Coordinator



Glogowska
Coordinator



Burns
Coordinator



Denis
Recruitment

PENN, Drexel, and WCUA



Riffey
CRA



Curtin
Post-doc



Tufanoglu
Engineer



Petro
Tech Support



Hartwell
Psychologist



Mendoza
CRNP

***** The Neurobiology of Relapse and Models to Support Recovery*****

***Unbridled passions:
Visualizing the brain substrates
of cue-induced drug motivation (“GO!”)
and its modulation (“STOP!”)
in addiction***

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Center for Studies of Addiction
3535 Market Street

June 11, 2026

Addiction



First ..let us consider.....



Are
YOU
having
a “GO!”
moment
?



We humans are exquisite reward detectors!



But hmmnnnn....is there a disadvantage, a “dark side” to our reward sensitivity?



**Not a
Reward**

***Yes -- a possible “dark side”
to reward sensitivity....***

***A brain that responds very quickly to reward
signals (even when “unseen” -- without our
awareness) may have greatly helped our early
species survival –***

***....BUT – ironically -- very rapid, almost
automatic, brain responses may NOT help in
the battle against relapse -->> greater reward
sensitivity may be a...relapse vulnerability !!***

*Our research efforts....
driven by our addicted patients' struggles with*



RELAPSE

Outline

Context :

- Two brain systems implicated in **relapse vulnerability**: “GO!” and **STOP!** circuits

Part 1 : Visible Drug Cues

- Can we image the brain response to **visible** drug cues ?
- Can we link the brain response to **relapse**?

Part II : “Unseen” Drug Cues

- Can we image the brain response to 33 msec “unseen” drug cues?
- Can we link the brain response to “unseen” cues to **relapse**?

Part III : Why are some more vulnerable to drug cues ?

- (**Genetics?** Epigenetics / **prior Trauma / Abuse**)?

Part IV : Is there hope? Can we impact the “cue-vulnerable” phenotype with a **medication**? With other new tools?

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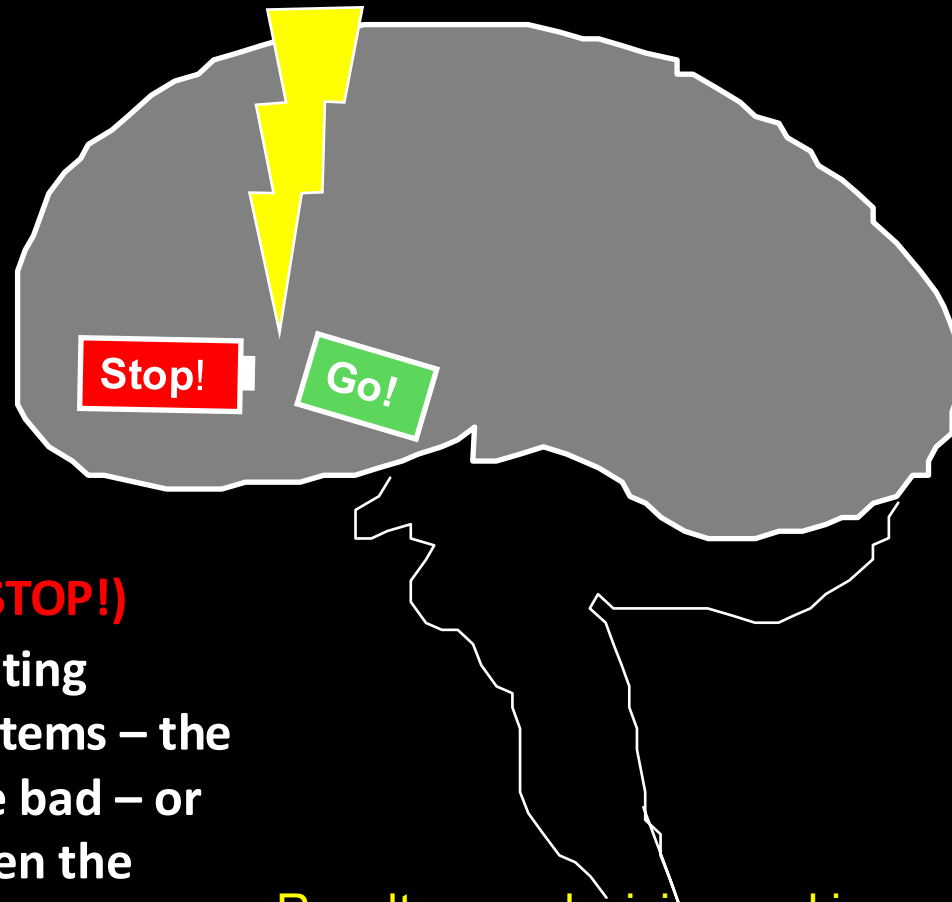
Part III : Why are some more vulnerable to drug cues ?

- (Genetics? Epigenetics / prior Trauma/Abuse)?

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In a vulnerable brain....



..the brain's frontal **(STOP!)** circuitry is not modulating downstream **(GO!)** systems – the “brain brakes” may be bad – or the connection between the brakes and the other regions may be “broken”.

Result: poor decision-making...poor impulse control...greater risk-taking...poor inhibition...an “over-reacting” brain

VULNERABILITY

“GO!”

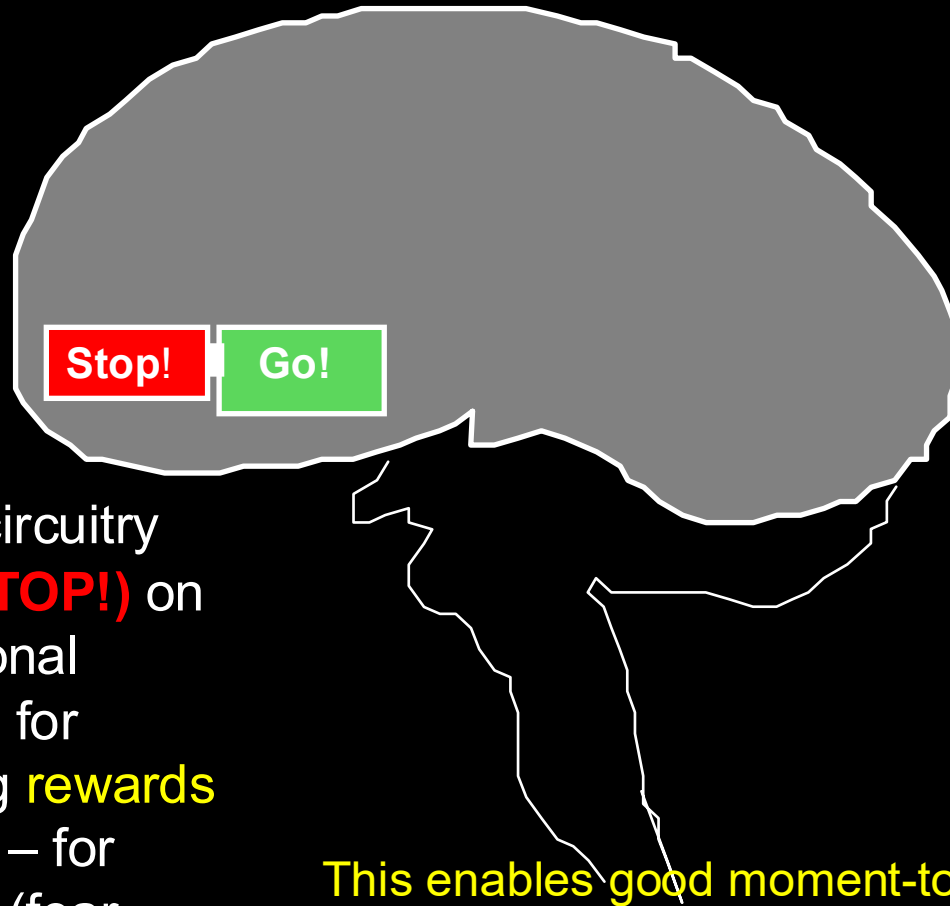
“STOP!”

*** A delicate balance ***





In a normal, adult brain....



...the brain's frontal circuitry acts as a “brake” **(STOP!)** on downstream motivational **(GO!)** systems critical for survival – for pursuing **rewards** such as food and sex – for responding to **danger** (fear and aggression).

This enables good moment-to-moment decision-making...good evaluation of risk...good impulse control.

Vulnerability:
Can “GO!” and “STOP!”
Differences in the Brain
Help Us Explain

- WHY are some more vulnerable –**
- *To relapse?***
 - *To addiction itself?***

VULNERABILITY

“GO!”

*Brain substrates of
cue-induced **drug**
motivation.....*

“STOP!”

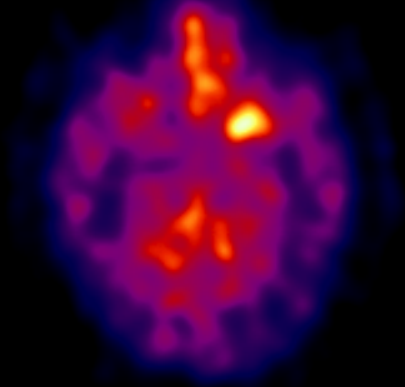
*....and its **regulation**
(or lack thereof :
deficits in frontal
modulatory circuits)*

*(a little Stopping
at the end !)*

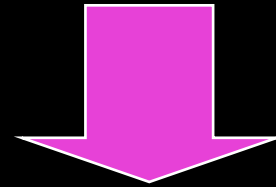
*Brain substrates of cue-induced **drug motivation (GO!”)***



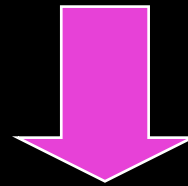
**Our initial focus was conscious
desire....to highly visible cues....**



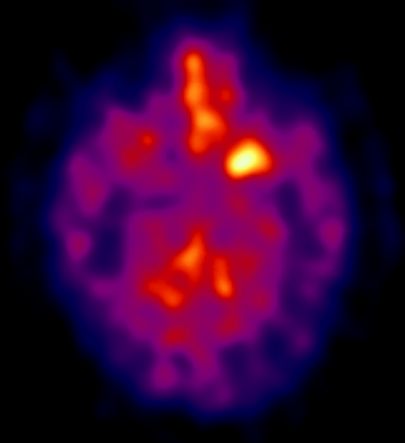
Drug cues



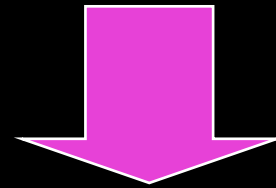
Throbbing, pulsating desire



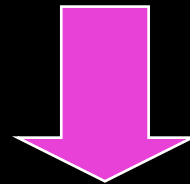
Relapse



Drug cues



“GO!”



Relapse

How Do Drug Cues Come to Trigger Drug Craving?

Drug Cues ---- signal --> Cocaine

Drug Cues



Desire
“Craving”
“GO!”

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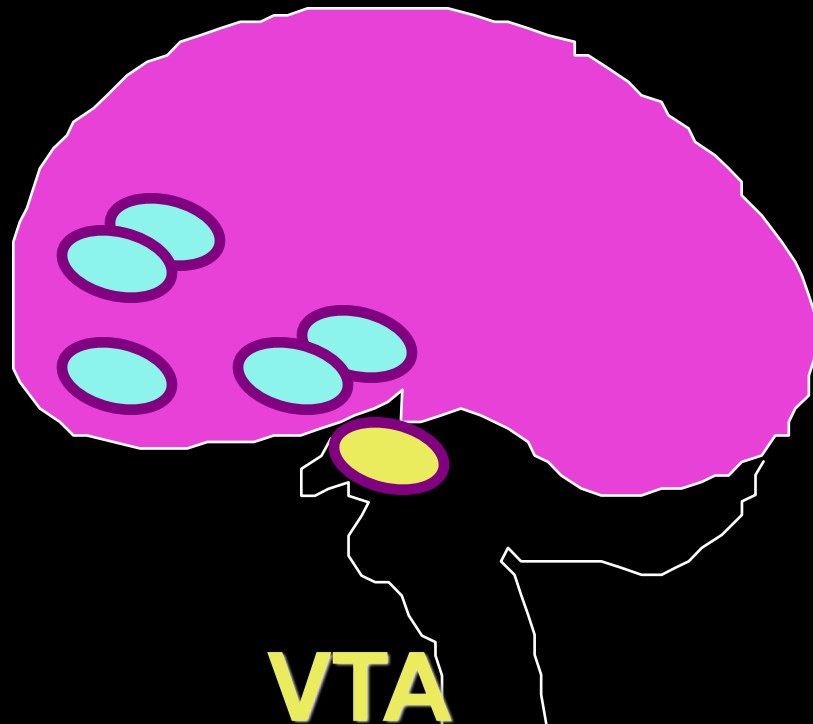
Part IV : Is there hope? Can we protect the “cue-vulnerable” phenotype with a medication?



What are the Brain Substrates of Cue-Induced Desire (GO!) ?

Limbic Structures as Candidate Circuit

Drug Cues Should Activate Mesocorticolimbic Circuitry



V. striatum (NAc) / pallidum amygdala
OFC mPFC insula Ant.cingulate

PET Session Timeline



Scan 1

Scans 2 & 3

Scan 4

Scans 5 & 6

Baseline

Neutral Videos

Resting

Cocaine Videos

0

Minutes

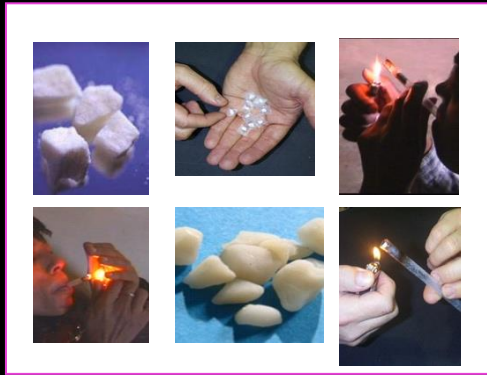
86



**Were we able
to elicit a**

“GO!” state?

***(under these hostile
Laboratory Conditions)?***

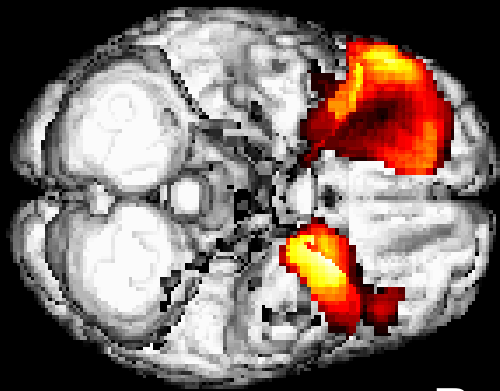


Did we find

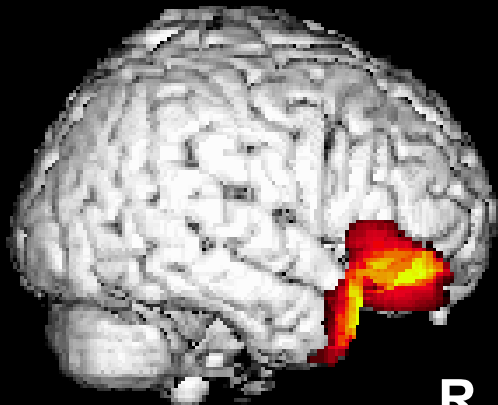
limbic

activation?

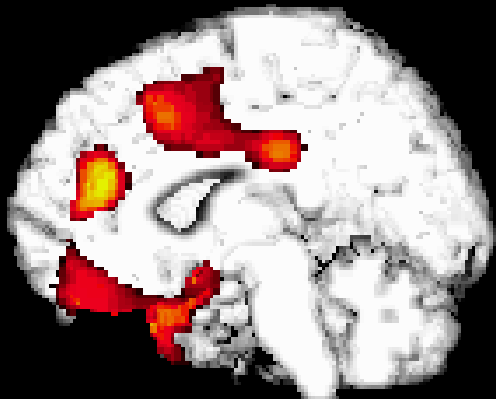
Brain Activation During Craving Triggered By Cocaine Cues



Bottom



R. Side



Middle

Three views of the brain's activity* in cocaine patients viewing a cocaine video which triggered desire for cocaine.

**Statistical parametric map showing brain regions differentially activated by a cocaine video as compared to a non-drug (nature) video.*

*Childress, et al. American Journal of Psychiatry
1999*

Nicotine Cues



Marijuana Cues



Prescription Opioid Cues



Examples of **VISUAL** drug cues used in our current fMRI projects.

Patients view cues reflecting the way(s) their particular problem drug is used in the **Philadelphia region**.

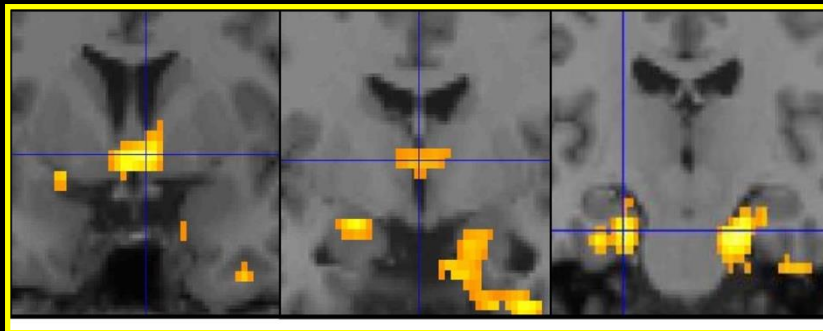
In our early polygraph studies of cue reactivity, we included not only visual, but sound, smell, taste and tactile cues!

Limbic activation

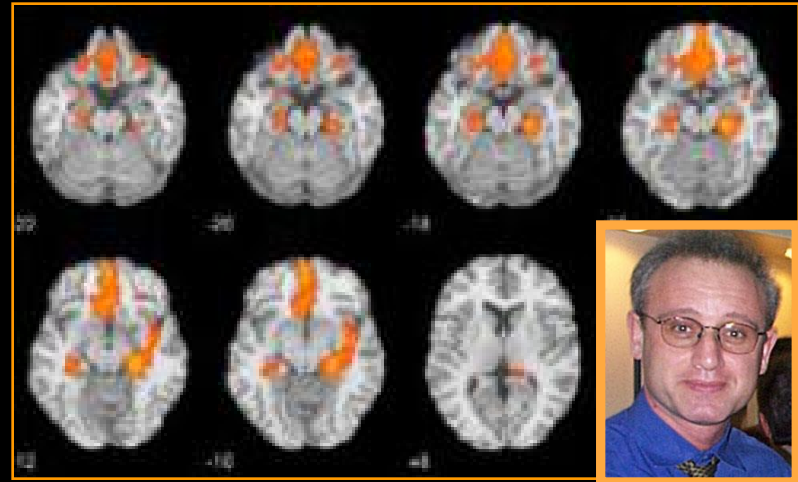
(*amyg, striatum / v. pallidum, insula, OFC / VMPFC, ant. cing.*) by cocaine cues &

opiate, cigarette smoking, and marijuana cues * (*expectancy*)

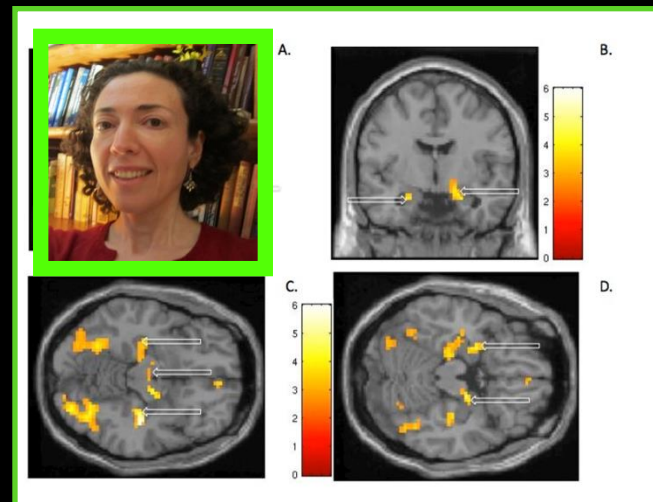
Cigarette cues (independent of nicotine withdrawal)
Teri Franklin, et. al, Neuropsychopharm. 2007



Opiate cues (in stable MM patients, prior to daily dose) *Daniel Langleben, et al. Am. J. Psychiatry, 2007*

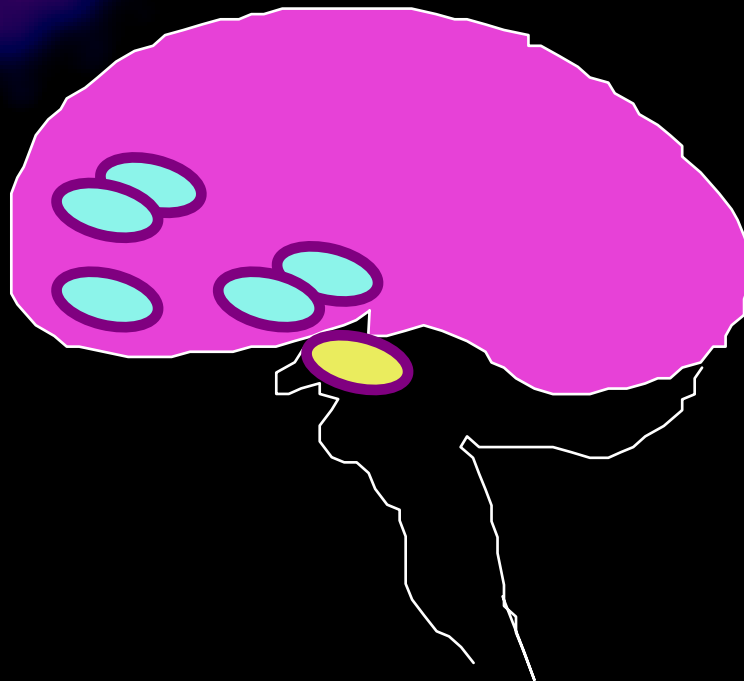


MJ cues *Marina Goldman, et al. 2013*



Desire Substrates

Other Desires?



Nicotine SEX Marijuana
Chocolate COCAINE
Alcohol Opiates

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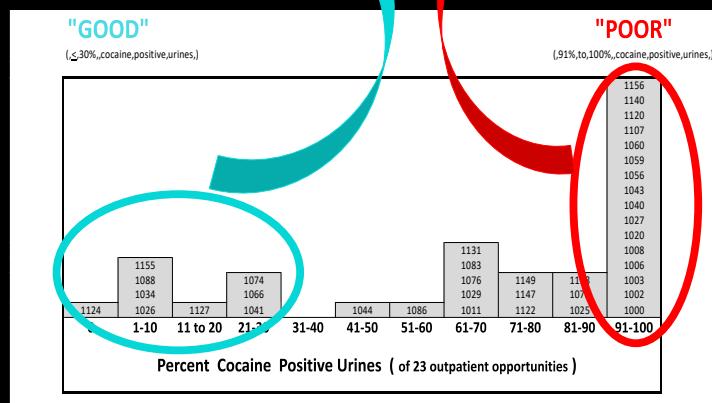
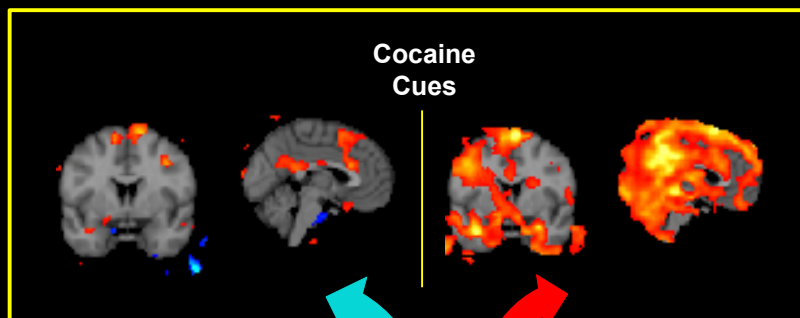
Part IV : Is there hope? Can we protect the “cue-vulnerable” phenotype with a medication?

Cocaine patients with future **POOR** drug use outcomes have a heightened brain response to **6 second Cocaine (v. Neutral)** VIDEO clips – in our Craving Inhibition task.

OUTCOME Phenotype

“GOOD”

“POOR”



Top Figure. Dramatic brain response to 6 second cocaine (v. neutral) video clips for cocaine patients with POOR (n=12) v. GOOD (n=9) outcomes. SPM8 Images (t maps) thresholded at $2 < T < 5$ for display on MNI template.

[Images above based on first half of the 6-second task, to minimize impact of carryover on the neutral referent. Data available for n=9 from “GOOD”; n=12 from the “POOR” subgroup].

Bottom Figure. Histogram of two phenotypic extremes chosen to examine the brain response to 6 sec cocaine videos: **“GOOD”**, $\leq 30\%$ cocaine positive/missing urines during 12 outpatient weeks, and **“POOR”** ($> 90\%$ cocaine positive or missing urines during the 12 outpatient weeks).

Yes –

We **can** link the brain response to (visible) cocaine cues to **relapse**.

Individuals who will proceed to **“POOR”** urine outcomes ($> 90\%$ cocaine-positive or missing) have a heightened brain response to cocaine cues...whereas those proceeding to **“GOOD”** outcome have a low response.

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Struggling against an “unseen” foe

Cocaine patients often describe conscious craving as **volcanic -- erupting rapidly -- seemingly “out of nowhere”**:

“...I don’t know what happened -- one second I was OK -- and the next -- I was on a mission -- out the door, looking for cocaine...”

By the time our patients can “label”
the state triggered by exposure to drug
cues...they are already in big trouble!



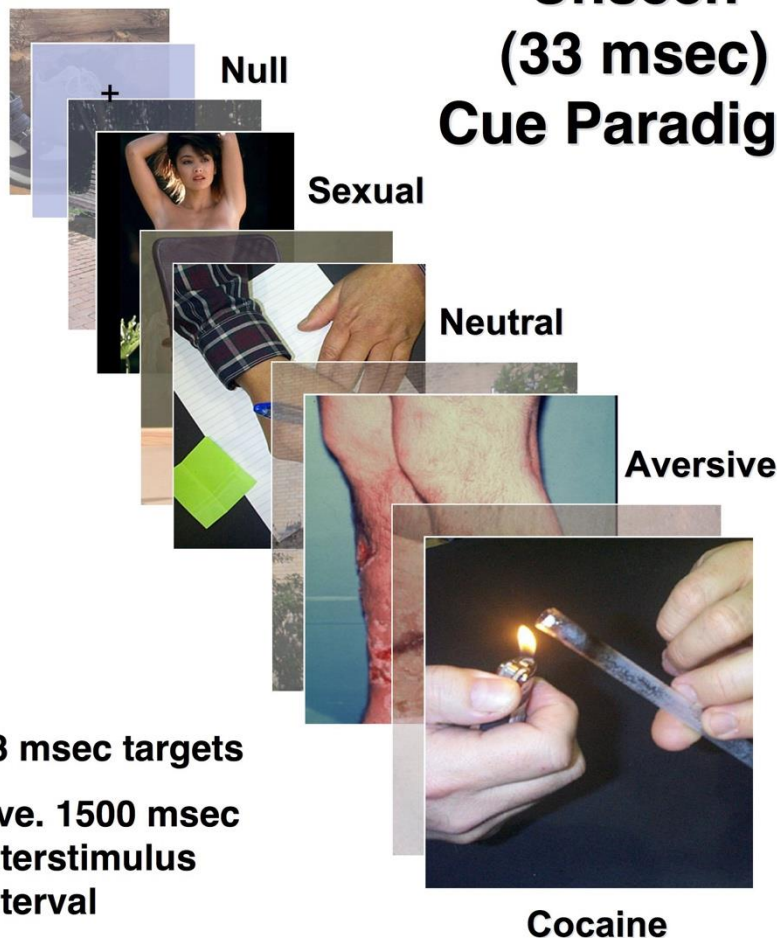
Does the “trouble” -- the brain response
to drug cues -- begin instantly.....even
outside awareness?

Struggling against the “unseen” foe

To test this, we used:

Exceedingly Brief
(33 msec-
backward-masked)
“Unseen” Cues...

“Unseen” (33 msec) Cue Paradigm



“Unseen” cue paradigm. 24 randomly-presented 33 msec targets in each of four categories (cocaine, sexual, aversive and neutral, interspersed with grey-screen nulls) were immediately followed by a 467 msec neutral “masking” stimulus”. Under these conditions, the **33 msec stimuli escape conscious detection**; only the 467 msec masks are later recognized at levels above chance. (From *PLoS ONE*, 2008).

“Unseen” Cue Paradigm

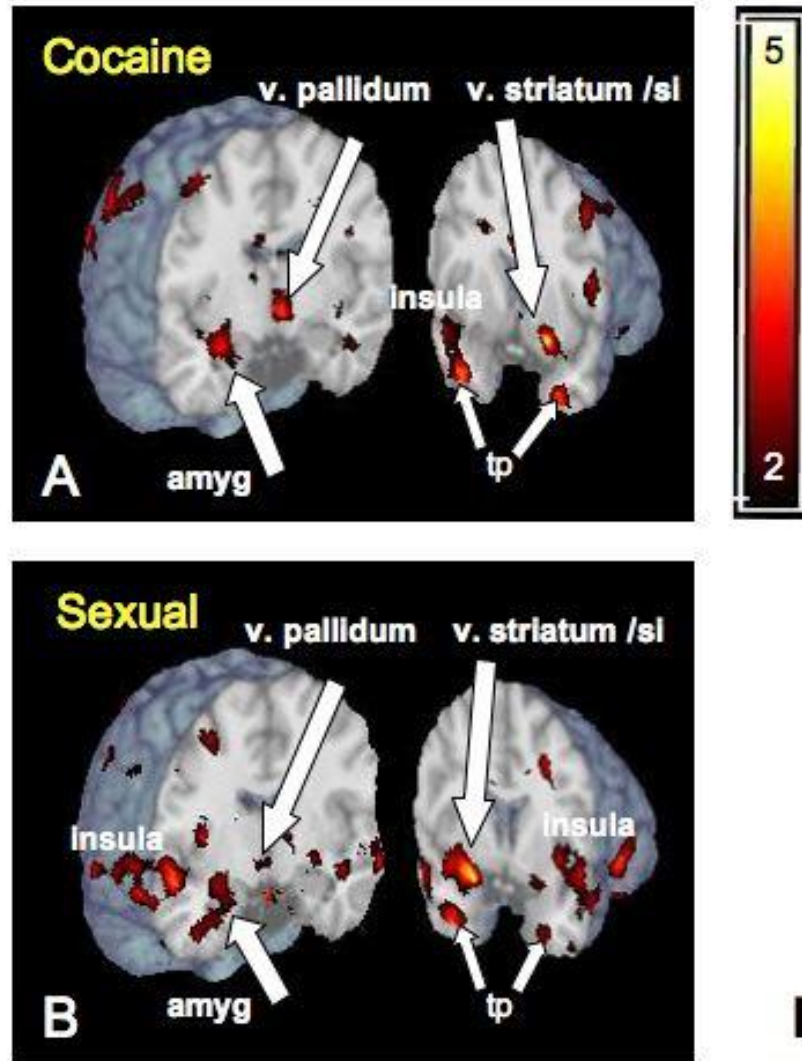
33 msec targets

467 msec “masks”

Advantages:

- A “**demand-free**” measure of the reward circuitry (*unaffected by secondary responses of regret, shame, embarrassment...or by attempts to modulate the response*)....
- thus – it reveals the early, “uncontaminated” response of the reward circuitry...

Activations



“Unseen”
Reward Cues

trigger

limbic
activation:

amygdala

v. striatum

v. pallidum

insula

OFC

OK, Anna Rose --- nice demo
of the brain response to
“unseen” drug cues –
but

.... does this brain response
have any thing do with “**real-
world” vulnerability to
relapse?**

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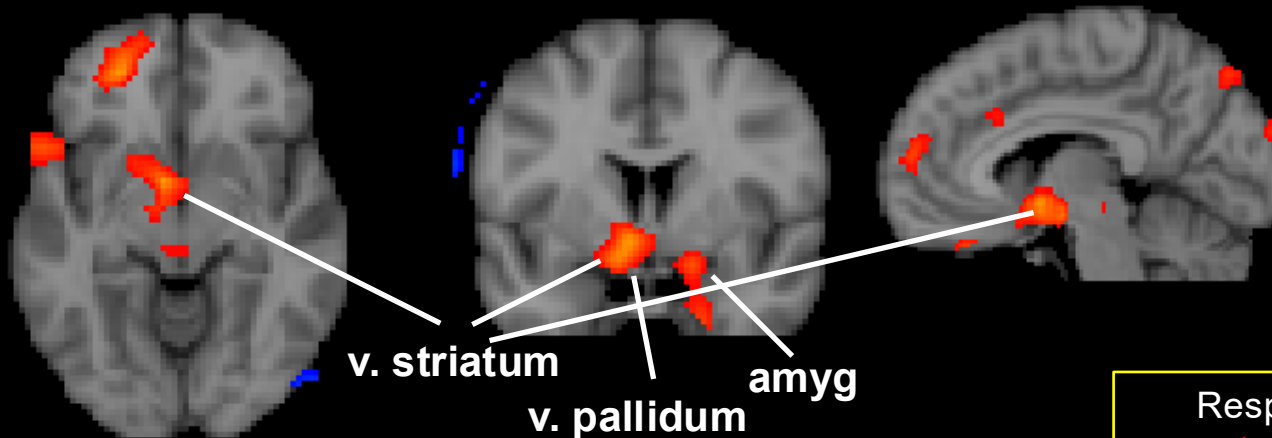
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“Unseen” (subliminal) **33 msec COCAINE** cues trigger
“GO!” – and the brain response predicts future **relapse!**

Rapid > Delayed Relapse

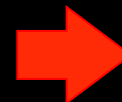


↑
v. striatum
v. pallidum
amygdala

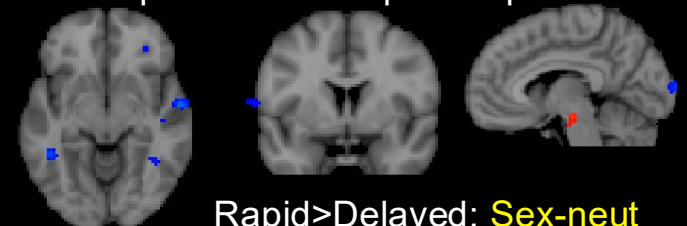
($N=17$ v. 8) drug- neut, $2 < t < 5$ for display

(MonteCarlo corrected, $k=533$; voxel=.005, cluster=.05)

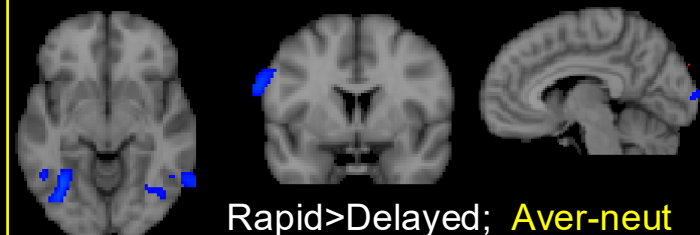
Is the rapid relapse prediction
specific to cocaine cues? Yes



Response to **sexual** or **aversive** cues
not predictive of rapid relapse



Rapid>Delayed; **Sex-neut**



Rapid>Delayed; **Aver-neut**

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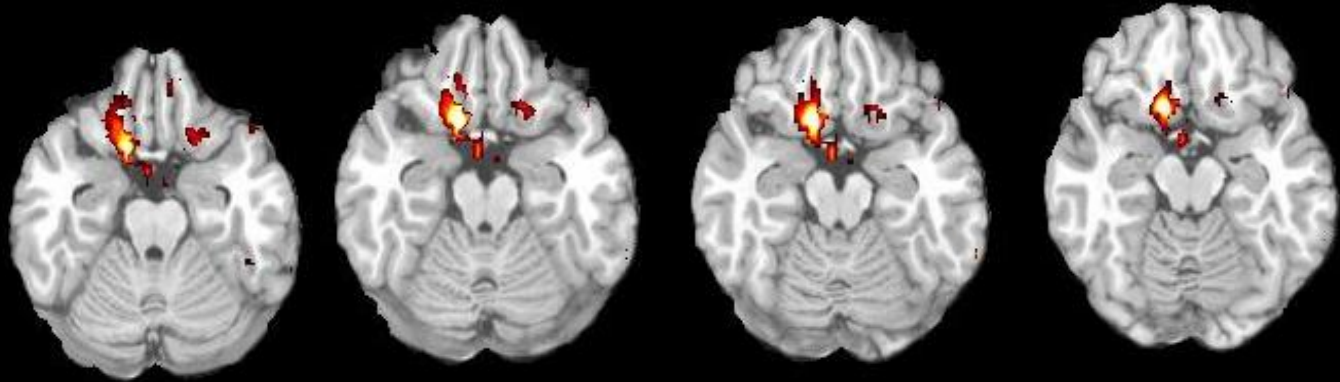
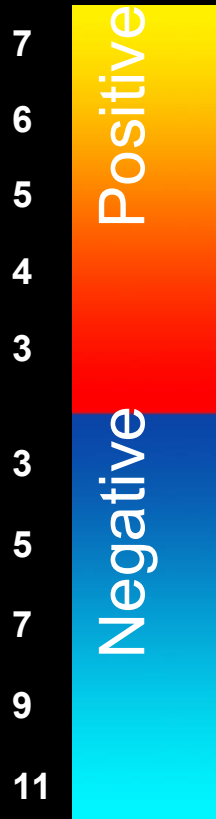
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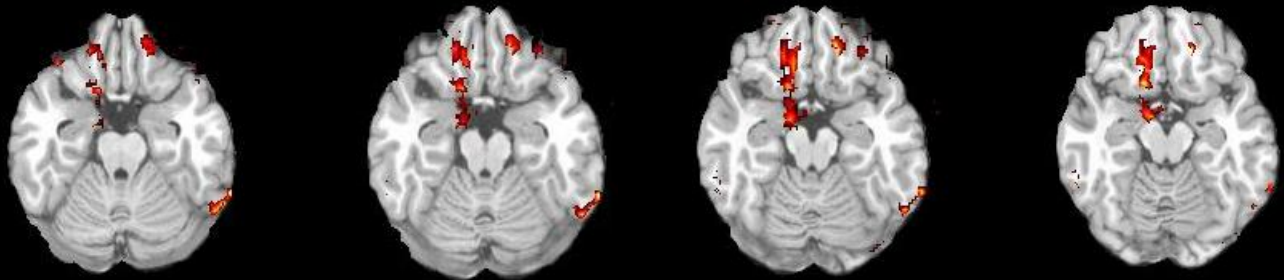
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Smoking vs. Non-smoking Cues

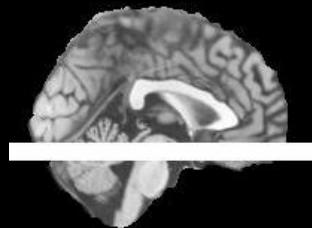
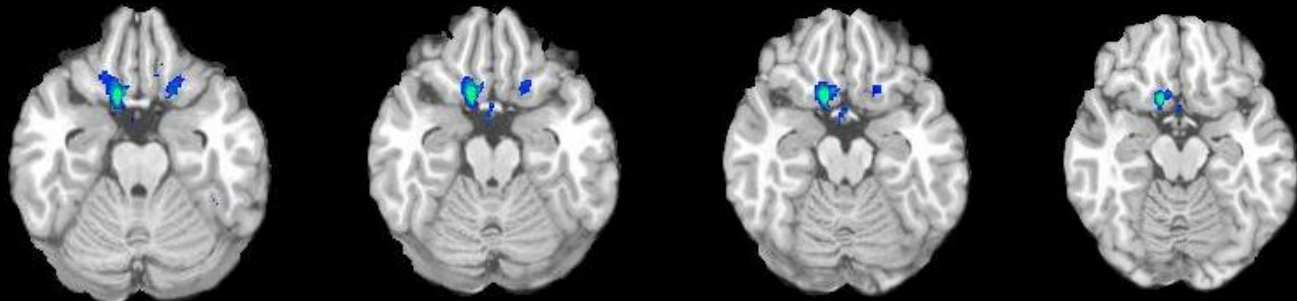
DAT 9 carriers vs. DAT 10 homozygotes



DAT 9 carriers

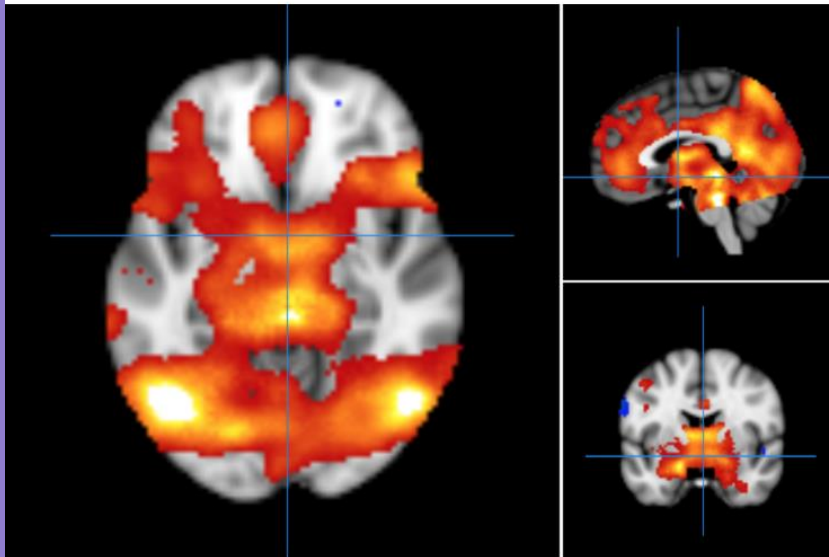


DAT 10 homozygotes



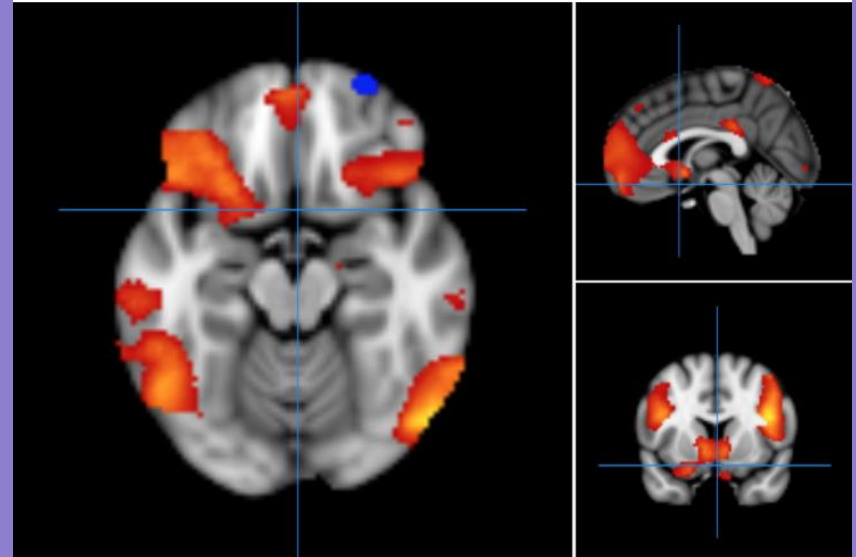
Cocaine and **Opioid** patients (left panel) carrying the G-allele variant of FKBP5 SNP **rs 3800373** (associated with heightened cortisol signaling, as compared to TT homozygotes) demonstrate a **heightened limbic** (striatum, v. pallidum, amygdala, midbrain, anterior insula) and **extra-limbic** (p. cingulate, visual cortex) **brain response to DRUG cues**, (n=56 total)

Drug1-Neut1_Unmasked_RFX_TG_GG_N_coc_opi_31_01-May-2019



T/G and G/G subgroup (n=31)

Drug1-Neut1_Unmasked_RFX_TT_N_coc_opi_25_01-May-2019



T/T homozygotes (n=25)

500 msec fast-event-related fMRI task; first half (Drug1—Neut1); SPM 8 parametric maps thresholded $2 < t < 5$ for display.

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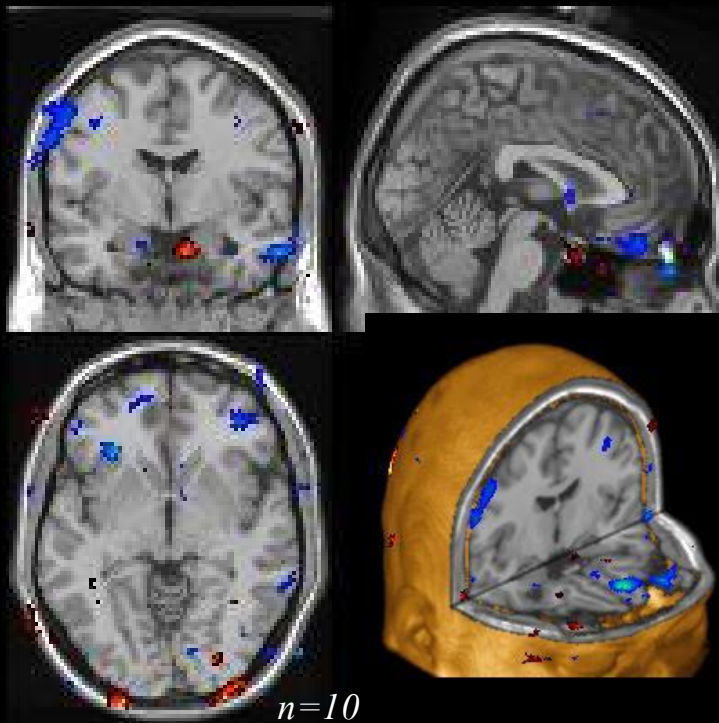
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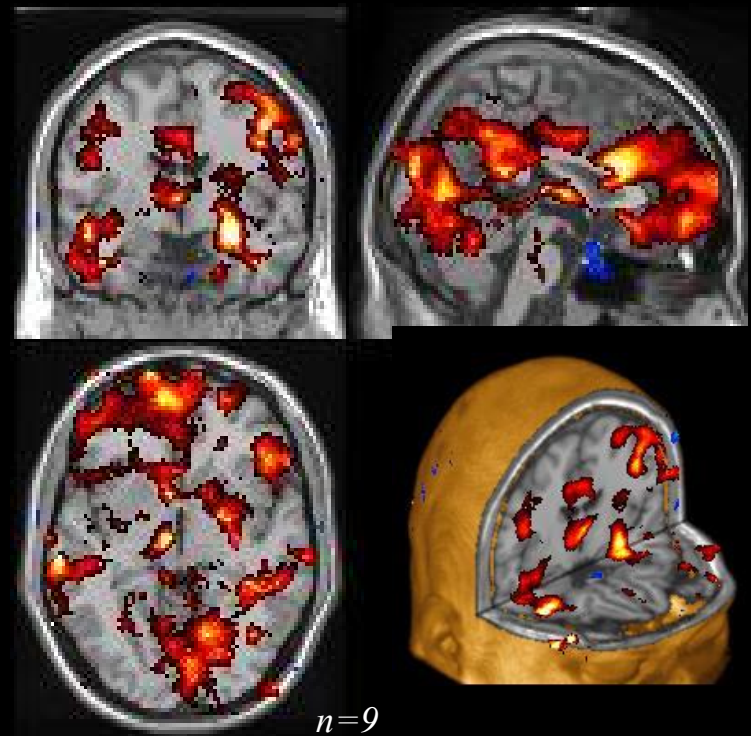
Dr. Jesse J. Suh

Enhanced Limbic Reactivity to 33 msec “Unseen” COCAINE Cues In CocainePatients with Prior Trauma

No Prior Trauma



Prior Trauma



Drug1-neut1

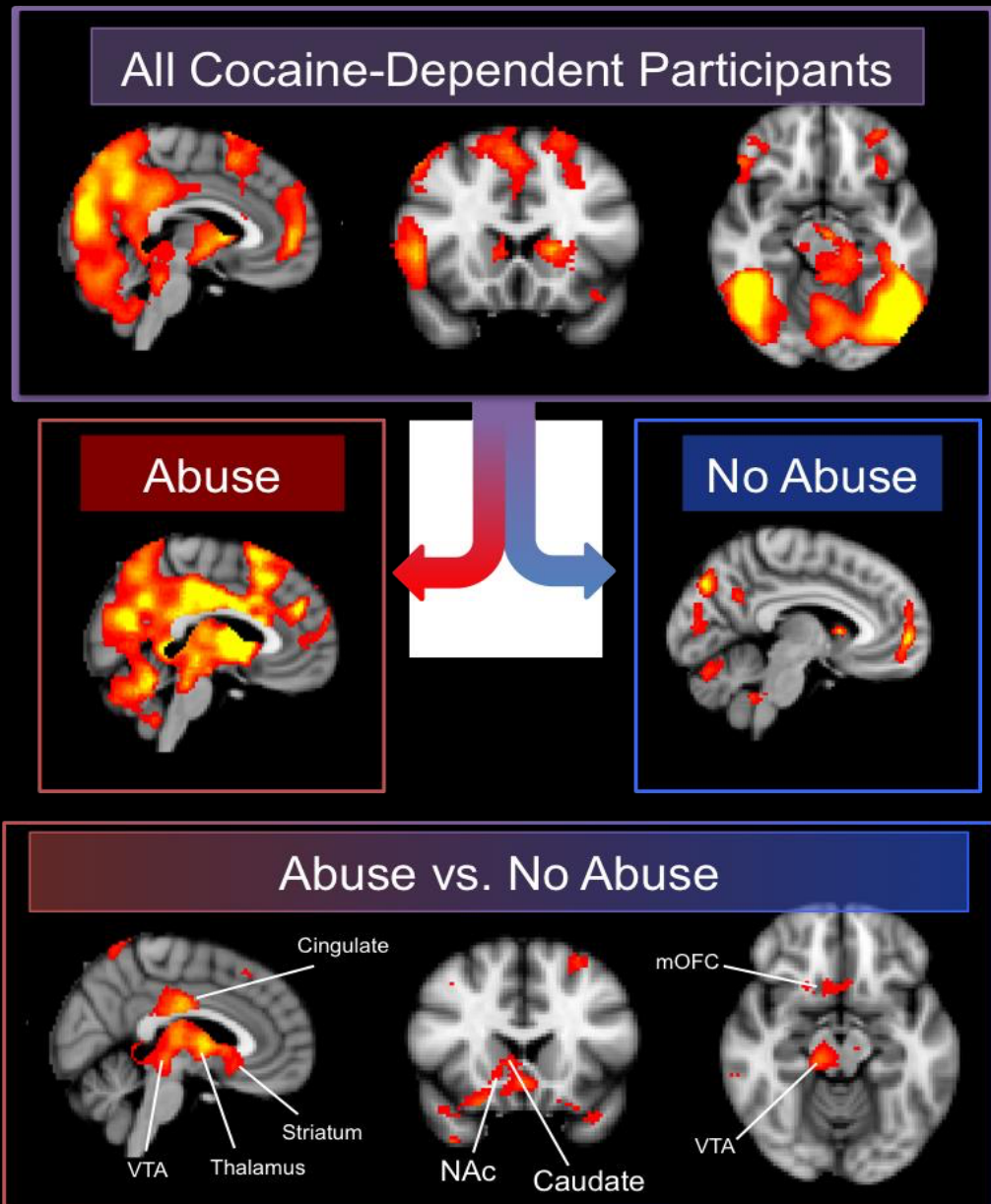
New results with our 500 msec cue task: An Abuse Endophenotype



Paul Regier, Ph.D.
NIDA T32 Fellow

Cocaine patients with a prior history of **abuse** (physical, emotional, or sexual) have a heightened brain response to **500 msec cocaine cues**.

Addiction Biology,
2016.



WHEW – Cues are everywhere – sometimes even “unseen” – thus very difficult to battle....

.... what can we do to protect against this vulnerability -- to assist our patients in their struggle? **Is there hope?**

Yes: ongoing intervention studies

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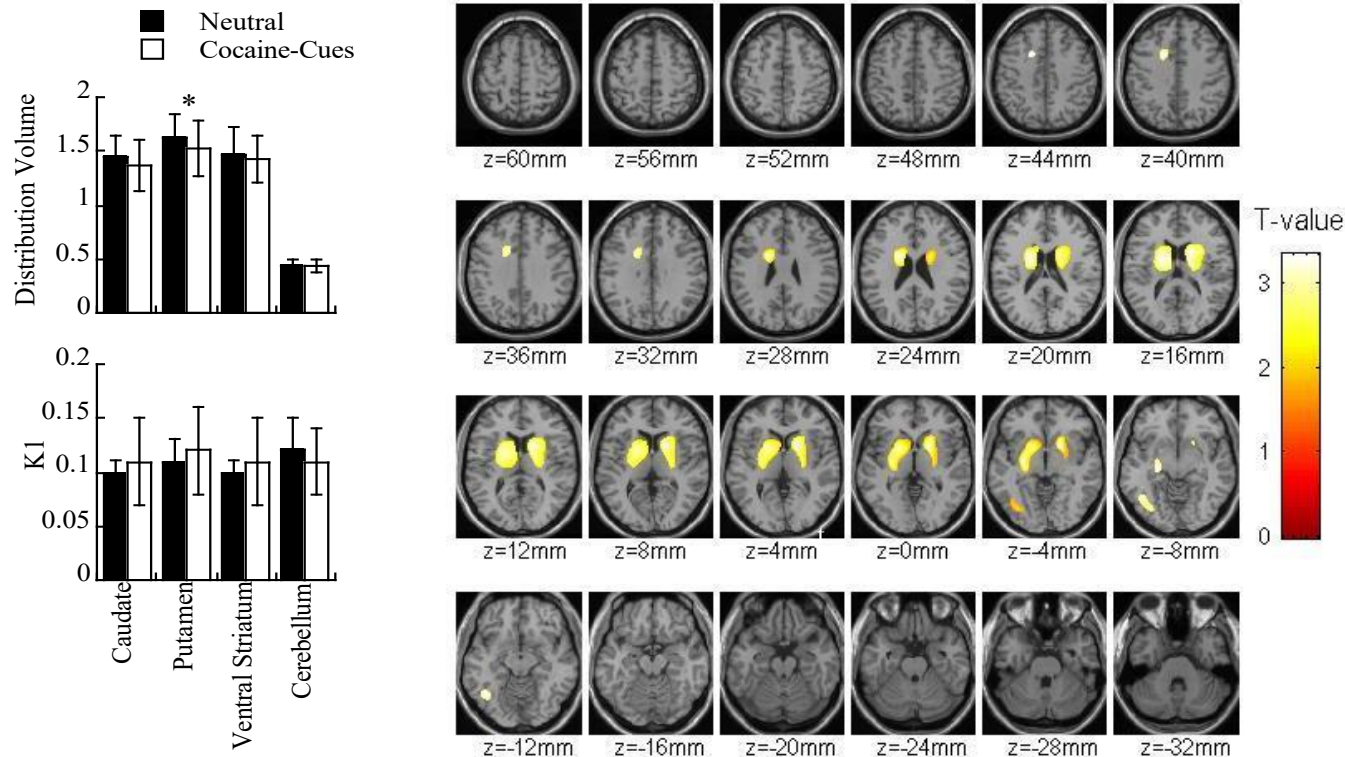
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Evidence of striatal DA release to cocaine cues in humans



B. Brain maps obtained with SPM showing the difference in the distribution volume images of [¹¹C]raclopride between the neutral and the cocaine cue condition ($p < 0.05$, uncorrected, threshold > 100 voxels). Note that there were no differences in the ventral striatum (-4 and -8 planes).

Can we modulate the “GO!” substrates with a Medication?

Hypothesis:

If limbic DA release is one substrate for cue-induced cocaine craving, then a GABA B agonist medication might help blunt both subjective and brain responses to cocaine cues.

Protection against the “unseen” foe...

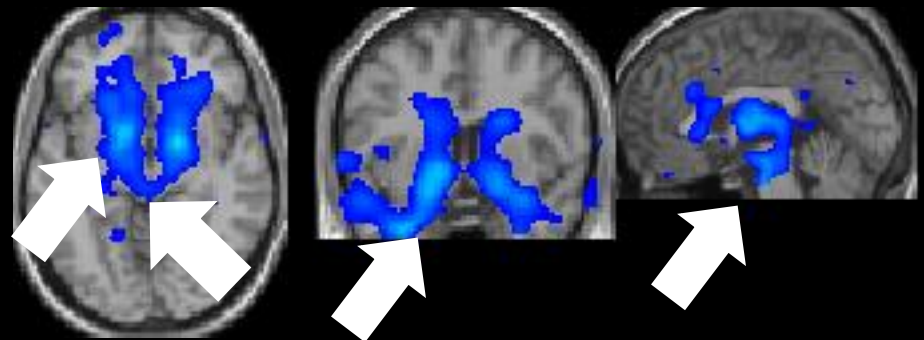
Baclofen for

“GO!”

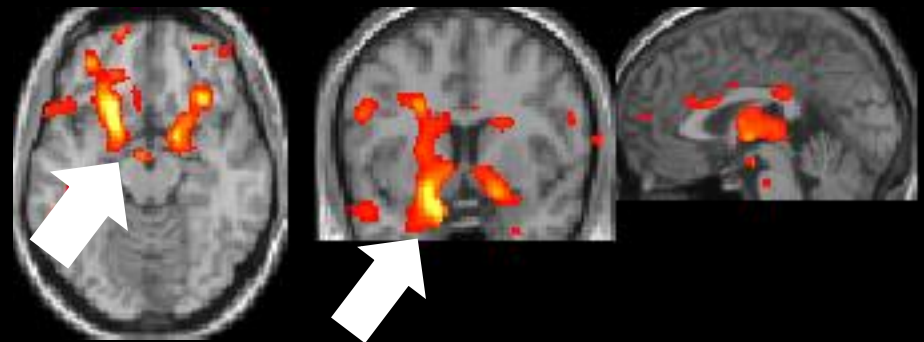


- GABA B agonist baclofen protects against “unseen” cues....
- **Dr. Kim Young**, a NIDA T-32 Fellow, has been energizing our baclofen efforts!

Baclofen – Placebo (n=20)



Placebo (n=10)

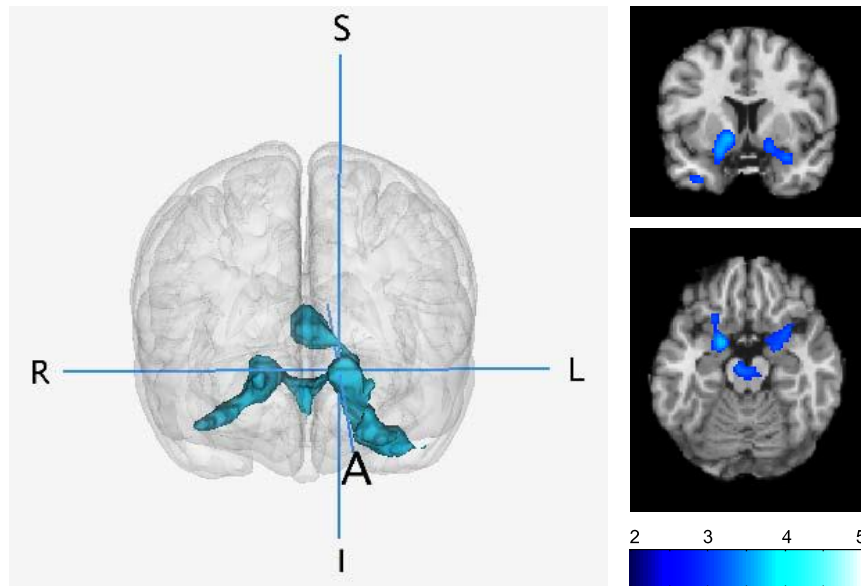


Baclofen reduces limbic activation to “unseen” cocaine cues (drug-neutral contrast; t-maps thresholded at $2 > t < 5$ for display.)

The GABA B agonist baclofen blunts limbic activation to 33 msec cocaine cues

A

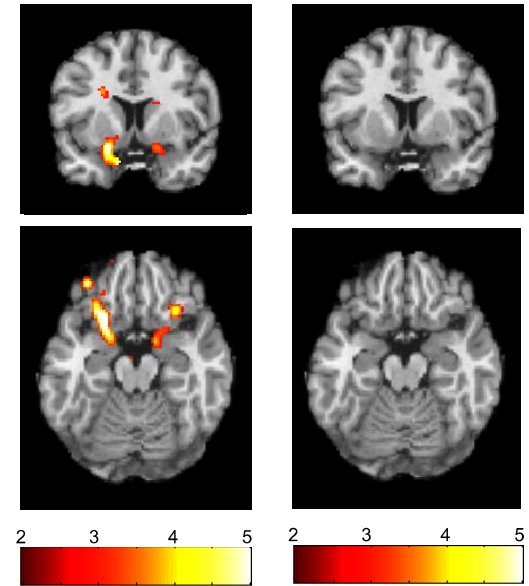
Baclofen < Placebo



B

Placebo

Baclofen



(A) Baclofen-as compared to placebo-treated cocaine patients (B) show blunted activation in a large inter-connected cluster spanning v. striatum, v.pallidum, amygdala, midbrain and OFC. Our “unseen” cue probe is sensitive to a DA-modulating medication.
Young et al. *Journal of Neuroscience*, 2014.

Coming Attractions: New Tools, New Targets

Much of our anti-relapse work has targeted the “cue-reactive” subcortical circuitry – and we are continuing to do this with new brain tools: LIFU... and... DBS....

New Interventions



Psychedelics

(Psilocybin, 5MeODMT,
Ketamine)



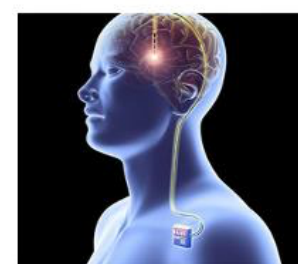
LIFU

Low-intensity Focused Ultrasound



TMS

Transcranial Magnetic
Stimulation



DBS

Deep Brain Stimulation

However, we are expanding our targets to include ‘neurocognitive flexibility’, helping patients get ‘unstuck’ from vicious relapse cycles, and to support sustained recovery. We have two ongoing psilocybin trials in our patients struggling with OUD.

SUMMARY

Context :

- Two brain systems implicated in **relapse vulnerability**: “GO!” and **STOP!** circuits

Part 1 : Visible Drug Cues

- Can we image the brain response to **visible** drug cues ?
- Can we link the brain response to **relapse**?

Part II : “Unseen” Drug Cues

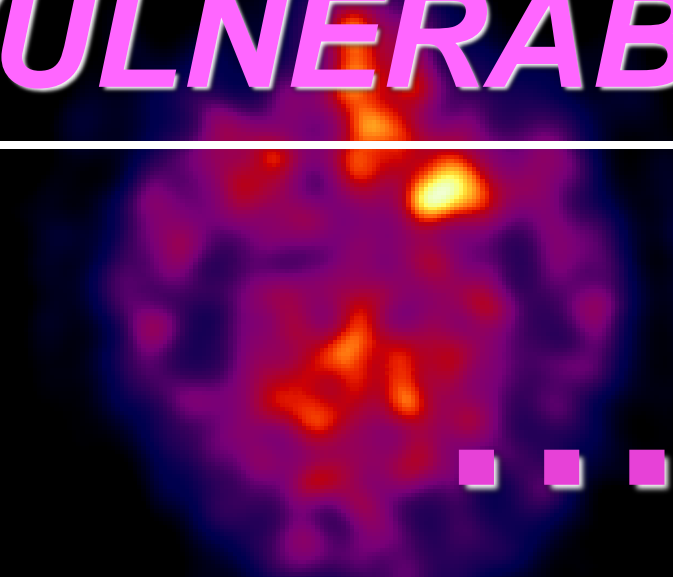
- Can we image the brain response to 33 msec “unseen” drug cues?
- Can we link the brain response to “unseen” cues to **relapse**?

Part III : Why are some more vulnerable to drug cues ?

- (**Genetics?** Epigenetics / **prior Trauma / Abuse**)?

Part IV : Is there hope? Can we impact the “cue-vulnerable” phenotype with a new tools? (LIFU; DBS) ? New targets: getting “**unstuck**”: **Psilocybin**

VULNERABILITY



....but
is “GO!” the
whole story?

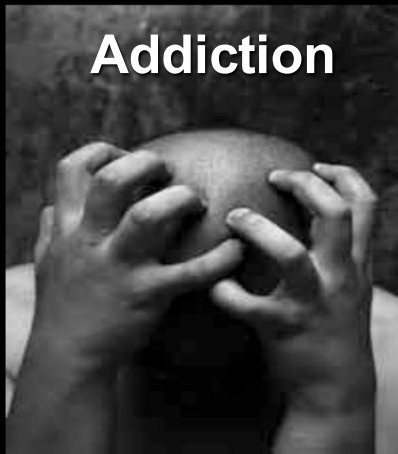
VULNERABILITY

“GO!”

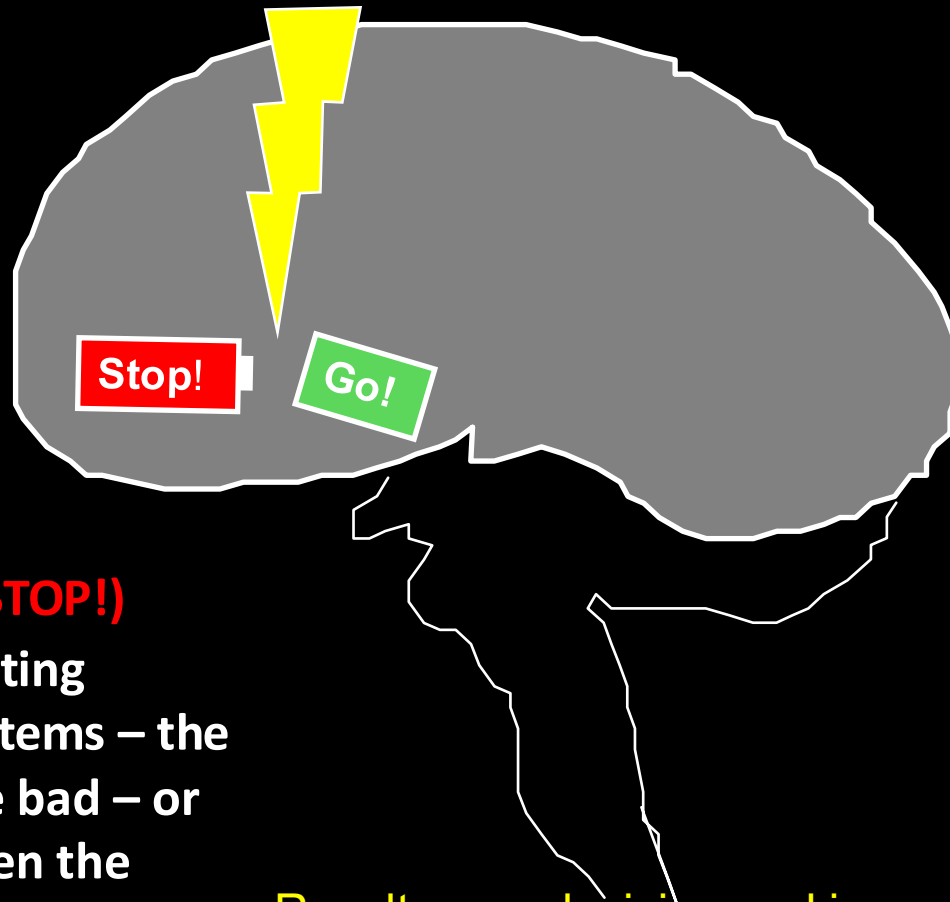
*Brain substrates of
cue-induced **drug**
motivation.....*

“STOP!”

*....and its **regulation**
(or lack thereof :
deficits in frontal
modulatory circuits)*



In a vulnerable brain....



..the brain's frontal **(STOP!)** circuitry is not modulating downstream **(GO!)** systems – the “brain brakes” may be bad – or the connection between the brakes and the other regions may be “broken”.

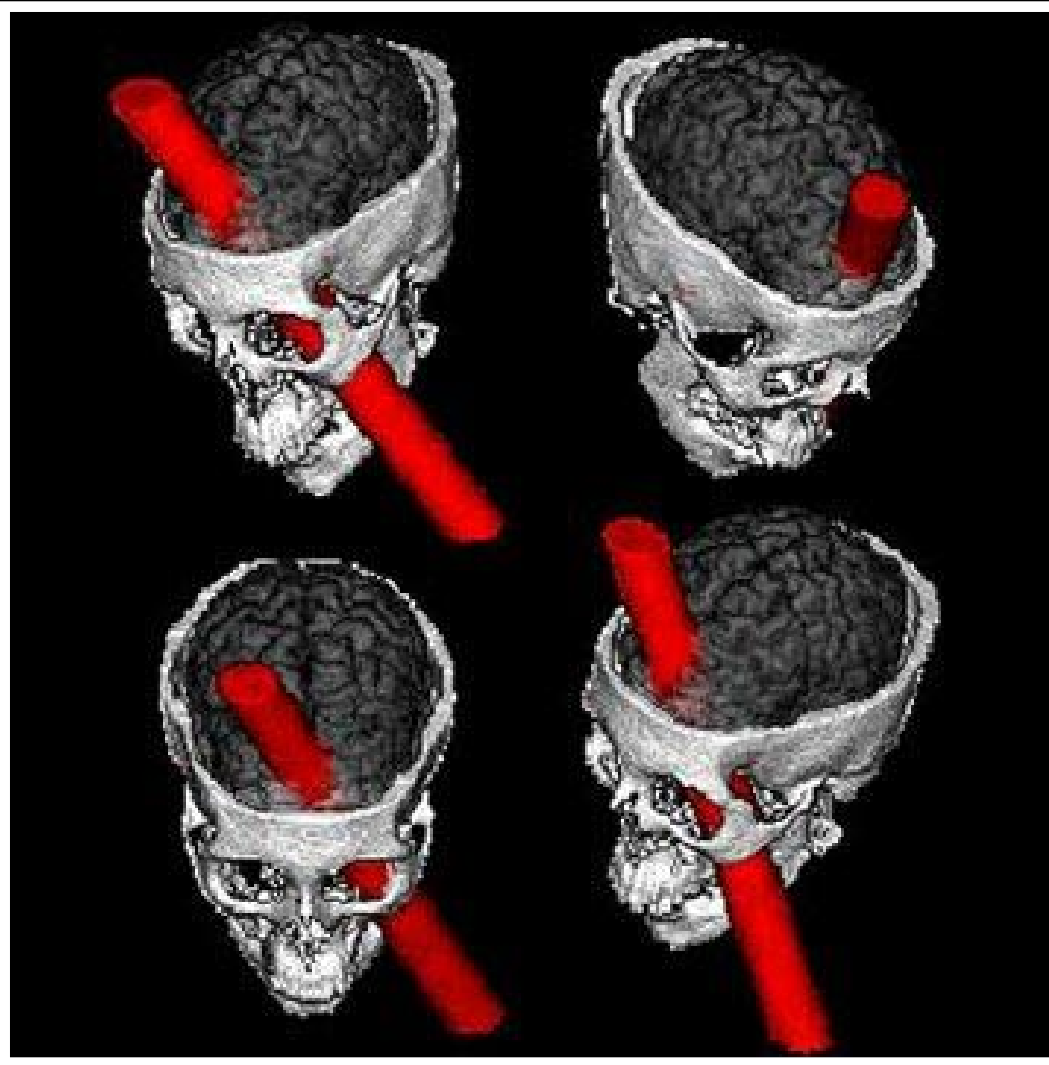
Result: poor decision-making...poor impulse control...greater risk-taking...poor inhibition...an “over-reacting” brain

What if some patients have a deficit in their frontal (“STOP!”) circuitry?

Why it’s hard to say NO.....



***“Bad Brakes?”
(Poor Frontal Endowment)***



In 1994, the D'Amasios (Iowa) reconstructed the likely trajectory of the tamping iron, noting the similarity of his deficits to those of their patients with lesions of VMPFC....

....Though Phineas suffered NO speech, motor, memory, or intellectual deficits....he was no longer able to make **good life decisions** ...to be responsible, to plan ahead, **to consider consequences**...he was **disinhibited**, profane, took to drink...was never able to again hold the same level job. Suffered profound decline. Died in 1861.

Cocaine Patients

PET O-15

Hypoactivity



Reduced

Gray Matter



NEURO targets: Relapse Prevention

“GO!”

“STOP!”

Scan Day 1

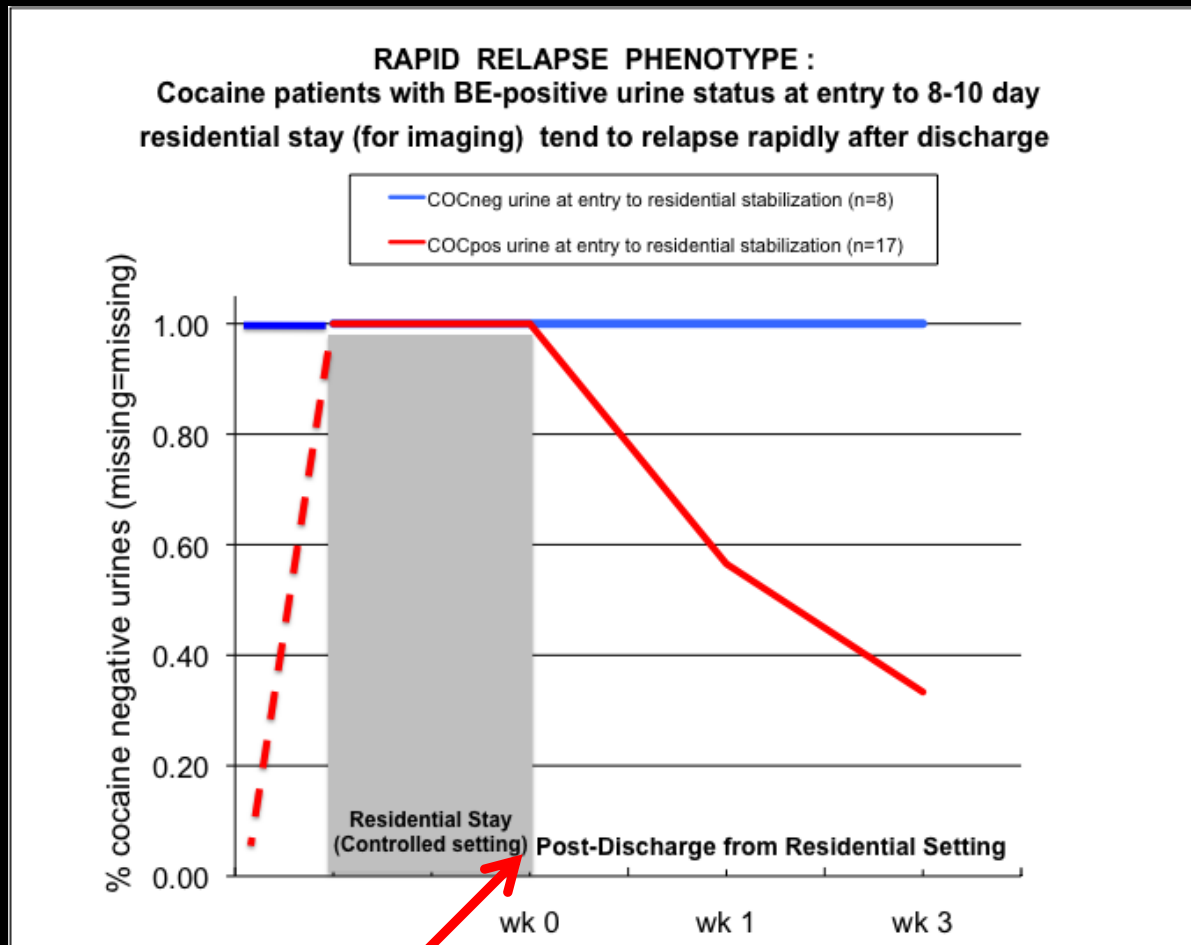
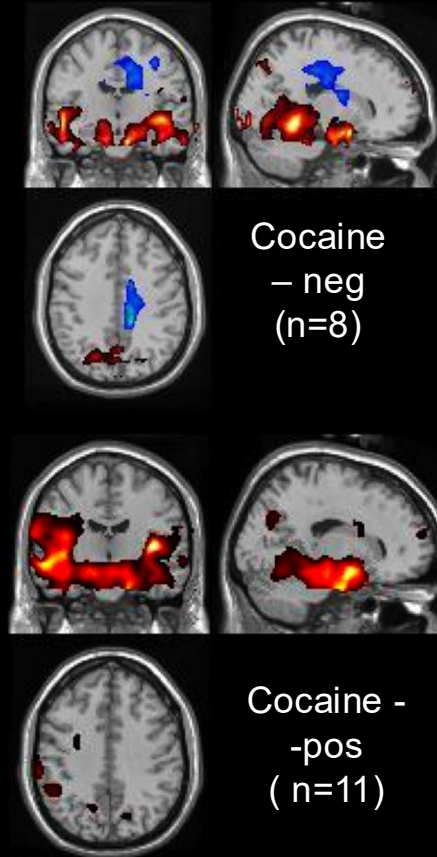
Scan	Anatomical Localizer	ASL Resting Scan	BOLD Resting Scan	Brief Cue I (33 msec)	Go-NoGO	Brief Cue II (500 msec)	High Res. Structural	De-Brief
Minutes	30 sec	5	5	8.25	5.5	8.25	3	

Scan Day 2

Scan	Anatomical Localizer	ASL Resting Scan	BOLD Resting Scan	Craving- Inhibition	High Res. Structural	Affect Regulation	De-Brief	Task Day Behavioral Tasks (60 min)	De-Brief
Minutes	30 sec	5	5	7.5	3	7.5			

As a group, cocaine patients were poor inhibitors...but when divided according to a prognostic clinical phenotype (**cocaine +** vs. **cocaine - urine** at entry) --- the “good prognosis” phenotype showed an **inverse relationship between amygdala (seed) and r. dorsal cingulate**; this pattern was absent in the “poor prognosis” subgroup.

Attempted “STOP!”



Resting	Neutral 1	go	Neutral 2	GO	Neutral 3	STOP	Neutral 4
video	video	video	video	video	video	video	video

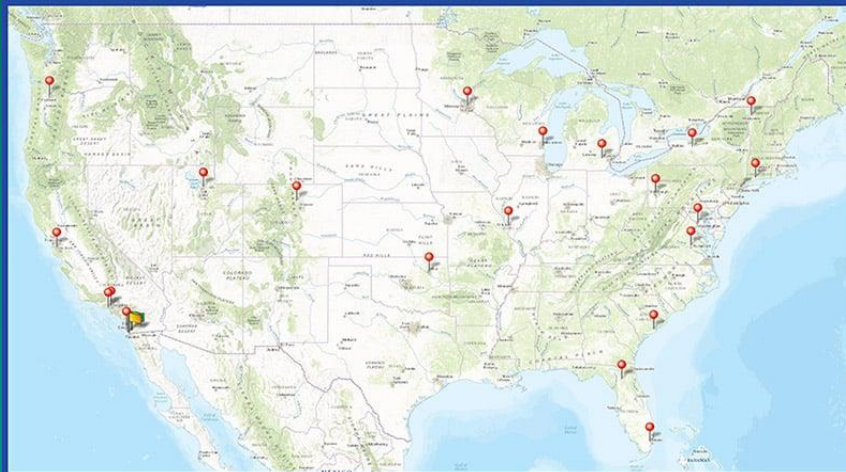
fMRI session prior to discharge

How can we determine whether these brain differences we see in adults were present even **before** drug exposure? Important, in terms of what we target – or can expect – from treatment!





>11,000 9-10 year olds to be assessed regularly for at least a decade (2018-2028), with many MANY phenotypic measures and brain probes for **reward, inhibition, working memory and emotion recognition** (along with structural and resting brain function).



<https://abcdstudy.org/es/publications/>

A great research opportunity -- with no data collection required !



Importantly -- most of our work on “GO” and “STOP” substrates has been conducted in adults with addiction – and has documented **differences** in the brain response to probes for reward and inhibition – **but – we still need to understand the big “chicken-egg” question:** which differences are the ‘result’ of drug use/exposure, and which may have pre-dated, and even predisposed the progression to addiction?

*The goal is to identify the strongest brain, behavioral and environmental predictors of **vulnerability to later mental health and drug use problems** – toward early intervention and improved (even novel) treatments.*

Summary

Context :

- Two brain systems implicated in **relapse vulnerability**: “GO!” and **STOP!** circuits

Part 1 : Visible Drug Cues

- Can we image the brain response to **visible** drug cues ? **Yes**
- Can we link the brain response to **relapse**? **Yes**

Part II : “Unseen” Drug Cues

- Can we image the brain response to 33 msec “unseen” drug cues? **Yes**
- Can we link the brain response to “unseen” cues to **relapse**? **Yes**

Part III : Why are some more vulnerable to drug cues ?

- Genetics and Epigenetics, e.g., **prior Trauma may modulate**

Part IV : Is there hope? **Yes** We may be able to protect the “cue-vulnerable” phenotype with **DA-modulating** or other **medications**. And a **NEW** target: **Neurocog Flexibility: Psilocybin**

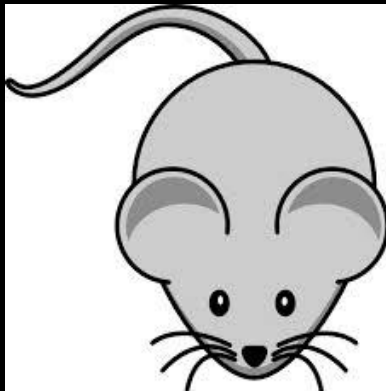
Part V: The ‘**STOP**’ system (poor recruitment – and/or compensatory **over**-recruitment of the ‘stop’ circuitry) may also be a relapse vulnerability....and some individuals may be more vulnerable, **even before drug exposure**.

Our new NEURO tools will help us move us forward...

Animal Models

NEURO tools

Clinical Trials



Adding NEURO targets and tools to the development pipeline promises to accelerate the pace of discovery.

These NEURO measures will allow us to embrace the heterogeneity within the disorders -- and make use of the heterogeneity itself as a powerful tool for medication development in the addictions.....



...offering new hope
for managing our “old” brain’s
GO! and **STOP!** circuitry...



....thus new hope for the addictions ...
...ahem....and what about managing our day-to-day
“limbic moments” that don’t quite qualify as “addiction”
?

Acknowledgements

NIDA R61DA061206-02 Psilocybin: Capturing brain mechanisms of motivation and neurocognition in individuals with opioid use disorder

A Wellcome Leap for the Opioid Crisis: Can the 5HT2A agonist Psilocybin improve brain, behavioral, and clinical outcomes in opioid use disorder (OUD)?

NIDA UG1 DA050209 Clinical Laboratories with

NIDA U54 Cooperative Cocaine Medication Development Ctr.

NIDA R01DA039215 (Targeting Dopamine D3 Receptors in Cocaine)

NIDA P50 DA12756 (Cocaine Medication Development Ctr.)

NIDA P60 DA 005186 (Improving Treatment of Drug Abuse)

NIDA RO1 DA 10241 (Coc Cue + Inhibition)

NIDA RO1 DA 12162 (Coc Cue + Baclo)

NIDA RO1 DA 15149 (Coc Cue – ASL fMRI)

NIDA RO3 I-Start – J. Suh

NIDA KO1 (Nic Cue, Franklin)

NIDA K23 (Opiate Cue, Langleben)

NIDA CSP #1021 (Baclofen Multi-site Clinical Trial)

NIDA R01 DA025906 (“Unseen” Coc Cue Extinction)

NIDA R21/R33 DA026114 (Coc Cue + Real-time fMRI)

NIDA T32 Translational Addiction Research (Childress/Blendy)

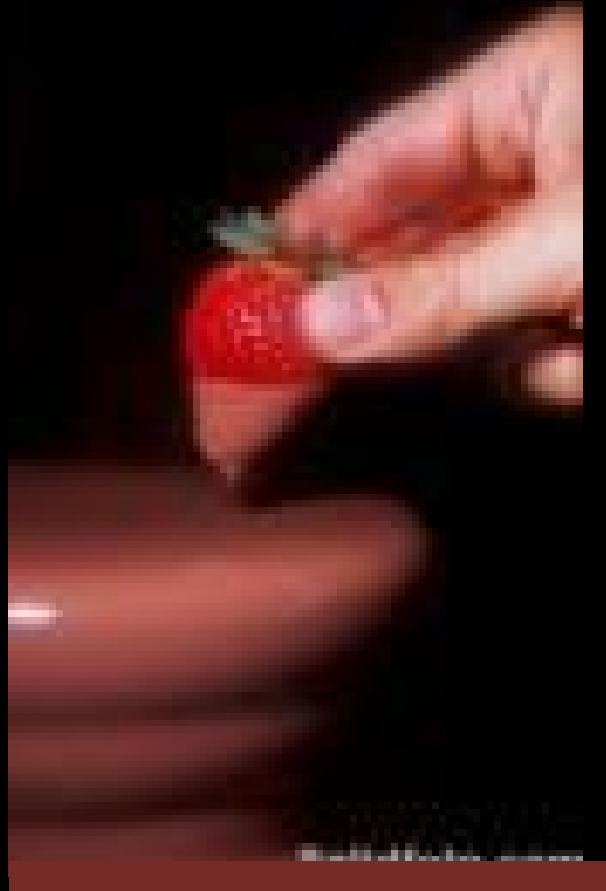
CURE Addiction Center of Excellence (Childress)

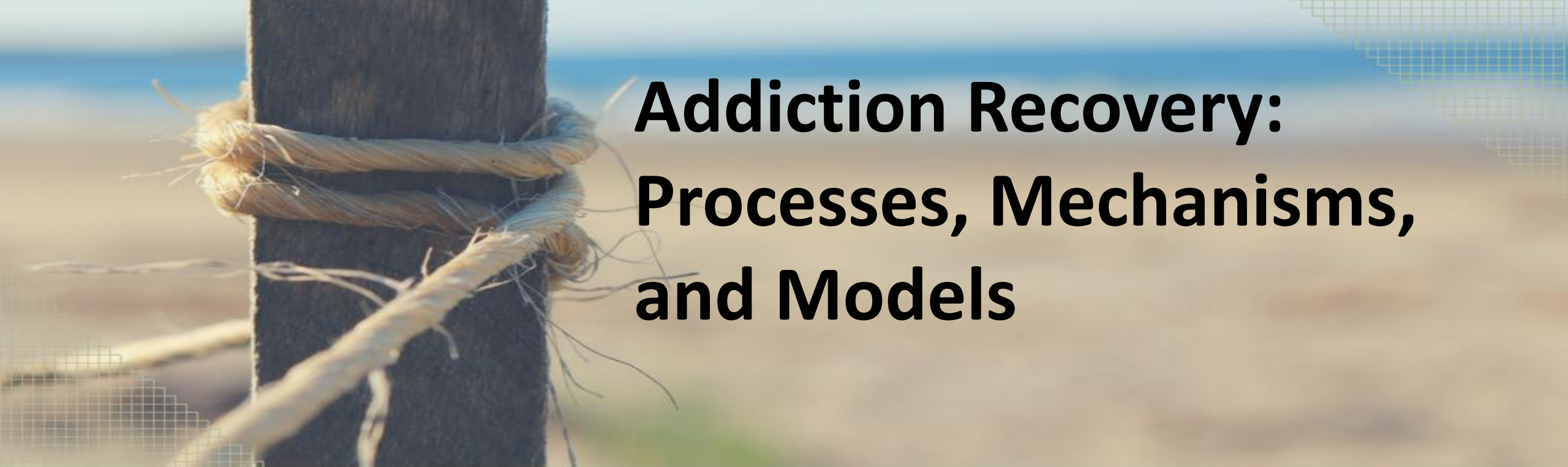
VA Medical Research Division / MIRECC

DANA Foundation



Thank you!





Addiction Recovery: Processes, Mechanisms, and Models

JUNE 2026

Addiction Policy Forum

John F. Kelly, PhD, ABPP



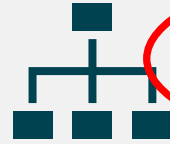
RECOVERY
RESEARCH
INSTITUTE



Massachusetts General Hospital
Founding Member, Mass General Brigham

HARVARD MEDICAL SCHOOL
TEACHING HOSPITAL

Outline



What is “recovery”: Taxonomy and terminology



How to conceptualize recovery



Models: How to achieve stable recovery and reduce and prevent long-term relapse risk

Healing Terminology Used in the Addiction Field

Different Terms. Different Focus. Shared Goal.

All represent positive change toward improved health and well-being.

REDUCTION



Decreasing the amount of substance use and related consequences.

Focus

- Safer use
- Fewer harms
- Improved stability
- Step toward change

*"Less use. Less harm.
More control."*

CESSATION



Stopping substance use, which may be short-term or longer-term.

Focus

- Ending use
- Gaining control
- Beginning a new chapter
- May or may not be sustained

*"Stopping use—
a powerful first step."*

ABSTINENCE



The complete avoidance of alcohol and other drugs.

Focus

- No use of any mind-altering substances
- Often chosen for health, personal, or spiritual reasons
- Can be time-limited or ongoing

"Choosing not to use."

RESOLUTION



A sustained state in which substance use no longer controls or defines a person's life.

Focus

- Long-term stability
- Peace and clarity
- Freedom from the grip of addiction
- Forward movement

*"The issue is resolved. Freedom
and a new normal."*

REMISSION



A period of improved health where symptoms of substance use disorder are absent or significantly reduced.

Focus

- Fewer or no symptoms
- Improved functioning
- May or may not include abstinence

*"Symptoms improve.
Life gets better."*

RECOVERY



A voluntary, ongoing process of change through which individuals improve their health and well-being, live a self-directed life, and strive to reach their full potential.

Focus

- Whole-person wellness
- Growth and purpose
- Quality of life
- Includes many pathways

*"Living a meaningful
and fulfilling life."*



These terms are not one-size-fits-all. People's goals, paths, and definitions of success are unique.
Respect. Person-Centered. Strengths-Based. Hope-Focused.



Whatever term is used, the goal is the same: improved health, well-being, and quality of life.



DIVERSE PATHS

INDIVIDUAL CHOICE

ONGOING PROCESS

MEANINGFUL LIFE

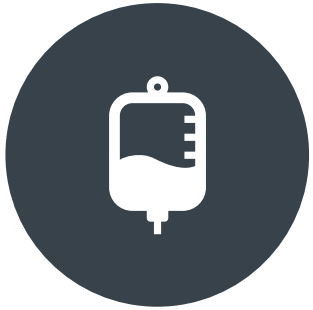
Recovery Definitions

Source	Definition
SAMHSA	A process of change through which individuals improve their health and wellness, live a self-directed life, and strive to reach their full potential.
ASAM	Recovery from addiction is an active process of continual growth that addresses the biological, psychological, social and spiritual disturbances inherent in addiction...
Recovery Science Research Collaborative	Recovery is an individualized, intentional, dynamic, and relational process involving sustained efforts to improve wellness.
UK Drug Policy Commission	The process of recovery from problematic substance use is characterized by voluntarily-sustained control over substance use which maximizes health and wellbeing and participation in the rights, roles and responsibilities of society.
William L. White	Recovery is the experience (a process and a sustained status) through which individuals, families, and communities impacted by severe alcohol and other drug (AOD) problems utilize internal and external resources to voluntarily resolve these problems, heal the wounds inflicted by AOD-related problems, actively manage their continued vulnerability to such problems, and develop a healthy, productive, and meaningful life.
National Institute on Drug Abuse	Research on the science of addiction and the treatment of substance use disorders has led to the development of research-based methods that help people to stop using drugs and resume productive lives, also known as being in <i>recovery</i>.
Betty Ford Institute	Recovery is a “voluntarily maintained lifestyle characterized by sobriety, personal health, and citizenship.”
Alexandre B. Laudet	“...recovery goes well beyond abstinence ...experienced as a bountiful ‘new life’, an ongoing process of growth, self-change and of reclaiming the self.”

Most recognize
that recovery is
a process and
is both
dimensional
and a
category....

- “Recovery” describes a process of salubrious change that occurs as individuals attempt to resolve AOD problems
- “Recovery” also a category (“in recovery”); at some point people consider themselves “in recovery” but previously did not
- Like “remission”, also potentially different degrees of “recovery” that may be marked by time (alone) that suggest more stable “recovery” with greater time, but...
- Currently missing - markers of resilience beyond just “time”; which remission-based factors are indicative of continued remission vs elevated risk of relapse/reinstatement (e.g., resiliency/vulnerability)?

Consisting of different intersecting constituent parts
(e.g., SAMHSA)



HEALTH



HOME



COMMUNITY



PURPOSE

Many people with broad array of mental health challenges define recovery often as consisting of five key elements ... (C.H.I.M.E)



CONNECTION



**HOPE AND
OPTIMISM**



**POSITIVE SOCIAL
IDENTITY**



**MEANING AND
PURPOSE**



EMPOWERMENT



Addiction Recovery = diminished or
absent disease + resilience

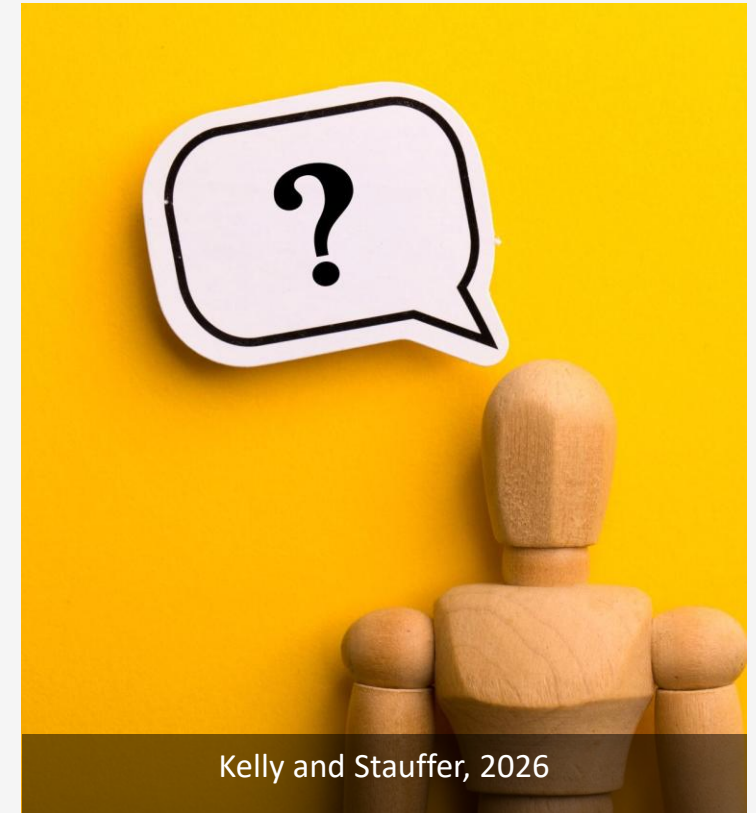
Recovery: Weatherproofing remission

Lingering questions on “recovery”

- Do we want to leave “recovery” as a broad organizing concept without defining it operationally?
- Do we want to allow people to subjectively self-define “recovery” however they want (“You’re in recovery when you say you are”)?
- Do we want to obtain standardized “recovery” prevalence rates (i.e., that are not self-defined) like we do for diagnostic remission?
- Do we want to construct “recovery” and duration of recovery, in a standardized objective way to help inform and determine socio-legal decisions (e.g., child custody cases)

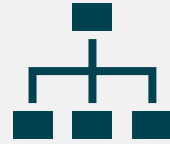
Similar changes face the definition of “health/ healthy” and how best to define/**operationalize**...

- WHO (1948) “**state of complete** physical, mental, social **well-being, not merely the absence of disease** and infirmity”
- WHO (1984): “The extent to which an individual or group is able to realize aspirations and satisfy needs and to **change or cope with the environment**. Health is a **resource** for everyday life, not the objective of living; it is a positive concept, emphasizing social and personal resources, as well as physical capacities.”

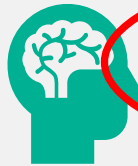


Kelly and Stauffer, 2026

Outline



What is “recovery”: Taxonomy and terminology



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Models: How to achieve stable recovery and reduce and prevent long-term relapse risk

RECOVERY

Successful healing from addiction involves the dynamic interplay of the psychobiological factors **within the person** with the socio-ecological factors **in the environment**.

PSYCHOBIOLOGICAL *Inside the person*

INTERNAL PROCESSES

Internal processes that shape healing, recovery, and well-being.

BRAIN & NEUROBIOLOGY

Healing brain circuits, reducing craving, improving response to stress.

EMOTIONS & MENTAL HEALTH

Managing emotions, building resilience, treating co-occurring conditions.

IDENTITY & PURPOSE

Rebuilding self-worth, meaning, and a hopeful future.

HEALTH & WELL-BEING

Improving sleep, nutrition, movement, and overall physical health.

COPING & SKILLS

Developing skills to handle triggers, solve problems, and make healthy choices.

SOCIO-ECOLOGICAL *Outside the person*

ENVIRONMENTAL CONDITIONS

Environmental conditions and supports that enable recovery to flourish.

RELATIONSHIPS & SOCIAL SUPPORT

Connection with family, peers, mentors, and supportive networks.

COMMUNITY & CULTURE

Belonging, cultural inclusion, and community acceptance and engagement.

EDUCATION & EMPLOYMENT

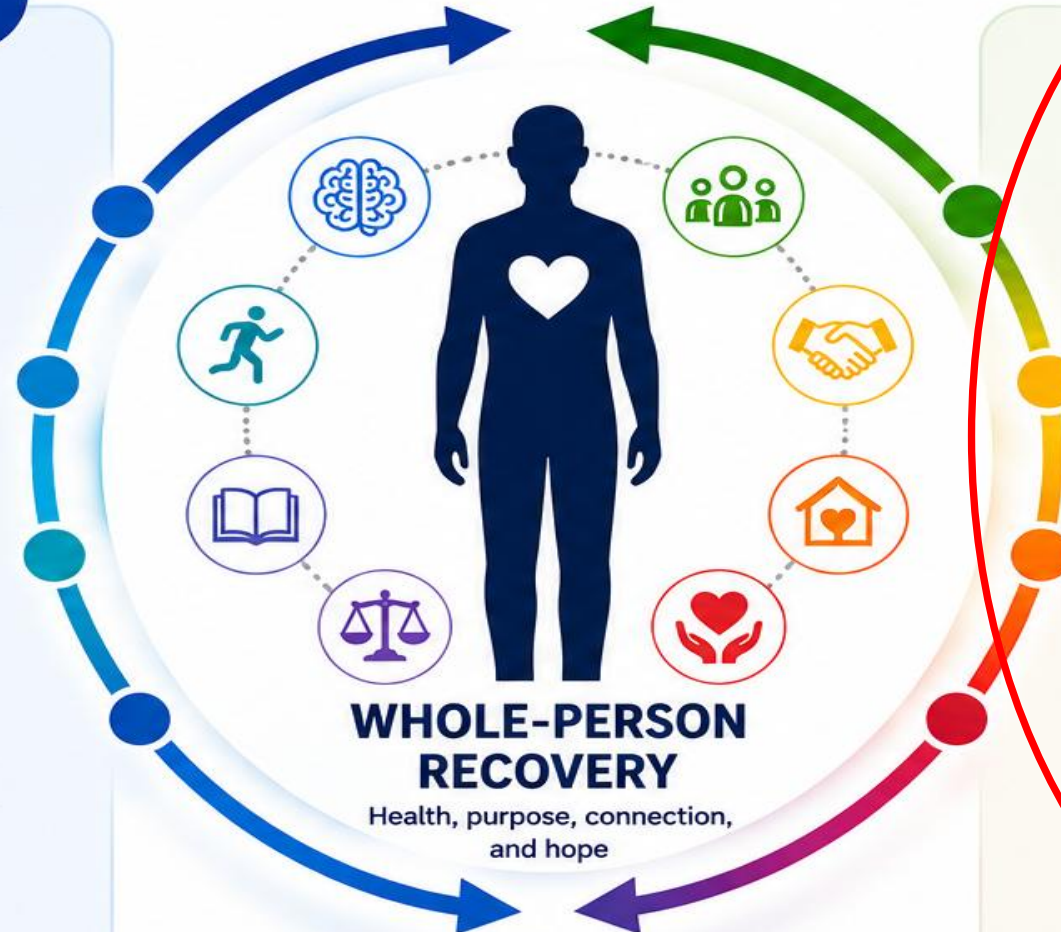
Meaningful opportunities that promote stability and purpose.

POLICY & SYSTEMS

Equitable access to healthcare, housing, harm reduction, justice reform, and recovery supports.

SAFE & STABLE ENVIRONMENTS

Safe housing, financial security, and neighborhoods that support health and recovery.

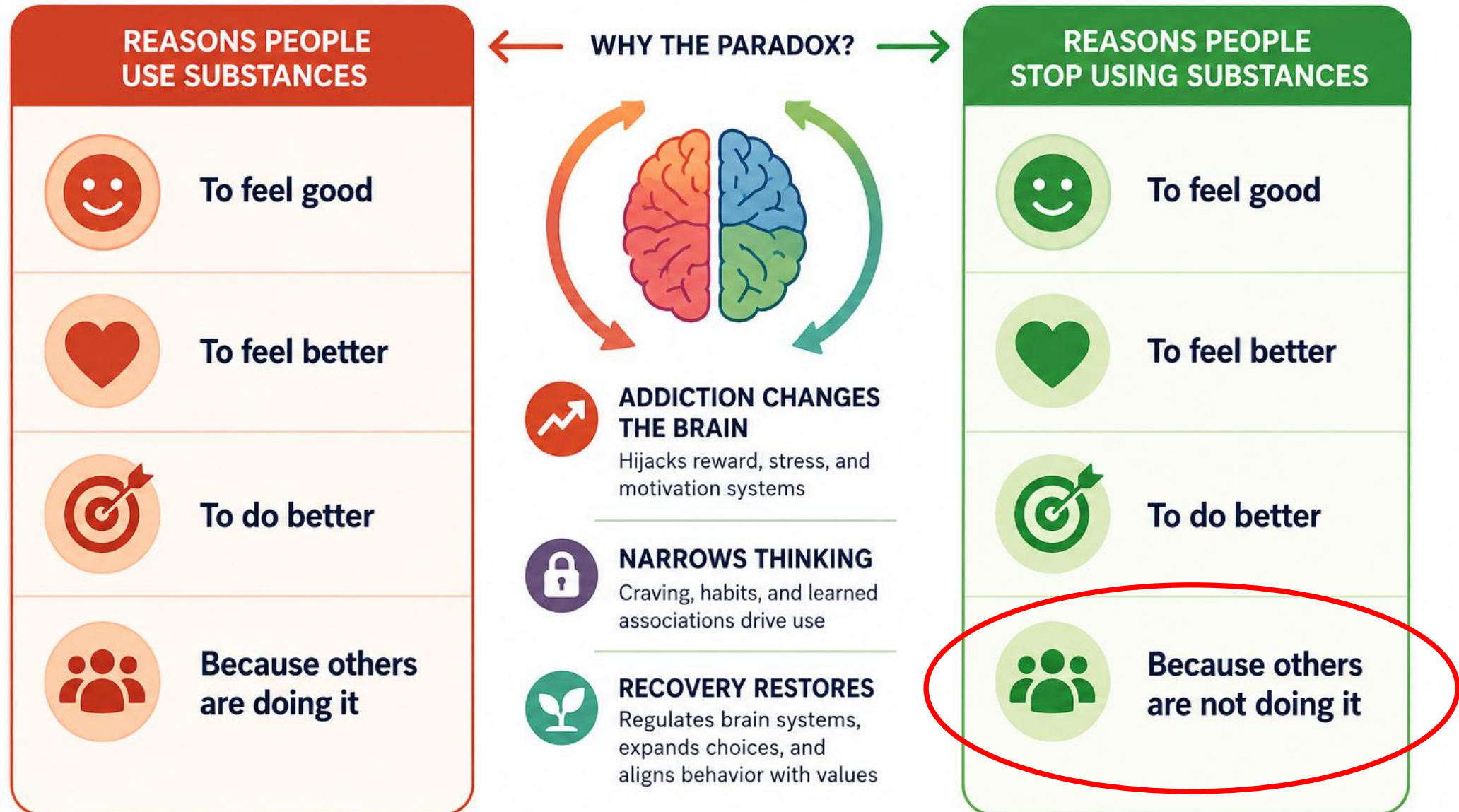


WHOLE-PERSON RECOVERY

Health, purpose, connection,
and hope

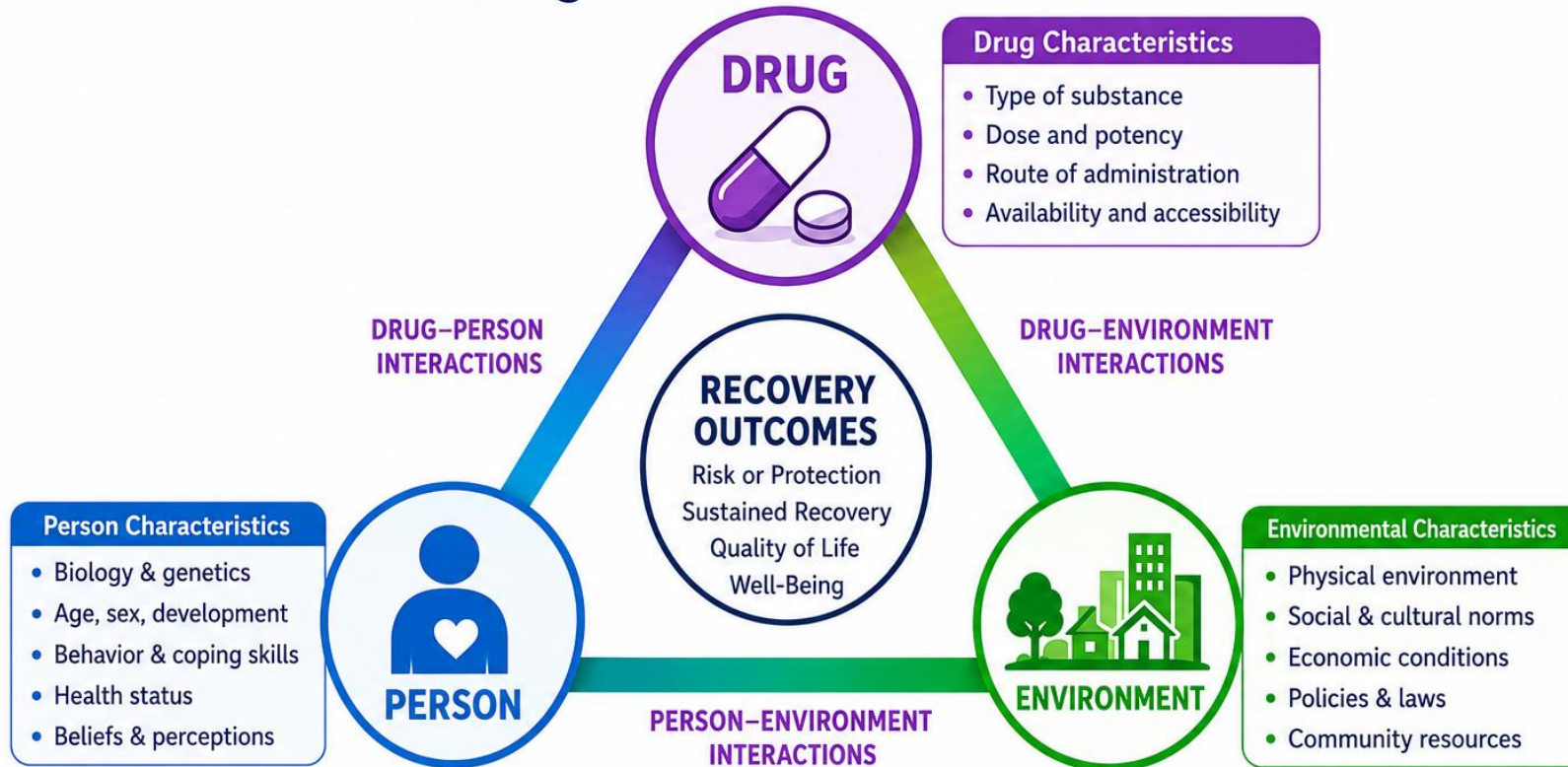
The Paradox of Addiction:

The same four Reasons are responsible for getting in to and out of addiction



RECOVERY PUBLIC HEALTH PARADIGM

Drug • Person • Environment



RECOVERY IS SHAPED BY THE INTERPLAY OF DRUG, PERSON, AND ENVIRONMENT.

Understanding and addressing all three elements leads to better prevention, treatment, and healthier communities.

How Organisms Heal



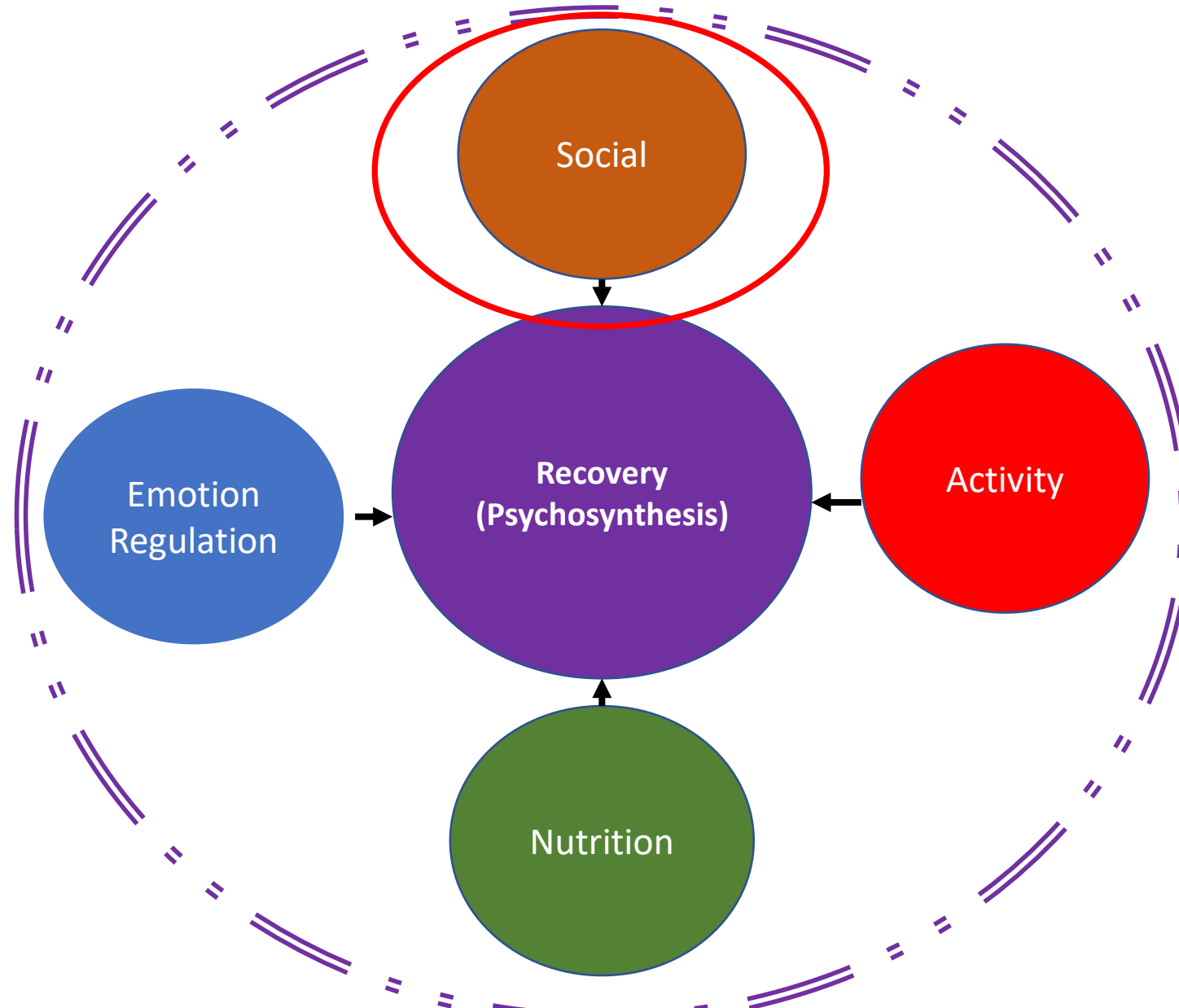
Photosynthesis



Psychosynthesis



A Social Activity Nutrition Emotion Regulation (SANER) Approach to Recovery



Social

Activity

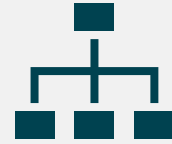
Nutrition

Emotion

Regulation

(**SANER**)

Outline



What is “recovery”: Taxonomy and terminology



How to conceptualize recovery



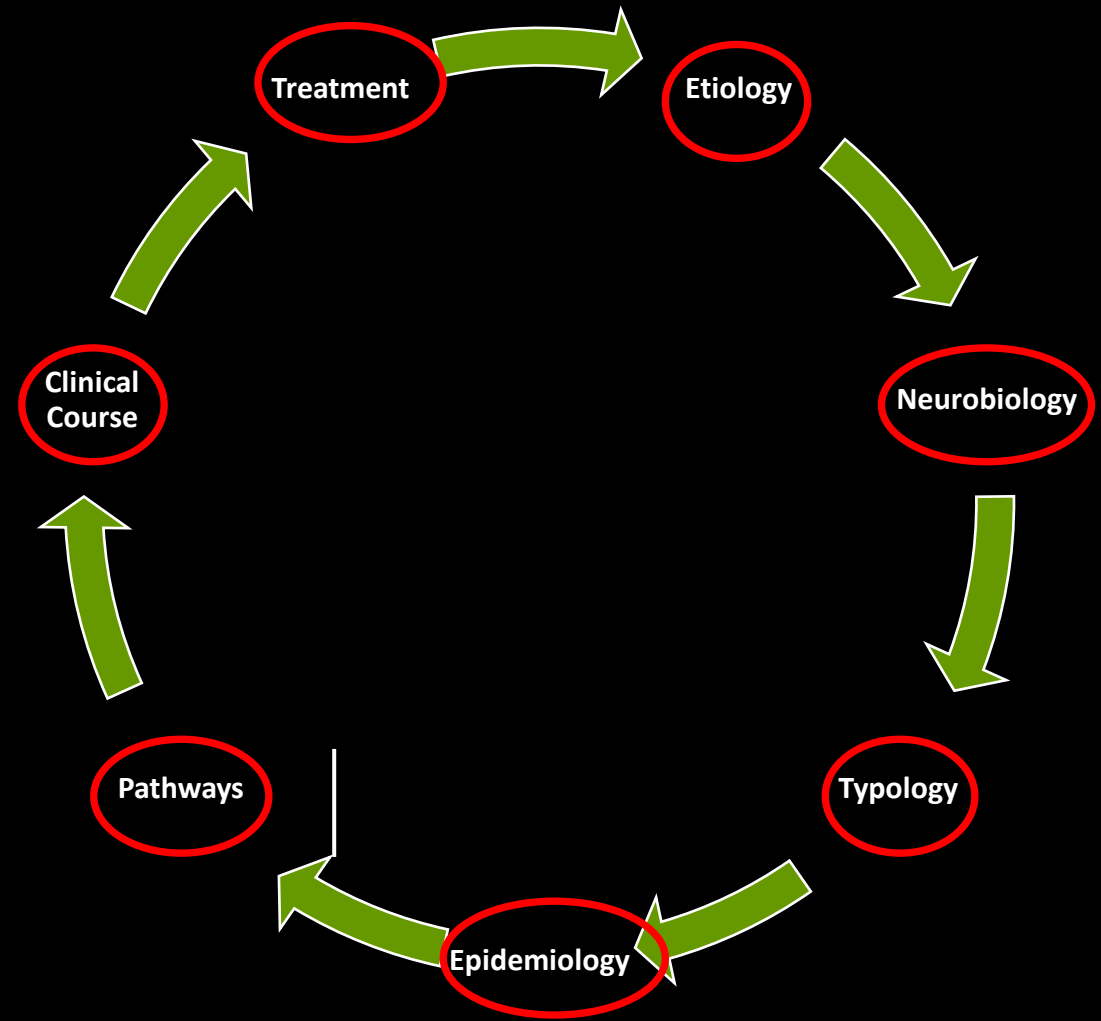
Models: How to achieve stable recovery and reduce and prevent long-term relapse risk

50 years

of treatment and public health advances facilitated largely by NIH and VA funded research has led to progress in many areas addressing addiction ...

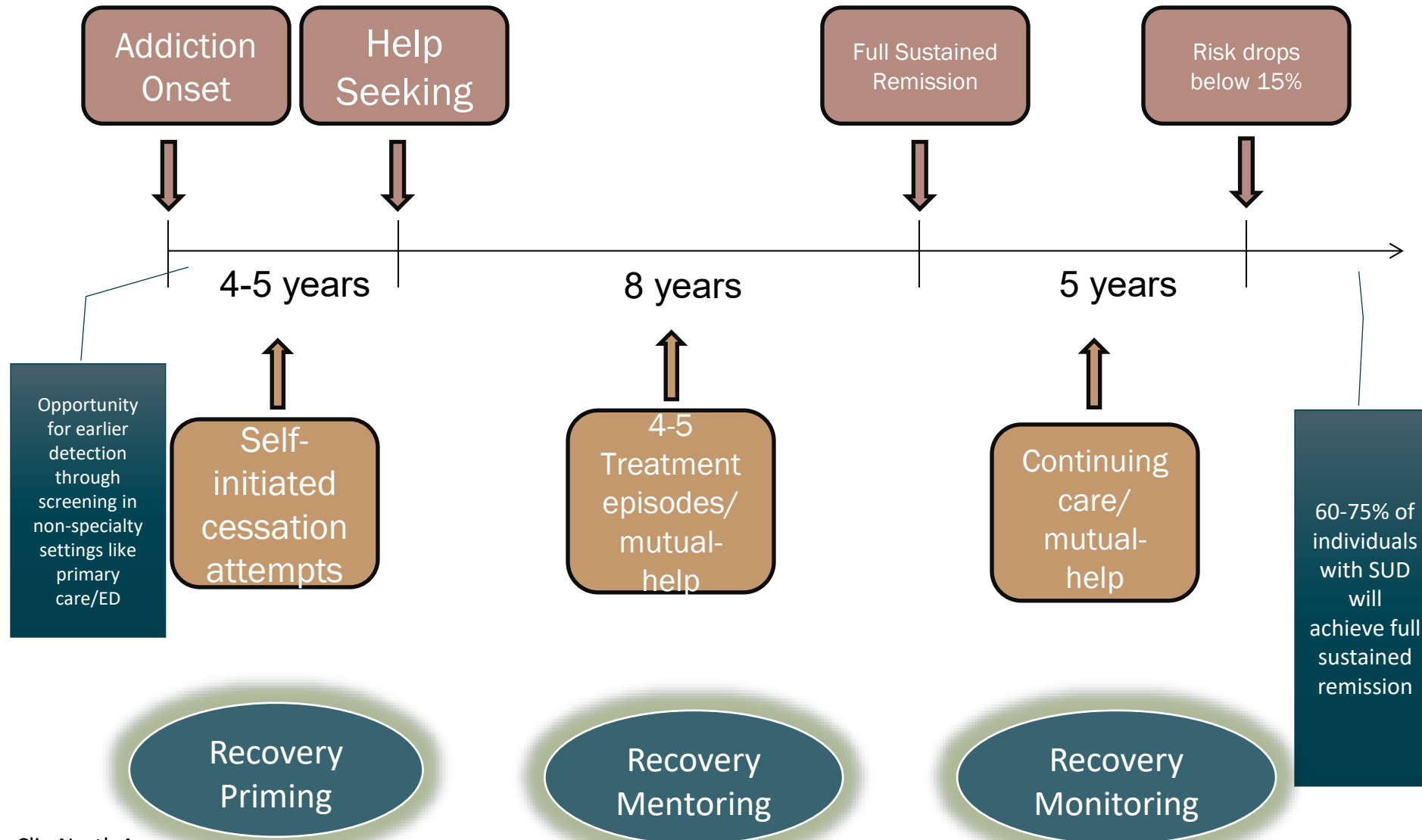


Research has led to a number of paradigm shifts... including different taxonomies and terminologies and contributed to decreased stigma...



The clinical course of addiction and achievement of stable recovery can take a long time ...

Can we speed this up?



50 years of Progress:
Burning building
analogy...

- **Putting out the fire** –addressing acute clinical pathology - good job
- **Preventing it from re-igniting** (RP) - emphasized - pragmatic disconnect...
- **Building materials** (recovery capital) – mostly neglected
- **Scaffolding** (building skills and support beyond acute stabilization)
- **Granting “rebuilding permits”** - (removing barriers - neglected)



Recovery Capital

Individual

(coping, motivation, self-efficacy)

Social

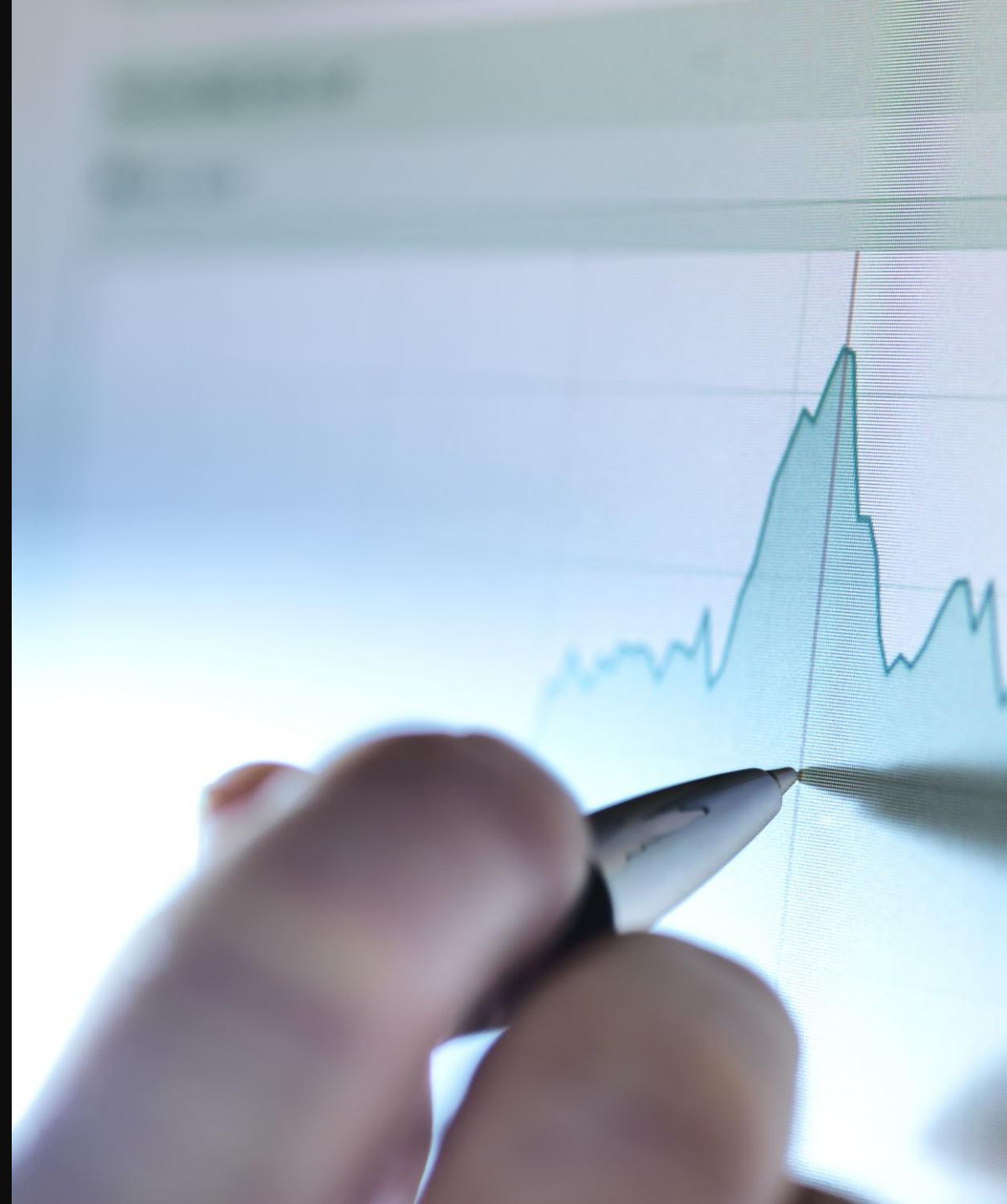
(recovery-specific/family, friends)

Financial

(income, resources)

Cultural

(identity, values)



Multiple Pathways to Recovery

Acknowledges myriad ways in which individuals can recover:

- **Clinical pathways** (provided by a clinician or other medical professional – both medication and psychosocial interventions)
- **Nonclinical pathways** (services not involving clinicians such as Alcoholics Anonymous)
- **Self-management pathways** (recovery change processes that involve no formal services, sometimes referred to as “natural recovery”)



Recovery support services have grown intended to facilitate access to conducive and supportive environments and recovery capital ...



Advantages of
recovery support
services in
disease/recovery
management....

Available

Accessible

Flexible

Enduring

Low/no cost

Recovery support services have grown intended to facilitate access to conducive and supportive environments and recovery capital ...





Cochrane Database of Systematic Reviews

Alcoholics Anonymous and other 12-step programs for alcohol use disorder (Review)

Kelly JF, Humphreys K, Ferri M

Kelly JF, Humphreys K, Ferri M.
Alcoholics Anonymous and other 12-step programs for alcohol use disorder.
Cochrane Database of Systematic Reviews 2020, Issue 3. Art. No.: CD012880.
DOI: [10.1002/14651858.CD012880.pub2](https://doi.org/10.1002/14651858.CD012880.pub2).

www.cochranelibrary.com

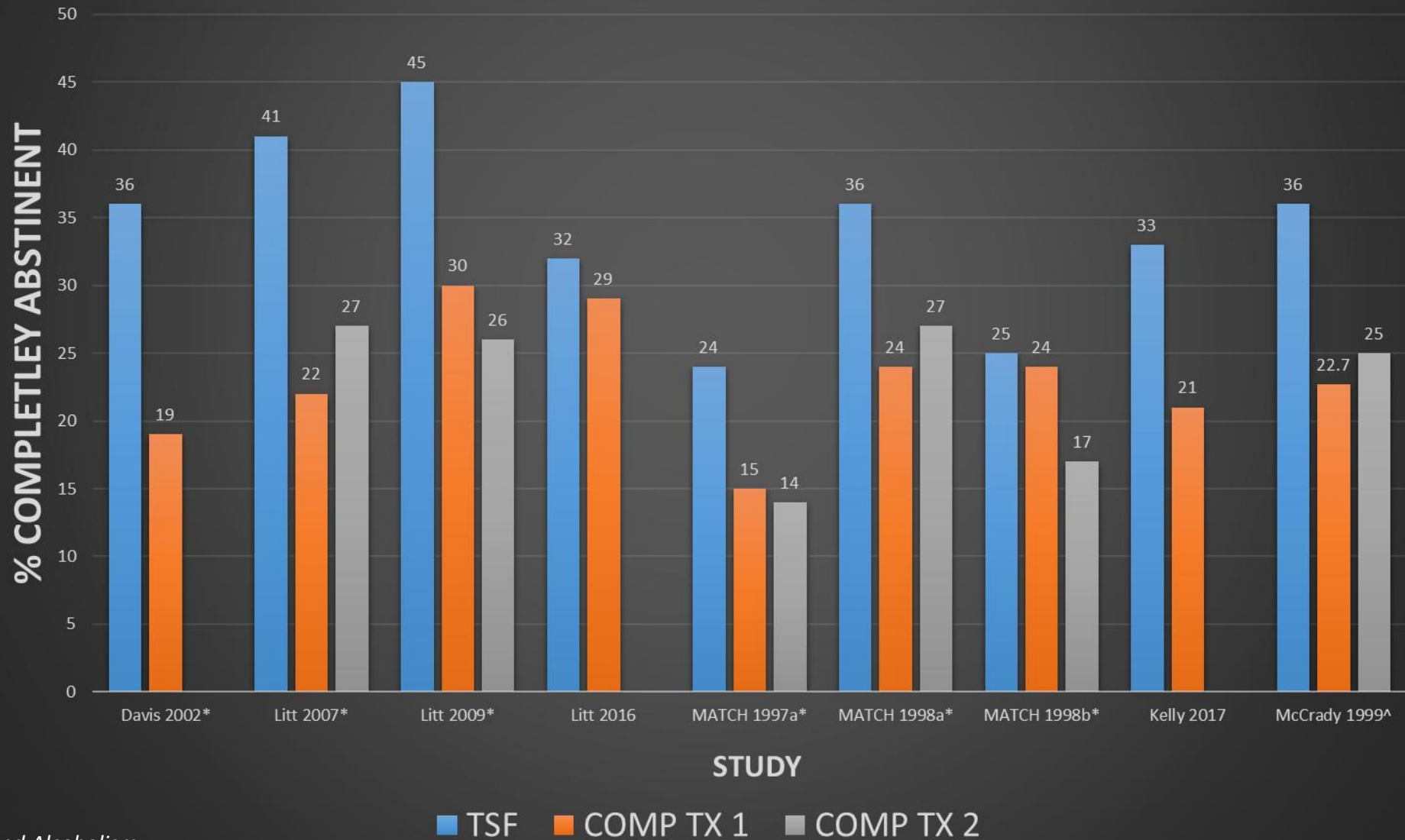
Alcoholics Anonymous and other 12-step programs for alcohol use disorder (Review)
Copyright © 2020 The Cochrane Collaboration. Published by John Wiley & Sons, Ltd.

WILEY

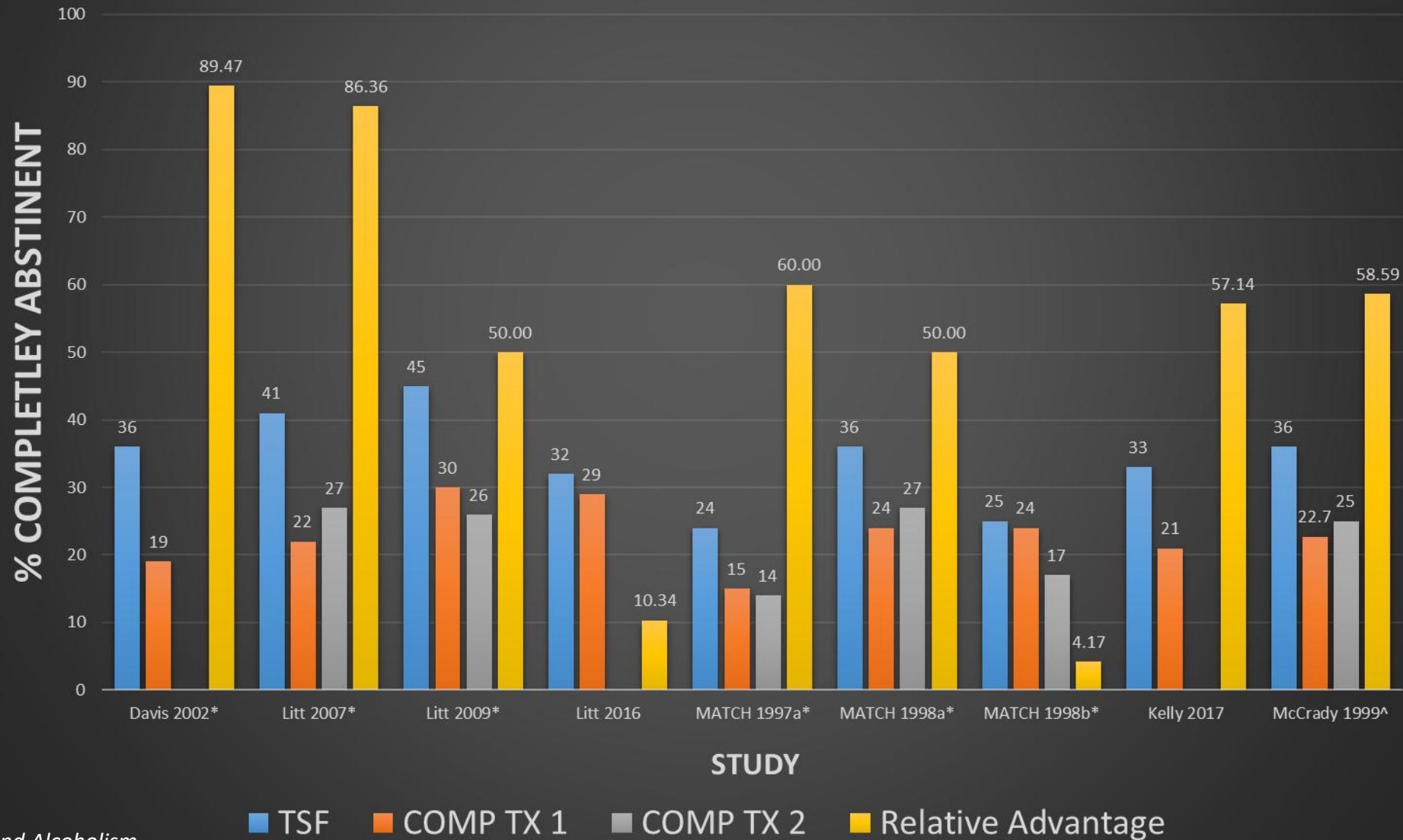
Cochrane Systematic Review on AA/TSF (2020)

- Kelly, JF
- Humphreys, K
- Ferri, M

TSF Compared to Different Theoretical Orientation Treatments (RCTs all Manualized)



TSF Compared to Different Theoretical Orientation Treatments (RCTs all Manualized)



Economic Studies

Healthcare Cost Savings

- 3/4 included studies in this category (n reports = 4/5; found sig. health care cost saving in favor of the AA/TSF condition.

- While AA is proven to help, not everyone wants to use AA
- Increasing the menu of recovery mutual-help support options is likely to engage more individuals in the recovery process

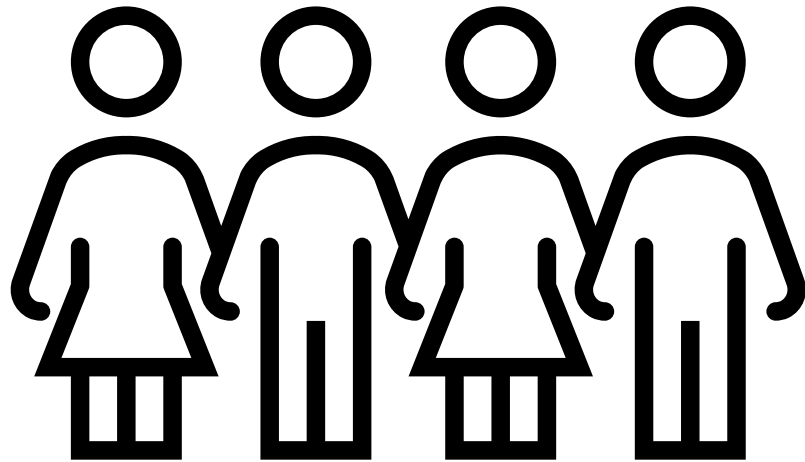
Do Fitness Centers Keep people fit?



- Of course!
- If you go and if you work out regularly
- Ongoing challenge is engaging and retaining people in some kind of ongoing exercise regimen...
- Fitness Centers therefore provide not just one, but an array, of different classes, spaces, equipment, pools, and courts, so that people can find something appealing...
- ...and move toward increasing physical fitness

Emerging Evidence for Additional Mutual-Help Organizations....

J Subst Abuse Treat. 2017 February ; 73: 16–26. doi:10.1016/j.jsat.2016.10.004.



Comparison of 12-step Groups to Mutual Help Alternatives for AUD in a Large, National Study: Differences in Membership Characteristics and Group Participation, Cohesion, and Satisfaction

Sarah E. Zemore, Ph.D., Lee Ann Kaskutas, Dr.P.H., Amy Mericle, Ph.D., and Jordana Hemberg, MPH

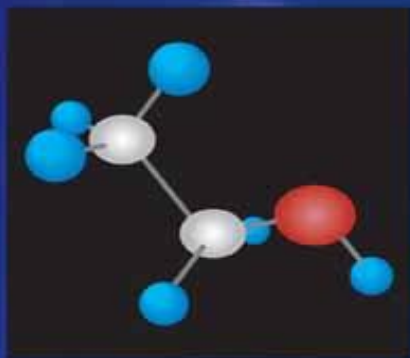
Alcohol Research Group, Emeryville, CA

Abstract

Background—Many studies suggest that participation in 12-step groups contributes to better recovery outcomes, but people often object to such groups and most do not sustain regular involvement. Yet, research on alternatives to 12-step groups is very sparse. The present study aimed to extend the knowledge base on mutual help group alternatives for those with an alcohol use disorder (AUD), sampling from large, active, abstinence-focused groups including Women for Sobriety (WFS), LifeRing, and SMART Recovery (SMART). This paper presents a cross-sectional

ALCOHOL

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RESEARCH ARTICLE



Who affiliates with SMART recovery? A comparison of individuals attending SMART recovery, alcoholics anonymous, both, or neither

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Abstract

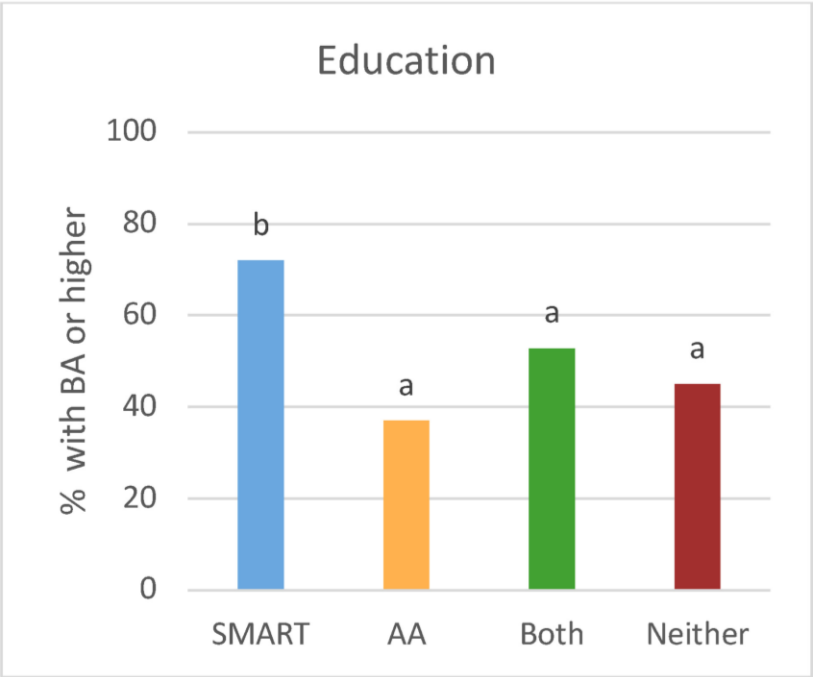
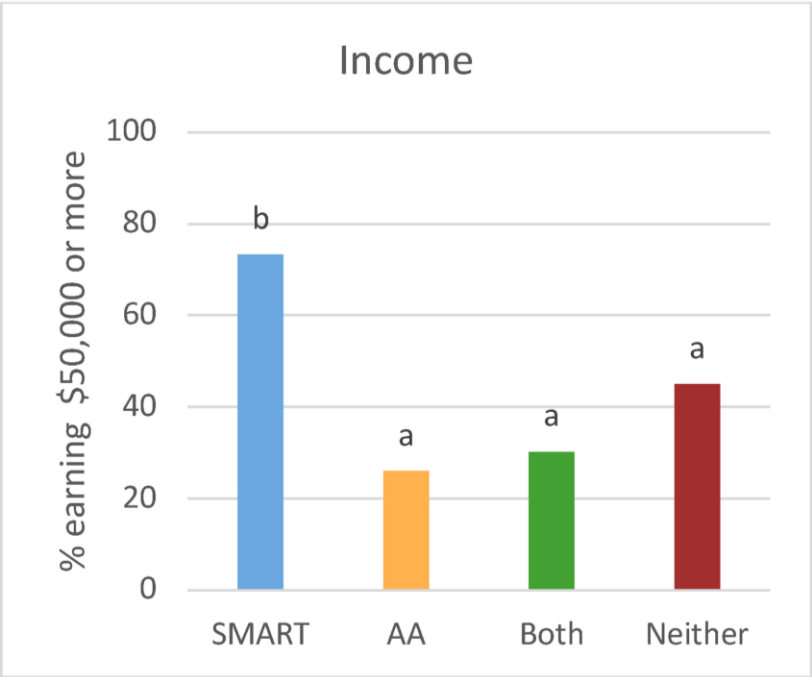
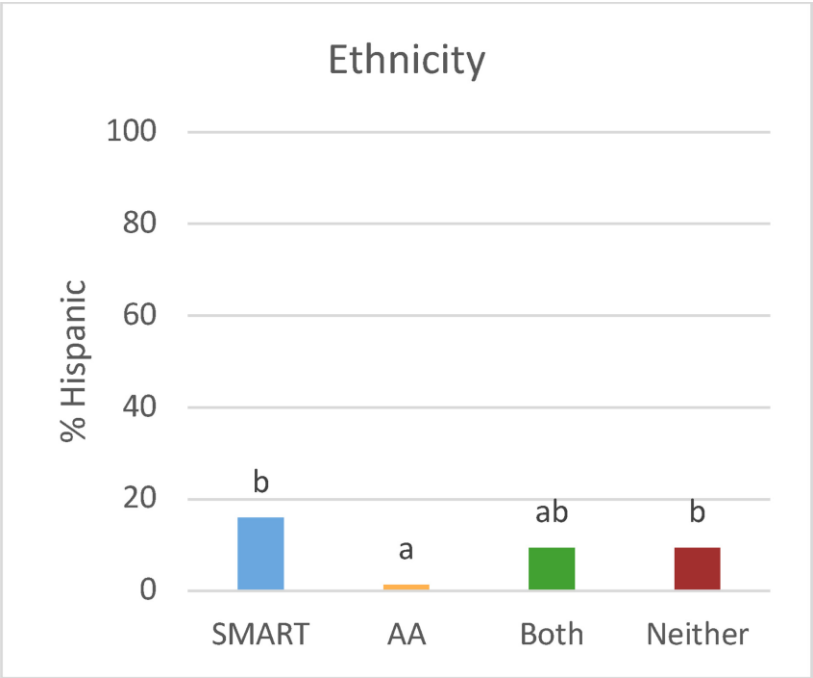
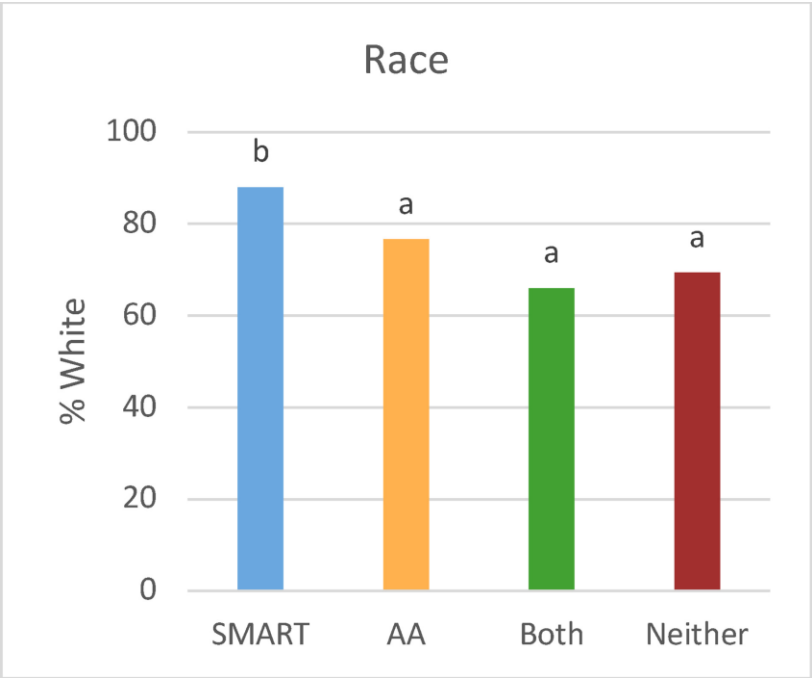
Background: Mutual-help organizations (MHOs) play a crucial role for many individuals with alcohol use disorder (AUD) or other substance use disorders in achieving stable remission. While there is now substantial research characterizing who uses 12-step MHOs, very little is known about who becomes affiliated with newer and rapidly growing MHOs, such as Self-Management and Recovery Training ("SMART" Recovery). More research could inform knowledge regarding who may be best engaged by these differing pathways.

Methods: We conducted a cross-sectional analysis of participants ($N = 361$) with AUD recruited mostly from the community who were starting a new recovery attempt and self-selected into one of four different recovery paths: (1) SMART Recovery ("SMART-only"; $n = 75$); (2) Alcoholics Anonymous ("AA-only"; $n = 73$); (3) Both SMART and AA ("Both"; $n = 53$); and (4) Neither SMART nor AA ("Neither"; $n = 160$). We compared the groups on demographics, clinical history, treatment and recovery support service use, and indices of functioning and well-being. We computed descriptives and conducted inferential analyses according to the data structure.

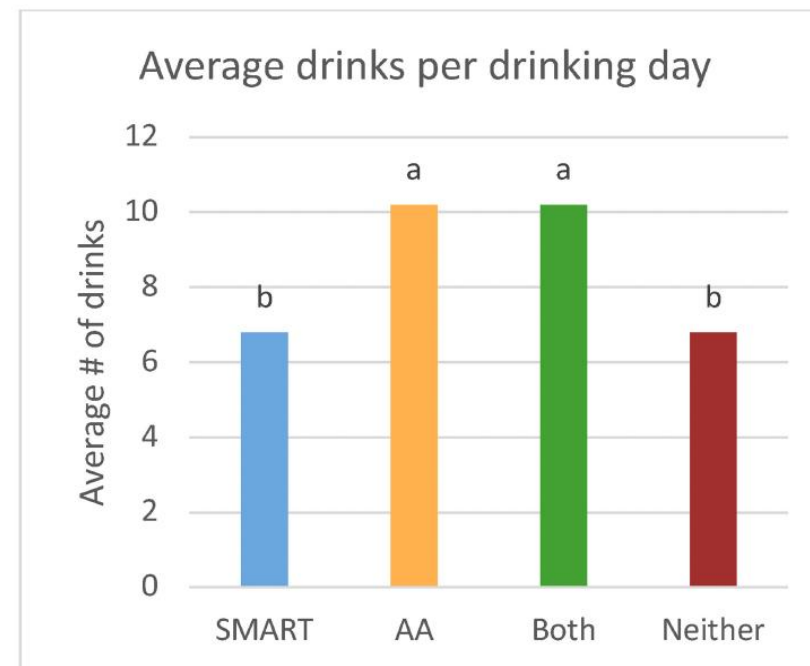
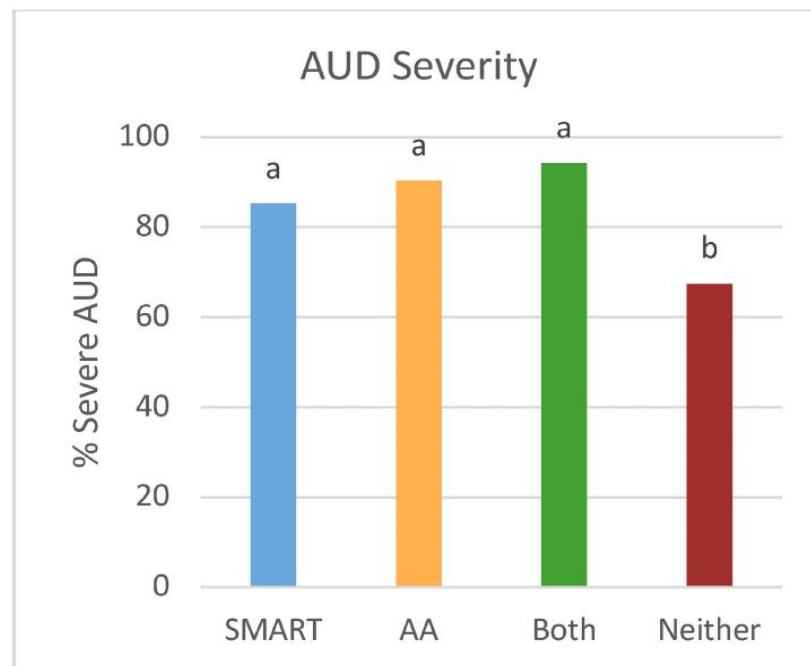
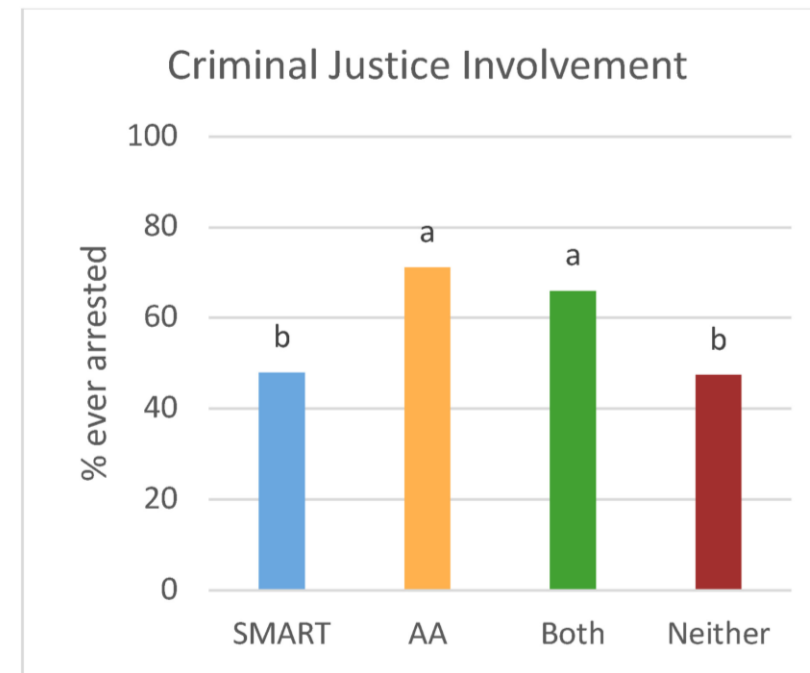
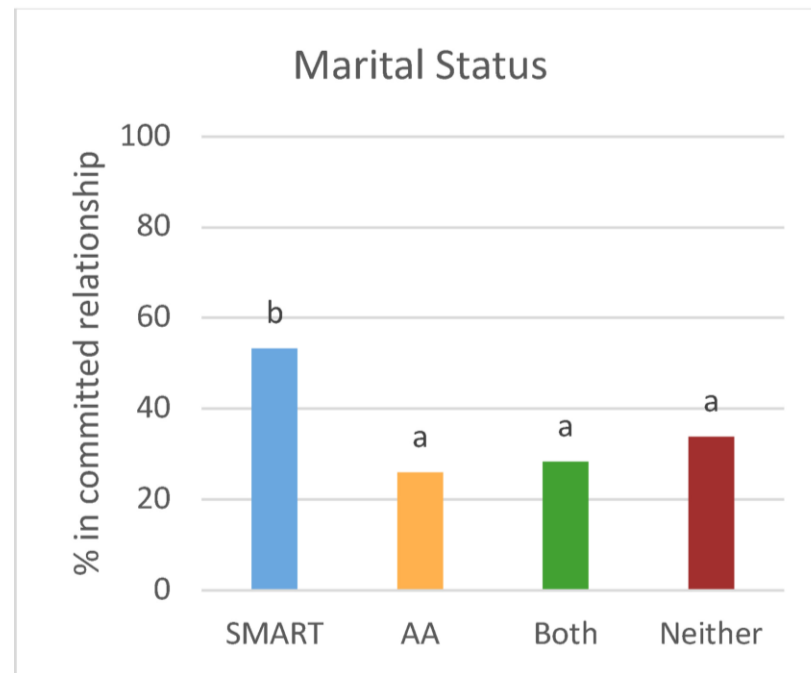
Results: Compared to study participants choosing AA-only or Both, SMART-only participants were more likely to be White, married, have higher income and more education, be full-time employed, and evince a pattern of lower clinical severity characterized by less lifetime and recent treatment and recovery support services usage, lower alcohol use intensity and fewer consequences, and less legal involvement. AUD symptom levels, lifetime psychiatric diagnoses, psychiatric distress, and functioning were similar across MHO-engaged groups.

Conclusion: SMART Recovery appears to attract individuals with greater psychosocial stability and economic advantage and less severe histories of alcohol-related impairment and legal involvement. Findings suggest that certain aspects specific to the SMART Recovery group approach, format, and/or contents may appeal to individuals exhibiting this type of profile. As such, SMART appears to provide an additional

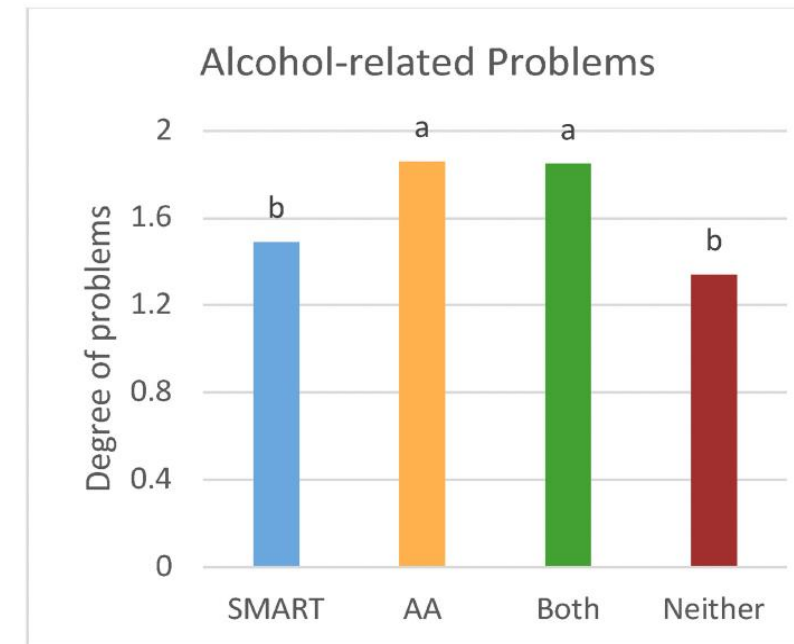
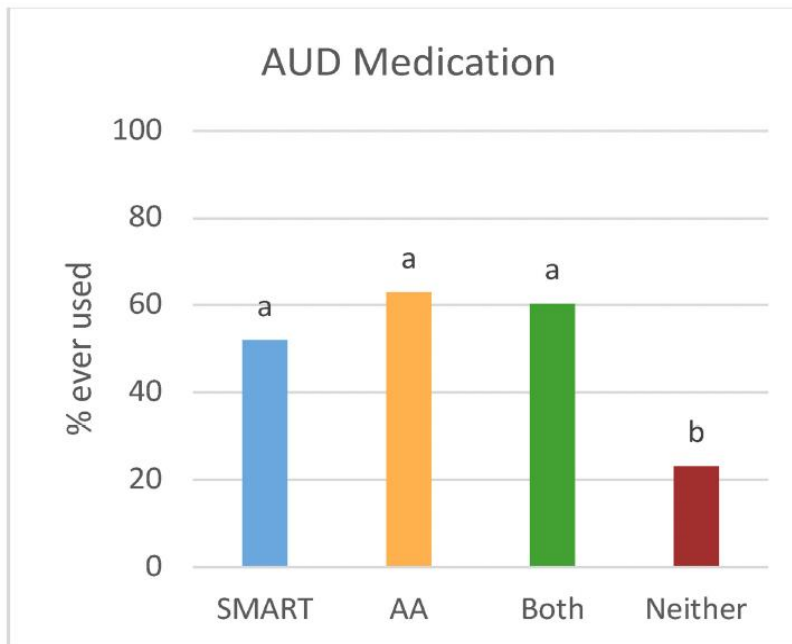
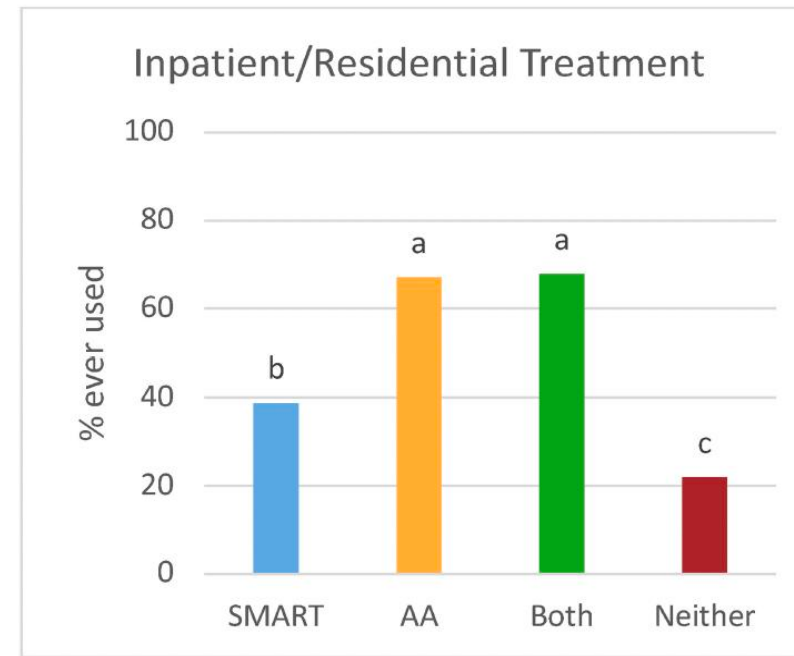
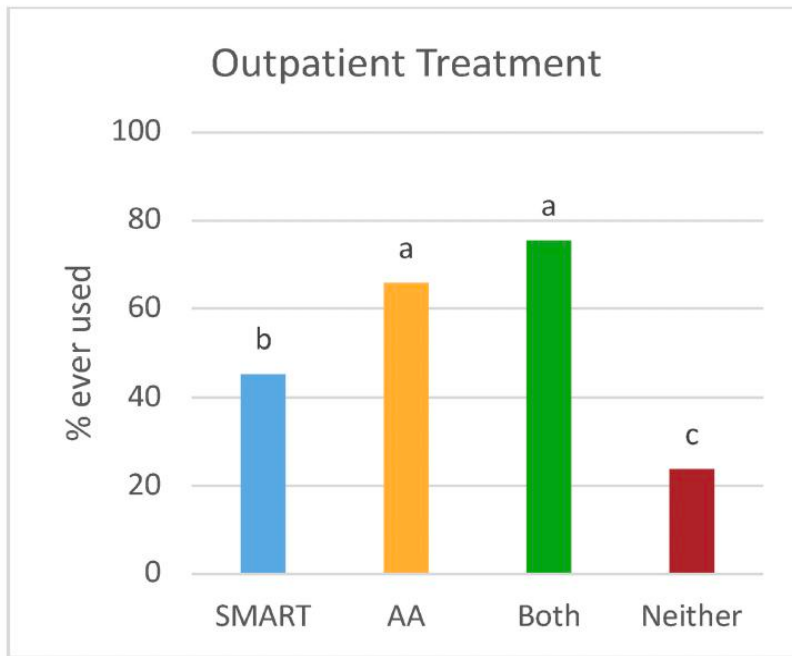
Results



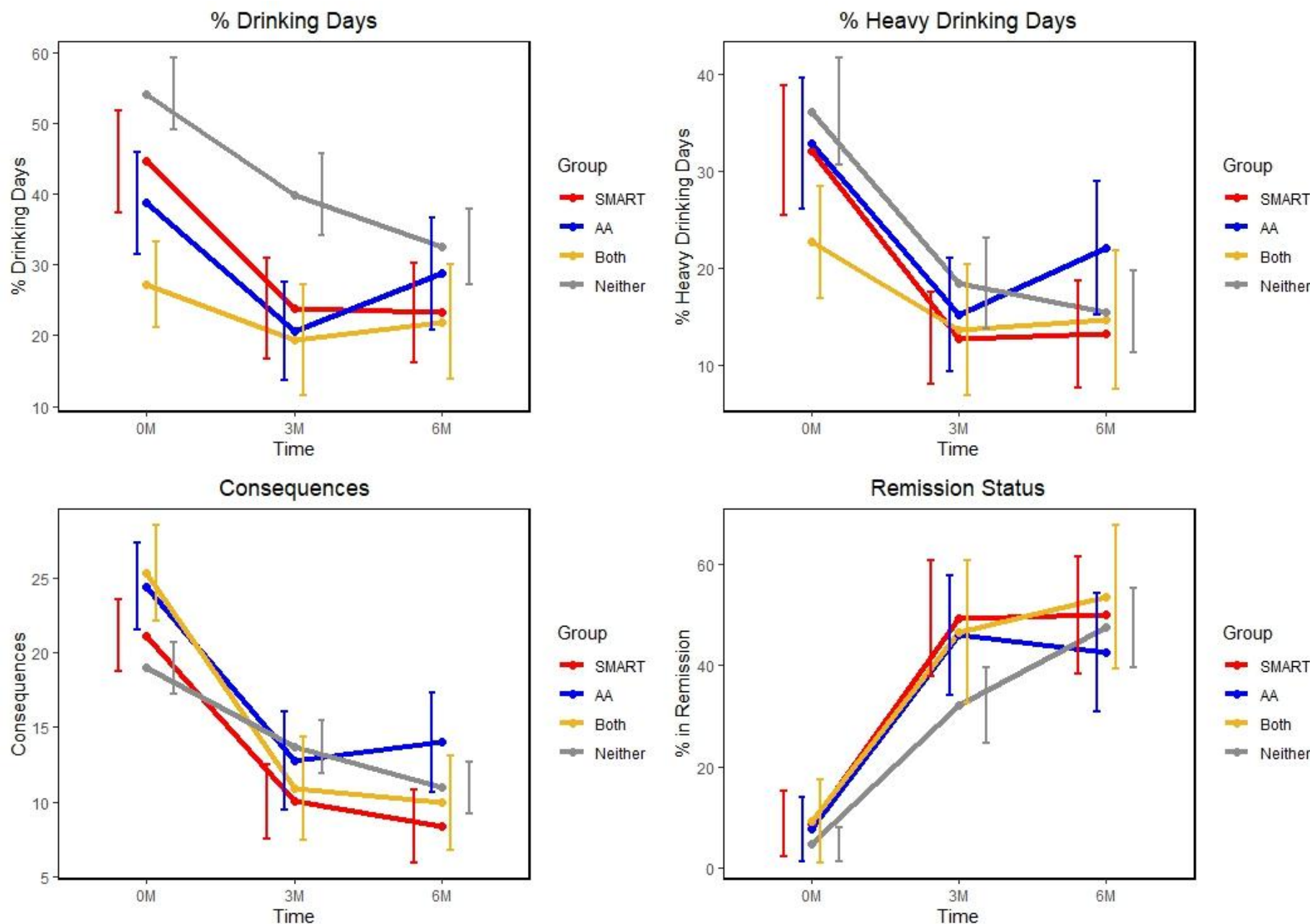
Results



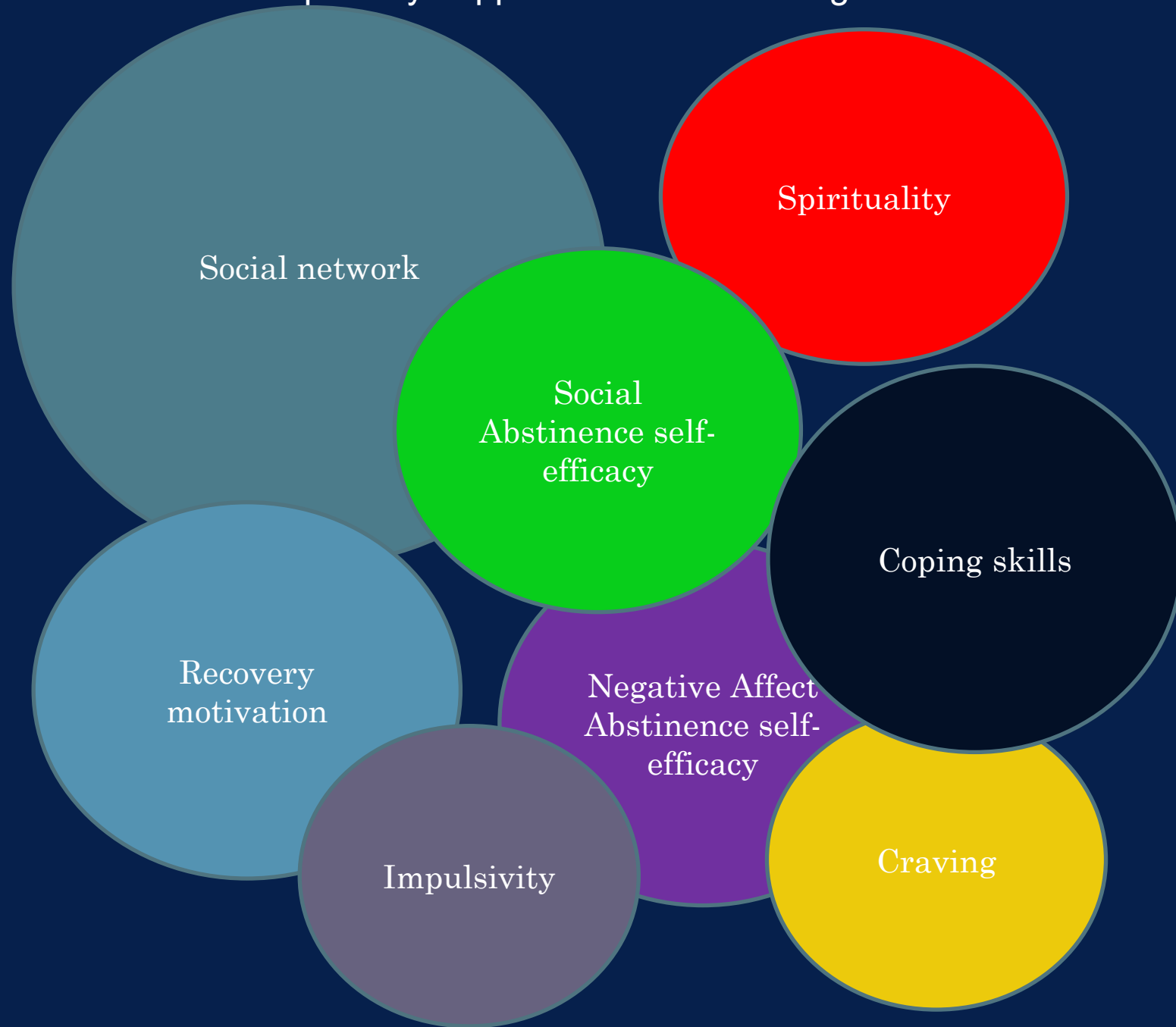
Results



SMART vs AA vs Both vs Neither: Group Effects over Time on four outcomes across 6 months

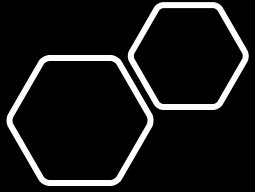


Empirically-supported MOBCs through which AA confers benefit



Recovery support services have grown intended to facilitate access to conducive and supportive environments and recovery capital ...

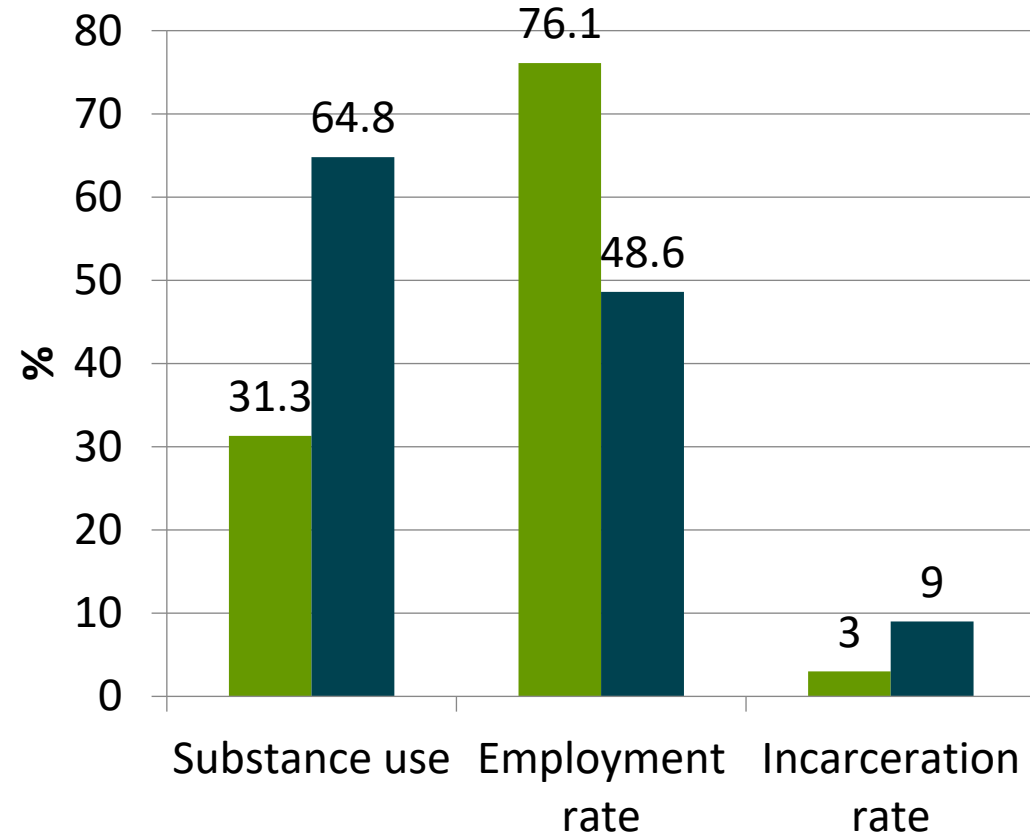




Oxford House vs. Usual Care

Recovery Residences had –

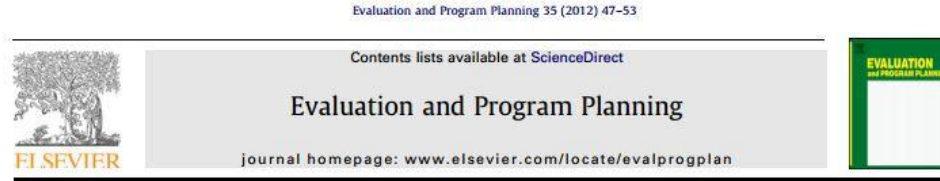
- half as many using substances across 2 yrs
- 50% more employed
- 1/3 re-incarceration rate



■ Oxford House

■ Usual Care

Cost-benefit analysis of the Oxford House Model



Benefits and costs associated with mutual-help community-based recovery homes: The Oxford House model

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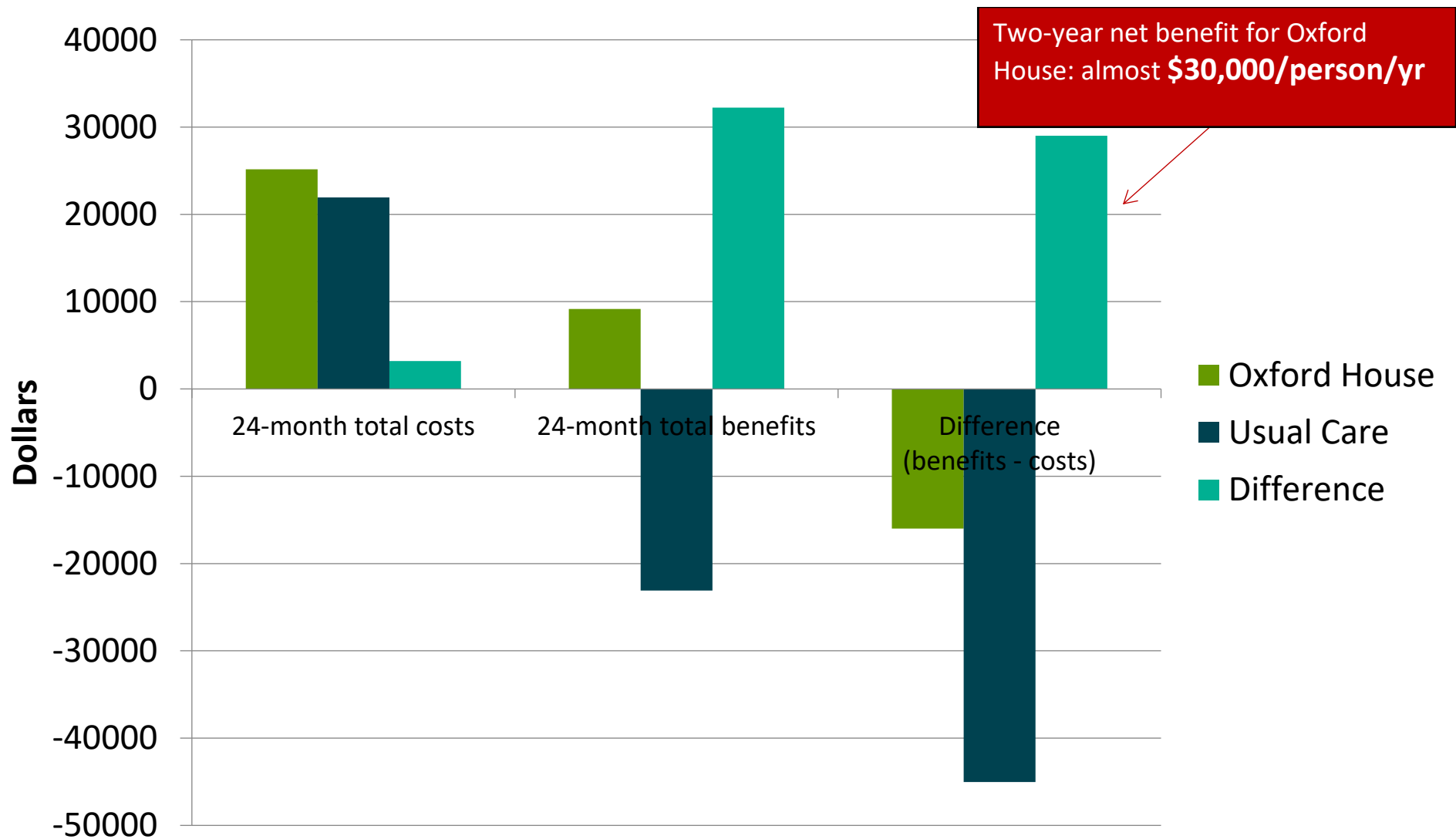
ABSTRACT

We used data from a randomized controlled study of *Oxford House* (OH), a self-run, self-supporting recovery home, to conduct a cost-benefit analysis of the program. Following substance abuse treatment, individuals that were assigned to an OH condition ($n = 68$) were compared to individuals assigned to a usual care condition ($n = 61$). Economic cost measures were derived from length of stay at an Oxford House residence, and derived from self-reported measures of inpatient and outpatient treatment utilization. Economic benefit measures were derived from self-reported information on monthly income, days participating in illegal activities, binary responses of alcohol and drug use, and incarceration. Results suggest that OH compared quite favorably to usual care: the net benefit of an OH stay was estimated to be roughly \$29,000 per person on average. Bootstrapped standard errors suggested that the net benefit was statistically significant. Costs were incrementally higher under OH, but the benefits in terms of reduced illegal activity, incarceration and substance use substantially outweighed the costs. The positive net benefit for Oxford House is primarily driven by a large difference in illegal activity between OH and usual care participants. Using sensitivity analyses, under more conservative assumptions we still arrived at a net benefit favorable to OH of \$17,830 per person.

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- **Sample:** 129 adults leaving substance use treatment between 2002 and 2005
- **Design:** Cost-benefit analysis using RCT data
- **Intervention:** Oxford House vs. usual continuing care
- **Follow-up:** 2 years
- **Outcome:** Substance use, monthly income, incarceration rates

Mean per-person societal benefits and costs



Recovery support services have grown intended to facilitate access to conducive and supportive environments and recovery capital ...





One-Stop Shopping for Recovery: An Investigation of Participant Characteristics and Benefits Derived From U.S. Recovery Community Centers

John F. Kelly , Robert L. Stout, Leonard A. Jason, Nilofar Fallah-Sohy, Lauren A. Hoffman, and Bettina B. Hoepfner

Background: Recovery community centers (RCCs) are the “new kid on the block” in providing addiction recovery services, adding a third tier to the 2 existing tiers of formal treatment and mutual-help organizations (MHOs). RCCs are intended to be recovery hubs facilitating “one-stop shopping” in the accrual of recovery capital (e.g., recovery coaching; employment/educational linkages). Despite their growth, little is known about who uses RCCs, what they use, and how use relates to improvements in functioning and quality of life. Greater knowledge would inform the field about RCC’s potential clinical and public health utility.

Methods: Online survey conducted with participants ($N = 336$) attending RCCs ($k = 31$) in the northeastern United States. Substance use history, services used, and derived benefits (e.g., quality of life) were assessed. Systematic regression modeling tested *a priori* theorized relationships among variables.

Results: RCC members ($n = 336$) were on average 41.1 ± 12.4 years of age, 50% female, predominantly White (78.6%), with high school or lower education (48.8%), and limited income (45.2% < \$10,000 past-year household income). Most had either a primary opioid (32.7%) or alcohol (26.8%) problem. Just under half (48.5%) reported a lifetime psychiatric diagnosis. Participants had been attending RCCs for 2.6 ± 3.4 years, with many attending <1 year (35.4%). Most commonly used aspects were the socially oriented mutual-help/peer groups and volunteering, but technological assistance and employment assistance were also common. Conceptual model testing found RCCs associated with increased recovery capital, but not social support; both of these theorized proximal outcomes, however, were related to improvements in psychological distress, self-esteem, and quality of life.

Conclusions: RCCs are utilized by an array of individuals with few resources and primary opioid or alcohol histories. Whereas strong social supportive elements were common and highly rated, RCCs appear to play a more unique role not provided either by formal treatment or by MHOs in facilitating the acquisition of recovery capital and thereby enhancing functioning and quality of life.

Key Words: Recovery Community Centers, Recovery, Addiction, Support Services, Recovery Coaching, Addiction, Substance Use Disorder.

PROFESSIONAL TREATMENT SERVICES often play a vital role in addressing substance use disorders in the United States and around the world. Such clinical services can provide life-saving medically managed detoxification and stabilization as well as deliver medications and psychosocial interventions that can alleviate cravings and help prevent relapse. Extending the framework and benefits of these professional treatment efforts, peer-led mutual-help

organizations (MHOs), such as Alcoholics Anonymous (AA), Narcotics Anonymous (NA), SMART Recovery, and many others are commonly used to provide additional long-term free recovery support over time in the communities in which people live (Bog et al., 2017; Kelly, 2017; Kelly et al., 2017a). Adding to these resources in recent years has been a new dimension of recovery support services that are neither professional treatment nor MHOs. These new services (e.g., recovery community centers [RCCs], recovery residences, recovery coaching, recovery high schools, and collegiate recovery programs; Kelly et al., *in press*; White et al., 2012, 2012) combine voluntary, peer-led initiatives, with professional activities, and are intended to provide flexible community-based options to address the psychosocial barriers to sustained remission (White et al., 2012, 2012).

RCCs are one of the most common of these new additions to recovery support infrastructure and are growing rapidly (Cousins et al., 2012; Kelly et al., *in press*; Kelly et al., 2017b). RCCs are literally and metaphorically, “new kids on the block,” as these novel entities are most often located on

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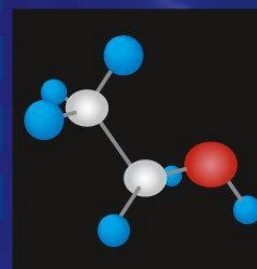
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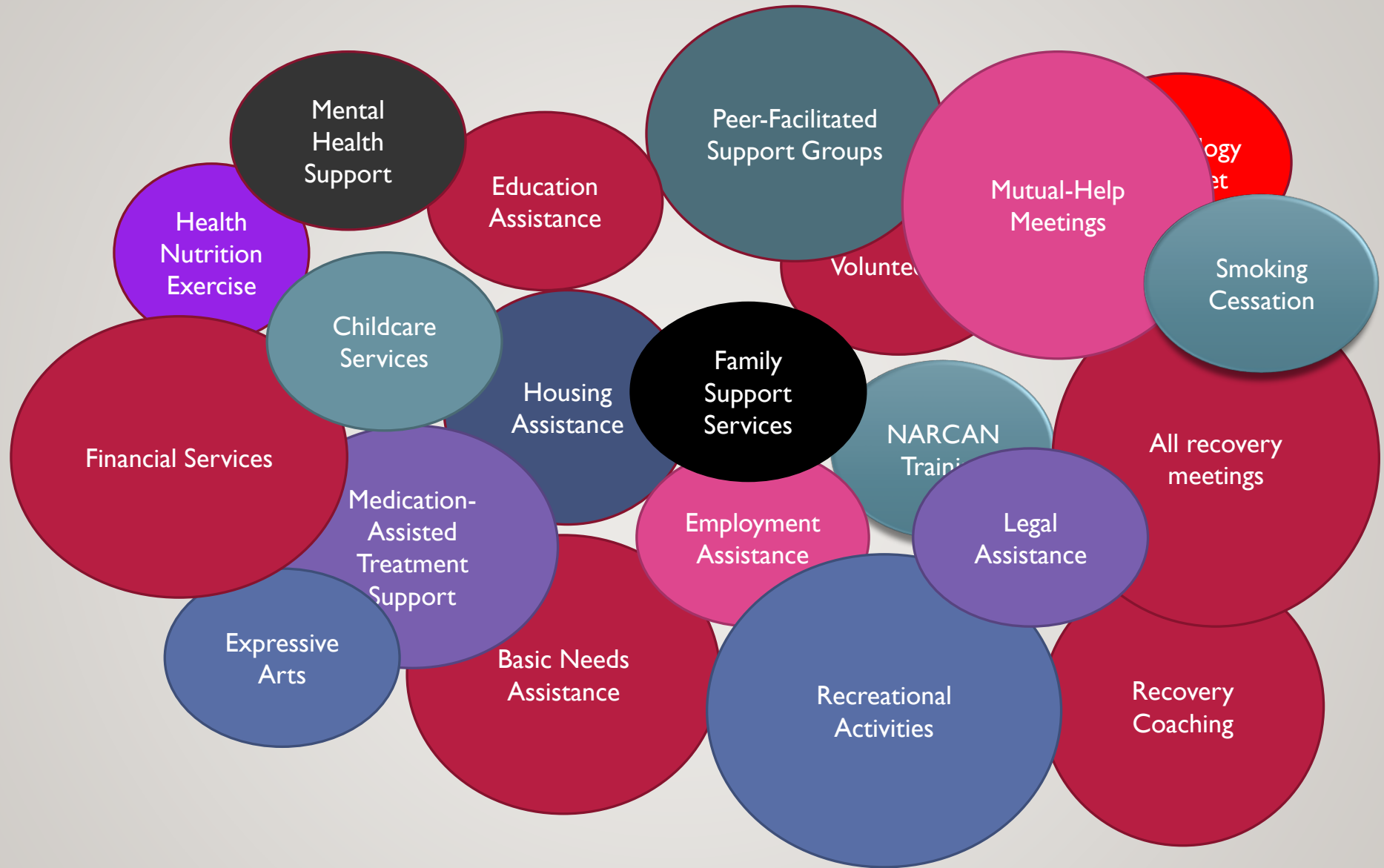
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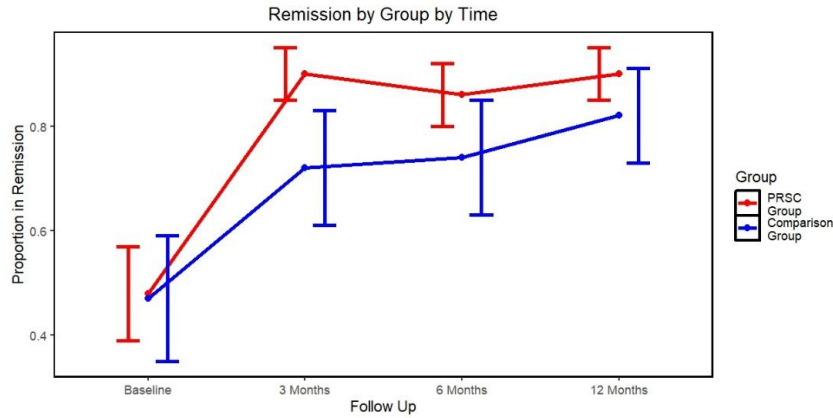


Founded in 1977 by the National Council on Alcoholism
(Now National Council on Alcoholism and Drug Dependence, Inc.)

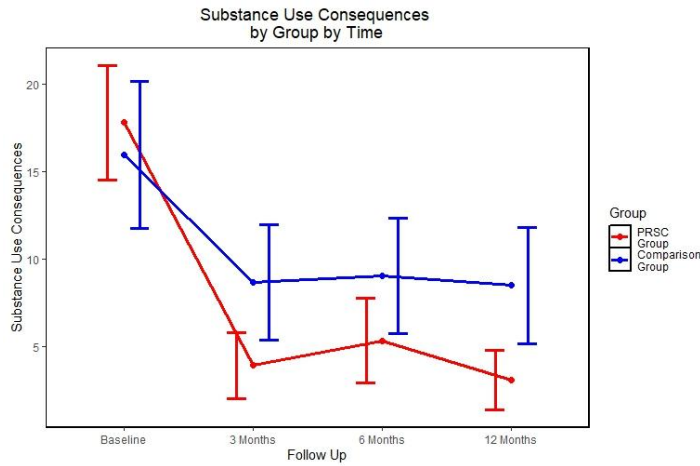
SERVICES PROVIDED



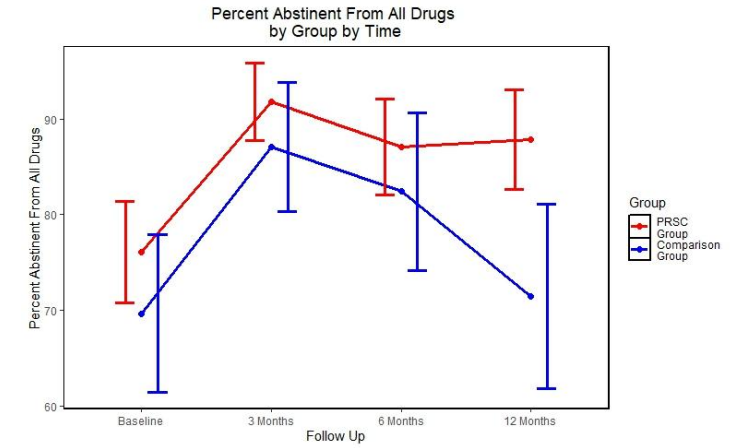
Higher Remission Rates



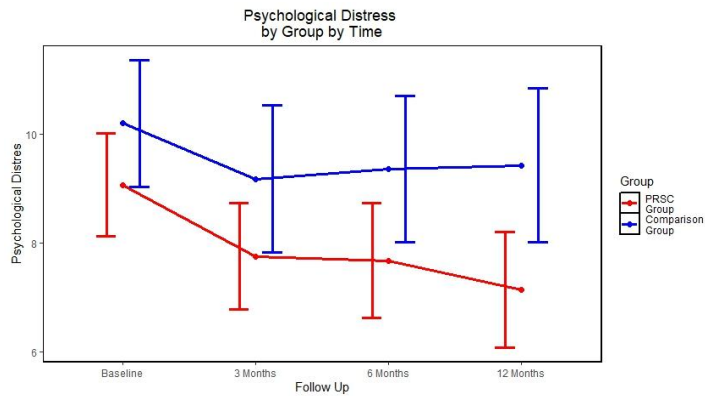
Fewer Substance-Related Consequences



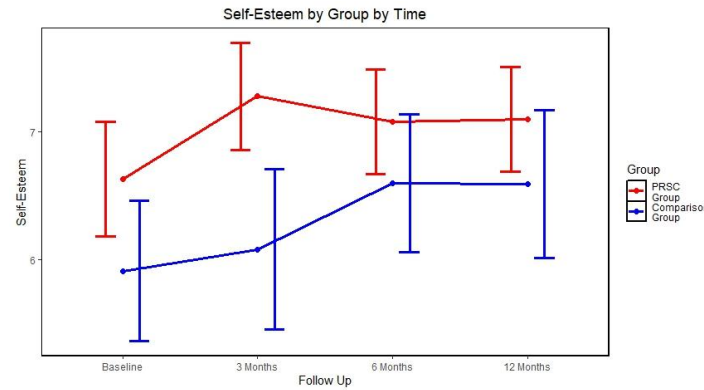
Higher Abstinence Rates



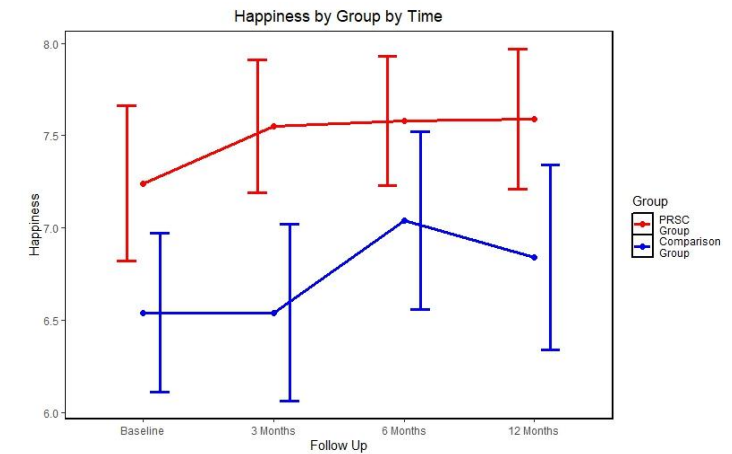
Fewer Psychiatric Problems



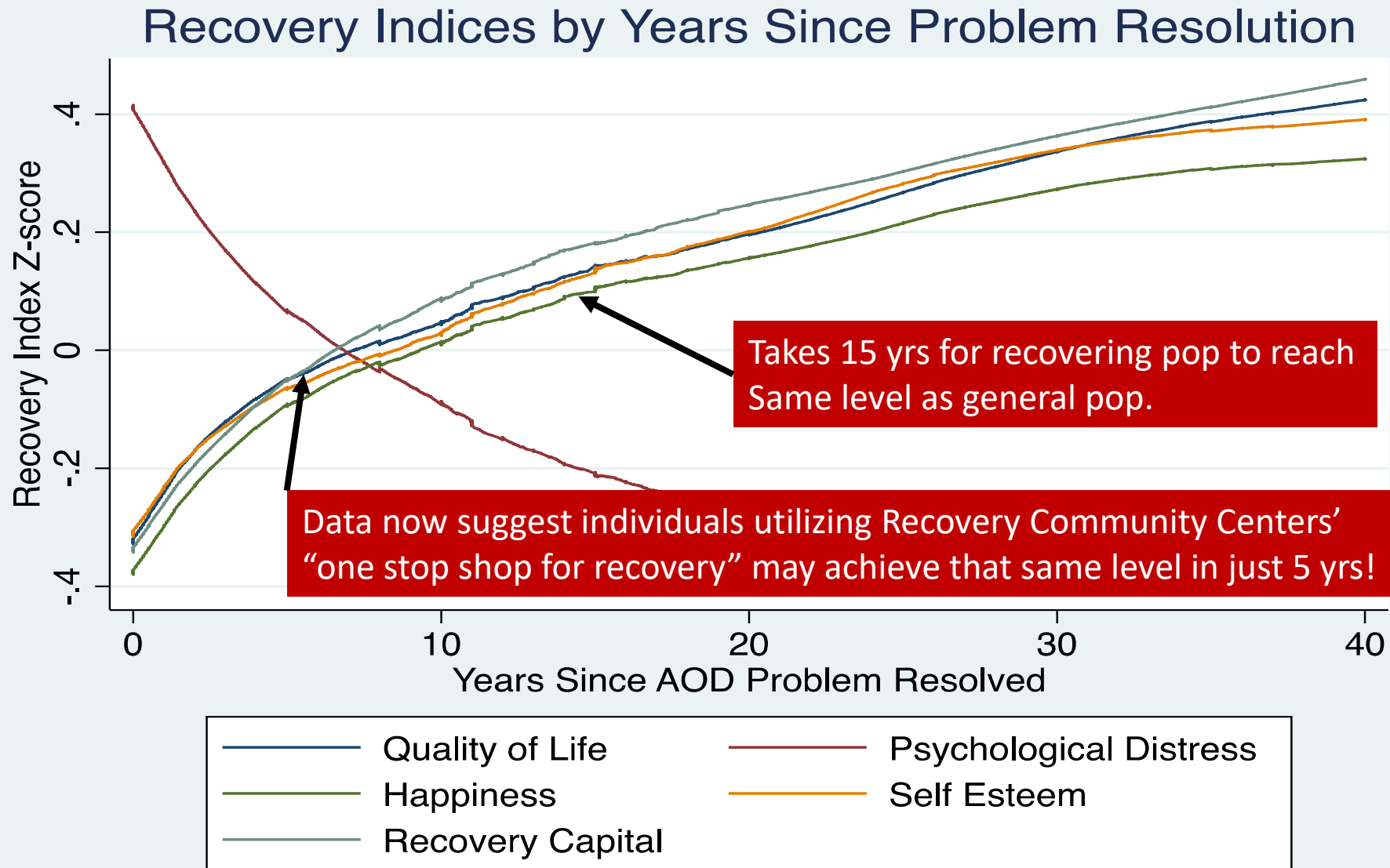
Higher Self-Esteem and Pos. Soc. Identity



Higher Life Satisfaction



40-Year Time Frame of Recovery Trajectories

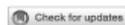


National
Recovery Study
(NRS)
N=2,002

How do people sustain change over the long-term?

Relapse + recurrence vs resilience + recovery

Long-Term Relapse - Markers, Mechanisms, and Implications for Disease Management



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Long-term relapse: markers, mechanisms, and implications for disease management in alcohol use disorder

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and Alexandra Abry

Department of Psychiatry, Recovery Research Institute, Massachusetts General Hospital, Harvard
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Objective: Much has been theorized and documented about factors involved in alcohol use disorder (AUD) relapse during the early months following a recovery attempt where biobehavioral classical conditioning ("cues/triggers") and neurophysiological explanatory theories predominate. Little has been documented, however, about long-term relapse (LTR) factors following sustained AUD remission where self-regulation and stress and coping theories may predominate because LTR precursors are centered less around neurophysiological dysregulation and cue reactivity and more around factors such as lowered recovery vigilance, avoidant coping, or changes in recovery-support services (RSS) usage. Greater knowledge of factors involved in LTR could sensitize and empower clinicians to deliver more effective disease management protocols to monitor and intervene upon such risks prior to AUD recurrence.

Methods: Cross-sectional, retrospective study of individuals in recovery from primary AUD ($N = 50$; 44% Female; 50% White) who had experienced LTR within the past 5 years following at least 1 year of remission (M years remitted prior to relapse = 3.6; range = 1–23) and assessed for any change in bio-psycho-social domains or RSS usage during the year prior to LTR, along with their attributions of factors' contribution to relapse (risk "potency"; i.e., didn't contribute, possibly, probably, or definitely, contributed). Research questions focused on the year preceding the LTR assessing: (1) prevalence and nature of the bio-psycho-social and RSS use changes and degree of attributed LTR risk potency; (2) number and type of definitely-contributing relapse factors within participants; (3) dynamic temporal onset and nature of high-risk LTR precipitants; (4) single most influential LTR risk factor.

Results: Several bio-psycho-social and RSS changes occurred during the year preceding LTR varying in prevalence and potency. Some were prevalent, but not potent, in terms of definitely contributing to LTR (e.g., sleep, energy); others occurred infrequently, but were potent (e.g., physical pain, recreational drug use); others were both highly prevalent and highly potent (e.g., change in recovery vigilance). Within participants, median number of definitely contributing LTR factors = 4, covering 2 different domains, on average. Temporal accumulation of LTR risks tended to intensify toward the relapse horizon over the preceding year. The single most important relapse factor tended to cluster in psychological (e.g., recovery vigilance, mental health) and social domains.

Conclusions: Findings have implications for long-term disease management during AUD recovery providing a set of potential preliminary markers and mechanisms that might be assessed, monitored, and, when necessary,

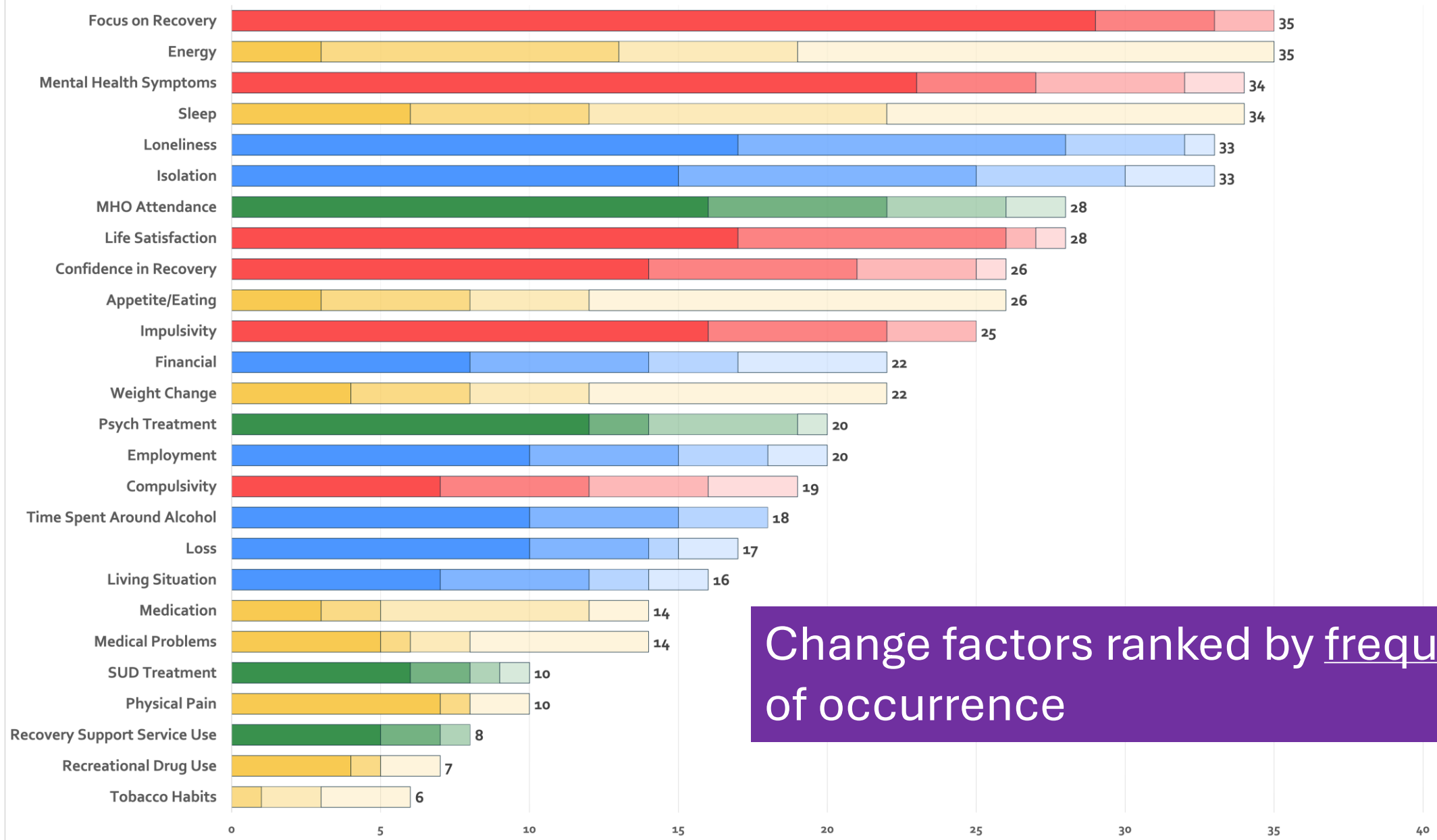


frontiers



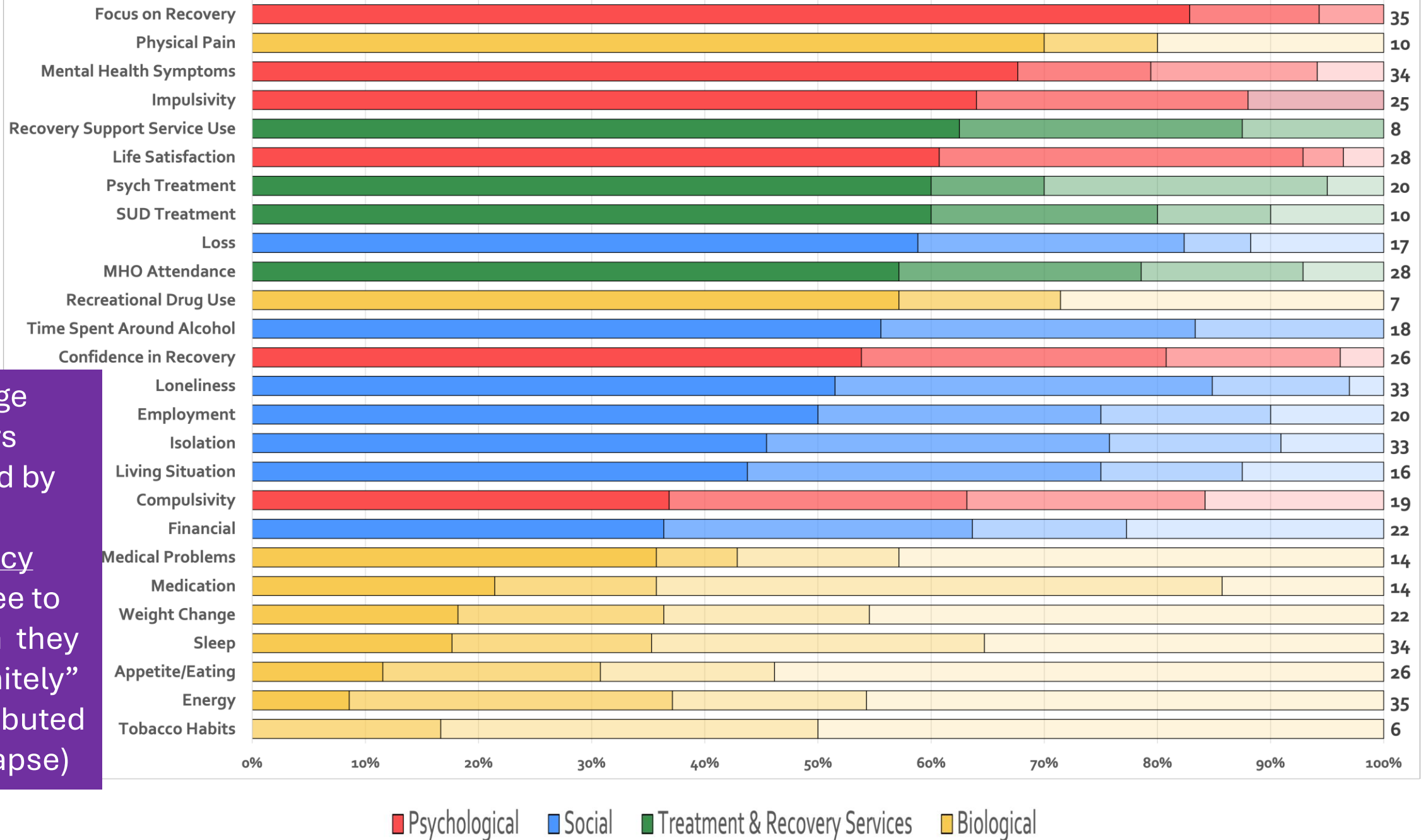
frontiers in Public Health

SUMMARY OF FACTORS PRECIPITATING RETURN TO USE



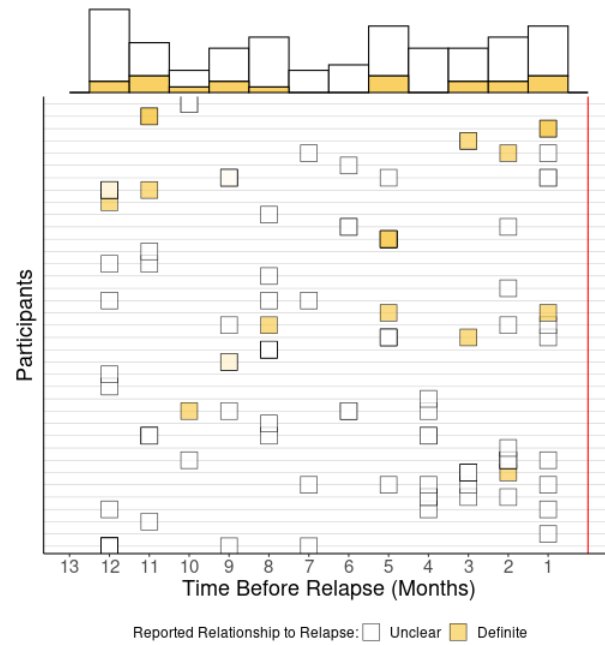
Change factors ranked by frequency of occurrence

FACTORS PRECIPITATING RETURN TO USE

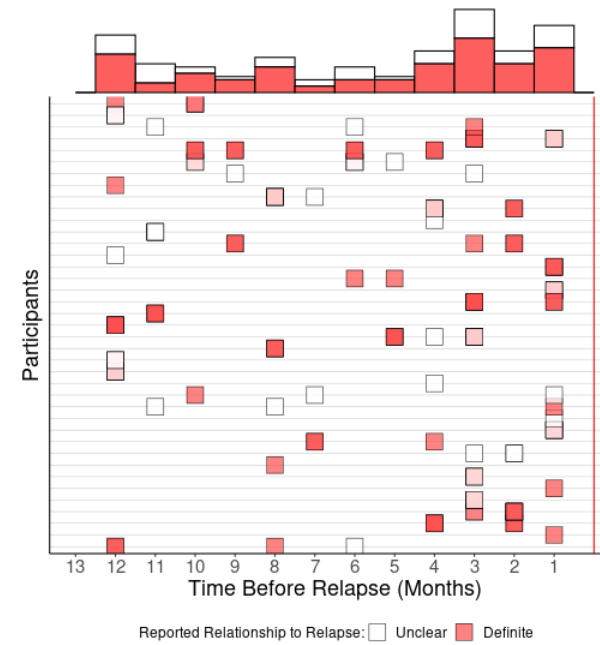


Change factors ranked by risk potency (degree to which they “definitely” contributed to relapse)

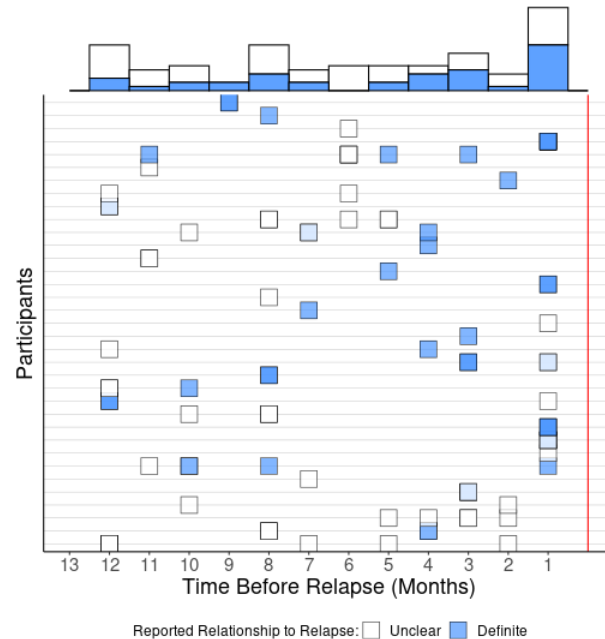
Biological Factors



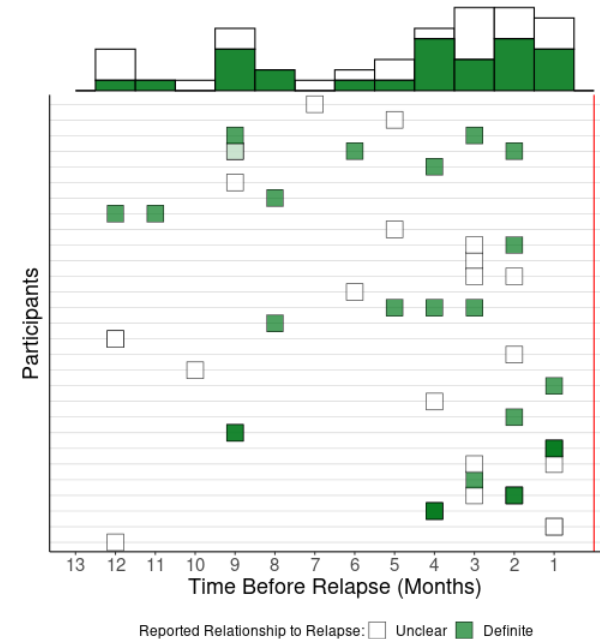
Psychological Factors



Social Factors



Recovery Service Factors



Marginal graphs show change in frequency of reported change factors across 12 months prior to relapse horizon - darker colors represent proportion that “definitely contributed” across the entire sample

Note: boxes represent factors reported to have changed in the year prior to relapse.
Darker boxes result from an overlap of multiple changes indicated on the same time point.

Clinical Implications for Long-Term Disease Management

- **Checklist** for patients to complete prior to ongoing visits involved in long-term disease management
 - In addition to the great need for more research into this important clinical and public health area, the array of psychological, social, recovery health services, and biological factor changes that occurred in the year preceding long-term AUD relapse detected in this study, highlight several markers and potential mechanisms that could be incorporated into a **checklist** for patients to complete prior to ongoing visits involved in long-term disease management.
- **Checklist may serve as stimulus and foundation for broader clinical discussion...**
 - Such a list as those factors reported in this study the presence of one or more of which, could **alert a clinician to focused discussion and intervention; or otherwise, serve as a stimulus and foundation for broader clinical discussion about the need for continued AUD recovery focus** and successfully facing and addressing recovery and life course challenges that ultimately may help prevent further morbidity and mortality that can arise from AUD recurrence

APPENDIX I

POTENTIAL DISEASE MANAGEMENT AND MONITORING QUESTIONNAIRE FOR USE IN CLINICAL RESEARCH ON ALCOHOL USE DISORDER RECOVERY AND LONG-TERM RELAPSE BASED ON STUDY FINDINGS

Rate your level of agreement with the following statements about significant changes you may have experienced since your last appointment

Biological

	Not at all	A little bit	Moderately	Quite a bit	A lot
	0	1	2	3	4
I have been having problems with sleep.					
I have been experiencing changes in my energy levels.					
I have been having problems with appetite and/or eating habits.					
I have been experiencing significant weight changes.					
I have been suffering from chronic/ ongoing pain.					
I have been using recreation drugs to get high or change how I am feeling.					
I have been making changes in my use of tobacco (started using or quitting tobacco).					
I have been having physical health problems.					
My medications have changed.					

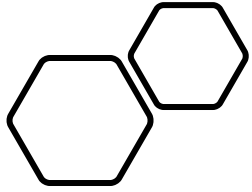
Psychological

	Not at all	A little bit	Moderately	Quite a bit	A lot
	0	1	2	3	4
I have been feeling more impulsive than usual					
I have been focusing less on my own recovery					
I have been feeling less confident I can sustain my recovery					
I have been feeling dissatisfied with my life					
I have been feeling more compulsive than usual					
I have been having trouble with mental health symptoms					

Social

	Not at all	A little bit	Moderately	Quite a bit	A lot
	0	1	2	3	4
I have been spending more time around alcohol.					
I have become more socially isolated.					
I have been feeling lonely.					
My employment situation has changed.					
I have been affected by the loss of someone close to me					

Such a clinical checklist might be deployed in long-term disease management (e.g., in recovery management check-ups)...



Connecting the Dots

Toward a Recovery-Oriented System of Care (ROSC)

A ROSC is a coordinated network of treatment and community-based services and supports that is person-centered and builds on the strengths and resiliencies of individuals, families, and communities to help achieve remission and improved health, wellness, and quality of life for those with or at risk of alcohol and drug problems

Thank you!

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