SUBSTANCE ABUSE AND TREATMENT

JONATHON LAZZARA, D.O.
DIRECTOR OF PSYCHIATRY, ATRIUM MEDICAL CENTER

OBJECTIVES

Describe the use of alcohol and drugs to self-medicate in the military culture with emphasis on before and after deployment.
Will be able to understand and apply the criteria for substance use disorder as outlined in the DSM-5
Adequately screen patients who show signs and symptoms of substance abuse and addiction including the more complex dual diagnosis with special consideration of post-trauma conditions.
Identify strategies for intervention which help shape treatment planning, including examining risk and protective factors.
Increase basic awareness of intensive case-management and evidence based therapy including cognitive processing therapy and referral resources.
Identify solutions for relapse prevention including positive alternatives and creating an improved living environment to help maintain sobriety.
Describe current pharmacotherapy recommendations for substance abuse and how benzodiazepines may delay the recovery process event further and may result in abuse, addiction, and legal issues.
Describe the epidemiology of opiate abuse and addition and its impact on productivity and rehabilitation programs.
Recognize the pathology of opiates on making pain worse in chronic pain and the cause of opiate hyperalgesia.
Identify the pharmacological agents available to help reduce cravings for opiates.
Define the requirements for a patient to qualify for admission to the Chillicothe Suboxone Clinic and the requirements to participate.
Assess what is needed for successful recovery outside of medication assisted treatment.

DSM-5

SUBSTANCE USE DISORDERS

SUBSTANCE USE DISORDER

Addiction

- No longer applied as a diagnostic term
- Common usage in many countries to describe severe problems related to compulsive and habitual use of substances.
- Eliminated from DSM-5 because of its uncertain definition and its potentially negative connotation

Substance Use Disorder

- Wide range of the disorder
- From mild form to a severe state of chronically relapsing, compulsive drug taking

SUBSTANCE USE DISORDER

Problematic pattern of substance use leading to clinically significant impairment or distress, as manifested by at least 2 of the following within a 12 month period

1. Substance is often taken in larger amounts or over a longer period than was intended
2. There is a persistent desire or unsuccessful efforts to cut down or control substance use
3. A great deal of time is spent in activities necessary to obtain the substance, use the substance or recover from its effects
4. Craving, or a strong desire or urge to use
5. Recurrent substance use resulting in a failure to fulfill major role obligations at work, school or home
6. Continued substance use despite having persistent or recurrent social or interpersonal problems caused or exacerbated by the effects

SUBSTANCE USE DISORDER

7. Important social, occupational, or recreational activities are given up or reduced because of substance use
8. Recurrent substance use in situations in which it is physically hazardous
9. Substance use is continued despite knowledge of having a persistent or recurrent physical or psychological problem that is likely to have been caused or exacerbated by the substance
10. Tolerance, as defined by either of the following:
    - A need for markedly increased amounts of the substance to achieve intoxication or desired effect
    - A markedly diminished effect with continued use of the same amount of the substance
11. Withdrawal

SPECIFIERS

Early remission

- After full criteria for substance use disorder were previously met, none of the criteria for substance use disorder have been met for at least 3 months but for less than 12 months

In sustained remission

- After full criteria for substance use disorder were previously met, none of the criteria for substance use disorder have been met at any time during a period of 12 months or longer
- Craving may still be met

In a controlled environment

- This additional specifier is used if the individual is in an environment where access to the substance is restricted

SEVERITY

Mild

- Presence of 2-3 symptoms

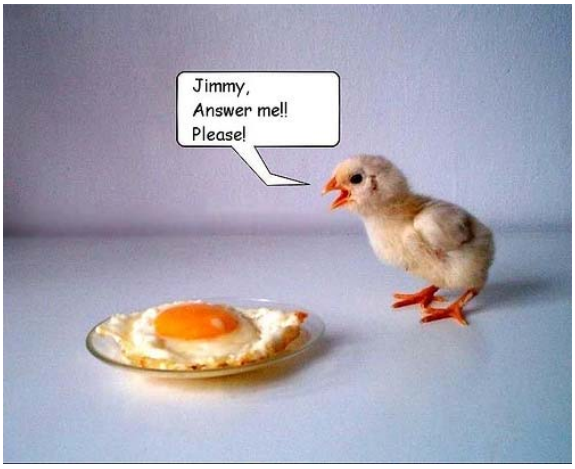
Moderate

- Presence of 4-5 symptoms

Severe

- Presence of 6 or more symptoms

DUEL DIAGNOSIS -- WHICH CAME FIRST????



http://farm3.static.flickr.com/2009/2017878606_c72967b08f.jpg?v=0

SYMPTOMS OF SUBSTANCE ABUSE

- Withdrawal from friends and family
- Sudden changes in behavior
- Using substances under dangerous conditions
- Engaging in risky behaviors when drunk or high
- Loss of control over use of substances
- Doing things you wouldn't normally do to maintain your habit
- Developing tolerance and withdrawal symptoms
- Feeling like you need the drug to be able to function

https://www.nami.org/Learn-More/Mental-Health-Conditions/Related-Conditions/Dual-Diagnosis

TRAUMATIC ABUSE, DEPRESSION AND ADDICTIVENESS

1. **Co-morbidity is an expectation not an exception**
2. **Successful treatment requires most importantly the creation of welcoming, empathetic, hopeful, continuous treatment relationships, in which integrated treatment and coordination of are sustained through multiple treatment episodes.**
3. **Within the context of the continuous integrated treatment relationship, case management and care-taking must be balanced with empathetic detachment and confrontation in accordance with the individual's level of functioning, disability and capacity for treatment adherence.**
4. **When mental illness and substance disorder co-exist, both disorders should be considered primary, and integrated dual primary treatment is required.**
5. **Both psychiatric illnesses and substance dependence are examples of chronic, biological mental illness that can be understood using a disease and recovery model. Each disorder is characterized by parallel recovery phases: acute stabilization, engagement and motivational enhancement, active treatment, prolonged stabilization, rehabilitation and recovery.**
6. **There is no single correct dual diagnosis intervention. Appropriate practice guidelines require that interventions must be individualized, according to the subtype of dual disorder, specific diagnosis of each disorder, phase of recovery/stage of change, and level of functional capability or disability.**
7. **Within a managed-care system, any of the individualized phase-specific interventions can be applied at any level of care. Consequently, a separate multidimensional level of care assessment is required.**

Minkoff, K. (2000). An Integrated Model for the Management of Co-Occurring Psychiatric and Substance Disorders in Managed Care Systems. Dis Manage Health Outcomes, Nov: 8 (5): 251-257.

CASE MANAGEMENT AND PSYCHOTHERAPY

CASE MANAGEMENT

Coordination of community services for mental health patients by allocating a professional to be responsible for the assessment of need and implementation of care plans.

For patients who need ongoing support in areas such as housing, employment, social relationships, and community participation

Underlying tasks include:

- Assessment of need
- Care planning
- Implementation
- Regular review

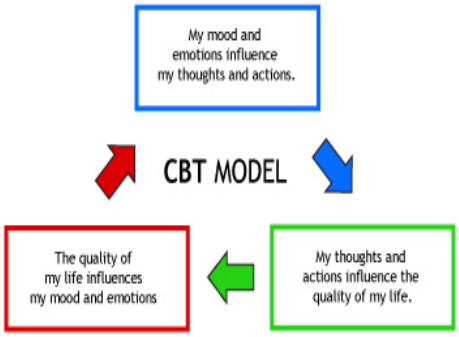
GROUP THERAPY APPROACHES

COGNITIVE BEHAVIORAL THERAPY
MOTIVATIONAL INTERVIEWING
RATIONAL EMOTIVE BEHAVIOR THERAPY

COGNITIVE BEHAVIORAL THERAPY

Emphasis on teaching participants self-management skills and how to restructure their thoughts.

Relies on the principles and procedures of the scientific method which are systematically appli to help effect change of maladaptive behaviors



RELAPSE PREVENTION

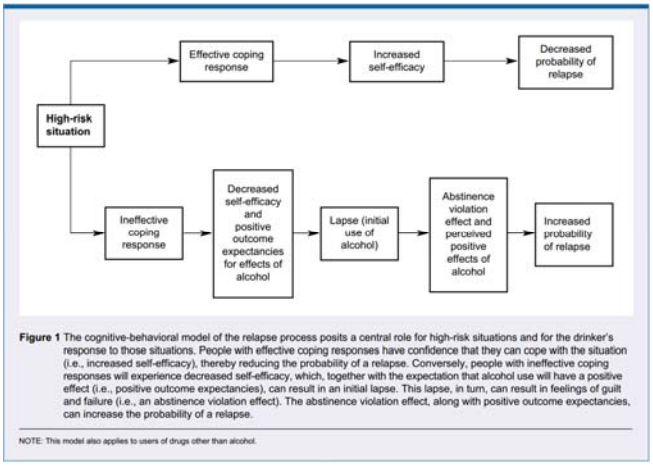
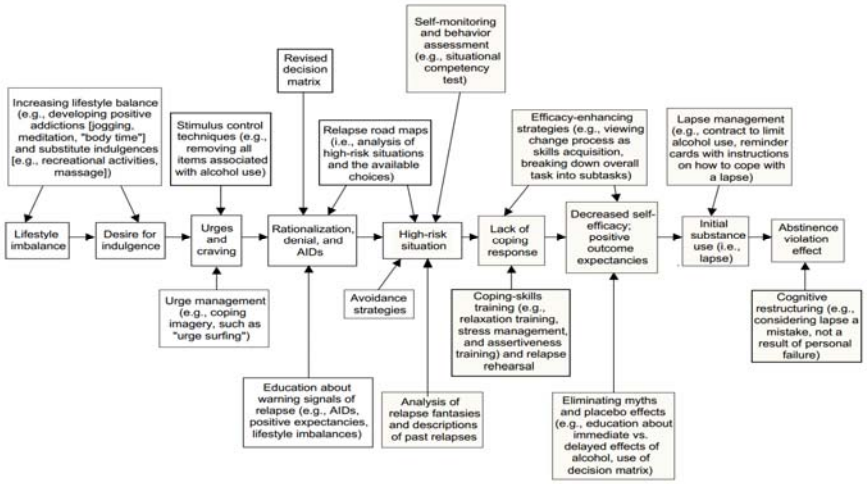


Figure 1 The cognitive-behavioral model of the relapse process posits a central role for high-risk situations and for the drinker's response to those situations. People with effective coping responses have confidence that they can cope with the situation (i.e., increased self-efficacy), thereby reducing the probability of a relapse. Conversely, people with ineffective coping responses will experience decreased self-efficacy, which, together with the expectation that alcohol use will have a positive effect (i.e., positive outcome expectancies), can result in an initial lapse. This lapse, in turn, can result in feelings of guilt and failure (i.e., an abstinence violation effect). The abstinence violation effect, along with positive outcome expectancies, can increase the probability of a relapse.

NOTE: This model also applies to users of drugs other than alcohol.

Larimer ME, Palmer RS, Marlatt GA. Relapse prevention An overview of Marlatt's cognitive-behavioral model. Alcohol Res Health. 1999;23(2):151-60. PubMed PMID: 10890810.

RELAPSE PREVENTION

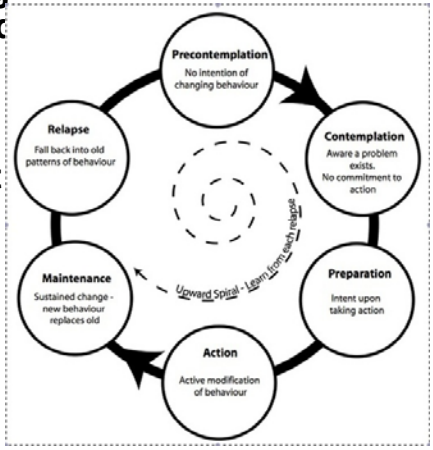


Larimer ME, Palmer RS, Marlatt GA. Relapse prevention An overview of Marlatt's cognitive-behavioral model. Alcohol Res Health. 1999;23(2):151-60. PubMed PMID: 10890810.

MOTIVATIONAL INTERVIEWING

Motivational Interviewing focuses on exploring and resolving ambivalence and centers on motivational processes within the individual that facilitate change.

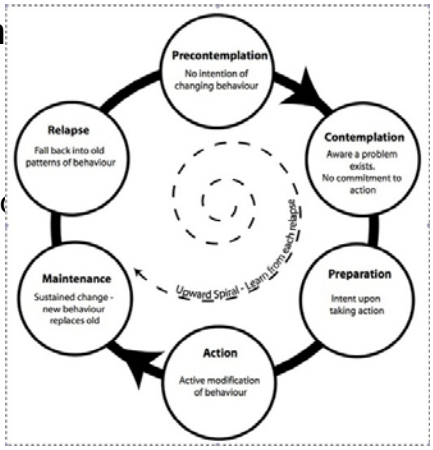
Supports change in a manner congruent with the person's own values and concerns.



MOTIVATIONAL INTERVIEWING

There are four distinct principles that guide th practice of MI:

- Express Empathy with clients
- Support Client Self-Effica
- Roll with Resistance of clients to effect change
- Develop Discrepancy between clients current state and desired goals



RATIONAL EMOTIVE BEHAVIOR THERAPY

Stresses the reciprocal interactions among cognition, emotion, and behavior

A-B-C Theory:

Activating Event occurs

Irrational Beliefs one develops regarding the event

Results in Emotional Consequence regarding the event – i.e guilt, shame, etc.

RATIONAL EMOTIVE BEHAVIOR THERAPY

Clients learn to confront Irrational Beliefs

Key Goals:

- Achieving Unconditional Self-Acceptance
- Unconditional Other Acceptance

BENZODIAZEPINES AND DELAYED RECOVERY

Benzodiazepines are now taught to not be prescribed in the PTSD protocol and used in treatment refractory anxiety, yet benzodiazepines are still heavily prescribed.

With the use of benzodiazepines, patient become dependent on the medication and end up with worsened anxiety if they do not have the medication.

Body can become dependent on benzodiazepines and if a patient does not get these medications abruptly, can withdrawal and die from seizures

Interfere with the extinction of fear conditioning or potentiate the acquisition of fear responses actually worsening recovery from trauma and interfering with first-line recommended PTSD therapies

MEDICATION ASSISTED TREATMENT

MEDICATION ASSISTED TREATMENT

Alcohol Use Disorder

Cocaine Use Disorder

- Amphetamine use disorder
- Ecstasy use disorder

Opiate use disorder

DISCLAIMER

Medication assisted therapy ONLY works when a patient is self motivated in a recovery program that is goal directed toward sobriety. No medication alone will ever magically make a person stay sober

It is only one wheel on a car

***Medication does not keep a person sober, it helps a person stay in their recovery*.**

ALCOHOL USE DISORDER

28 y/o male comes in seeking help for his cravings for alcohol. He has a history of binge drinking on weekends consuming alcohol to the point of blackouts. He drinks 3 nights a week usually on weekends. For the past year this was not interfering in his life but it has resulted now in missed work the day after and family has discussed mood changes in the patient.

His review of symptoms is negative except for high anxiety with difficulty sleeping at night.

Patient is not sure if he is committed to a 12 step program at this time because of his discomfort with religion.

Patient does not have any comorbid diagnosis other than substance induced mood disorder exhibiting anxiety.

ALCOHOL USE DISORDER

12 Steps

(1) We admitted we were powerless over alcohol – that our lives had become unmanageable.
(2) Came to believe that a power greater than ourselves could restore us to sanity.
(3) Made a decision to turn our will and our lives to the care of God as we understood him.
(4) Made a searching and fearless moral inventory of ourselves.
(5) Admitted to God, to ourselves, and to another human being the exact nature of our wrongs.
(6) Were entirely ready to have God remove all these defects of character.
(7) Humbly asked Him to remove our shortcomings.
(8) Made a list of all persons we had harmed and became willing to make amends to them all.
(9) Made direct amends to such people wherever possible, except when to do so would injure them or others.
(10) Continued to take personal inventory and when we were wrong promptly admitted it.
(11) Sought through prayer and meditation to improve our conscious contact with God as we understood him, praying only for knowledge of His will and the power to carry that out.
(12) Having had a spiritual awakening as the result of these steps, we tried to carry this message to alcoholics and to practice these principles in all our affairs.

These steps are from the book, "Alcoholics Anonymous."

MEDICATION ASSISTED TREATMENT

Alcohol Use Disorder

- Baclofen

Cocaine Use Disorder

- Amphetamine use disorder
- Ecstasy use disorder

Opiate use disorder

BACLOFEN

NON-FDA APPROVED

BACLOFEN

Addolorato G, Caputo F, Capristo E, et al. Baclofen Efficacy in Reducing Alcohol Craving and Intake: A preliminary Double-Blind Randomized Controlled Study. Alcohol and Alcoholism (2002) 37(5): 504-508.

39 alcohol dependent patients were consecutively enrolled in the study. 20 patients were treated with baclofen and 19 with placebo. Both were administered doses of baclofen 15mg/day for first 3 days and then 30mg/day for 27 days divided into 3 daily doses.

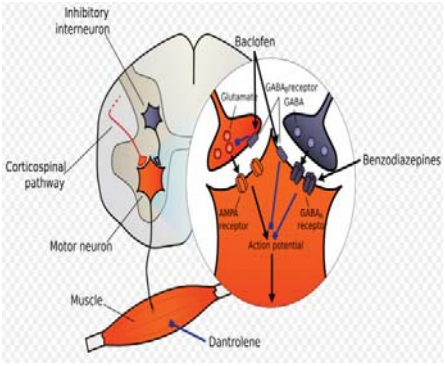
Higher percentage of subjects totally abstinent from alcohol and a higher number of cumulative abstinence days throughout the study period were found in the baclofen compared to the placebo, group. A decrease in the obsessive and compulsive components of craving was found in the baclofen compared to the placebo group. Alcohol intake was reduced in the baclofen group as well. No significant difference was found between the two groups in terms of current depressive symptoms. Baclofen proved to be easily manageable and no patient discontinued treatment due to presence of side effects.

Conclusion

- Baclofen proved to be effective in inducing abstinence from alcohol and reducing alcohol craving and consumption in alcoholics.

BACLOFEN

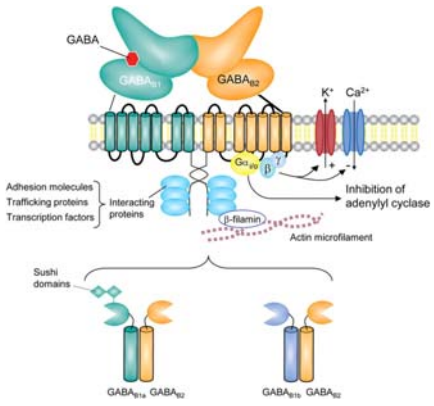
- **GABA-mimetic drug which acts as a GABA agonist at $GABA_B$ receptors**
- **Its actions results from an action at spinal level where it inhibits both monosynaptic & polysynaptic reflexes**
- **It also reduces pain associated with spastic conditions as it inhibits the release of substance-P in the spinal cord**



http://pharma-munvar.blogspot.com/2010/04/skeletal-muscle-relaxants.html

BACLOFEN

- **$GABA_B$ involved in behavioral actions of ethanol, GHB and pain**
- **Non-FDA used in reducing alcohol cravings.**
- **Used in chronic pain to reduce opiate cravings and pain with high success**



http://www.neurology.org/content/78/8/578/F1.large.jpg

BACLOFEN

Off label use to reduce alcohol and opiate cravings

- Initiate at 5mg TID
- Increase to 10mg TID
- Do not give as PRN as we are targeting cravings and not pain symptoms!
- If patients do not respond to 10mg TID then I have not found they respond to higher doses (personal experience).
- Doses over 60mg per day require a titration if discontinuation of the drug is to occur of there is a risk of seizure

MEDICATION ASSISTED TREATMENT

Alcohol Use Disorder

- Baclofen
- Naltrexone

Cocaine Use Disorder

- Amphetamine use disorder
- Ecstasy use disorder

Opiate use disorder

NALTREXONE

NALTREXONE

Volpicelli J, Alterman A, Hayashida M, O'brien C. Naltrexone in the Treatment of Alcohol Dependence. Arch Gen Psychiatry. 1992; 49(11): 876-880.

70 male alcohol dependent patients participated in a 12 week, double-blind, placebo controlled trial of naltrexone as an adjunct to treatment following alcohol detoxification.

Subjects taking naltrexone reported significantly less alcohol craving and days in which any alcohol was consumed.

During 12 week study, only 23% of naltrexone-treated subjects met criteria for a relapse, whereas 54.3% of the placebo-treated subjects relapsed.

NALTREXONE

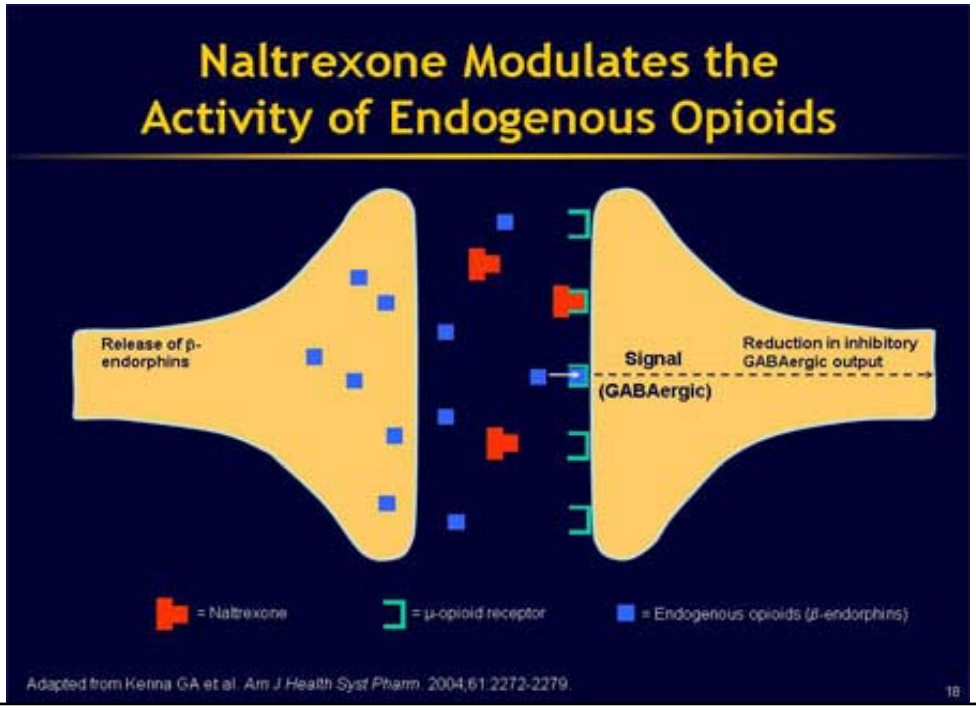
FDA Approved for Opiate use disorder and alcohol use disorder to reduce cravings

Non-FDA for Cocaine Use Disorder

Opiate receptor antagonist

Mechanism of action

- Reduces cravings by antagonizing Mu and Kappa receptors



NALTREXONE

Dosing

- Initiate first dose at 25mg on first day 7 days after last use of opiate medication
  - If NO withdrawal symptoms
- Initiate regular dose of 50mg every day

MEDICATION ASSISTED TREATMENT

Alcohol Use Disorder

- Baclofen
- Naltrexone
- Gabapentin

Cocaine Use Disorder

- Amphetamine use disorder
- Ecstasy use disorder

Opiate use disorder

GABAPENTIN

NON-FDA APPROVED, ALCOHOL, BENZODIAZEPINE AND MARIJUANA CRAVINGS

GABAPENTIN

Brower K, Kim H, Strobbe S, Karam M, et al. A Randomized Double-Blind Pilot Trial of Gabapentin Versus Placebo to Treat Alcohol Dependence and Comorbid Insomnia. Alcoholism: Clinical and Experimental Research. Aug 2008(32)8. pp 1429-1438

21 subjects who met criteria for alcohol dependence and insomnia and expressed a desire to abstain from alcohol were recruited to the study. 11 were assigned to placebo and 10 were assigned to gabapentin for 6 weeks with a titrated total dose of 1500mg at bedtime. Assessment was done at 6 weeks following treatment.

Gabapentin significantly delayed the onset to heavy drinking, an effect which persisted for 6 weeks after treatment ended. Insomnia improved in both treatment groups during the medication phase, but gabapentin had no differential effects on sleep as measured by either subjective report or polysomnography.

Because gabapentin is a short-acting medication that was taken at nighttime in this study it may possibly exert a nocturne effect that prevents relapse to heavy drinking by a physiological mechanism not measured in this pilot study.

GABAPENTIN

Mason BJ, Quello S, Goodell V, Shadan F, Kyle M, Begovic A. Gabapentin treatment for alcohol dependence: A randomized clinical trial. JAMA Intern Med. 2014 Jan; 174(1):70-1.

12 week, double blind, placebo=controlled, randomized dose-ranging trial of 150 men and women older than 18 years with current alcohol dependence, conducted from 2004 through 2010 at a single-site, outpatient clinical research facility adjoining a general medical hospital.

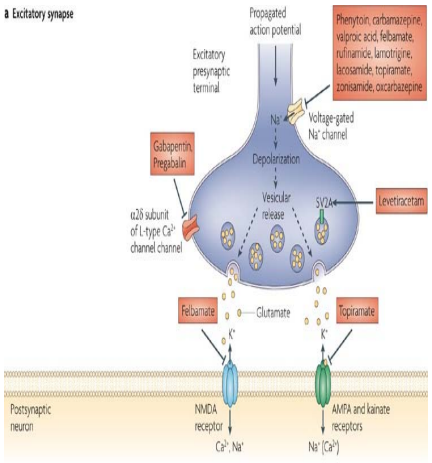
Gabapentin, particularly in the 1800mg dosage range was effective in treating alcohol dependence an relapse-related symptoms of insomnia, dysphoria and cravings with a favorable safety profile.

GABAPENTIN

GABA analog

Originally used to treat epilepsy (FDA approved for partial seizures)

Currently used to treat neuropathic pain

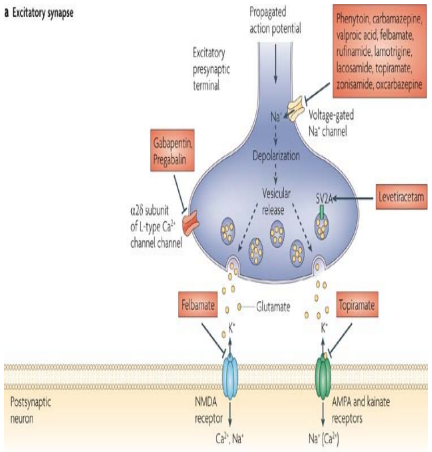


GABAPENTIN

MOA – Interacts with Voltage-sensitive calcium channels in cortical neurons

Increases the synaptic concentration of GABA and enhances GABA responses at non-synaptic sits in neuronal tissues, and reduces the release of mono-amine neurotransmitters

Reduction of axon excitability measured as an amplitude change in the area of the hippocampus through its binding to presynaptic NMDA receptors



MEDICATION ASSISTED TREATMENT

Alcohol Use Disorder

- Baclofen
- Naltrexone
- Gabapentin
- Topiramate

Cocaine Use Disorder

- Amphetamine use disorder
- Ecstasy use disorder

Opiate use disorder

TOPIRAMATE

NON-FDA APPROVED

TOPIRAMATE

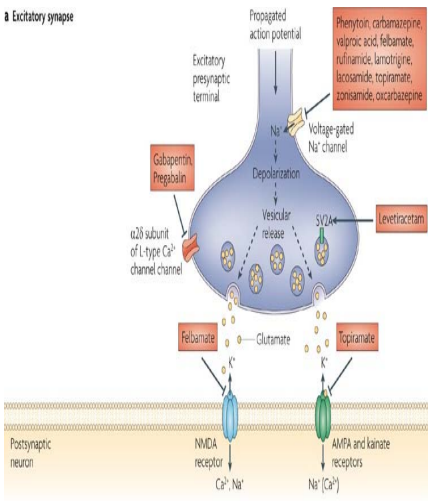
Johnson BA, Rosenthal N, Capece JA, Wiegand F, et al. Topiramate for treating alcohol dependence: a randomized controlled trial. JAMA. 2007 Oct 10; 298(14):1641-51.

Double-blind, randomized, placebo-controlled, 14-week trial of 371 men and women aged 18 to 65 years diagnosed with alcohol dependence, conducted between January 27, 2004 and August 4, 2006 at 17 US sites. Up to 300mg/day of topiramate (n=183) or placebo (n=188) along with a weekly compliance enhancement intervention.

Treated all dropouts as relapse. More efficacious than placebo at reducing the percentage of heavy drinking to week 14. Mean difference of 16.19%.

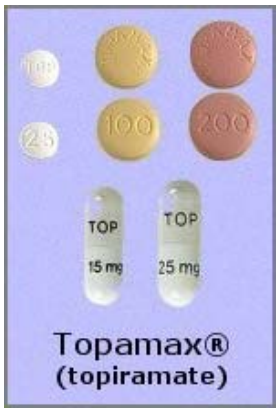
TOPIRAMATE (TOPAMAX)

- **Anticonvulsant**
- **Blocks voltage-sensitive sodium channels by an unknown mechanism**
- **Inhibits release of glutamate**
- **Potentiates activity of GABA**
- **Carbonic anhydrase inhibitor**



TOPIRAMATE

- **Partial Onset seizures**
- **Primary generalized tonic-clonic seizures**
- **Seizures associated with Lennox-gastaut syndrome**
- **Migraines**
- **Bipolar Disorder (adjunctive)**
- **Binge Eating**



TOPIRAMATE

Side Effects

- Sedation
- Dizziness
- Ataxia
- Tremor
- Appetite loss, weight loss
- **Problems concentrating**
- *Taste perversion*
- Psychomotor retardation
- **Memory problems**

TOPIRAMATE -- DOSING

25mg Twice a day dosing

- Personal antidotal experience

Migraines

- Titrate dosing 25mg qhs then 25mg BID.. Increase by 25mg/day weekly to desired effect.

Personally keep dosing low for cravings. Patients have had a positive response on 25mg BID and I make great lengths to avoid all side effects except for the taste preservation as it is a side effect that can be seen as helpful especially in binge drinking when alcohol is the drink of choice.

Why is this side effect beneficial or not beneficial????

MEDICATION ASSISTED TREATMENT

Alcohol Use Disorder

- Baclofen
- Naltrexone
- Gabapentin
- Topiramate
- Acamprosate

Cocaine Use Disorder

- Amphetamine use disorder
- Ecstasy use disorder

Opiate use disorder

ACAMPROSATE (CAMPRAL)

FDA APPROVED

THE DEVIL'S MEDICATION!!!

ACAMPROSATE

Dranitsaris G, Selby P, Negrete JC. Meta-analyses of placebo-controlled trials of acamprosate for the treatment of alcohol dependence: impact of the combined pharmacotherapies and behavior interventions study. J Addict Med. 2009 Jun; 3(2): 74-82.

26 studies were evaluated looking at acamprosate. Findings revealed that acamprosate was superior to placebo in the mean number of cumulative abstinent days and abstinence rates.

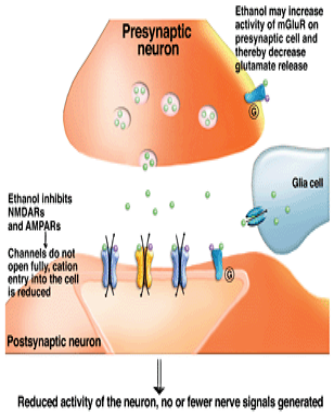
STAGES OF WITHDRAWAL

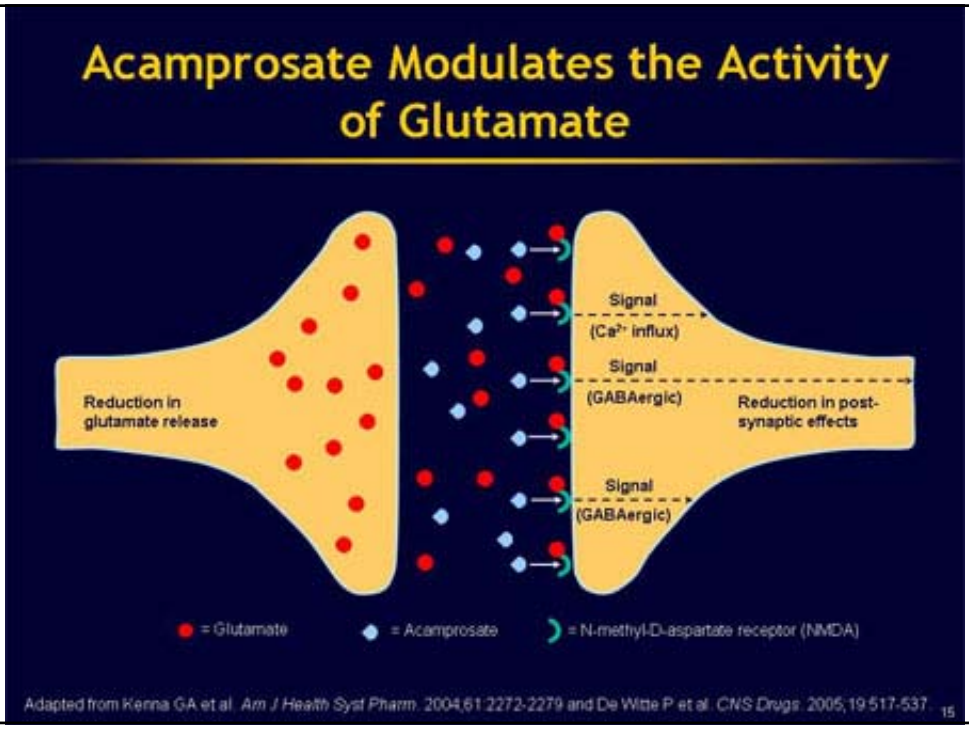
ACAMPROSATE

Antagonizes glutamatergic NMDA and $GABA_A$ receptors

Benefits

- Metabolized by kidneys
- Why ?????





ACAMPROSATE

Dosing

- 666mg TID

Start as soon as possible after withdrawal when abstinence is achieved.

Continue even if relapse

ALCOHOL USE DISORDER

28 y/o male comes in seeking help for his cravings for alcohol. He has a history of binge drinking on weekends consuming alcohol to the point of blackouts. He drinks 3 nights a week usually on weekends. For the past year this was not interfering in his life but it has resulted now in missed work the day after and family has discussed mood changes in the patient.

His review of symptoms is negative except for high anxiety with difficulty sleeping at night.

Patient is not sure if he is committed to a 12 step program at this time because of his discomfort with religion.

Patient does not have any comorbid diagnosis other than substance induced mood disorder exhibiting anxiety.

CASE STUDY – ALCOHOL USE DISORDER

Topiramate 25mg BID

Hydroxyzine 50mg TID for breakthrough anxiety and sleep

SMART Recovery

- Self-empowering addiction recovery support group with a 4 point program.
  1. Building and Maintaining Motivation
  2. Coping with Urges
  3. Managing Thoughts, Feelings and Behaviors
  4. Living a Balanced Life

http://www.smartrecovery.org/

BENZODIAZEPINE USE DISORDER????

Limited studies…..

Gabapentin????

Baclofen????

MEDICATION ASSISTED TREATMENT

Alcohol Use Disorder

- Baclofen
- Naltrexone
- Gabapentin
- Topiramate
- Acamprosate

Cocaine Use Disorder

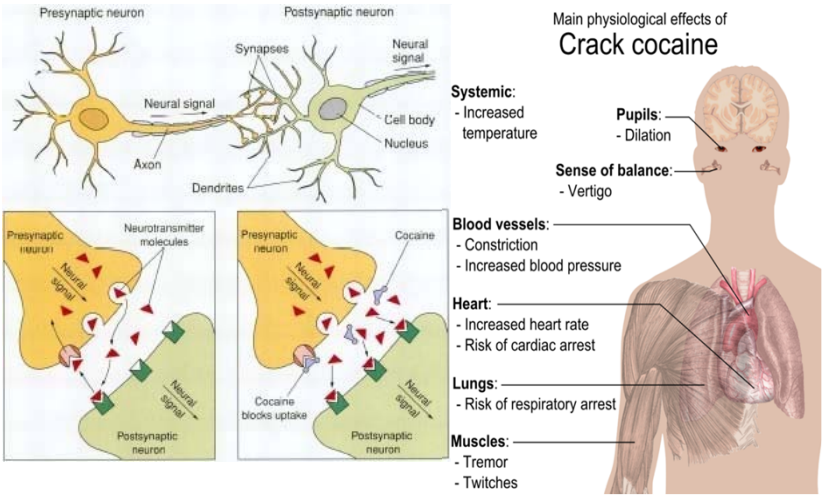
Opiate use disorder

COCAINE USE DISORDER

56 year old male is in substance abuse treatment. This is his 8th time in treatment. He states he is having problems because he keeps using due to the fact he is not being treated for his attention deficit disorder. He was never diagnosed in while in school and never had issues before he started abusing drugs. His drug of choice is crack cocaine and used daily until admission. He is currently 13 days sober at this time.

Review of symptoms are negative except for poor concentration and depression.

COCAINE/CRACK



MEDICATION ASSISTED TREATMENT

Alcohol Use Disorder

- Baclofen
- Naltrexone
- Gabapentin
- Topiramate
- Acamprosate

Cocaine Use Disorder

- Topiramate

Opiate use disorder

TOPIRAMATE

Nuijten M, Blanken P, van den Brink W, Hendriks V. Treatment of crack-cocaine dependence with topiramate: A randomized controlled feasibility trial in the Netherlands. Drug Alcohol Depend. 2014 Feb 26. pii: S0376-8716

74 Crack-cocaine dependent outpatients participated in an open-label, randomized feasibility trial. They were randomized to receive either 12-week CBT plus topiramate (200mg/day) or 12-week CBT only. The primary outcome measure was treatment retention.

Adherence to topiramate treatment was low. Topiramate neither improved treatment retention nor reduced cocaine or other substance use. Efficacy was not supported probably due to low acceptance of the treatment

TOPIRAMATE

Johnson BA, Ait-Daoud N, Wang XQ, Penberthy JK, et al. Topiramate for the treatment of cocaine addiction: a randomized clinical trial. JAMA Psychiatry. 2013 Dec; 70(12): 1338-46.

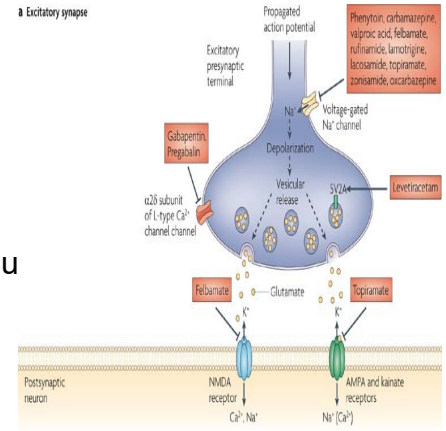
71 patients were given topiramate and 71 patients were on placebo. 12 week trial. Target maintenance dose of 300mg/d were given.

Topiramate was more efficacious than placebo at increasing the weekly proportion of cocaine nonuse days, irrespective of whether missing data were not or were imputed conservatively to the baseline. Topiramate also was associated, significantly more than placebo, with increasing the likelihood of urinary cocaine fee-weeks as well as decreasing cravings and improving observer-rated global functioning.

TOPIRAMATE -- MOA

Hypothesized MOA

- Dual modulation of the mesocorticolimbic dopamine system by topiramate-a glutamate receptor antagonist and GABA agonist would resu in efficious treatment for cocaine dependence



TOPIRAMATE -- DOSING

25mg Twice a day dosing

- Personal antidotal experience

Migraines

- Titrate dosing 25mg qhs then 25mg BID.. Increase by 25mg/day weekly to desired effect.

Personally keep dosing low for cravings. Patients have had a positive response on 25mg BID and I make great lengths to avoid all side effects. Make great efforts at conservative titration if goal dosing to 200mg as presented in literature.

Why would side effects to topiramate especially cause cocaine/crack users to stop the medication?

MEDICATION ASSISTED TREATMENT

Alcohol Use Disorder

- Baclofen
- Naltrexone
- Gabapentin
- Topiramate
- Acamprosate

Cocaine Use Disorder

- Topiramate
- Bupropion

Opiate use disorder

BUPROPION

NON-FDA APPROVED

BUPROPION

Stoops WW, Lile JA, Glaser PE, Hays LR, Rush CR. Influence of acute bupropion pre-treatment on the effects of intranasal cocaine. Addiction. 2012 Jun; 107(6):1140-7

Eight subjects completed 9 experimental session in which they were pre-treated with 0, 100 ore 200mg oral immediate release bupropion. 90 minutes later they sampled intranasal cocaine dose and made 6 choices between that dose and an alternative reinforcer.

Active bupropion reduced choice of 45mg of cocaine to 2.13 or 4 out of six drug choices on average.

Bupropion Acutely appears to reduce preference for intranasal cocaine versus a small amount of money but to increase reported positive experiences of the drug.

BUPROPION (WELLBUTRIN)

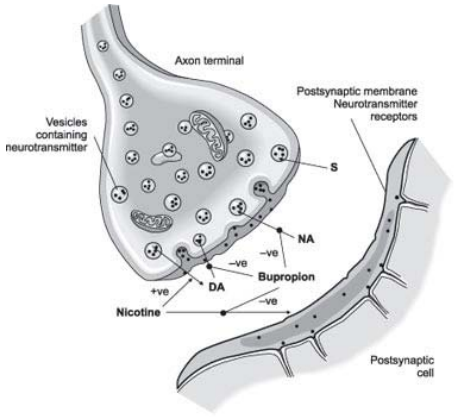
Atypical antidepressant

- Smoking cessation
- Adult attention deficit disorder

Norepinephrine-dopamine reuptake inhibitor

Dopamine is inactivated by norepinephrine reuptake in frontal cortex, which largely lacks dopamine transporters, bupropion can increase dopamine neurotransmission in this part of the brain

Nicotinic acetylcholine receptor antagonist



BUPROPION

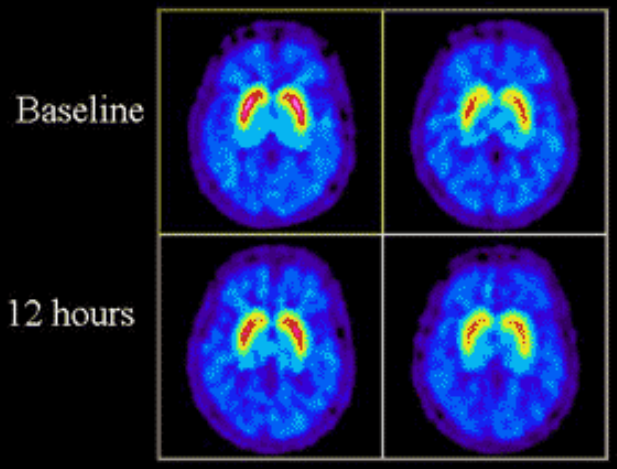
Dosage

- Extended Release
- 200mg qAM
- 100mg qPM (1500hr)
  - *Why????*
- Max 400mg Daily

Adult ADHD dosing

Bupropion Dopamine Transporter *In Vivo* Binding (^{11}C-bCIT-FE) in Humans

Bupropion SR 150 mg bid at steady state



Learned-Coughlin, *Biol Psychiatry*, 2003. In Press.

COCAINE USE DISORDER

56 year old male is in substance abuse treatment. This is his 8th time in treatment. He states he is having problems because he keeps using due to the fact he is not being treated for his attention deficit disorder. He was never diagnosed in while in school and never had issues before he started abusing drugs. His drug of choice is crack cocaine and used daily until admission. He is currently 13 days sober at this time.

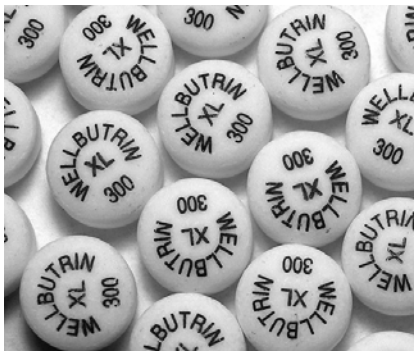
Review of symptoms are negative except for poor concentration and depression.

COCAINE USE DISORDER – CASE STUDY

Bupropion ER

- 150mg at 0700hr and 1500hr

Strongly re-enforce CA meetings



COCAINE ANONYMOUS

12 STEPS

1. We admitted we were powerless over cocaine and all other mind-altering substances — that our lives had become unmanageable.
2. Came to believe that a Power greater than ourselves could restore us to sanity.
3. Made a decision to turn our will and our lives over to the care of God as we understood Him.
4. Made a searching and fearless moral inventory of ourselves.
5. Admitted to God, to ourselves, and to another human being the exact nature of our wrongs.
6. Were entirely ready to have God remove all these defects of character.
7. Humbly asked Him to remove our shortcomings.
8. Made a list of all persons we had harmed, and became willing to make amends to them all.
9. Made direct amends to such people wherever possible, except when to do so would injure them or others.
10. Continued to take personal inventory and when we were wrong promptly admitted it.
11. Sought through prayer and meditation to improve our conscious contact with God as we understood Him, praying only for knowledge of His will for us and the power to carry that out.
12. Having had a spiritual awakening as the result of these steps, we tried to carry this message to addicts, and to practice these principles in all our affairs.

12 TRADITIONS

1. Our common welfare should come first; personal recovery depends upon C.A. unity.
2. For our group purpose there is but one ultimate authority — a loving God as He may express Himself in our group conscience. Our leaders are but trusted servants; they do not govern.
3. The only requirement for C.A. membership is a desire to stop using cocaine and all other mind-altering substances.
4. Each group should be autonomous except in matters affecting other groups or C.A. as a whole.
5. Each group has but one primary purpose — to carry its message to the addict who still suffers.
6. A C.A. group ought never endorse, finance, or lend the C.A. name to any related facility or outside enterprise, lest problems of money, property and prestige divert us from our primary purpose.
7. Every C.A. group ought to be fully self-supporting, declining outside contributions.
8. Cocaine Anonymous should remain forever nonprofessional, but our service centers may employ special workers.
9. C.A., as such, ought never be organized; but we may create service boards or committees directly responsible to those they serve.
10. Cocaine Anonymous has no opinion on outside issues; hence the C.A. name ought never be drawn into public controversy.
11. Our public relations policy is based on attraction rather than promotion; we need always maintain personal anonymity at the level of press, radio, television and

MEDICATION ASSISTED TREATMENT

Alcohol Use Disorder

- Baclofen
- Naltrexone
- Gabapentin
- Topiramate
- Acamprosate

Cocaine Use Disorder

- Topiramate
- Bupropion

Opiate use disorder

- Baclofen
- Naltrexone
- Suboxone

MEDICATION ASSISTED TREATMENT

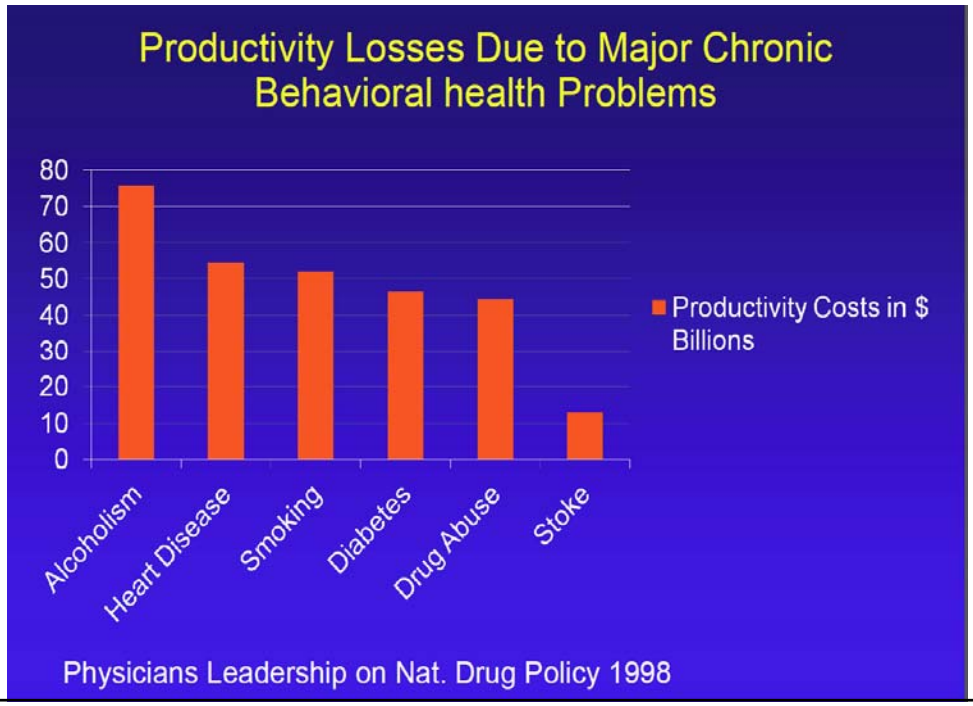
OPIATE USE DISORDER

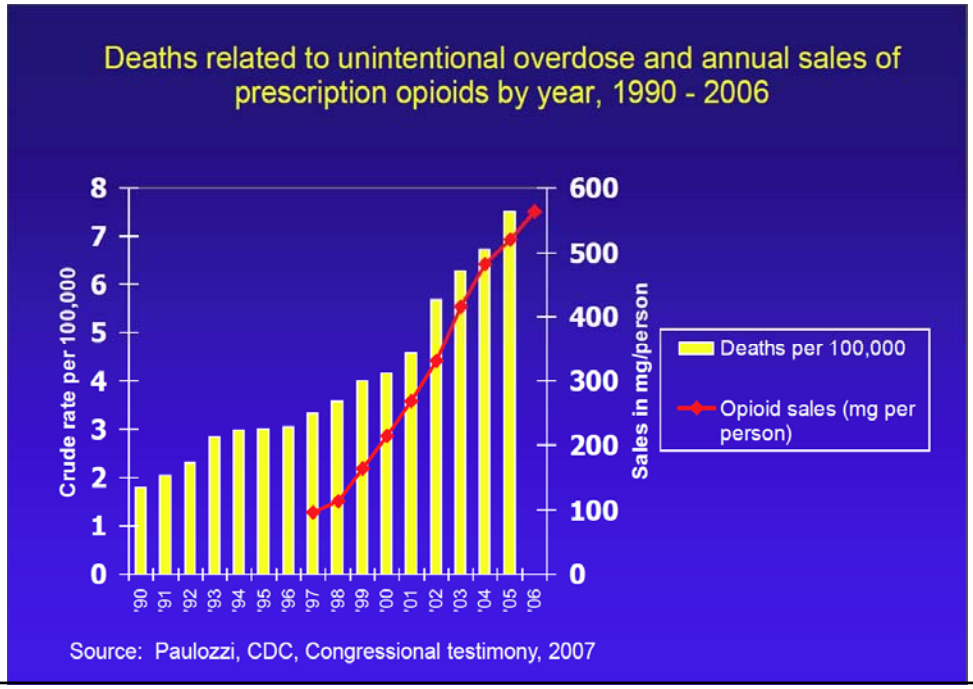
HEROIN AND OTHER OPIATE ADMISSIONS TO SUBSTANCE ABUSE TREATMENT

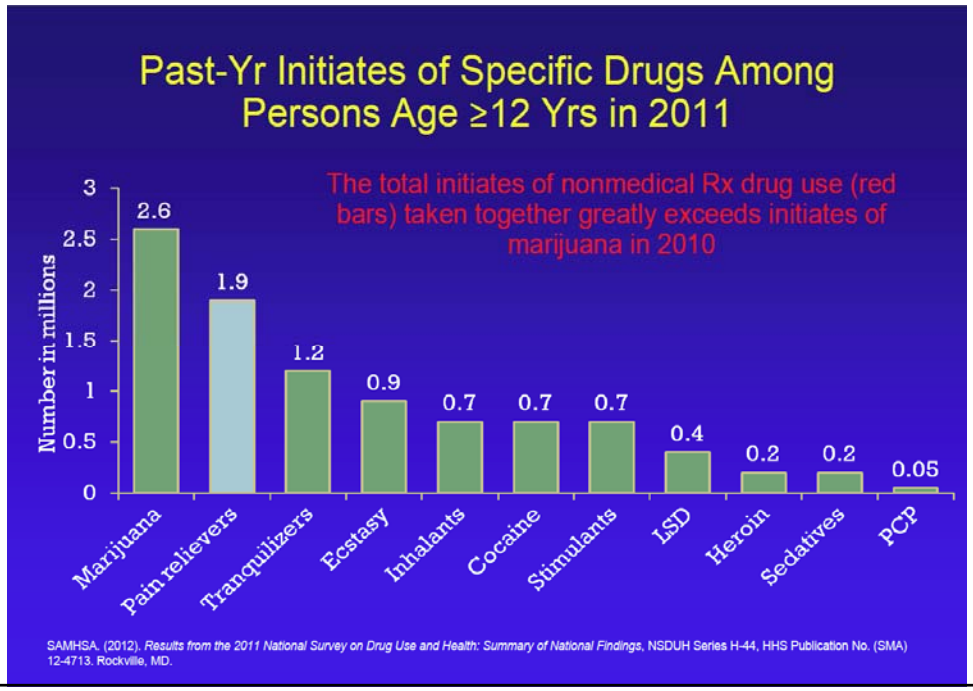
Opiates account for 19% of all substance abuse treatment admissions in 2007

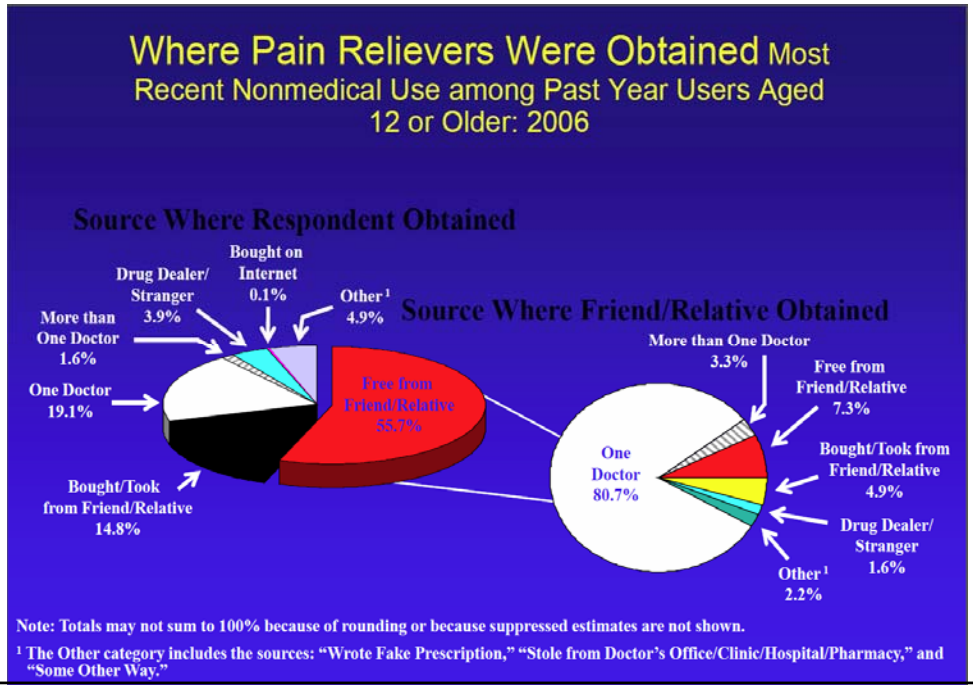
Heroin admission were 3x as like as admission for other opiates to report cocaine as a secondary substance of abuse

Heroin admission 3x as likely as other opiate admission to report five or more prior treatment admission (26 vs. 9%)


Productivity Losses Due to Major Chronic Behavioral health Problems
80
70
60
50
40
30
20
10
0
Alcoholism
Heart Disease
Smoking
Diabetes
Drug Abuse
Stoke
Productivity Costs in $ Billions
Physicians Leadership on Nat. Drug Policy 1998


Deaths related to unintentional overdose and annual sales of prescription opioids by year, 1990 - 2006
Crude rate per 100,000
Sales in mg/person
8
7
6
5
4
3
2
1
0
600
500
400
300
200
100
0
'90
'91
'92
'93
'94
'95
'96
'97
'98
'99
'00
'01
'02
'03
'04
'05
'06
Deaths per 100,000
Opioid sales (mg per person)
Source: Paulozzi, CDC, Congressional testimony, 2007


Past-Yr Initiates of Specific Drugs Among Persons Age ≥12 Yrs in 2011
The total initiates of nonmedical Rx drug use (red bars) taken together greatly exceeds initiates of marijuana in 2010
Number in millions
3
2.5
2
1.5
1
0.5
0
2.6
1.9
1.2
0.9
0.7
0.7
0.7
0.4
0.2
0.2
0.05
Marijuana
Pain relievers
Tranquilizers
Ecstasy
Inhalants
Cocaine
Stimulants
LSD
Heroin
Sedatives
PCP
SAMHSA. (2012). Results from the 2011 National Survey on Drug Use and Health: Summary of National Findings, NSDUH Series H-44, HHS Publication No. (SMA) 12-4713. Rockville, MD.

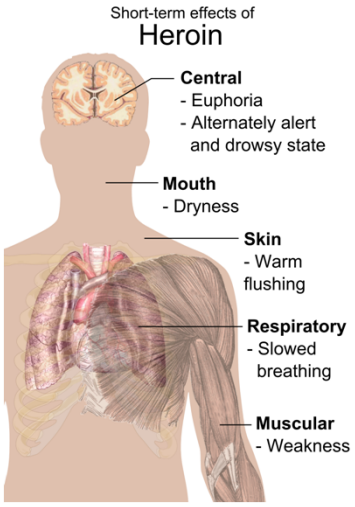

Where Pain Relievers Were Obtained Most Recent Nonmedical Use among Past Year Users Aged 12 or Older: 2006
Source Where Respondent Obtained
Drug Dealer/ Stranger 3.9%
Bought on Internet 0.1%
Other 1 4.9%
More than One Doctor 1.6%
One Doctor 19.1%
Free from Friend/Relative 55.7%
Bought/Took from Friend/Relative 14.8%
Source Where Friend/Relative Obtained
More than One Doctor 3.3%
Free from Friend/Relative 7.3%
Bought/Took from Friend/Relative 4.9%
Drug Dealer/ Stranger 1.6%
Other 1 2.2%
One Doctor 80.7%
Note: Totals may not sum to 100% because of rounding or because suppressed estimates are not shown.
1 The Other category includes the sources: "Wrote Fake Prescription," "Stole from Doctor's Office/Clinic/Hospital/Pharmacy," and "Some Other Way."

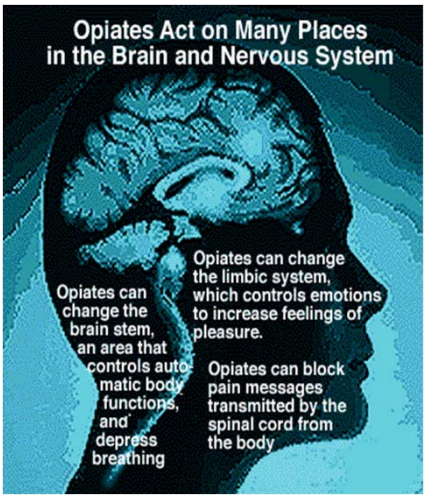
SECONDARY SUBSTANCE OF ABUSE

Secondary Substance of Abuse	Heroin	Other Opiates
Alcohol	18%	22%
Marijuana	11%	22%
Cocaine	51%	18%
Stimulants	3%	4%
Tranquilizers	4%	13%

Source: 2007 SAMHSA Treatment Episode Data Set (TEDS).

PATHOLOGY OF OPIATES





http://www.udel.edu/chem/C465/senior/fall00/DrugAddiction/brain3.gif

WHY DO WE HAVE AN EPIDEMIC OF OPIATE USE?

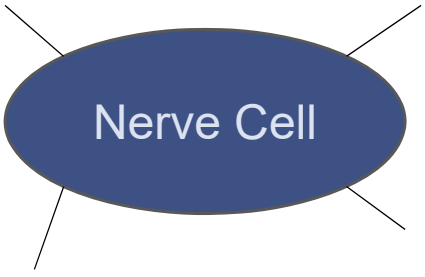
WHY ARE OPIATE INAPPROPRIATE FOR CHRONIC PAIN?

Up regulation of Pain Receptors

- 6 weeks of opiate use or abuse
- Seen as a symptom in opiate withdrawal
- Exaggerated pain symptom seen in patients with drug abuse history
  - Pain that is sometimes neglected due to those patients as being seen as seeking opiate pain medications.

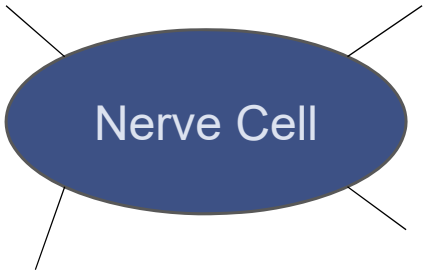
WHY ARE OPIATE INAPPROPRIATE FOR CHRONIC PAIN?

WHY ARE OPIATE INAPPROPRIATE FOR CHRONIC PAIN?



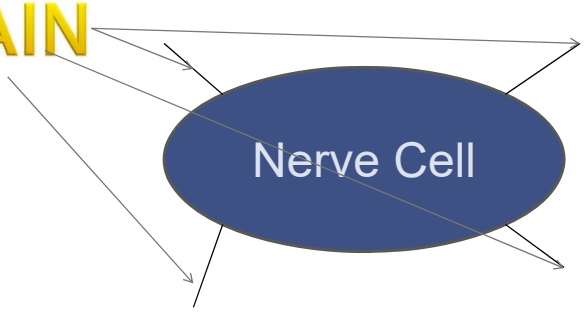
WHY ARE OPIATE INAPPROPRIATE FOR CHRONIC PAIN?

PAIN



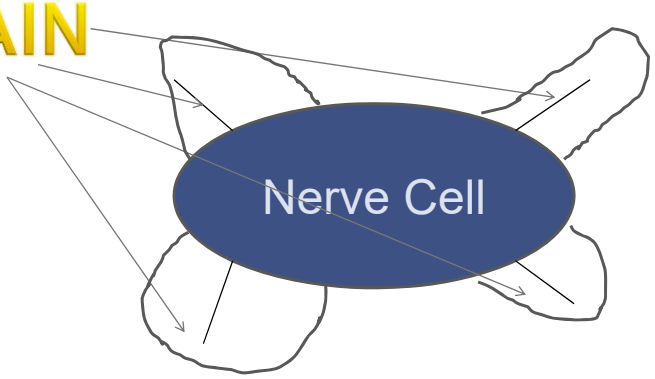
WHY ARE OPIATE INAPPROPRIATE FOR CHRONIC PAIN?

PAIN



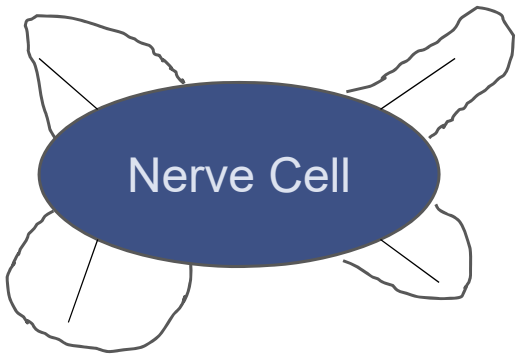
WHY ARE OPIATE INAPPROPRIATE FOR CHRONIC PAIN?

PAIN



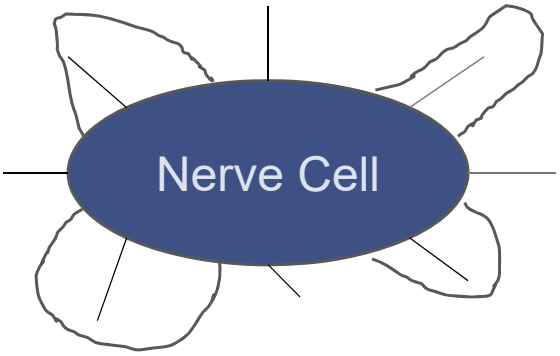
WHY ARE OPIATE INAPPROPRIATE FOR CHRONIC PAIN?

PAIN



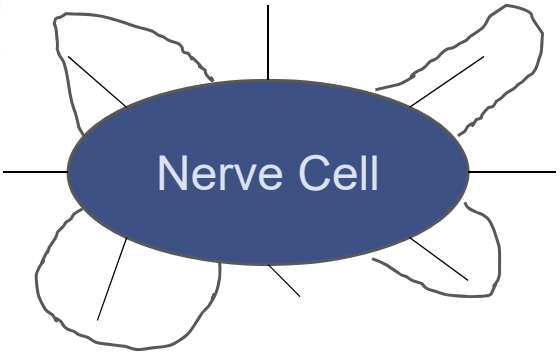
WHY ARE OPIATE INAPPROPRIATE FOR CHRONIC PAIN?

PAIN



WHY ARE OPIATE INAPPROPRIATE FOR CHRONIC PAIN?

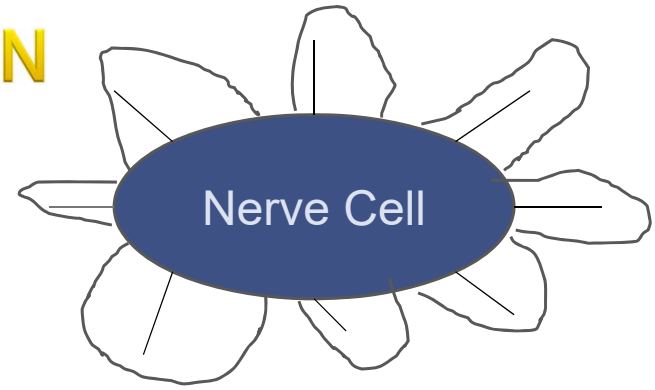
PAIN



↑OPIATES

WHY ARE OPIATE INAPPROPRIATE FOR CHRONIC PAIN?

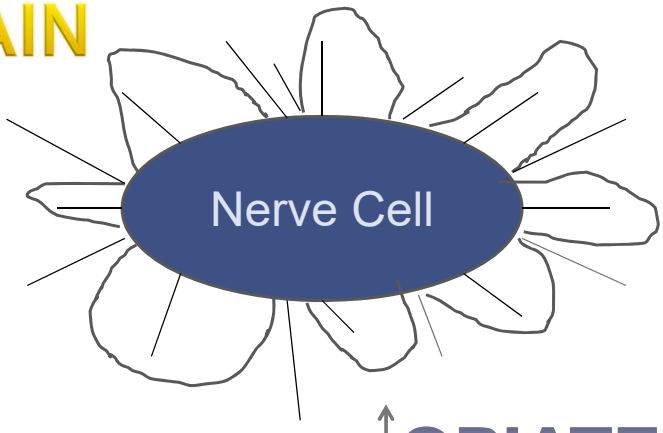
PAIN



OPIATES

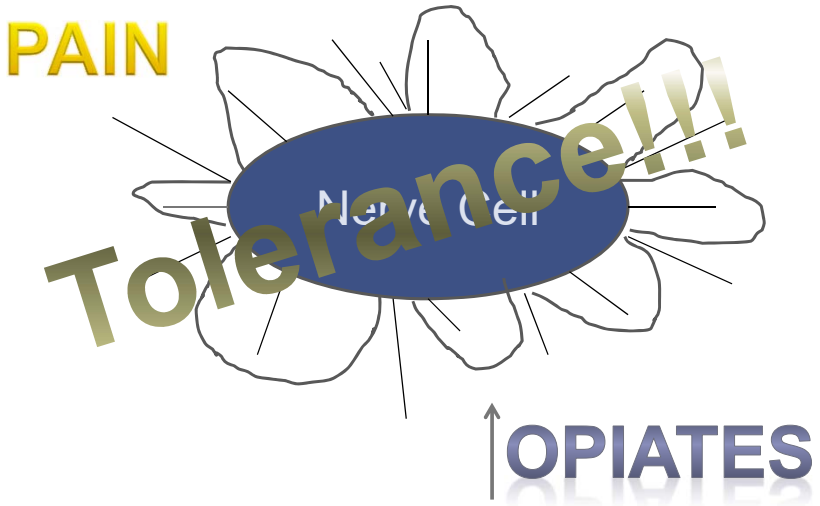
WHY ARE OPIATE INAPPROPRIATE FOR CHRONIC PAIN?

PAIN

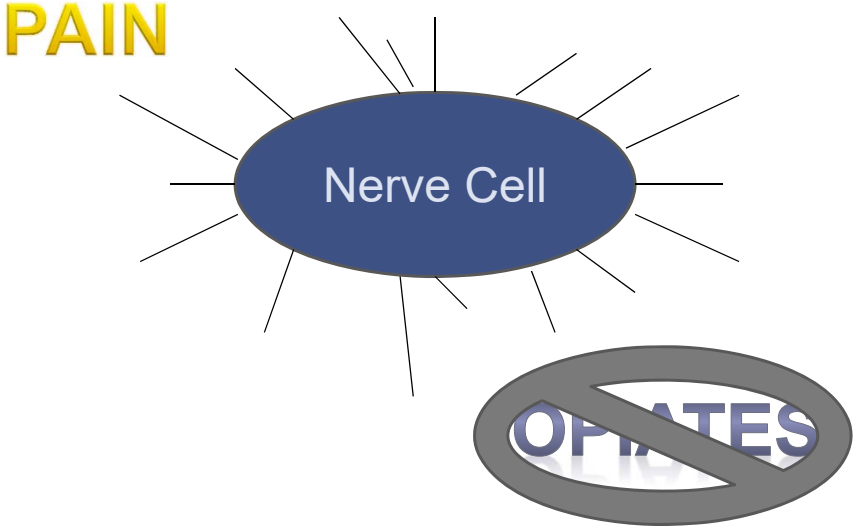


OPIATES

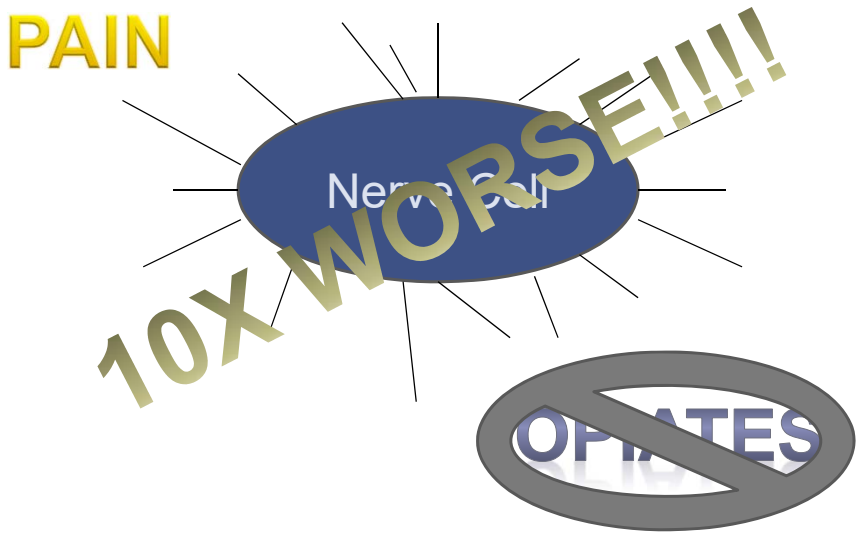
WHY ARE OPIATE INAPPROPRIATE FOR CHRONIC PAIN?



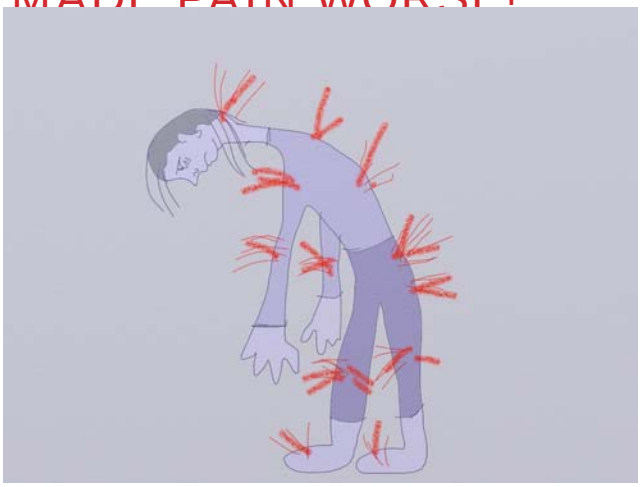
WHY ARE OPIATE INAPPROPRIATE FOR CHRONIC PAIN?



WHY ARE OPIATE INAPPROPRIATE FOR CHRONIC PAIN?



WE HAVE MADE PAIN WORSE!

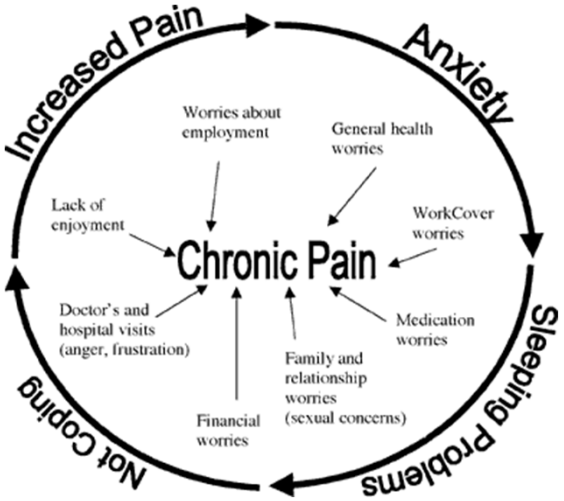


http://4.bp.blogspot.com/-HpYZw0M89o0/Ty2IqzGF9nI/AAAAAAAADLw/2TkffOaXWm0/s1600/Fibromyalgia%2B120204-771016.jpg

TREATMENT

http://www.pathoutofpain.com.au/tapes_cds/images/help.gif

TREATMENT



TREATMENT

Education

NSAID/Tylenol

Physical Therapy/OMT/Chiropractic Care

Drug Therapy

CBT

DRUG THERAPY

Gabapentin

- Pregabalin
  - Inappropriate for patients with Opiate Use Disorder

SNRI

- Venlafaxine
- Duloxetine

Tricyclic Antidepressants

- Amitriptyline

Baclofen

APAP (Scheduled)

Lidocaine Patches

Capsaisin

OPIATE USE DISORDER – CASE STUDY

36 year old female presents asking for medication for cravings. She specifically is asking for Suboxone. She is in substance abuse treatment programming at this time. This is her 3rd time through programming. Her drug of choice is IV heroin but she also uses nasal cocaine. She uses cocaine most of the time that she uses heroin. It helps her to be able to use more heroin because it will help her stay awake and prolong her high.

Review of symptoms is negative except of moderate anxiety, chronic low back pain and difficulty sleeping.

SUBOXONE

BUPENORPHINE/NALOXONE

WHY SUBOXONE (BUPRENORPHINE/NALOXONE)?

It is not a pure opiate replacement, a partial agonist.

Has two mechanisms of craving reduction

- Kappa Receptor
- Mu Receptor

Will block opiates so patients cannot get high.

Although it has a high potential for abuse, higher doses are required for a high.

Patient do not get high on therapeutic doses of Suboxone for addiction therapy.

HOW DOES SUBOXONE WORK?

Buprenorphine

- High affinity binding to μ opiate receptors
- Partial μ agonist and weak Kappa antagonist activity

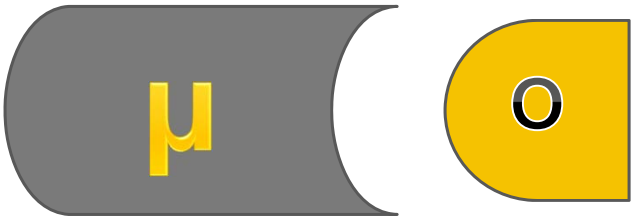
Naloxone

- Pure opioid antagonist that competes and displaces opioids at opioid receptor sites
- Used to prevent diversion of the drug

HOW DOES SUBOXONE WORK?

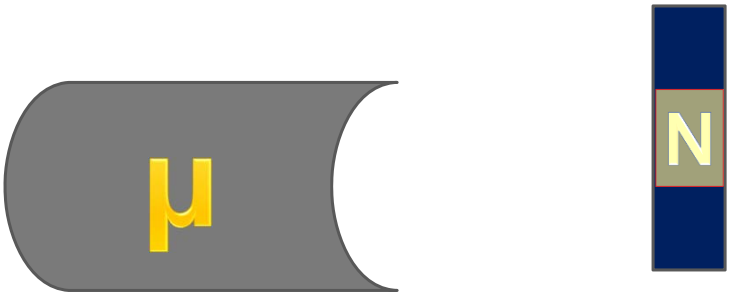
- **Patient takes medication sublingually**
- **Buprenorphine is absorbed but naloxone is not.**
- **Do not drink or eat while taking medication**
- **Can take 5-10 minutes to dissolve**

MECHANISM OF ACTION

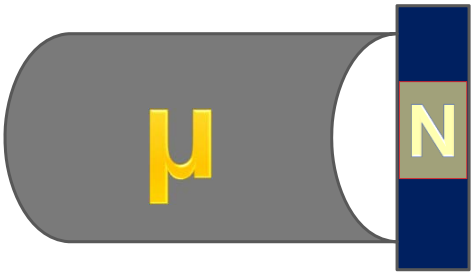


MECHANISM OF ACTION

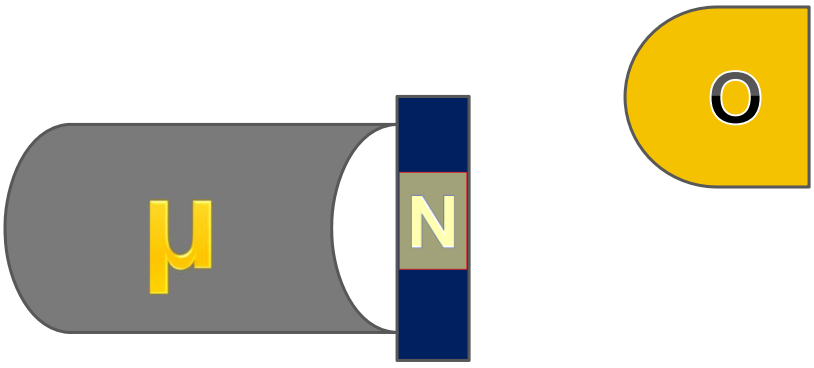
NALTREXONE



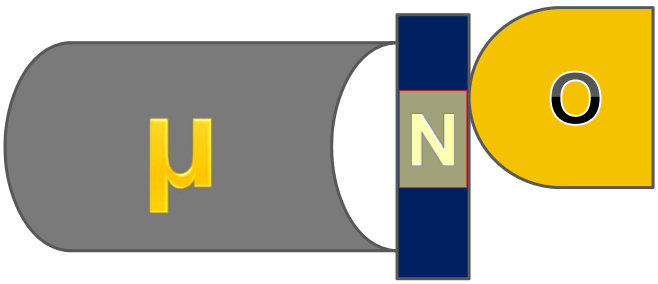
NALTREXONE



NALTREXONE



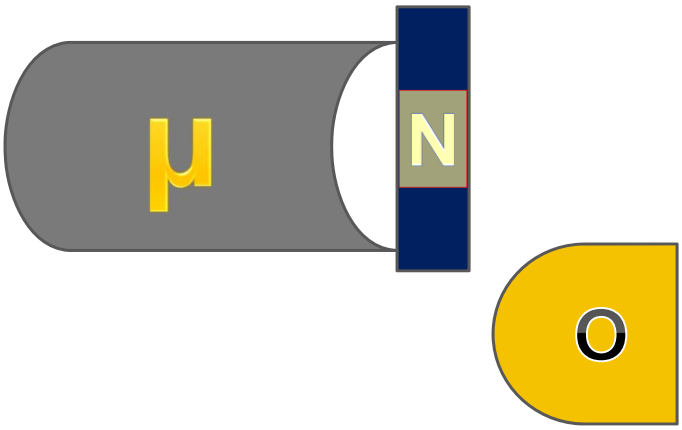
NALTREXONE



NALTREXONE

NALTREXONE

NALTREXONE



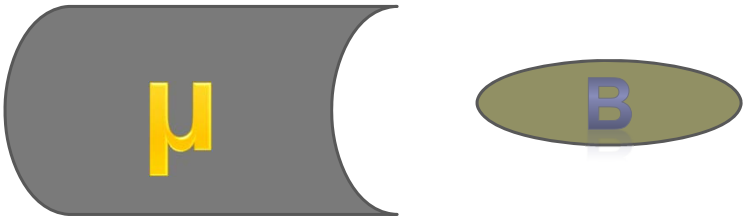
VIVITROL????

Once a month injection of extended release naltrexone.

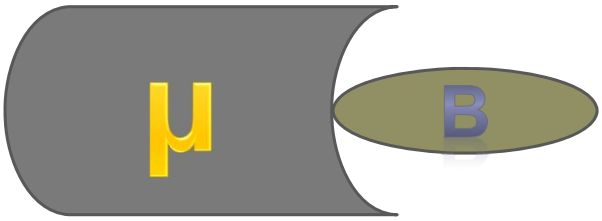
Downside

- Expense
- Medical emergencies requiring opiate use
- Controversy – If a person wants recovery why can't they take the pill?

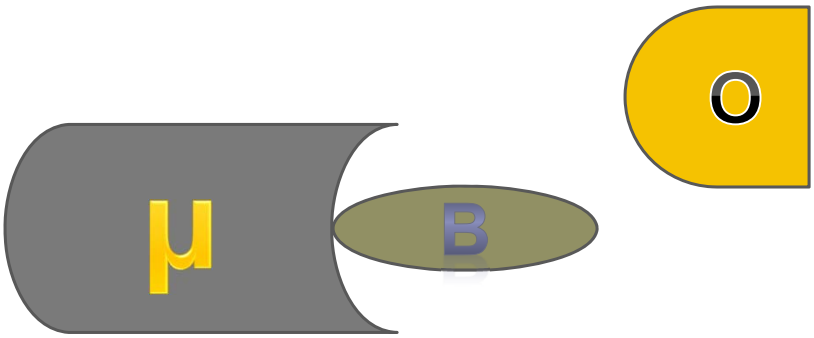
BUPRENORPHINE



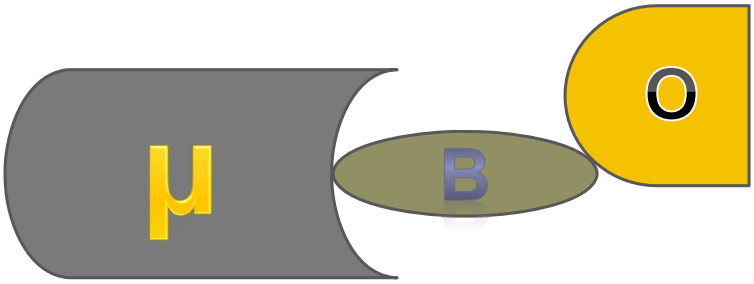
BUPRENORPHINE



BUPRENORPHINE

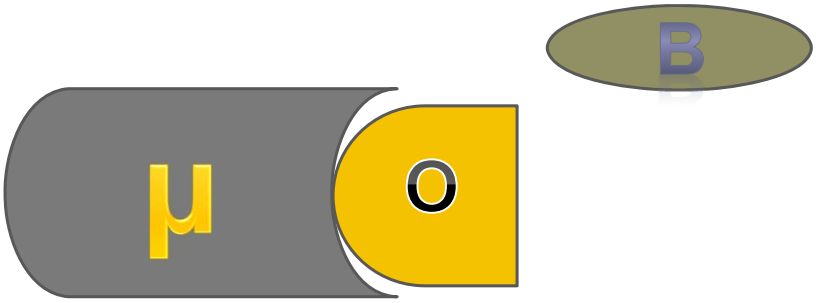


BUPRENORPHINE

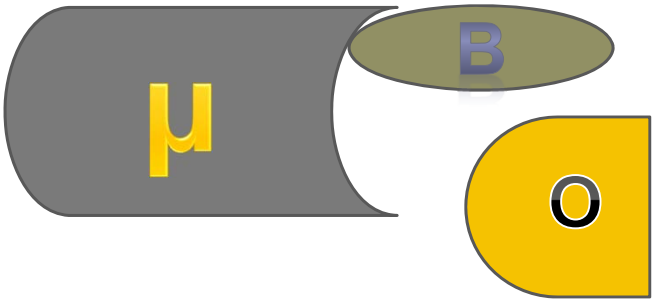


BUPRENORPHINE

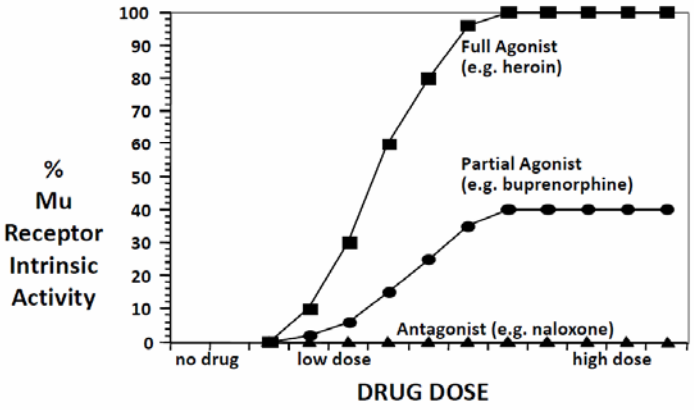
BUPRENORPHINE



BUPRENORPHINE



Buprenorphine is a Partial Agonist



PHARMACOLOGY OF FULL VS. PARTIAL AGONISTS

Buprenorphine can precipitate withdrawal if it displaces a full agonist from the mu receptors

- Buprenorphine only partially activates the receptors, therefore a net decrease in activation occurs and withdrawal develops

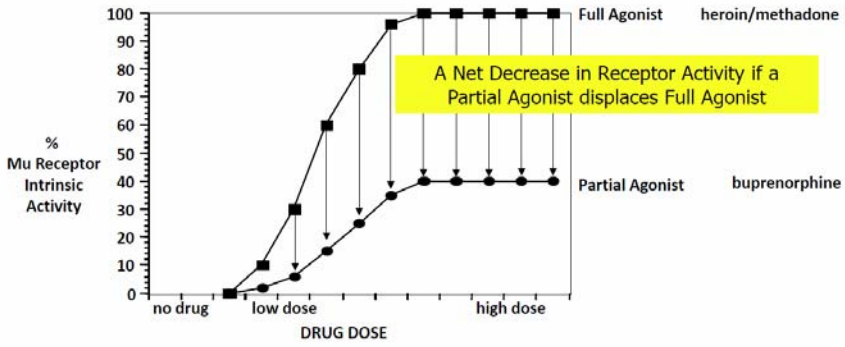


Table 2. Dose-dependent changes in Mu Opioid Receptor Availability by Sublingual Buprenorphine in Three Heroin-Dependent Volunteers

	BUP 0 mg	BUP 2 mg		BUP 16 mg			
Region	BP	BP	(% Δ from BUP 0)	BP	(% Δ from BUP 0)	F-value	p
Prefrontal cortex (BA 10)	1.38 ± 0.40	0.76 ± 0.33	(−47 ± 9)	0.23 ± 0.15	(−85 ± 9)	42.0	.002*
Prefrontal cortex (BA 11)	1.62 ± 1.00	0.79 ± 0.30	(−46 ± 16)	0.30 ± 0.20	(−82 ± 7)	6.2	.06
Anterior cingulate (BA 32)	1.67 ± 0.56	0.97 ± 0.39	(−43 ± 5)	0.23 ± 0.13	(−86 ± 5)	25.2	.005*
Anterior cingulate (BA 25)	1.65 ± 0.86	0.85 ± 0.33	(−46 ± 7)	0.30 ± 0.23	(−84 ± 8)	11.9	.02*
Anterior temporal cortex	1.55 ± 0.54	0.82 ± 0.41	(−49 ± 8)	0.24 ± 0.13	(−85 ± 5)	28.4	.004*
Caudate nucleus (dorsal)	2.29 ± 0.30	1.32 ± 0.32	(−42 ±12)	0.34 ± 0.17	(−85 ± 1)	61.5	.001*
Caudate nucleus (ventral)	2.79 ± 1.00	1.43 ± 0.60	(−50 ± 6)	0.49 ± 0.24	(−82 ± 6)	19.9	.008*
Putamen	1.82 ± 0.26	0.99 ± 0.23	(−46 ± 7)	0.32 ± 0.02	(−82 ± 2)	73.4	.0007*
Thalamus	2.70 ± 0.55	1.57 ± 0.40	(−42 ± 9)	0.56 ± 0.17	(−79 ± 8)	32.4	.003*
Hypothalamus	1.47 ± 0.76	0.71 ± 0.30	(−50 ± 9)	0.07 ± 0.21	(−95 ± 14)	8.7	.03*
Amygdala	1.82 ± 0.79	1.13 ± 0.49	(−37 ± 15)	0.23 ± 0.06	(−86 ± 3)	13.3	.02*
Midbrain	1.01 ± 0.22	0.55 ± 0.22	(−47 ± 11)	0.15 ± 0.09	(−85 ± 11)	27.0	.005*
Cerebellum	1.09 ± 0.40	0.72 ± 0.43	(−36 ± 21)	0.23 ± 0.18	(−82 ± 13)	16.3	.01*

Data represents the mean ± 1 s.d. of μOR binding potential (Bmax/Kd) in three heroin dependent volunteers studied during the sublingual administration of buprenorphine at 0 mg (placebo), 2 mg, and 16 mg. The percent change respect to the placebo condition is indicated in parenthesis (% Δ from BUP 0). Dose-dependency of receptor availability was examined for each individual region with one-way repeated measures ANOVA (df = 2,4). Their corresponding F-values and level of significance achieved (p) are shown in the right columns. BUP = buprenorphine; BP = binding potential.

*Statistically significant at $p < .05$.

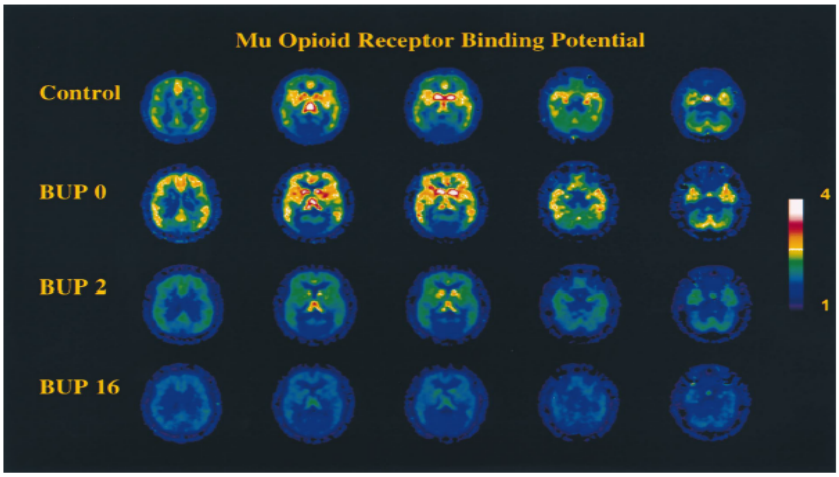


Figure 1. Parametric images of binding potential (Bmax/Kd) (extracted from Logan plot slopes with occipital cortex as input function) in two representative subjects: a non-dependent control (Control), and a matched heroin-dependent volunteer. The latter is shown under placebo conditions (BUP 0), during treatment with 2 mg (BUP 2) and 16 mg (BUP 16) sublingual liquid. Images are scaled so the occipital cortex binding potential, an area devoid of mu opioid receptors, is equal to 1. Five anatomical levels are shown, from superior (left) to inferior (right). The pseudocolor scale utilized is presented on the right side, using a color range depicting binding potential values from 1 to 4.

MU RECEPTOR BINDING AND BUPRENORPHINE

2mg Binds 36-50%

16mg Binds 79 to 95%

DRUG THERAPY FOR CHRONIC PAIN

Gabapentin

- Pregabalin
  - Inappropriate for patients with Opiate Use Disorder

SNRI

- Venlafaxine
- Duloxetine

Tricyclic Antidepressants

- Amitriptyline

Baclofen

APAP (Scheduled)

Lidocaine Patches

Capsaisin

SUBOXONE?!?!?!?

SUBOXONE INDUCTION

SUBOXONE enters the bloodstream from under the tongue.

- SUBOXONE takes a short time (about 5 to 10 minutes, but sometimes more) to dissolve completely. Don't chew or swallow the tablets, because less SUBOXONE will be absorbed into your bloodstream, it will not work as well, and your withdrawal symptoms could worsen
- Talking while the tablets are dissolving can interfere with how well SUBOXONE is absorbed
  - You may want to do something that doesn't require talking, such as reading or watching television, while waiting for the tablets to dissolve
  - Let family and friends know that you won't be able to answer them or talk on the phone during this time

http://www.quickstopclinic.com/pictures/suboxone_table5.jpg

CLINIC GOAL OF INDUCTION

Clinic goal will to be maintaining patients on a mediation dosage of 12-16mg daily. Doses higher than 16mg will be rare and unique circumstances.

Should be noted that MRI studies show that receptors in the brain are totally covered at 16mg of buprenorphine. Our goal to stay at 16mg is to help prevent tolerance and risk of chronic pain syndrome.

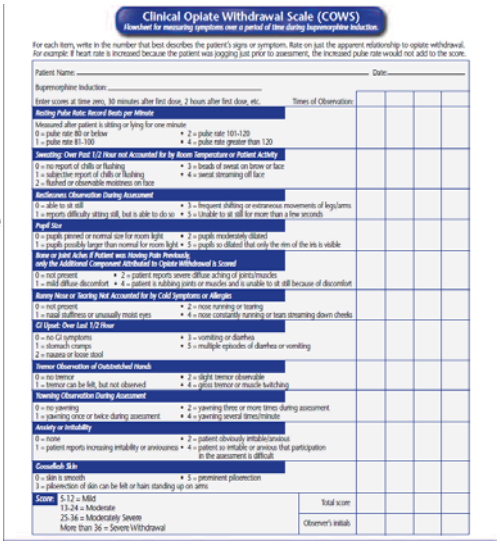
Withdrawal Symptoms will be assessed using the COWS score.

SUBOXONE INDUCTION DAY 1

Inducted with 2-4mg of buprenorphine Dose depending on COWS score

Observed 1-2 hours the given a second dose if withdrawal symptoms reappear.

8-12mg maximum dose recommended on day 1



Clinical Opiate Withdrawal Scale (COWS)

Flowsheet for measuring symptoms over a period of time during buprenorphine induction

For each item, write in the number that best describes the patient's signs or symptom. Rate on just the apparent relationship to opiate withdrawal. For example if heart rate is increased because the patient was jogging just prior to assessment, the increased pulse rate would not add to the score.

Patient Name: _____ Date: _____

Buprenorphine Induction: _____

Enter scores at time zero, 30 minutes after first dose, 2 hours after first dose, etc.	Times of Observation:				
Resting Pulse Rate: Record Beats per Minute Measured after patient is sitting or lying for one minute 0 = pulse rate 80 or below • 2 = pulse rate 101-120 1 = pulse rate 81-100 • 4 = pulse rate greater than 120					
Sweating: Over Past 1/2 Hour not Accounted for by Room Temperature or Patient Activity 0 = no report of chills or flushing • 3 = beads of sweat on brow or face 1 = subjective report of chills or flushing • 4 = sweat streaming off face 2 = flushed or observable moistness on face					
Restlessness / Observation During Assessment 0 = able to sit still • 3 = frequent shifting or extraneous movements of legs/arms 1 = reports difficulty sitting still, but is able to do so • 5 = unable to sit still for more than a few seconds					
Pupil Size 0 = pupils pinned or normal size for room light • 2 = pupils moderately dilated 1 = pupils possibly larger than normal for room light • 5 = pupils so dilated that only the rim of the iris is visible					
Bone or Joint Aches if Patient was Having Pain Previously, only the Additional Component Attributed to Opiate Withdrawal is Scored 0 = not present • 2 = patient reports severe diffuse aching of joints/muscles 1 = mild diffuse discomfort • 4 = patient is rubbing joints or muscles and is unable to sit still because of discomfort					
Runny Nose or Tearing Not Accounted for by Cold Symptoms or Allergies 0 = not present • 2 = nose running or tearing 1 = nasal stuffiness or unusually moist eyes • 4 = nose constantly running or tears streaming down cheeks					
GI Upset: Over Last 1/2 Hour 0 = no GI symptoms • 3 = vomiting or diarrhea 1 = stomach cramps • 5 = multiple episodes of diarrhea or vomiting 2 = nausea or loose stool					
Tremor Observation of Outstretched Hands 0 = no tremor • 2 = slight tremor observable 1 = tremor can be felt, but not observed • 4 = gross tremor or muscle twitching					
Yawning Observation During Assessment 0 = no yawning • 2 = yawning three or more times during assessment 1 = yawning once or twice during assessment • 4 = yawning several times/minute					
Anxiety or Irritability 0 = none • 2 = patient obviously irritable/anxious 1 = patient reports increasing irritability or anxiousness • 4 = patient so irritable or anxious that participation in the assessment is difficult					
Gooseflesh Skin 0 = skin is smooth • 5 = prominent piloerection 3 = piloerection of skin can be felt or hairs standing up on arms					
Score: 5-12 = Mild 13-24 = Moderate 25-36 = Moderately Severe More than 36 = Severe Withdrawal	Total score				
	Observer's initials				

SUBOXONE INDUCTION DAY 2

If there are withdrawal symptoms or cravings after 8-12mg on Day one, dose should be increased on Day 2

Initial 12-16mg on day 2.

Observed 1-2 hours and increase in 2mg increments when withdrawal symptoms return. Day 2 dose should not exceed 16mg

If withdrawal symptoms do not return within a few hours, you have established the patient's maintenance dose.

Most doses are between 12-16mg

SUBOXONE INDUCTION DAY 3

If the patient experiences withdrawal symptoms or cravings after taking a total of 16mg on Day 2, first assess whether patient is taking medication correctly (letting it dissolve under the tongue, not talking until it is dissolved, etc.)

If medication is administered correctly dose should be increased to 18-20mg.

Day 3 doses should not exceed 32mg/day.

- Many states have laws dictating maximum doses on Suboxone. Ohio is one of those states. Physicians are strongly encouraged to keep doses at 16mg/day

Doses higher than this will not harm the patient but will do little to decrease patient's cravings, due to ceiling effect.

Patients who require a high dose should be re-evaluated at the time of induction and/or monitored for diversion

OPIATE USE DISORDER – CASE STUDY

36 year old female presents asking for medication for cravings. She specifically is asking for Suboxone. She is in substance abuse treatment programming at this time. This is her 3rd time through programming. Her drug of choice is IV heroin but she also uses nasal cocaine. She uses cocaine most of the time that she uses heroin. It helps her to be able to use more heroin because it will help her stay awake and prolong her high.

Review of symptoms is negative except of moderate anxiety, chronic low back pain and difficulty sleeping.

OPIATE USE DISORDER – CASE STUDY

Start Naltrexone 25mg day one, if no withdrawal symptoms continue at 50mg daily

Start baclofen 10mg TID for muscle spasm and cravings.

Strongly encourage narcotics anonymous and aftercare

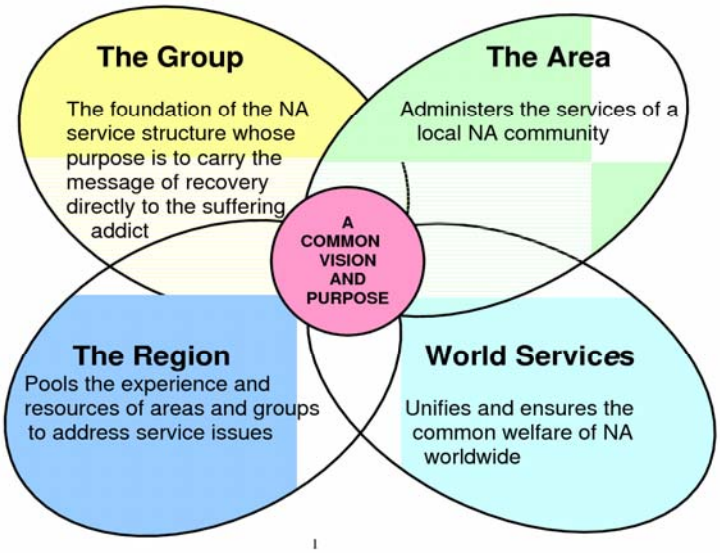
N.A. 12 STEPS

1. We admitted that we were powerless over our addiction, that our lives had become unmanageable.
2. We came to believe that a Power greater than ourselves could restore us to sanity.
3. We made a decision to turn our will and our lives over to the care of God as we understood Him.
4. We made a searching and fearless moral inventory of ourselves.
5. We admitted to God, to ourselves, and to another human being the exact nature of our wrongs.
6. We were entirely ready to have God remove all these defects of character.
7. We humbly asked Him to remove our shortcomings.
8. We made a list of all persons we had harmed, and became willing to make amends to them all.
9. We made direct amends to such people wherever possible, except when to do so would injure them or others.
10. We continued to take personal inventory and when we were wrong promptly admitted it.
11. We sought through prayer and meditation to improve our conscious contact with God as we understood Him, praying only for knowledge of His will for us and the power to carry that out.
12. Having had a spiritual awakening as a result of these steps, we tried to carry this message to addicts, and to practice these principles in all our affairs.

Your Role as a Leader in Narcotics Anonymous

Worksheet 1 – *The NA Service Structure*

Each part of the NA structure has its own function, and in turn connects with the other parts to form a system with one primary purpose – to carry the message to the addict who still suffers.



CONCLUSION

Medication assisted treatment is a great tool to help a person work their recovery program but is not a magic pill for sobriety

Medication should be used as an individualized plan to target symptoms of patients they are exhibiting. Do not be blinded by what they are asking for and take time to educate the patients as to the best choice. Do not underestimate their knowledge.

Suboxone is has great potential in reducing cravings in patients with opiate use disorder, but is only available to a narrow population of patient who are addicted to opiates. Non-Suboxone providers should not be intimidated to initiate medications for cravings!!!!

REFERENCES

Addolorato G, Caputo F, Capristo E, et al. Baclofen Efficacy in Reducing Alcohol Craving and Intake: A preliminary Double-Blind Randomized Controlled Study. Alcohol and Alcoholism (2002) 37(5): 504-508.

BupPractice. *Standard Induction Protocol.* DATA 2000- Qualifying Buprenorphine Training. Accessed on 10/23/2013 at http://www.buppractice.com/howto/induction/protocol.

Brower K, Kim H, Strobbe S, Karam M, et al. A Randomized Double-Blind Pilot Trial of Gabapentin Versus Placebo to Treat Alcohol Dependence adnComorbid Insomnia. Alcoholism: Clinical and Experimental Research. Aug 2008(32)8. pp 1429-1438

Dranitsaris G, Selby P, Negrete JC. Meta-analyses of placebo-controlled trials of acamprosate for the treatment of alcohol dependence: impact of the combined pharmacotherapies and behavior interventions study. J Addict Med. 2009 Jun; 3(2): 74-82.

Diagnostic and statistical manual of mental disorders: DSM-5. (5th ed.). (2013). Washington, D.C.: American Psychiatric Association.

IOM (Institute of Medicine). 2010. *Returning Home from Iraq and Afghanistan: Preliminary Assessment of Readjustment Needs of Veterans, Service Members, and Their Families.* Washington, DC: The National Academies Press.

Johnson BA, Rosenthal N, Capece JA, Wiegand F, et al. Topiramate for treating alcohol dependence: a randomized controlled trial. JAMA. 2007 Oct 10; 298(14):1641-51.

Johnson BA, Ait-Daoud N, Wang XQ, Penberthy JK, et al. Topiramate for the treatment of cocaine addiction: a randomized clinical trial. JAMA Psychiatry. 2013 Dec; 70(12): 1338-46.

Larimer ME, Palmer RS, Marlatt GA. Relapse prevention An overview of Marlatt's cognitive-behavioral model. Alcohol Res Health. 1999;23(2):151-60. PubMed PMID: 10890810.

Mason BJ, Quello S, Goodell V, Shadan F, Kyle M, Begovic A. Gabapentin treatment for alcohol dependence: A randomized clinical trial. JAMA Intern Med. 2014 Jan; 174(1):70-1.

Nuijten M, Blanken P, van den Brink W, Hendriks V. Treatment of crack-coaine dependence with topiramate: A randomized controlled feasibility trial in the Netherlands. Drug Alcohol Depend. 2014 Feb 26. pii: S0376-8716

Stahl, S., & Stahl, S. (2009). *Stahl's essential psychopharmacology: The prescriber's guide.*(3rd ed.). New York: Cambridge University Press

Stoops WW, Lile JA, Glaser PE, Hays LR, Rush CR. Influence of acute buproprion pre-treatment on the effects of intranasal cocaine. Addiction. 2012 Jun; 107(6):1140-7

Targeting Self-Stigma in Returning Military Personnel and Veterans: A Review of Intervention Strategies . Journal Military Psychology Publisher APA Journals ISSN 0899-5605 (Print) 1532-7876 (Online) Collection PsycARTICLES® Issue Volume 22, Number 2 / April 2010 Category ARTICLES Pages 224-236

Volpicelli J, Alterman A, Hayashida M, O'brien C. Naltrexone in the Treatment of Alcohol Dependence. Arch Gen Psychiatry. 1992; 49(11): 876-880.

Weinick RM, et al. Programs Addressing Psychological Health and Traumatic Brain Injury Among U.S. Military Servicemembers and Their Families. Santa Monica: RAND Corporation, 2011.