Medication Assisted Treatment (MAT)
Medications Used in Substance Related Treatment and Recovery

Carl M. Dawson, M.S., MAC, LPC, Q-SAP

Independent Practice

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National Judicial College (NJC)

Reno, Nevada

National Drug Court Institute Faculty (NDCI)

Alexandria, Virginia

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Cdawson1028@yahoo.com

Disclaimer

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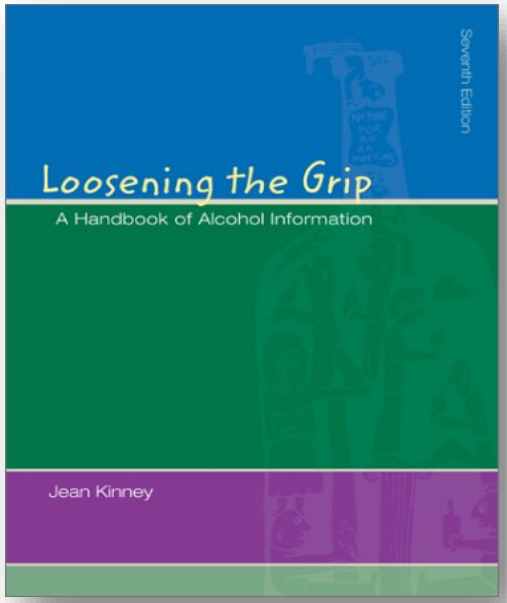
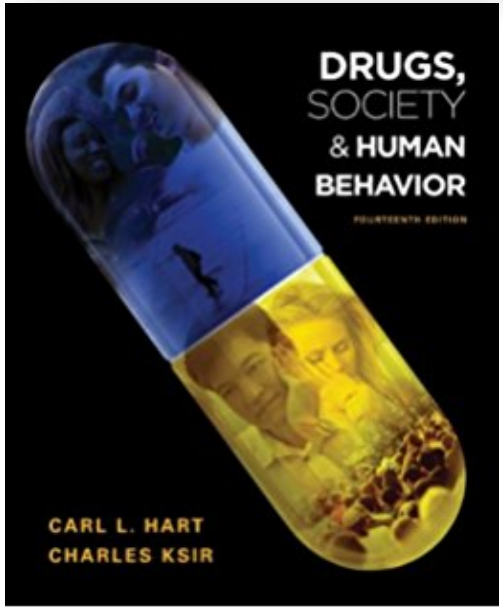
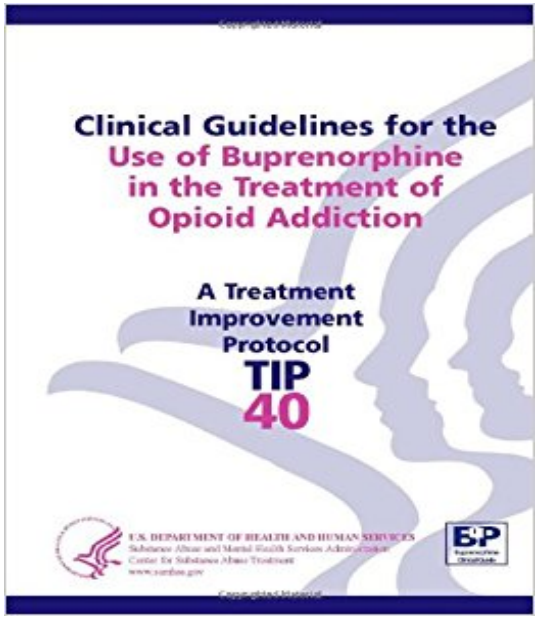
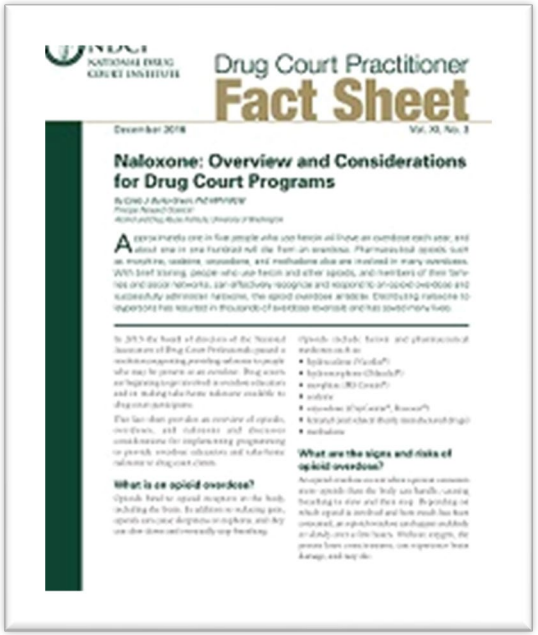
Recommended reading and reference material









A MESSAGE TO THE ATTENDEE:

"YOU WILL FIND THIS PRESENTATION DIFFICULT IF YOU DO NOT EMBRACE THE **BIOLOGICAL-PSYCHOLOGICAL-SOCIAL** MODEL OF ADDICTION SCIENCE"

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"**IN AN OTHER WORDS**, IF YOU HONESTLY BELIEVE RECOVERY IS A MATTER OF **WILL POWER** … THIS LECTURE IS NOT FOR YOU!"

OVERVIEW

After attending this session, the participant will be able to:

- UNDERSTAND MEDICATION-ASSISTED TREATMENT (MAT)
- IDENTIFY THE THERAPEUTIC TRIAD
- IDENTIFY HOW ANTI-DEPRESSANT MEDICATIONS ASSIST IN SUBSTANCE USE TREATMENT
- UNDERSTAND THE THREE (3) TYPES OF MAT TREATMENT OBJECTIVES
- EXPLORE ANTI-ALCOHOL MEDICATIONS
- UNDERSTAND WHAT MAKES OPIOID DRUGS DESIRABLE
- EXPLORE THE THERAPEUTIC DELEMIA ASSOCIATED WITH MAT
- EXAMINE THREE (3) MAT MEDICATIONS ASSOCIATED WITH OPIOID USE DISORDER AND THEIR CURRENT EFFECTIVENESS
- DISCUSS THE LENGTH OF TIME NECESSARY FOR MAT TO BE EFFECTIVE

Key terms and phrases in this section

- **Medicated Assisted Treatment (MAT):** MAT combines the use of medications and behavioral therapy to treat substance use disorders

- **The Therapeutic Triad:** Three necessary components for strong recovery

- **Relapse:** Resuming the use of a drug or drugs after a period of abstinence

- **Anti-depressant medications:** Psychiatric medications designed to improve the effectiveness of the brains neurotransmitters in treating depressive and anxious disorders

Medication-Assisted Treatment (MAT)

Medication-Assisted Treatment (MAT), combines the use of medications and behavioral therapy to treat substance use disorders

THE THERAPEUTIC TRIAD

MEDICTION-COUNSELING-EXERCISE

A frequently asked question:

“Isn’t Medication-Assisted Treatment, just switching one drug for another?”

THE THREE (3) MAIN OBJECTIVES OF MEDICATION-ASSISTED TREATMENT

- **FIRST (1ST) OBJECTIVE: STOP** THE **ILLICIT** DRUG USE!
- **SECOND (2ND) OBJECTIVE:** TO OBSTAIN FROM THE USE OF ALL MOOD-ALTERING SUBSTANCES INCLUDING ALCOHOL
- **THIRD (3RD) OBJECTIVE**: TO ELIMINATE THE OBSTICALES THAT LEAD TO **RELAPSE**

The biggest obstacle to overcome in the treatment of addiction disorders is

RELAPSE!

Do you know the number one cause of relapse?

Do you know the average number times most people relapse before finding solid recovery?

ANTI-DEPRESSANT MEDICATIONS

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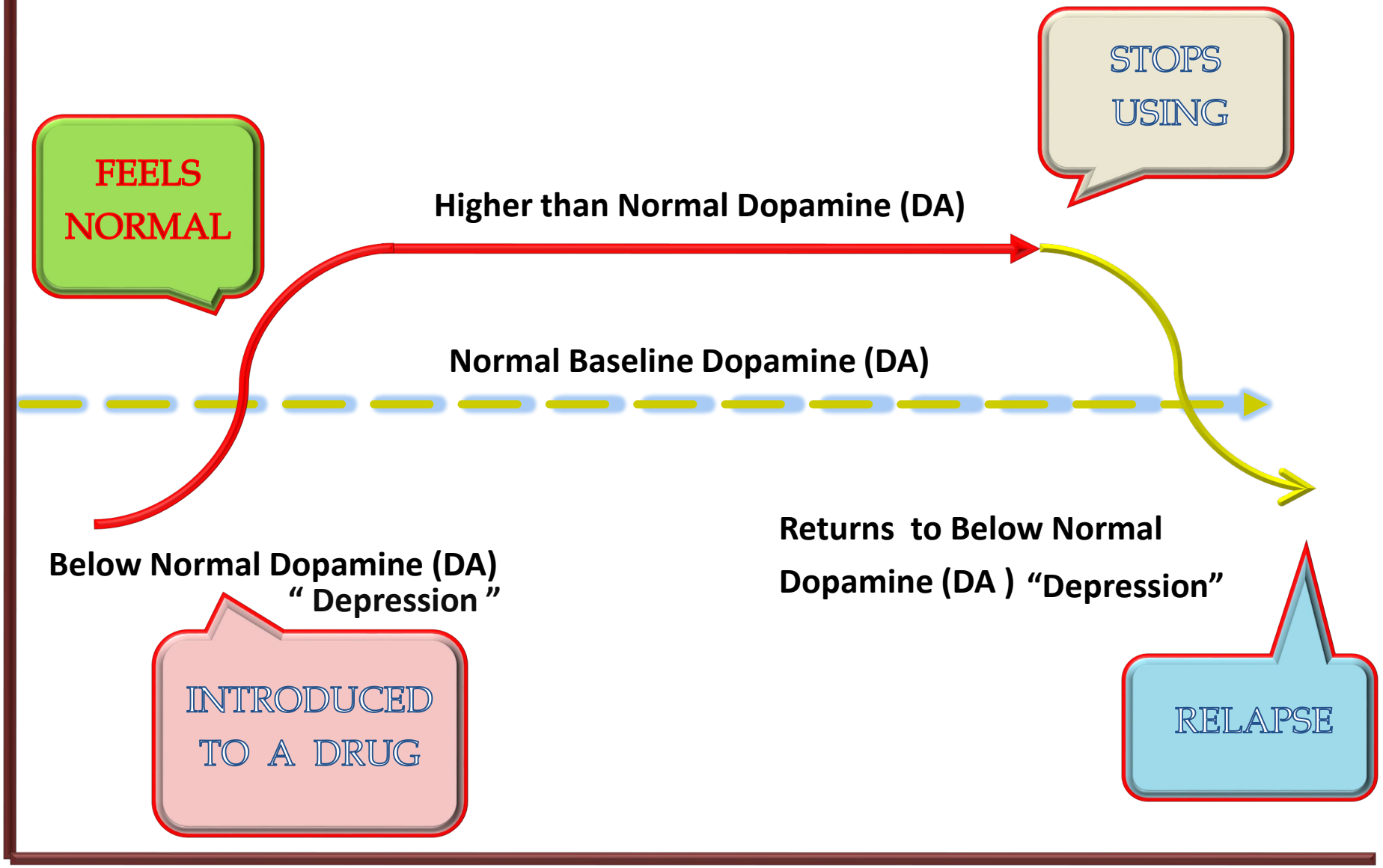
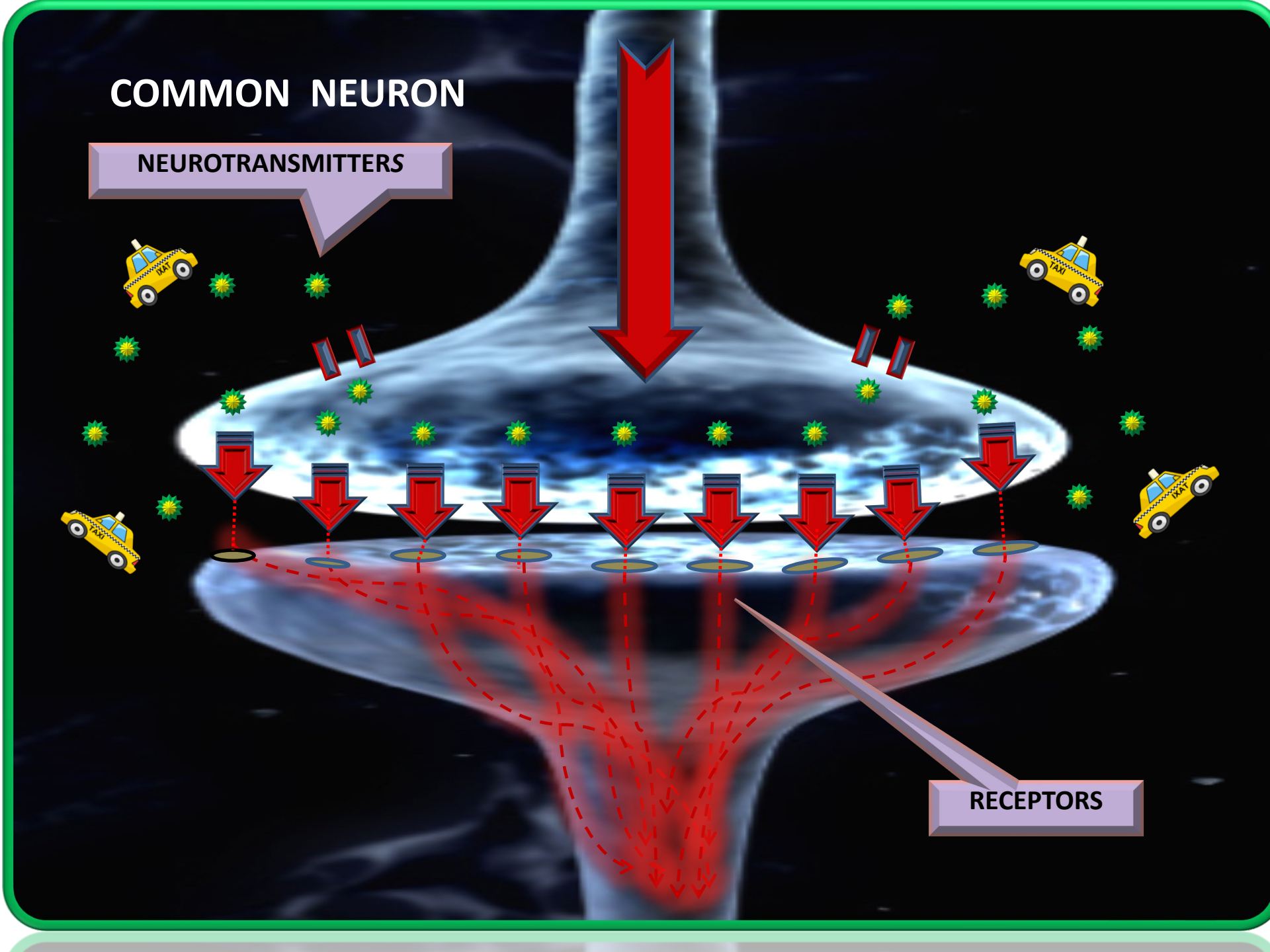
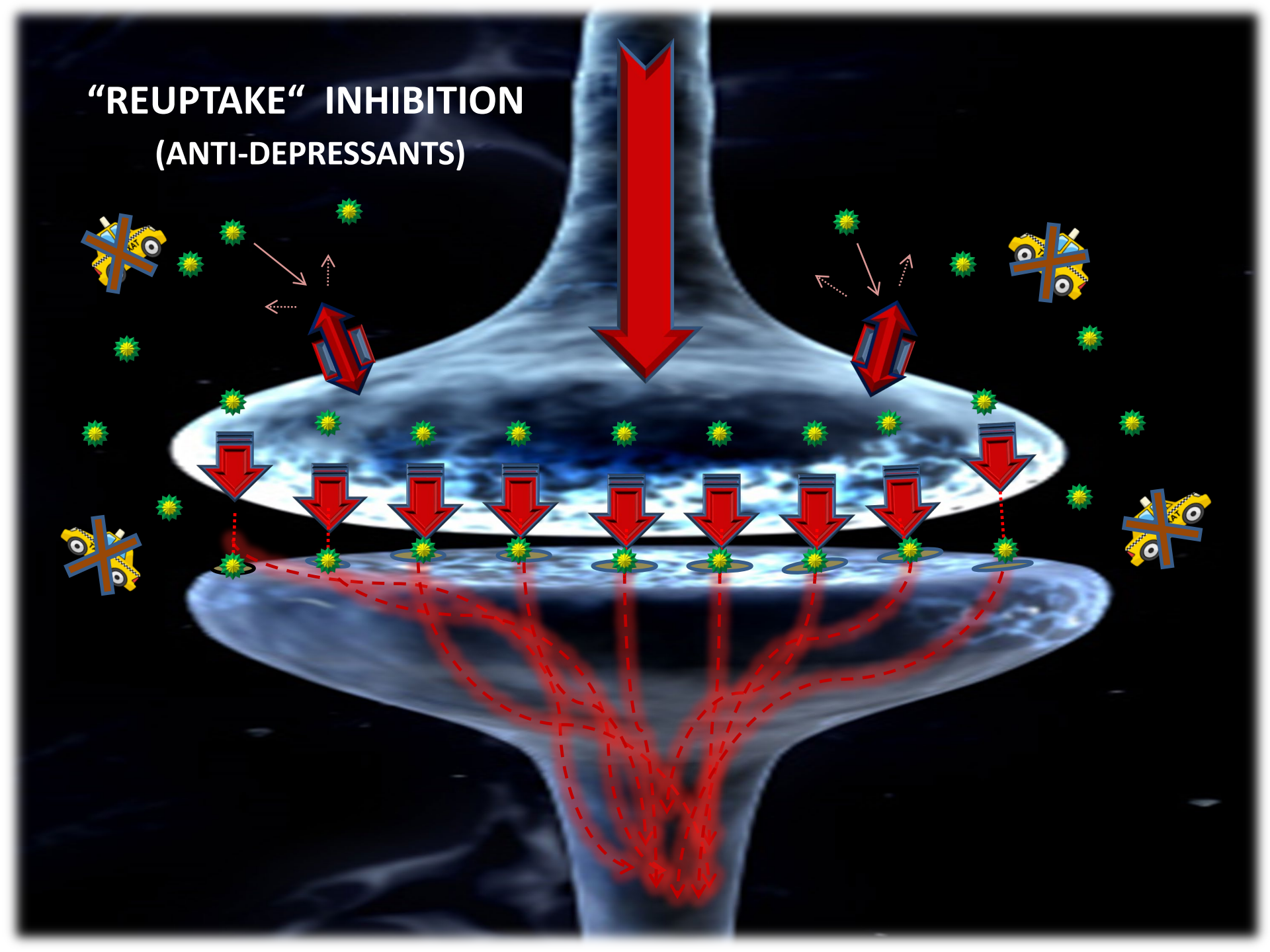
WHY CONSIDER ANTI-DEPESSANT MEDICATIONS IN SUBSTANCE ABUSE TREATMENT?

PSYCHIATRIC - “ALCOHOL“ MOOD DISORDERS




COMMON NEURON
NEUROTRANSMITTERS
RECEPTORS


"REUPTAKE" INHIBITION
(ANTI-DEPRESSANTS)

- **“DESIGNER“ ANTI-DEPRESSANT MEDICATIONS:**
  THESE MEDICATIONS ARE SPECIFICALLY DESIGNED TO EFFECT EITHER SERIOTONIN (5ht) OR NOREPINEPHRINE (NE) AND TO A LESSER EXTENT DOPAMINE (DA) NEUROTRANSMITTERS

  THESE MEDICATIONS INCLUDE :

  1 . PROZAC (5ht)
  2 . LEXAPRO (5ht)
  3 . CELEXA (5ht)
  4 . ZOLOFT (5ht)
  5 . CYMBALTA (5ht & NE)
  6 . WELLBUTRIN (NE & DA)

- THESE MEDICATIONS ARE EFFECTIVE FOR THE TREATMENT OF **“REACTIVE“** AND **“CLINICAL“** DEPRESSION

ANTI-ALCOHOL MEDICATIONS

Key terms and definitions used in this section

- **Anti-alcohol medications:** Medications used in the treatment of alcohol abuse and recovery

- **Anti-craving medications:** Medications specifically designed to reduce the biological and psychological craving for alcohol or other drugs

- **Homeostasis:** Keeping the internal activity of the body balanced

- **Glutamate:** Most plentiful neurotransmitter. Glu is known to stimulate and agitate brain activity. The excessive rebound production of Glu is considered the cause for the harsh physical symptoms associated alcohol withdrawal

- **GABA:** A neurotransmitter known to relax the activities of the brain, similar to Xanax and Alcohol

- **Endorphins:** Body produced opioids

Key terms and definitions continued

- **Tapering:** Progressively reducing the dosage or strength of a drug until the body no longer requires that drug in order to maintain normal functioning

- **Maintenance:** Providing a medically supervised alternative substance, for chronically drug dependent individuals who are unable or unwilling to stop using a drug

- **Harm Reduction:** Providing a medically supervised alternative substance, aimed at reducing the harmful consequences associated with drug use

POINTS OF REFERENCE . . .

- THE BODY DOES NOT HAVE SPECIFIC RECEPTORS FOR ALCOHOL; THEREFORE, ALCOHOL MUST MASQUERADE ITSELF AS EITHER A PAIN KILLER **(OPIOID)** OR AS AN ANTI-ANXIETY AGENT **(GABA)** IN ORDER TO GAIN ADMISSION INTO THE BODY

- THAT IS WHY SUBSTANCES THAT BLOCK THE UPTAKE OF OPIOID AND ANTI-ANXIETY DRUGS IN THE BODY ARE ALSO USED FOR THE TREATMENT OF ALCOHOL ABUSE AND RELAPSE


ANTABUSE
500

ANTI-ALCOHOL MEDICATIONS

- **ANTABUSE (DISULFIRAM):** IS A MEDICINE DESIGNED TO STOP THE NORMAL BREAK DOWN OF ALCOHOL IN THE BODY

- **ANTABUSE*:* STOPS THE BREAKDOWN OF ALCOHOL AT THE ACETALDEHYDE STAGE FOR APPROXIMATELY THREE (3) to FOURTEEN (14) DAYS**

- IT CAN BE DESPENSED AT **125mg** AND **500mg** DOSAGES

- **Should only be prescribed to individuals seeking complete abstinence!**

- **Should not be prescribed to individuals with advanced liver disease**

THE NORMAL ALCOHOL BREAKDOWN SEQUENCE

ADH

ACETALALHIDE IS VERY TOXIC TO THE BODY

ALDH

ACETATE

CO_2

H_2O

THE ALCOHOL BREAKDOWN SEQUENCE WITH "ANTABUSE"

ADH

ANABUSE CAUSES ACETALAHIDE TO ACCUMULATE IN THE BODY CAUSING SEVERE TOXIC REACTIONS

ANTABUSE AVERSE REACTIONS

THE FOLLOWING SYMPTOMS USUALLY OCCUR WITHIN FIVE (5) TO TEN (10) MINUTES AFTER COMBINING ALCOHOL WITH ANTABUSE:

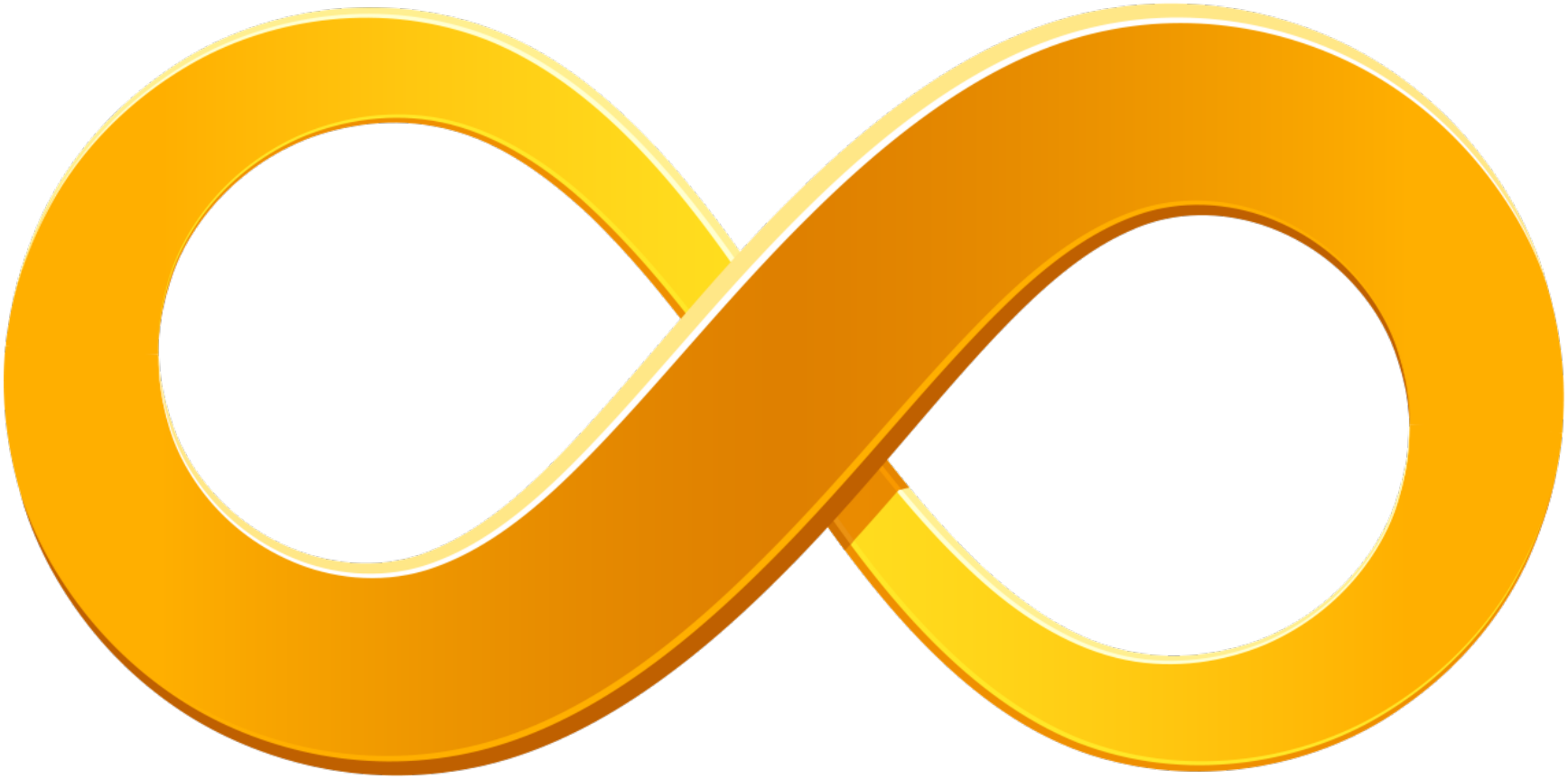
1. FLUSHING
2. SWEATING
3. THROBBING HEADACHE AND NECK PAIN
4. PALPITATIONS (HEART)
5. DYSPNEA (LABORED OR DIFFICULT BREATHING)
6. HYPERVENTILATION (INCREASED AND RAPID BREATHING)
7. TACHYCARDIA (FAST BEATING HEART)
8. HYPOTENSION (DECREASED BLOOD PRESSURE)
9. NAUSEA
10. VOMITING
11. POSSIBLE DEATH

YOUR BRAIN (hypothalamus) IS CONSTANTLY ATTEMPTING TO "RIGHT" ITSELF

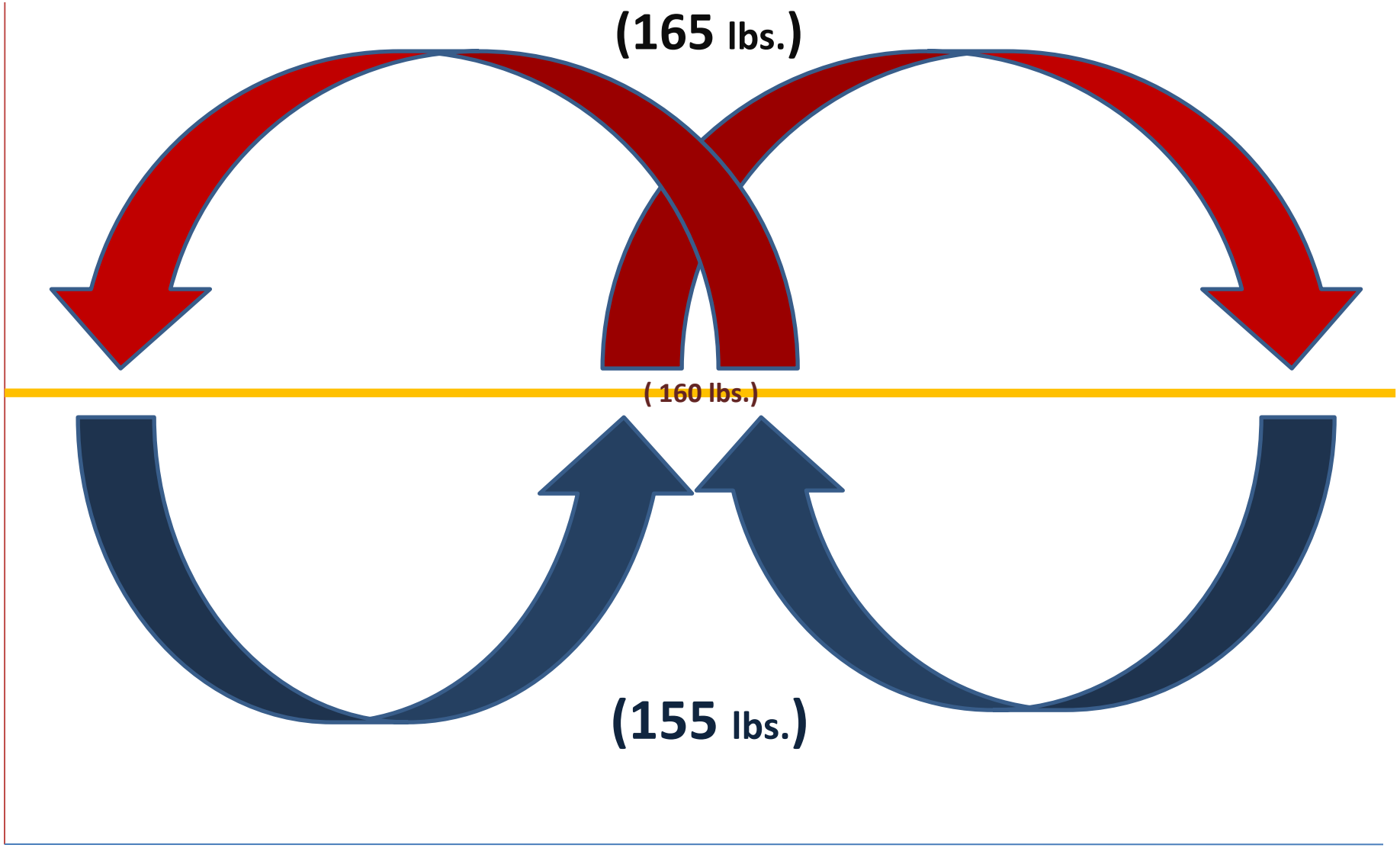
HOMEOSTASIS:

A TERM USED TO DESCRIBE THE BRAINS EFFORTS TO KEEP YOUR INTERNAL WORLD BALANCED!

OUR BODIES WORKS IN A CHRONIC "FEEDBACK" LOOP



“HOMEOSTASIS“ KEEPING EVERYTHING BALANCED



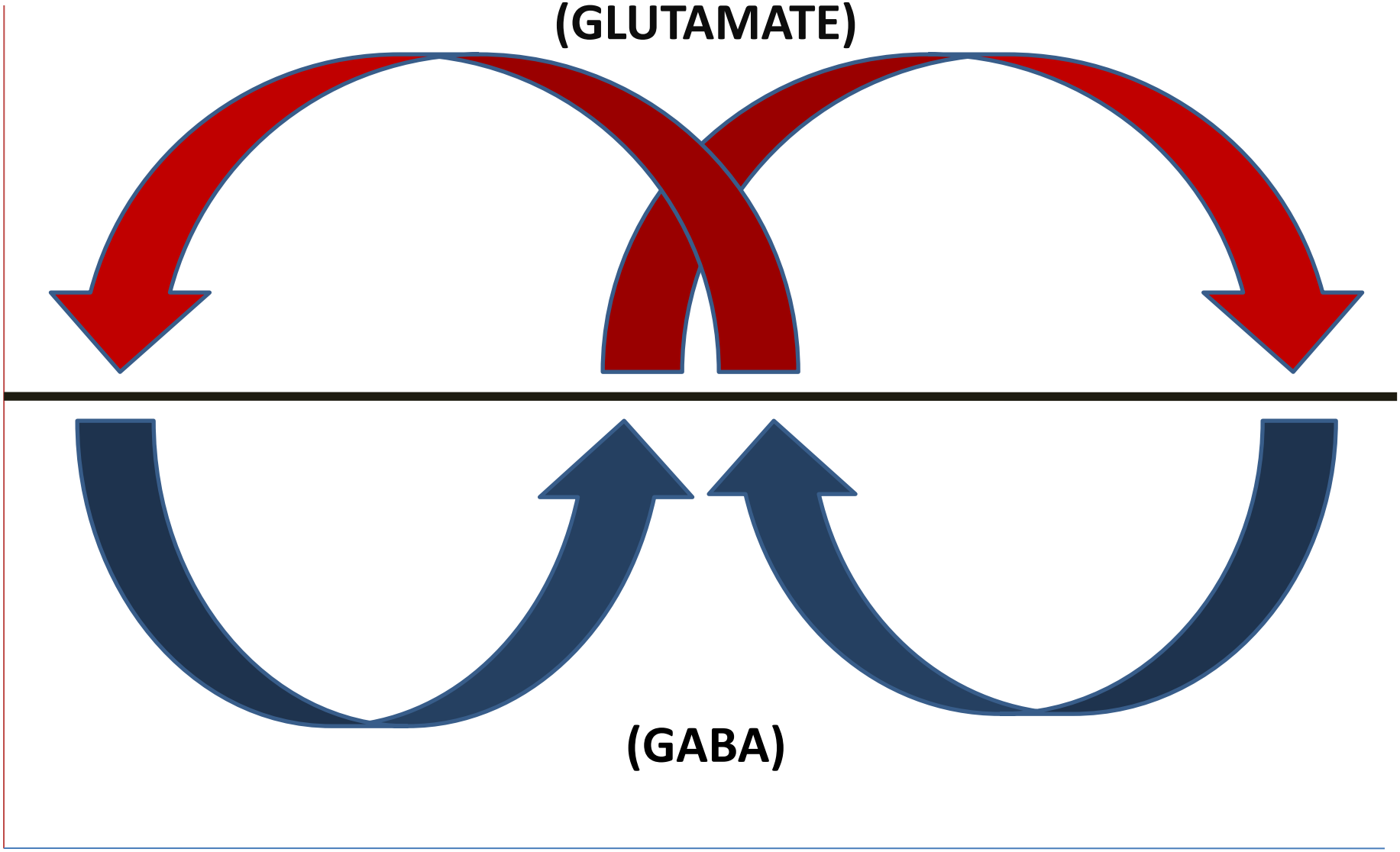
THE BIG THREE (3) NEUROTRANSMITTERS INVOLVED IN ANTI-CRAVING MEDICATIONS

Glutamate: Activates the **"under excited"** brain, producing the symptoms of a **"Hangover "**

GABA: Sedates the **"over excited"** brain, reduces anxiety, acts like Xanax or alcohol on the brain

Endorphins: "body produce morphine" Biological **"Pain Killers"** known as **Opioids**

“HOMEOSTASIS“ KEEPING EVERYTHING BALANCED



CAMPRAL (Acamprosate)

CAMPRAL (ACAMPROSATE): IS DESIGNED TO QUICKLY RESTORE THE GLUTAMATE SYSTEM AFTER DRINKING THUS, REDUCING THE NEED TO CONTINUE DRINKING IN ORDER TO AVOID THE UNPLEASANT WITHDRAWAL SYMPTOMS

BEST USED WITH INDIVIDUALS STRUGGLING WITH SOME SOBRIETY

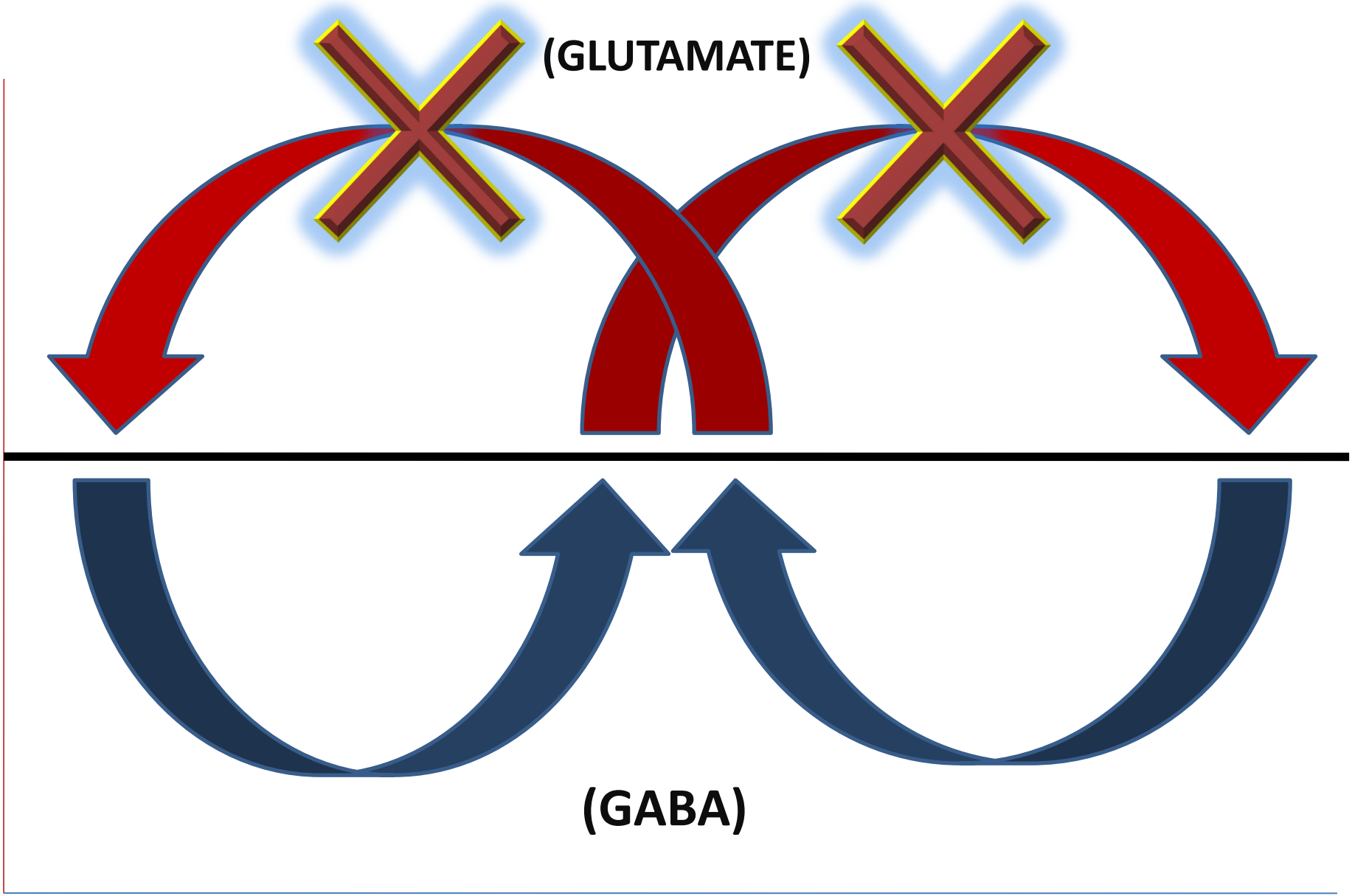
TARGETS ABSTINENCE, NOT HEAVY DRINKING

BEST USED WITH THE **"<u>BINGE</u>"** DRINKER

NOT METABOLIZED BY THE LIVER, AND CAN BE USED WITH POOR LIVER FUNCTIONS

PRESCRIBED AT 666 mg, THREE (3) TIMES PER DAY

“CAMPARAL” EFFECTS THE GLUTAMATE CYCLE



NALTREXONE

- **<u>NALTREXONE</u> (DON'T CONFUSE THIS WITH NALOXONE)** IS A FULL OPIOID **ANTAGONISTS** (BLOCKER)

- ORAL ADMINISTRATION IS LESS EFFECTIVE THAN TIME RELEASE INJECTIONS OR SUBCUTANIOUS IMPLANTS

- IS DESIGNED TO BLOCK THE RAPID RELEASE OF ENDORPHINS THAT SOME INDIVIDUALS EXPERIENCE USING ALCOHOL

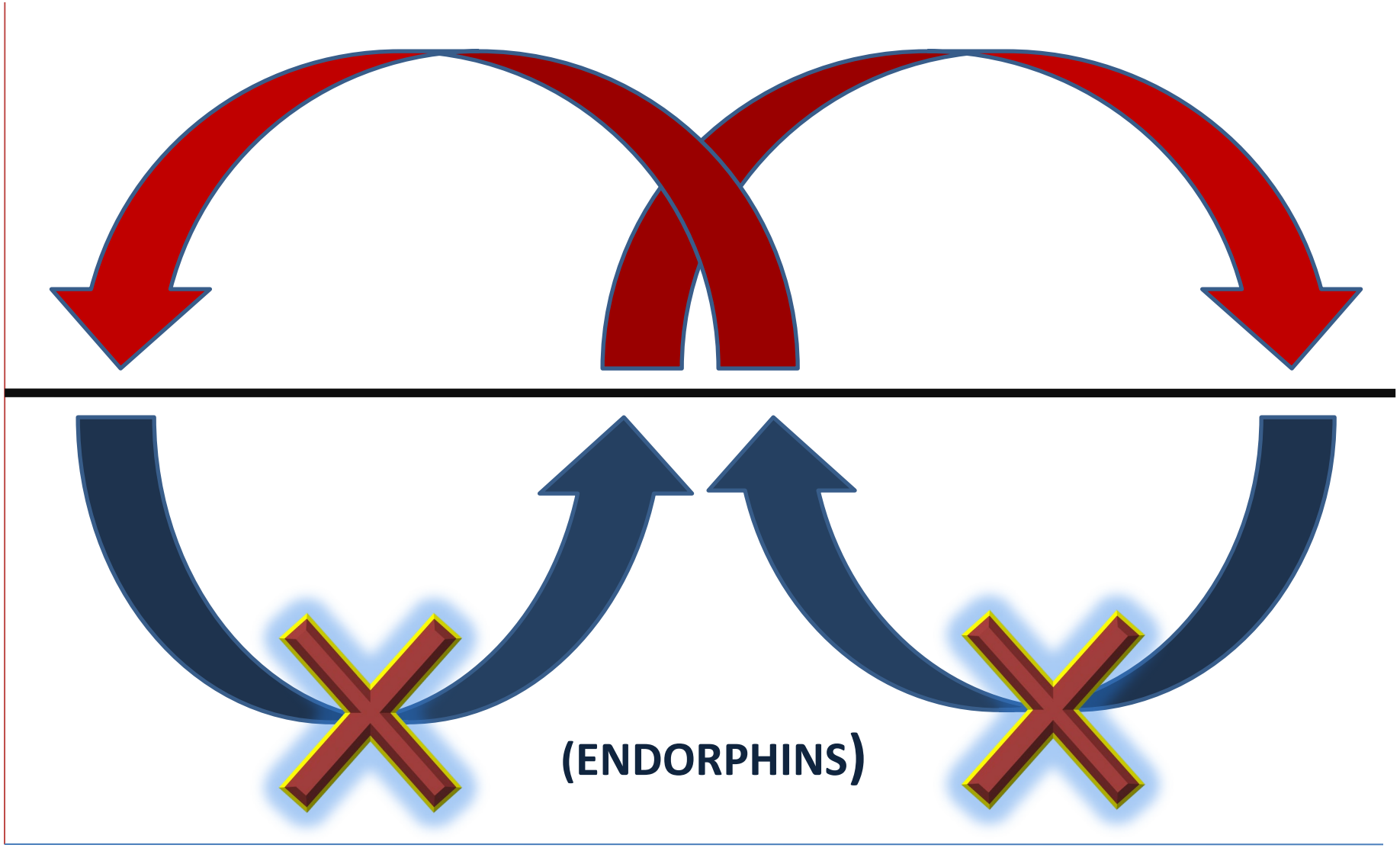
- IS APPROVED AS AN OPIOID BLOCKER

- **INJECTED NALTREXONE (VIVITROL "time released")** IS DESIGNED TO BLOCK THE ENDORPHIN **"RUSH"** AND REDUCES THE DESIRED EFFECTS OF ALCOHOL FOR APPROXIMATELY (30) DAYS

- MAY REDUCE ALCOHOL RELATED CRAVINGS

- IS BEST USED WITH **“CHRONIC”** DRINKERS
- RESEARCH SHOWS NALTREXONE DOES NOT REDUCE THE LIKEIHOOD OF ANY DRINKING, BUT DOES REDUCE THE LIKEIHOOD OF HEAVY DRINKING
- RESEARCH SHOWS NALTREXONE HELPS CONTAIN A **“SLIP”** RATHER THAN RESULTING IN FULL-BLOWN RELAPSE
- RESEARCH REPORTS THE MEDICATION DOES NOT WORK FOR EVERYONE
- IT IS EITHER VERY EFFECTIVE OR NOT AT ALL
- GENETICS MAY PLAY A FACTOR IN THE MEDICATIONS EFFECTIVENESS
- STARTING DOSAGE IS 25 mg TO 50 mg ONE (1) TIME DAILY
- REMAINS IN THE BODY APPROXIMATELY TWENTY-SIX (26) HOURS

“NALTREXONE” EFFECTS THE ENDORPHIN RELEASING CYCLES



Naltrexone (tablets, injection, subcutaneous implants)

AN INTRODUCTION TO OPIOID SUBSTANCES AND MEDICATIONS FOR OPIOID USE DISORDERS

Section overview

- What makes opioid drugs desirable
- What are **“Full Agonist”, “Partial Agonist”, “Antagonist”** and substances
- Explore **“Three (3) Therapeutic Objectives”** when treating Opioid Use Disorders
- Discuss **“The Therapeutic Dilemma”** when treating opioid abuse and dependence. When and when not to consider **Medication-Assisted Treatment**
- The use of medications in the treatment of opioid abuse, dependency and recovery **Methadone, Naltrexone, Buprenorphine**
- How long should a person remain on **MAT** for Opioid Use Disorders

What makes opioid drugs desirable

- Opioid substances are lipophilic **(fat-loving),** meaning they infiltrate high protein regions of the body quickly **(heroin vs. codeine)**

- Individuals' dependent on opioids become addicted to the **"RUSH"** of pleasure produced by the drug

- Opioid drugs that produce **"LESS OF A RUSH"** are less fat-loving and are more effective in treating opioid dependence **(methadone and buprenorphine)**

- Opioid overdose can be lethal, either when used alone or with other CNS depressant drugs **(1+1=3) effect**

- The most common cause of opioid related death is due to **respiratory arrest**

THE THREE (3) PRIMARY OPIOID RECEPTORS

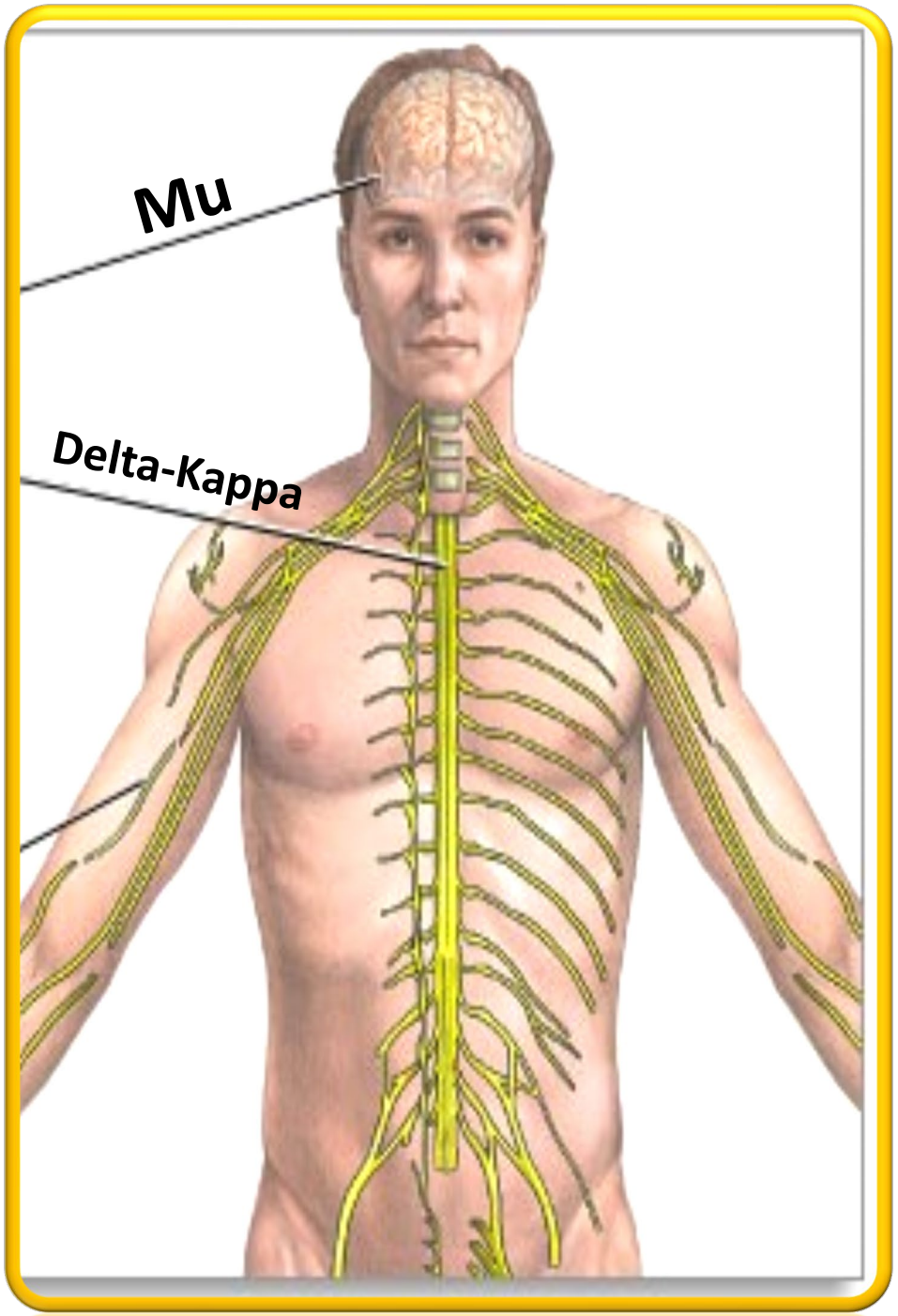
- **Mu RECEPTORS :**

  THE PRIMARY OPIOID RECEPTORS THAT HAVE THE STRONGEST ATTRACTION TO OPIOID SUBSTANCES . . . AND TRIGGER THE RELEASE OF PAIN AND PLEASURE PRODUCING CHEMICALS IN THE BRAIN

- **DELTA** AND **KAPPA** OPIOID RECEPTORS ARE LESS ATTRACTED TO OPIOID SUBSTANCES IN THE BRAIN

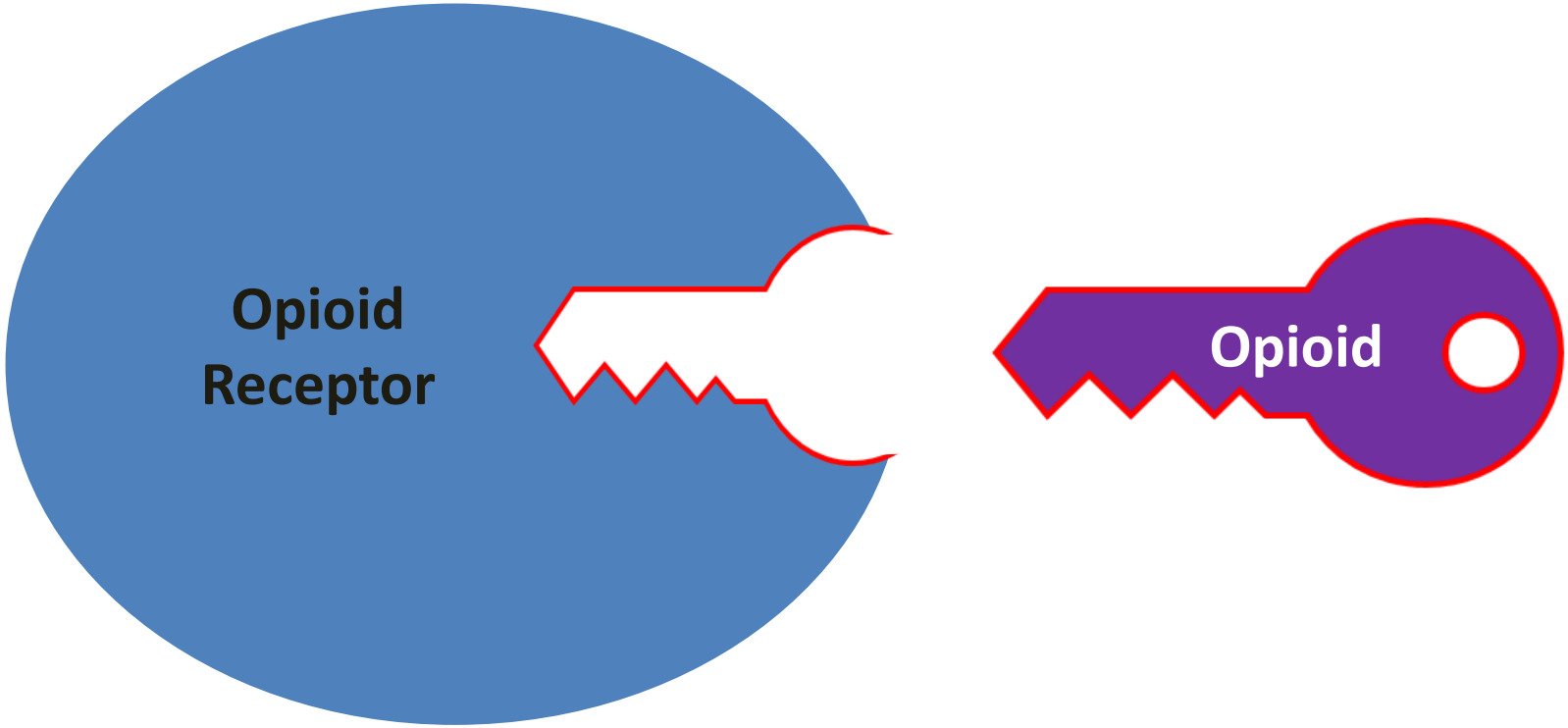
THE CENTRAL NERVOUS SYSTEM (CNS) CONSISTS OF THE BRAIN AND THE SPINAL CORD

THE GREATEST AMOUNT OF THE BODY'S OPIOID (Mu) RECEPTORS ARE LOCATED THROUGHOUT THE BRAIN, LESS IN THE SPINAL CORD.

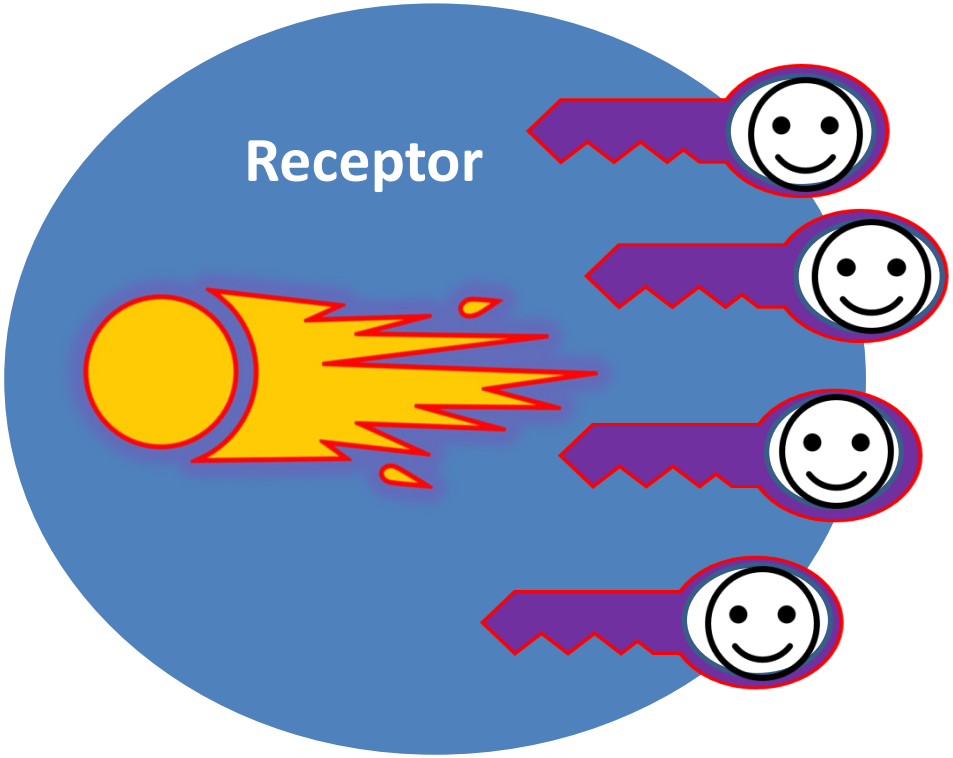


“Key into a Lock” Model

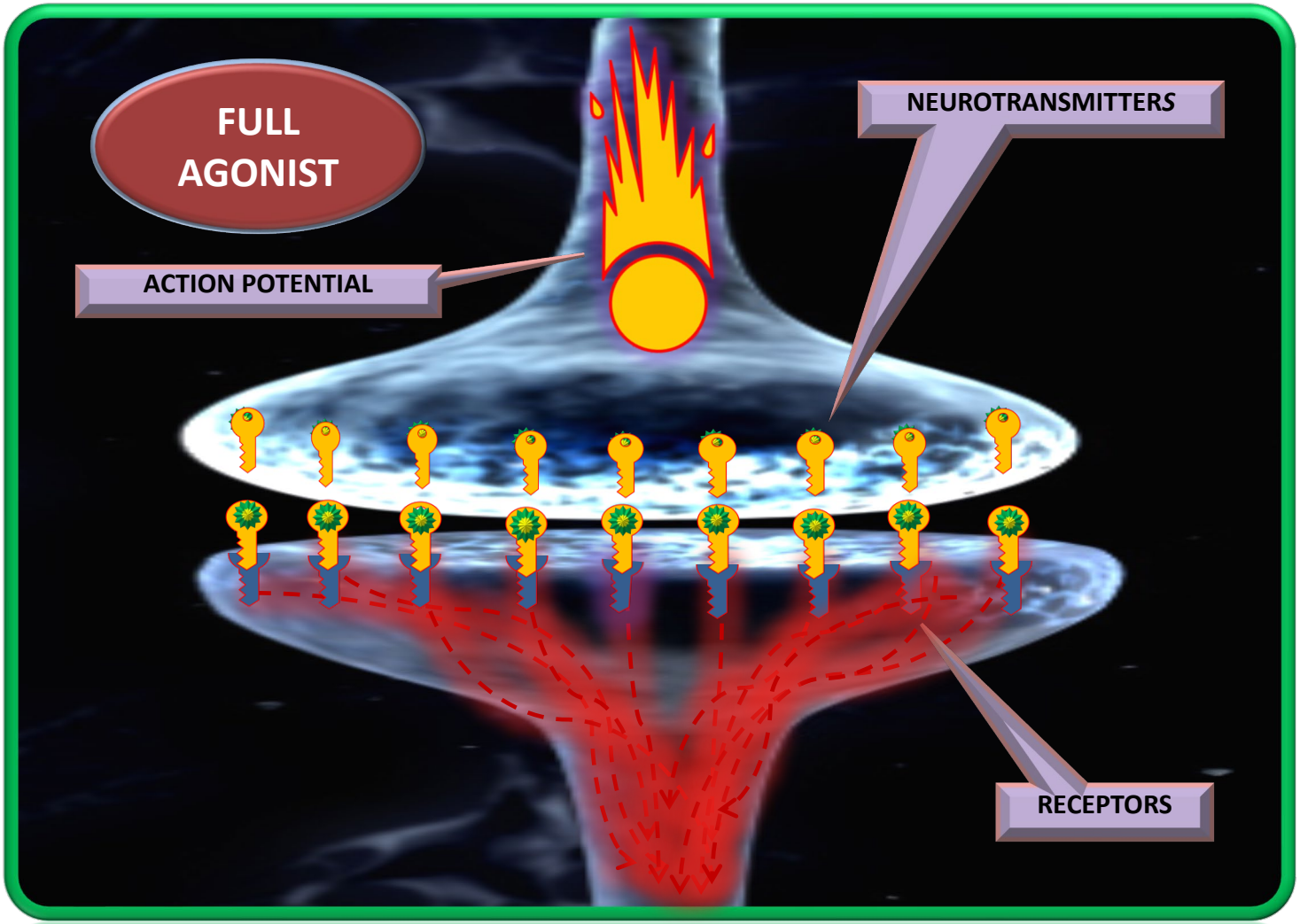
The key either turns **“On”** or turns **“Off”** the opioid receptor



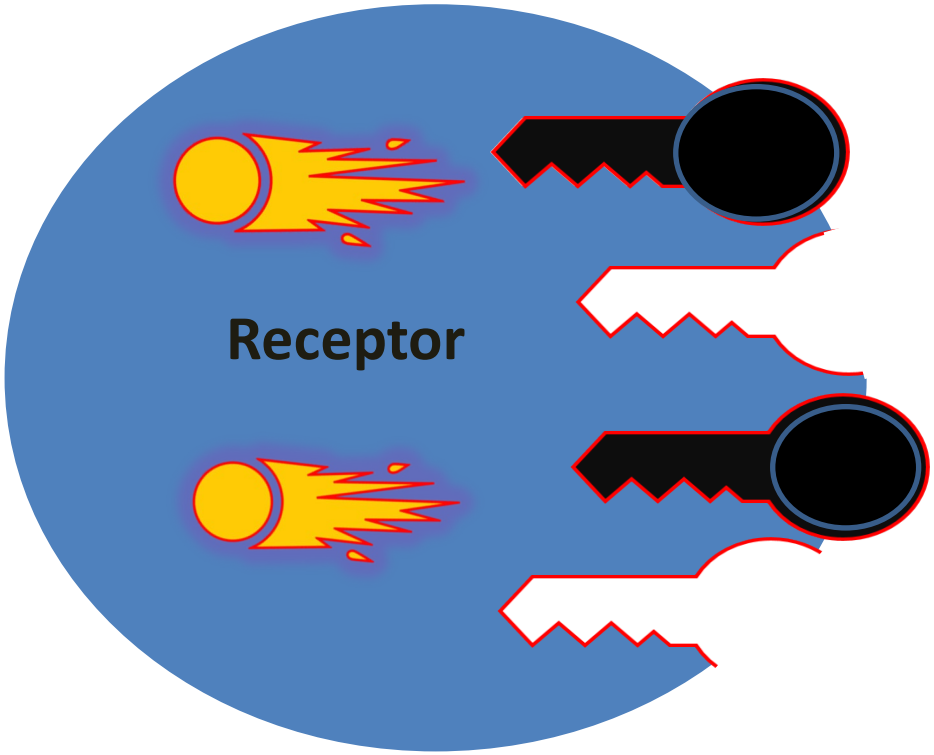
“Full” Agonists



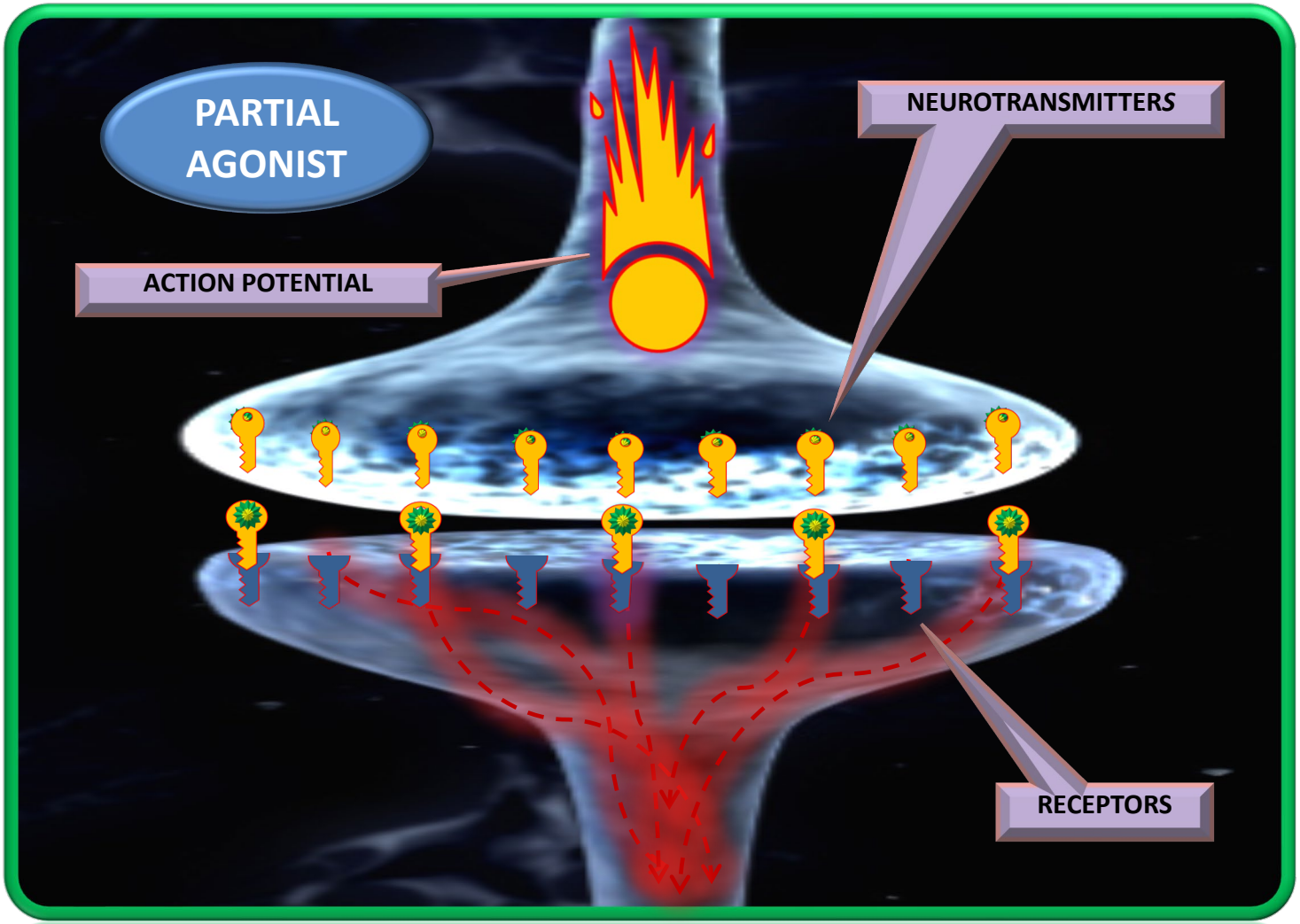
- **Full** agonists will occupy all the receptors
- **Full** agonists create a stronger potential than partial agonists
- **Full** agonists act like an **“On-Off”** light switch
- **Full** agonists, do not have a ceiling threshold which may result in single drug overdose
- **Methadone** is a **Full** agonist


FULL AGONIST
ACTION POTENTIAL
NEUROTRANSMITTERS
RECEPTORS

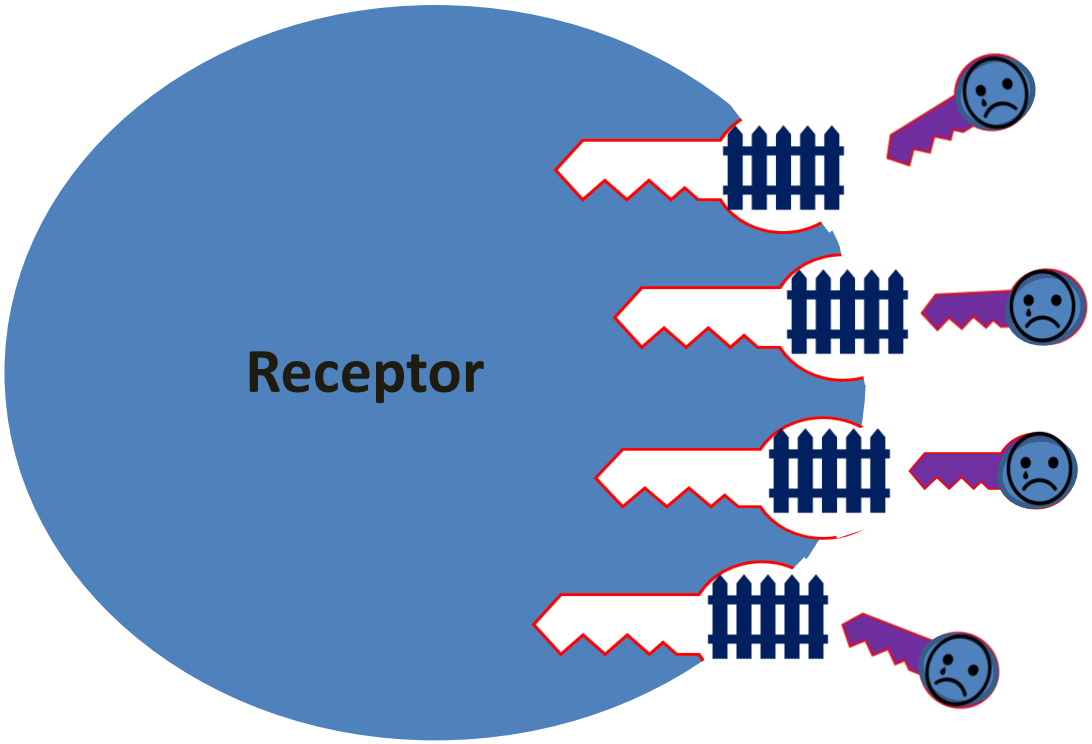
“Partial” Agonist



- **Partial** agonists are designed to stimulate only a portion of the receptor producing a weaker potential
- **Partial** agonists act like a light **“dimmer”** rather than an **“On-Off”** switch
- **Partial** agonists produce a **“ceiling”** limit and won’t permit single drug overdosing
- **Buprenorphine** is a **Partial** agonist


PARTIAL AGONIST
NEUROTRANSMITTERS
ACTION POTENTIAL
RECEPTORS

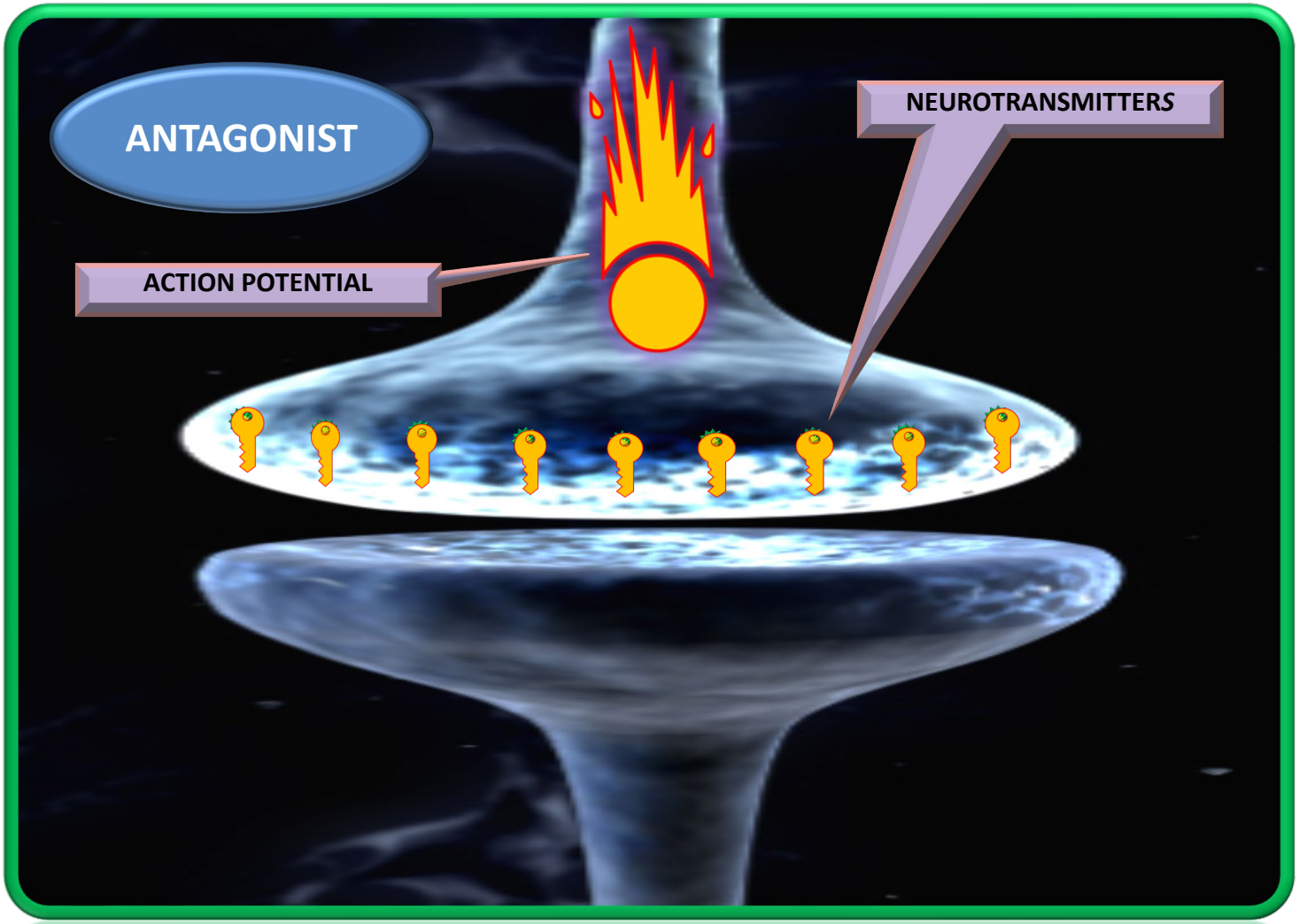
Antagonists



Antagonists are designed to block all chemicals **(full-partial agonist)** from entering the receptor

When an antagonist enters the NS, they will dislodge and remove any chemical on a receptor site resulting in spontaneous **(immediate)** drug withdrawal

Naltrexone-Naloxone are **Antagonist**


ANTAGONIST
NEUROTRANSMITTERS
ACTION POTENTIAL

THREE (3) THERAPEUTIC OBJECTIVES TO REMEMBER WHEN USING MEDICATION FOR AN OPIOID USE DISORDER

THE THERAPEUTIC DILEMMA

SINCE OPIOID DEPENDENCE IS
A MEDICAL / PHYSICAL DISORDER
IMPACTING THE BRAIN . . .

YOUR THERAPEUTIC DILEMMA IS…

. . . WILL THE INDIVIDUAL'S N.S. REBOUND ONCE THE OPIOID DRUGS ARE DISCONTINUED

-

OR HAS THE **LONG-TERM** USE OF OPIOID DRUGS CREATED A PERMINATE CHANGE IN THE BRAIN,

-

THUS, REQUIRING THE USE OF OPIOID REPLACEMENT MEDICATIONS IN ORDER TO FUNCTION WITHOUT PAIN OR PHYSICAL CRAVINGS?

YOU MUST CONSIDER THE FOLLOWING:

. . . ASSUMING THE BODY WILL
REBOUND NATURALLY AND RETURN TO NORMAL

-

THE USE OF OPIOID REPLACEMENT MEDICATIONS
MAY BE **COMPLETELY UNNECESSARY**

-

HOWEVER, IN LIMITED CIRCUMSTANCES OPIOID REPLACEMENT MEDICATIONS MAYBE AN IMPORTANT SHORT-TERM THERAPERUTIC OPTION, IN CONJUNCTION WITH CONVENTIONAL TREATMENT

-

IN THIS EVENT, OUR THERAPEUTIC MOTIVE WILL BE TO EVENTUALLY TAPER-DOWN AND DISCONTINUE THE USE OF OPIOID REPLACEMENT MEDICATIONS SAFELY OVER TIME

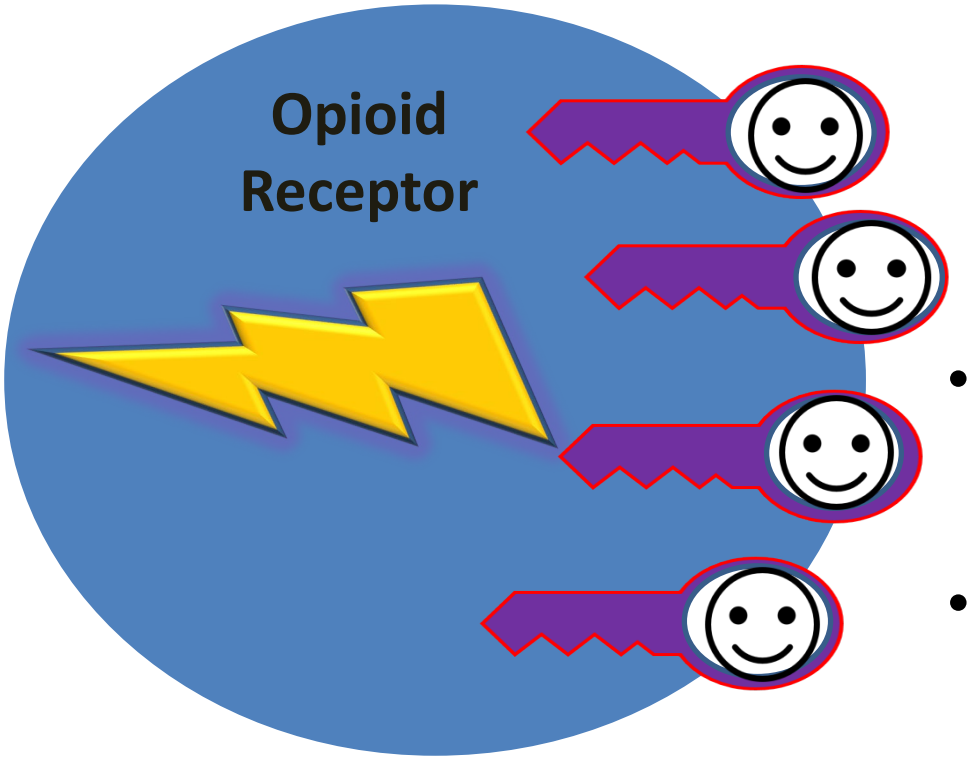
- **HOWEVER, . . .** IF AN INDIVIDUAL'S CHRONIC USE OF OPIOID SUBSTANCES HAS PRODUCED NEUROLOGICAL CHANGES THAT HAS RESULTED IN OPIOID RECEPTOR "DOWN REGULATION", THEN THE USE OF OPIOID REPLACEMENT "MAINTANENCE" MEDICATIONS MAY NEEDED IN ORDER TO AVOID RELAPSE AND THE EVENTUAL RETURN TO ILLICIT DRUG USING BEHAVIORS

- THE FOLLOWING MEDICATIONS ARE CURRENTLY BEING USED IN LONG-TERM OPIOID DEPENDENCE TREATMENT:

1. **METHADONE** (FULL AGONIST)
2. **NALTREXON:** (FULL ANTAGIONIST)
2. **BUPRENORPHINE** (PARTIAL AGONIST)
    a . **SUBUTEX** (STRAIGHT BUPRENORPHINE)
    b . **SUBOXONE** (BUPRENORPHINE WITH NALOXONE)

Methadone, Naltrexone, Buprenorphine

Methadone “Full-Opioid Agonists



- Methadone, like all full agonists will occupy the entire opioid receptor, producing a greater action potential and death from a single drug overdose
- Duration of action is **24 to 72** hours
- Dosages between **30 to 40 mg** will block withdrawal, not cravings
- Dosages of **80 to 100 mg** are more effective at reducing opioid use

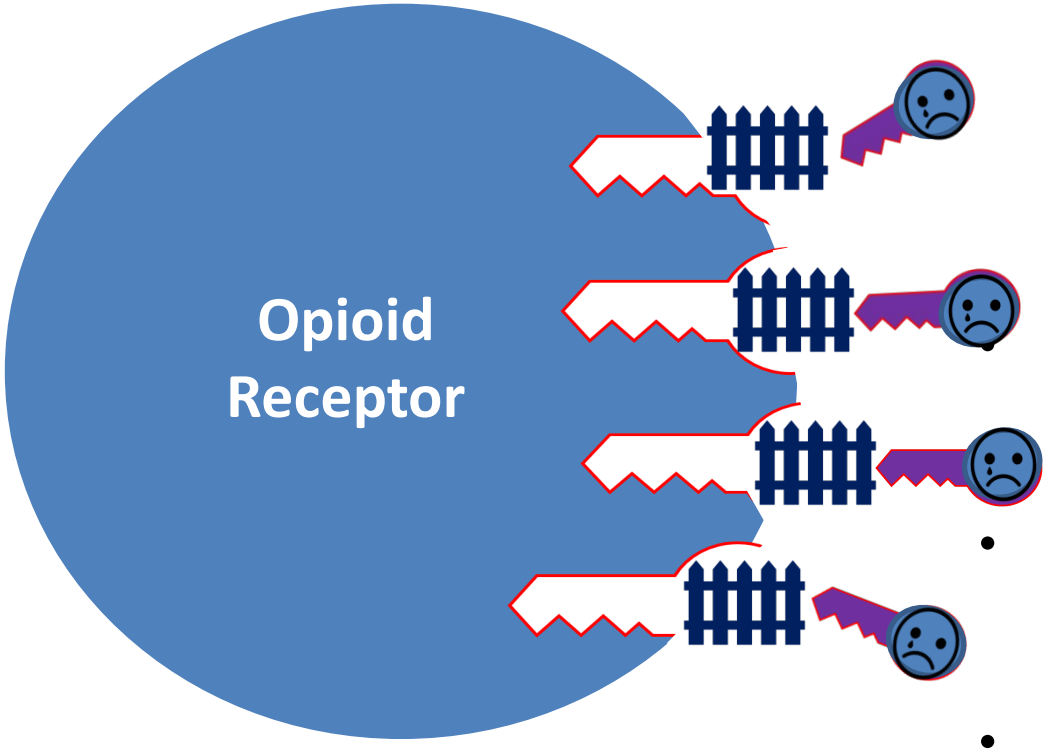
CAUTION:

THE MEDICATIONS **SUBUTEX** AND **SUBOXONE** ARE OPIOIDS

. . . AND. . .

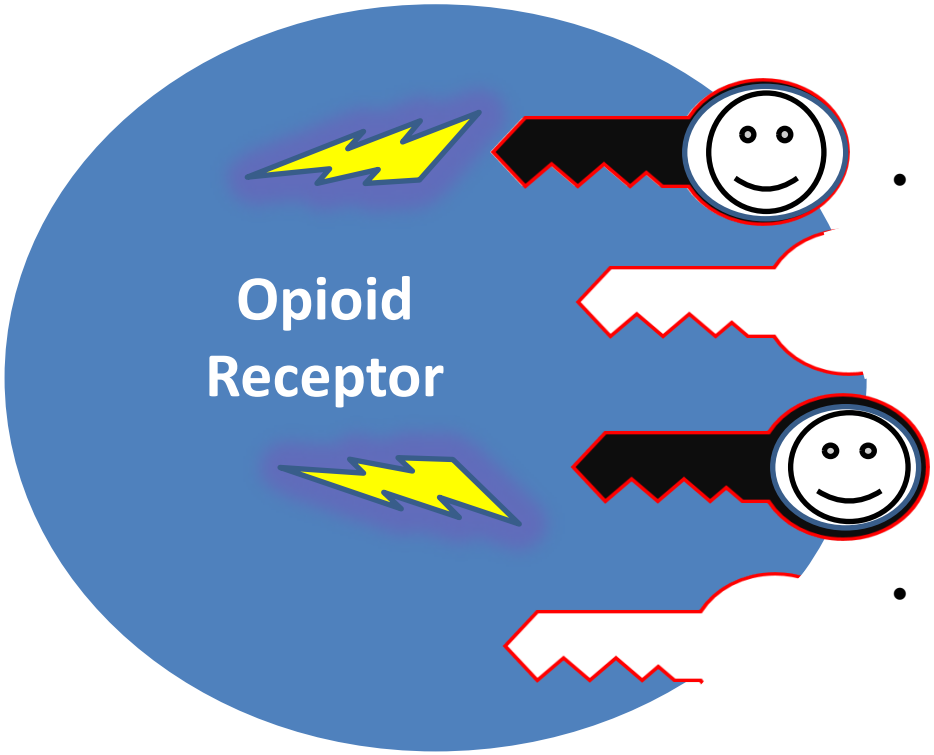
CAN BE ABUSED BY SIMPLY TAKING MORE THAN RECOMMENDED OR BY COMBINING THEM WITH OTHER CENTRALLY ACTIVATING DEPRESSING DRUGS (ALCOHOL OR BENZODIAZEPINES)

Naltrexone (Vivitrol) - Naloxone (Narcan),



- **Naloxone-Naltrexon**e are considered opioid and alcohol antagonists
- They are designed to block all opioid and ethanol chemicals **(full-partial agonist)** and removed any opioid or ethanol chemicals currently occupying an opioid receptor
- Naloxone lasts for only **30 to 90** minutes
- Naltrexone lasts for **1 day by tablet** or **30 days via injection**
- **Oral Naltrexone** 50 mg daily, poor adherence according to research
- **Injectable Naltrexone** resulted in 36% abstinence vs. 23% for placebo

Buprenorphine (Subutex)



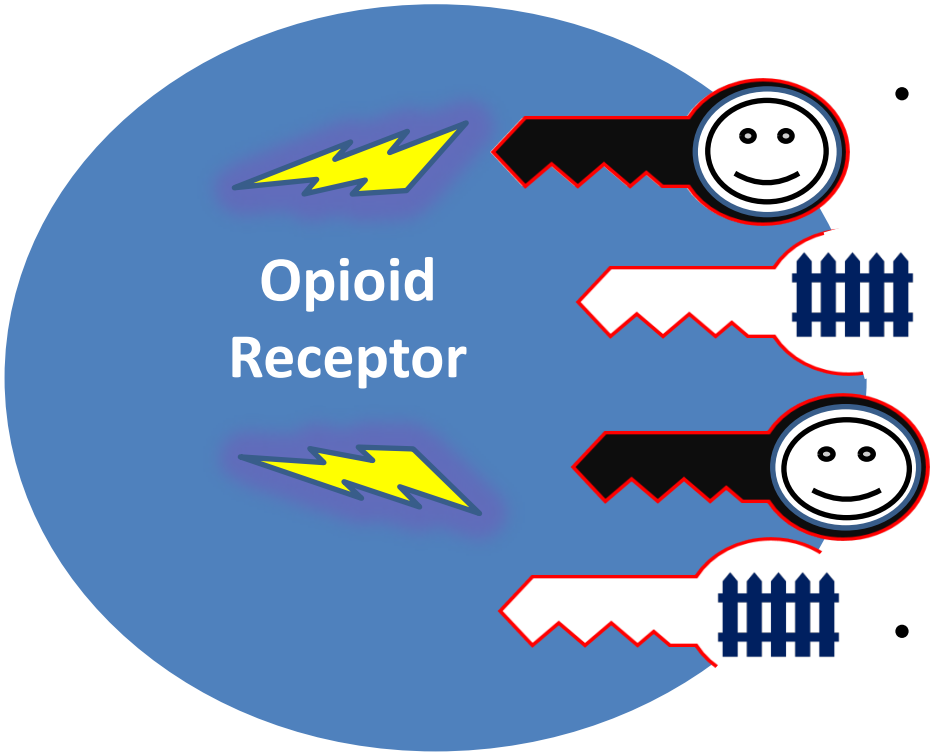
- Partial agonists are designed to only stimulate a portion of the opioid receptor, producing a lesser action potential and less opportunity for a single drug overdose
- Subutex is straight **buprenorphine**

  Subutex is less lipophilic than morphine

  Dosages range from **8-32 mg**
- Duration of action is **24 to 36** hours

  Buprenorphine has a **"Ceiling"** limit at **35 mg**
- **Sustained-Release Injectable Buprenorphine** effectiveness 28% to 29% in comparison to 2% placebo

Buprenorphine (Suboxone)



- Suboxone is **buprenorphine** and **naloxone** combined
- The addition of naloxone to the buprenorphine will ensure that any additional use of an opioid drug or any attempt to **divert** the drug will result in an immediate removal of all opioid substances, including buprenorphine, causing spontaneous drug withdrawal
- Dosages from **2mg-0.5mg, 8mg-2mg**
- **Injectable Extended-Release Buprenorphine-Naloxone** effectiveness 65%
- **Sublingual Buprenorphine-Naloxone** effectiveness 57%

HOW LONG SHOULD A PERSON REMAIN ON OPIOID-ASSISTED TREATMENT

Length of time on opioid-assisted treatment

- Currently there is limited research documenting a specific length of time someone should remain on MAT
- Most reports are expert consensus and not empirically run trials
- Research that does exist has conducted comparisons of six (6) months, nine (9) months, fifteen (15) months and eighteen (18) month intervals
- Their research indicated MAT should be customized tailored to the needs of the individual
- Their research also indicated those individuals who participated in their study showed better results at the fifteen (15) and eighteen (18) month interval then those who discontinued MAT sooner

Presentation Review

- REVIEWED MEDICATION-ASSISTED TREATMENT (MAT)
- REVIEWED THE THERAPEUTIC TRIAD
- REVIEWED HOW ANTI-DEPRESSANT MEDICATIONS ASSIST IN SUBSTANCE USE TREATMENT
- REVIEWED THE THREE (3) TYPES OF MAT TREATMENT OBJECTIVES
- EXPLORED ANTI-ALCOHOL MEDICATIONS
- REVIEWED WHAT MAKES OPIOID DRUGS DESIRABLE
- EXPLORED THE THERAPEUTIC DELEMIA ASSOCIATED WITH MAT
- EXAMINED THREE (3) MAT MEDICATIONS ASSOCIATED WITH OPIOID ABUSE AND THEIR CURRENT EFFECTIVENESS
- DISCUSSED THE EFFECTIVE LENGH OF TIME NECESSARY FOR MAT

CONTACT INFORMATION:

CARL M. DAWSON, M.S., MAC, LPC

1320 E. KINGSLEY SUITE “A“
SPRINGFIELD, MO. 65804

e-mail: (Cdawson1028@yahoo.com)