DEFINITIONS

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OPIATES

THE GOOD (Use)

THE BAD (Misuse)

THE UGLY (Abuse)

OPIATES

Opium Poppies in Northern Thailand
Photo By: John W. Allen

PAPAVER SOMNIFERUM
Photo By: Eric Clausen

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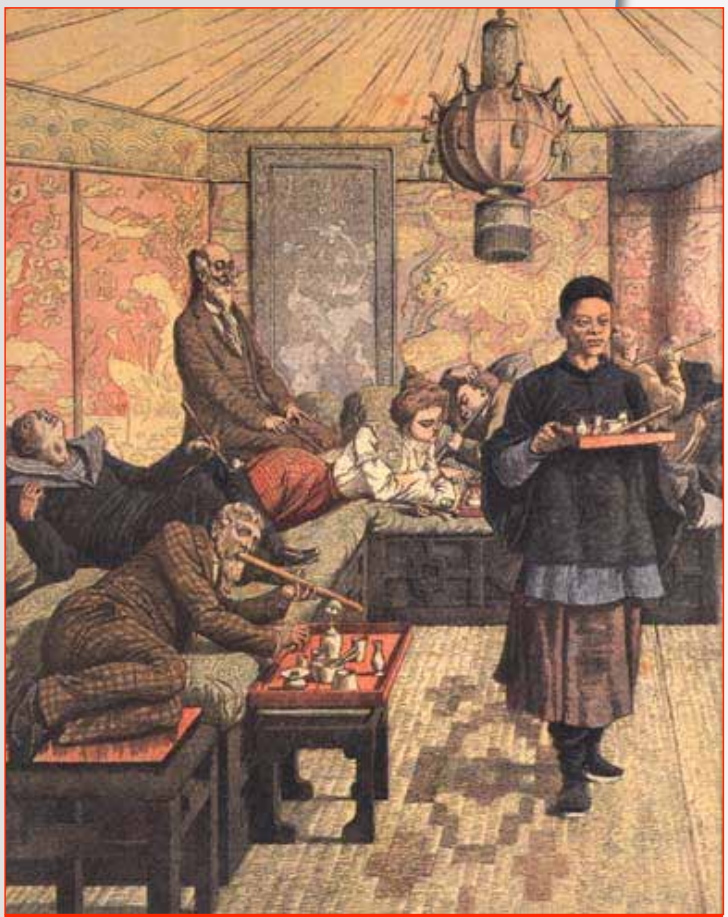
THE HISTORY OF OPIUM



1855 Opium Den

- 1850 – 1865 thousands of Chinese laborers immigrated to the US and brought the habit of opium smoking with them *(Opium Den shown on left)*
- Civil war soldiers became opioid dependent through medical treatment – referred to as “army disease” or “soldier’s disease”
- It was estimated that the total number of opium users in the U.S. in 1868 was 100,000
- Heroin was first synthesized in 1874 by the chemist, C.R. Alder Wright
  - First commercial production in 1898 by the Bayer Pharmaceutical Company
  - 1898: Heinrich Dreser announced that tests confirmed heroin was ideal for treating bronchitis, emphysema, asthma, tuberculosis, and was a cure for opium and morphine dependence

DEFINITIONS

PAPAVER SOMNIFERM

OPIUM

- Fluid obtained from the poppy plant

OPIATE

- A substance derived from opium

OPIOID

- Substance with morphine-like actions, but not derived directly from the poppy plant

HISTORIC OPIATE PRODUCTS

Stickney & Poor's Pure Paregoric

This bottle of Stickney and Poor's "Pure Paregoric" was distributed much like the spices for which the company is better known. McCormick also manufactured and sold paregoric, which is a mixture of opium and alcohol. Doses for infants, children, and adults are given on the bottle. At 46% alcohol, this product is 92 proof which is pretty potent in itself.

HISTORIC OPIATE PRODUCTS

Bayer Aspirin Ad

Bayer Aspirin Ad

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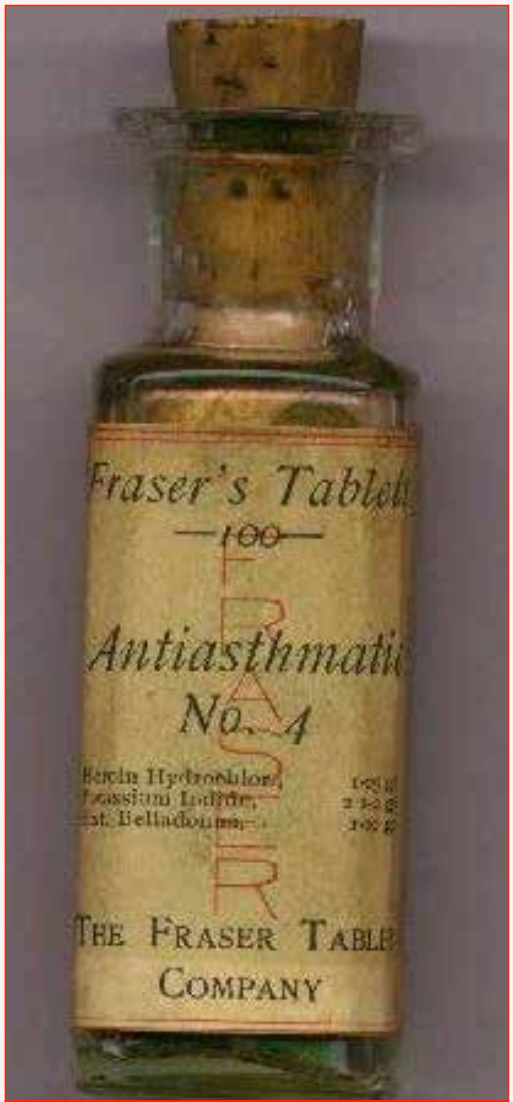

Fraser's Tablets for Asthma

HISTORIC OPIATE PRODUCTS

These heroin tablets, manufactured by the Fraser Tablet Company, were marketed for the relief of asthma.

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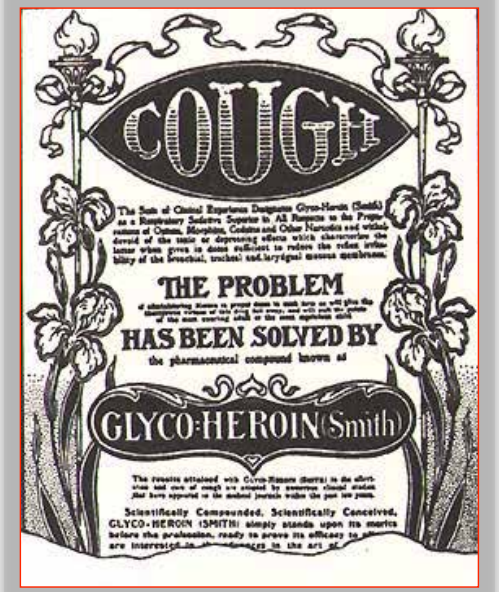
1903 ADS



- These magazine advertisements are for **Glyco-Heroin manufactured by Martin H. Smith Company** (New York).
- Heroin was widely used not only as an analgesic, but also as a remedy for asthma, coughs, and pneumonia.
- Mixing heroin with glycerin (and often adding sugar or spices) made the bitter-tasting opiate more palatable for oral consumption.

(From International Medical Magazine, January, 1902.)

THE PAST

1974 – NARCOTIC ADDICT TREATMENT ACT

- Required separate DEA registrations for physicians who want to use approved narcotics

1986 – EXECUTIVE ORDER 12564

- Mandated a drug-free workplace program

1988 – ANTI DRUG ABUSE ACT

- Established the OFFICE OF NATIONAL DRUG CONTROL POLICY (ONDCP) in the executive office of the President of the United States to oversee all federal policies regarding research about control of drug abuse

2000 – CHILDREN'S HEALTH ACT

- A section of this act dealt with drug addiction treatment (DATA)
  - Allowed qualified physicians to prescribe medications classified as schedule III, IV and V narcotics for treatment of addiction. This is the law that allows and regulates buprenorphine use in addiction treatment.

THE PROCESS

GROWERS

- Southeast Asia
- Middle East
- Latin and South America

SHIPPERS

MANUFACTURERS

LARGE WHOLESALE BUYERS

- Prices per kilo depend on the purchase amount

MID-RANGE BUYERS

- Dilute or “step on” the heroin using white substances that are not easily detected
  - These substances or “Cut” can be – lactose, mannitol and/or talc

CONTINUOUS DILUTION CAN OCCUR ALL THE WAY DOWN TO THE POINT OF SALE

- Bag = 1/10 to 1/15 gram
- 10 bags = bundle

THE PRESENT

- 1,000,000 -3,000,000* heroin addicted individuals in the United States (2012)
- 25% are involved in some type of treatment
- Opiate-dependent patients are not just using heroin, but other narcotic drugs as well!
- Heroin use in Chicagoland still high because the purity of heroin is extremely high (65%) and can be snorted instead of used intravenously.

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ASAM (American Society of Addiction Medicine) DEFINED **ADDICTION** IN 2001

- ADDICTION is a primary, chronic, neurobiologic disease, with genetic, psychosocial, and environmental factors influencing its development and manifestations. It is characterized by behaviors that include one or more of the following: impaired control over drug use, compulsive use, continued use despite harm, and craving

Savage et al., 2001

DSM 4 CRITERIA FOR DRUG USE

- Significant impairment or distress resulting from use
- Failure to fulfill roles at work, home, or school
- Persistent use in physically hazardous situations
- Recurrent legal problems related to use
- Continued use despite interpersonal problems

DSM 4 CRITERIA FOR DRUG DEPENDENCE

1. Desire or unsuccessful efforts to cut down on use
2. Large amount of time spent obtaining drugs, using drugs, or recovering from drug effects
3. Social, occupational, or recreational activities reduced because of drug use
4. Drug use continued despite knowledge that a physical or psychological problem is being caused or exacerbated by use
5. Use of drug in larger amounts or for longer periods of time than originally anticipated
6. Tolerance: Need for increased amounts of drugs to achieve desired effect; or diminished effect with continued use of the same amount of drug

TOLERANCE DEVELOPS NORMALLY WITH REPEATED USE OF SOME DRUGS

DSM 5 CRITERIA FOR OPIATE USE DISORDER

≥ 3 of the following occurring in the same 12- month period

1. Opioids are taken in large amounts or over a longer periods than was intended.
2. Persistent desire or unsuccessful efforts to cut down or control opioid use.
3. Great deal of time spent in activities necessary to obtaining opioid, using opioid, or recover from its effects.
4. Cravings or strong desire or urge to use opioids.
5. Recurrent opioid use resulting in failure to fulfill major role obligations at work, school or home.
6. Continued opioid use despite having persistent or recurrent social or interpersonal problems caused or exacerbated by the effects of opioids.
7. Important social, occupational, or recreational activities are given up or reduced because of opiate use.
8. Recurrent opioid use in situations in which it is physically hazardous.
9. Continued opioid use 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 the following:
    A need for markedly increased amounts of opioids to achieve intoxication or desired effect.
    A markedly diminished effect with continued use of the same amount of opioid.
11. Withdrawal: As manifested by either of the following
    The characteristics: opioid withdrawal syndrome; or opioids (or closely related substances) are taken to relieve or avoid withdrawal SX.

ADDICTION IS NOT:

- PHYSICAL DEPENDENCE – characteristic withdrawal syndrome emerges upon decreased blood level of substance or antagonist administration
- TOLERANCE - increasing amount of drug needed over time to induce the same effect

BOTH ARE *NEUROADAPTIVE STATES* RESULTING FROM CHRONIC DRUG ADMINISTRATION

PSEUDOADDICTION IS:

- Operationally defined as aberrant drug-related behaviors that make patients with chronic pain look like addicts.
- These behaviors stop if opioid doses are increased and pain improves *(Weissman and Haddox, 1989)*.
- This indicates that the aberrant drug-related behaviors were actually a search for relief
- LITTLE DATA ON THE SUBJECT, BUT EVIDENCE HAS BEEN SEEN IN RATS

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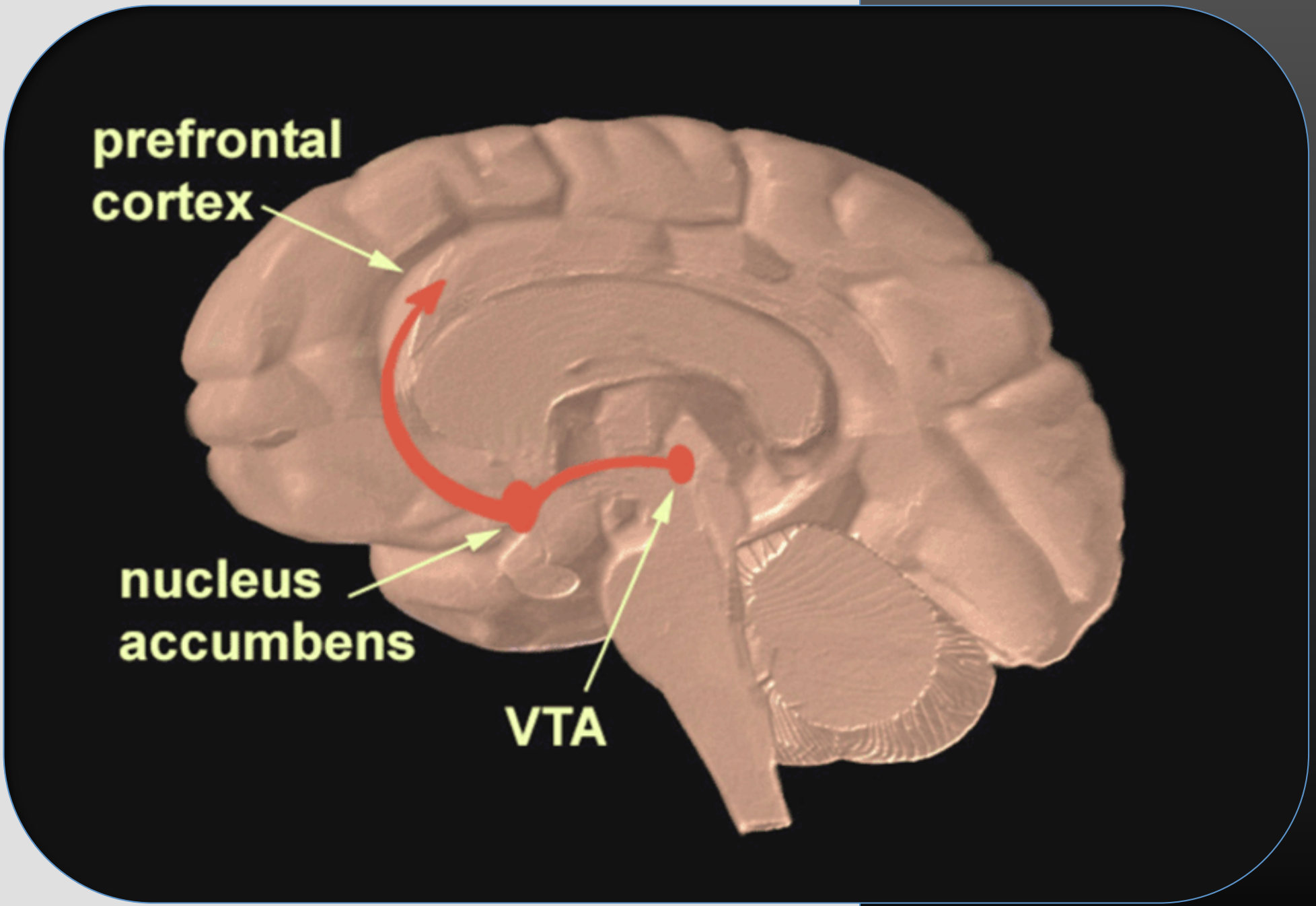

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WHAT IS ADDICTION

Chronically relapsing disorder that is characterized by 3 major elements:

I. Compulsion to seek and take the drug

II. Loss of control in limiting intake

III. Emergence of a negative emotional state when access to drug is prevented

FACTORS CONTRIBUTING TO ADDICTION

Chronically relapsing disorder that is characterized by 3 major elements:

- REINFORCEMENT Stimulus ↑ probability of response
- NEUROADAPTION Processes by which initial drug effects are either enhanced or attenuated

Together these factors motivate the acute response to a drug and establishment of a chronic craving

FACTORS CONTRIBUTING TO ADDICTION

REINFORCEMENT

POSITIVE REINFORCEMENT

Rewarding stimulus (euphoria) ↑ probability of response (drug use)

NEGATIVE REINFORCEMENT

Incentive-relief of pain or unpleasant state (withdrawal symptoms)

CONDITIONED REINFORCEMENT

Environmental conditions of administration elicit euphoria without a drug and places of abstinence produce symptoms of withdrawal

NEURAL CIRCUITS

CHEMICAL TRANSMITTERS

Pass information between neurons

NEURAL CIRCUIT

Group of connected neurons that pass info. Related to a specific function

AOD (alcohol & other drugs

ADO possess positive reinforcing effects because of their NT interactions within reward pathway

FACTORS CONTRIBUTING TO ADDICTION

NEUROADAPTATION

Modulatory process leading to ↑ reinforcement with repeated drug exposure.

1. **SENSITIZATION**
   - increased response to a drug effect after repeated drug administration
   - motivational states (cravings) ↑ after repeated exposure → relapse, compulsive drug use

2. **COUNTERADAPTION**

   Processes to counter the acute drug effects
   - TOLERANCE reduction in a drug's effect after repeated use
   - WITHDRAWAL processes to counter the initial drug effects when drug is removed-symptoms are opposite of drug effect

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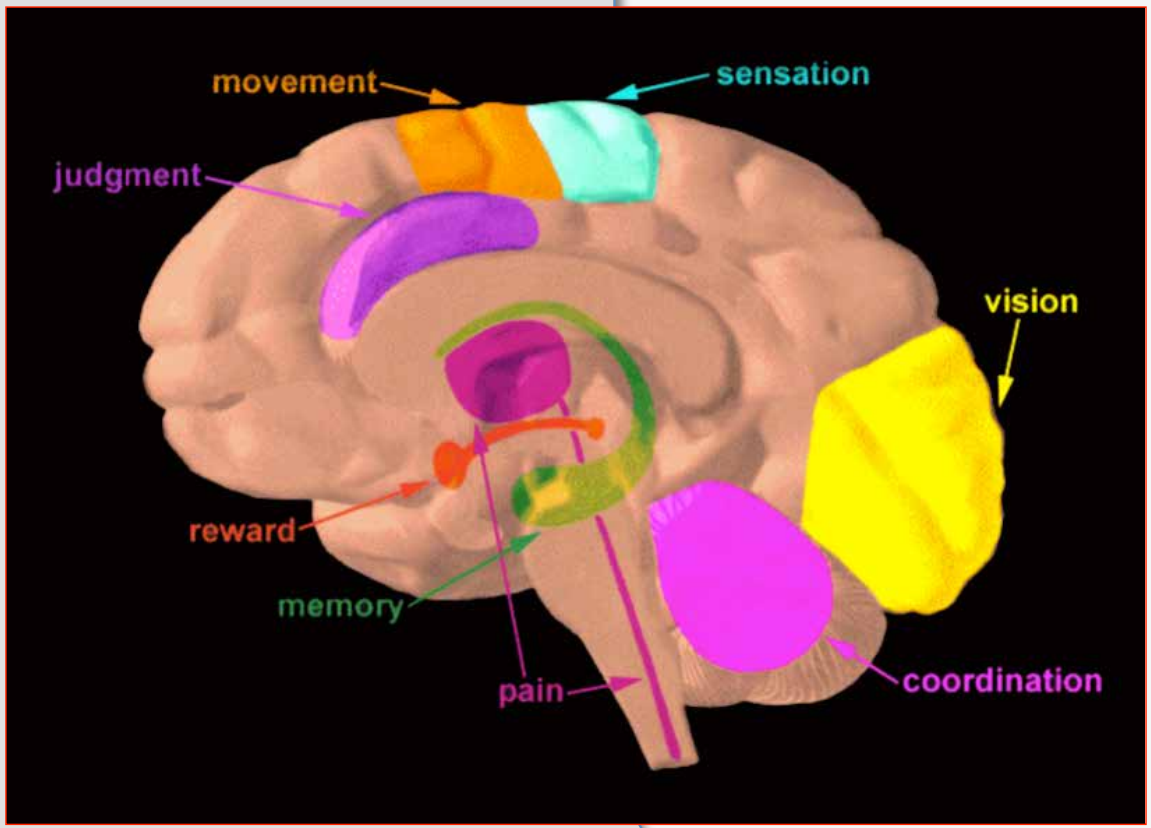
DIFFERENT REGIONS OF THE BRAIN ARE RESPONSIBLE FOR DIFFERENT FUNCTIONS



- LIGHT BLUE
  Primary Sensory Cortex
- ORANGE
  Primary Motor Cortex
- YELLOW
  Visual Cortex
- PINK
  Cerebellum: Coordination
- GREEN
  Hippocampus: Memory
- ORANGE
  Reward Pathway
- MAGENTA
  Thalamus & Pain Pathway

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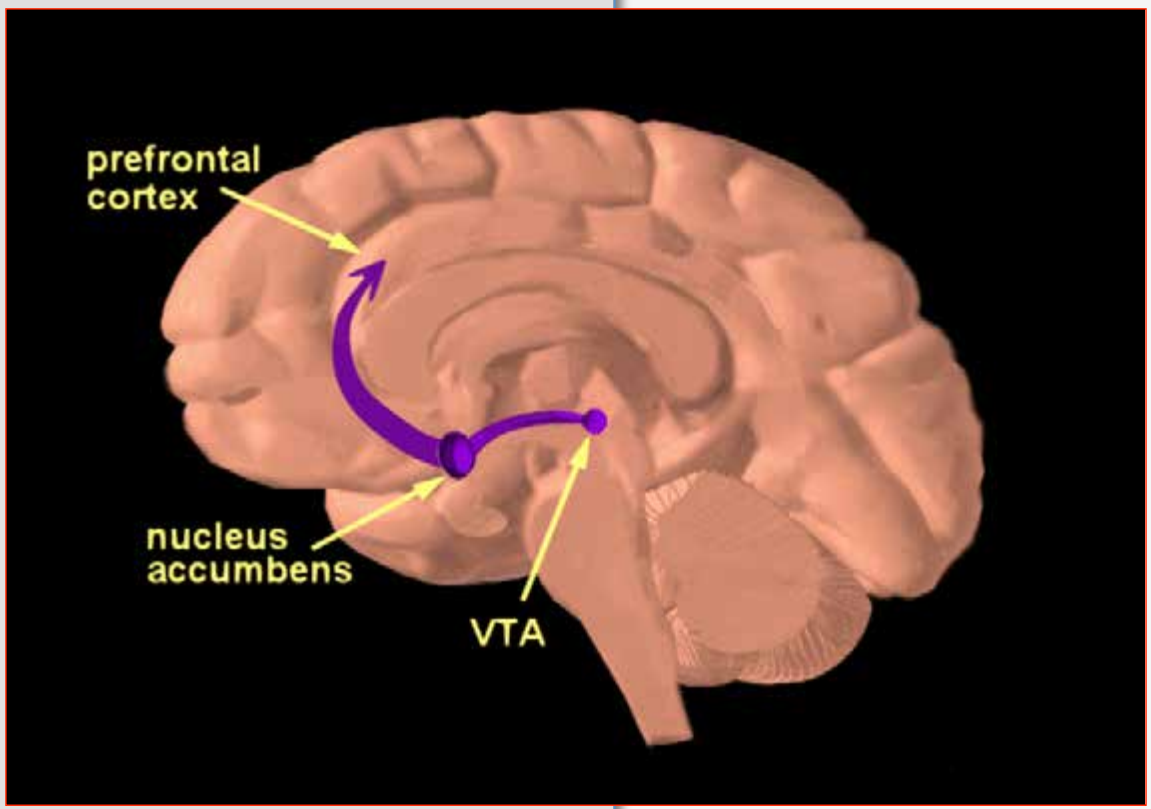
REWARD MEMORY



- Ventral tegmental area (VTA)
- Nucleus accumbens
- Prefrontal cortex
- VTA is connected to the nucleus accumbens and the prefrontal cortex via this pathway
- Sends information to these structures via its neurons

REWARD PATHWAY STRUCTURES

- Neurons of the VTA contain the neurotransmitter **dopamine** which is released in the nucleus accumbens and in the prefrontal cortex
- The reward pathway is activated when a person receives positive reinforcements (rewards) for a certain behavior - addictive drug is used

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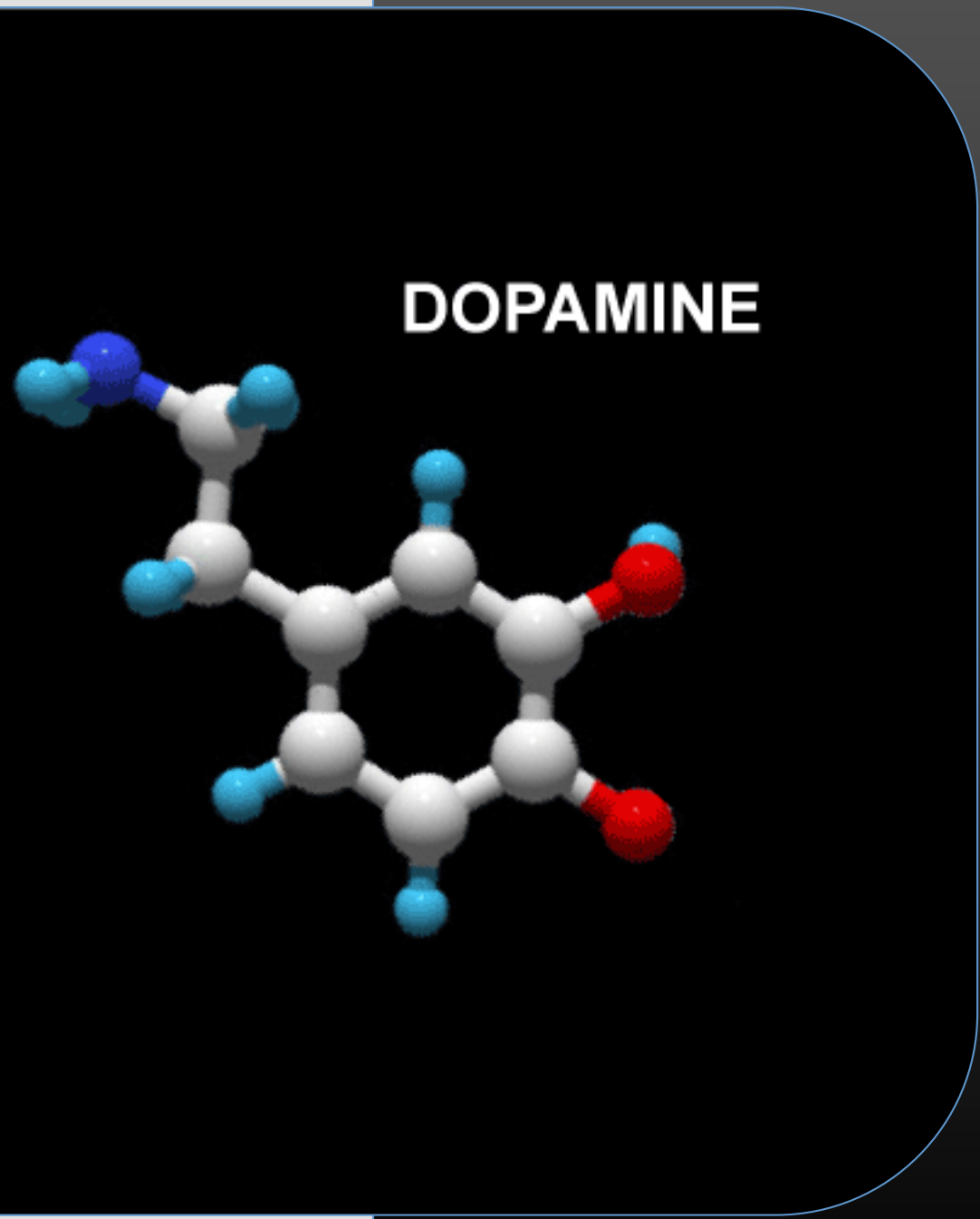
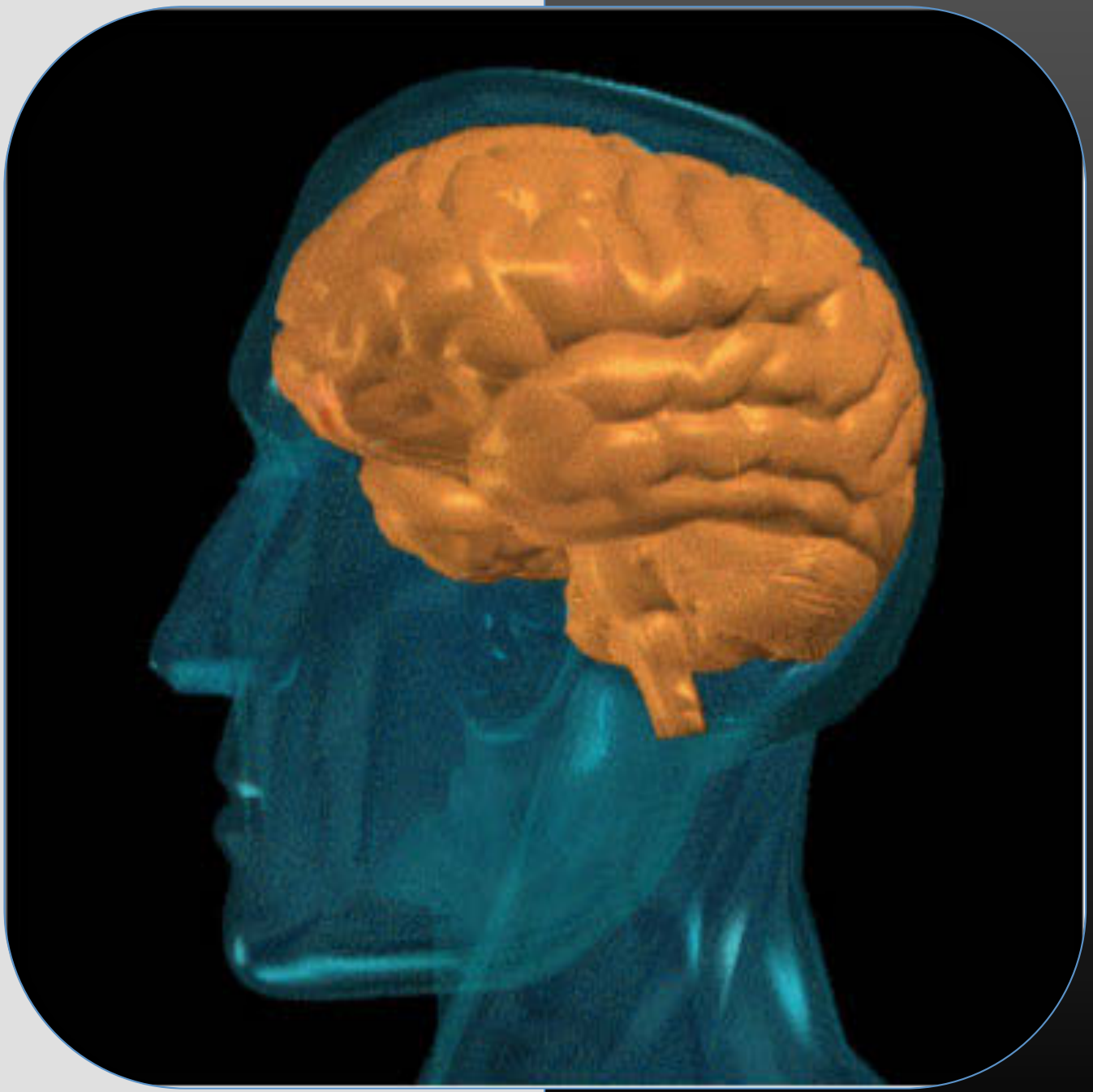
DOPAMINE (DA)

- One of the neurotransmitters playing a major role in addiction
- DA affects brain processes that control movement, emotional response, and ability to experience pleasure and pain



THE BRAIN AND THE ACTIONS OF COCAINE, OPIATES, AND MARIJUANA



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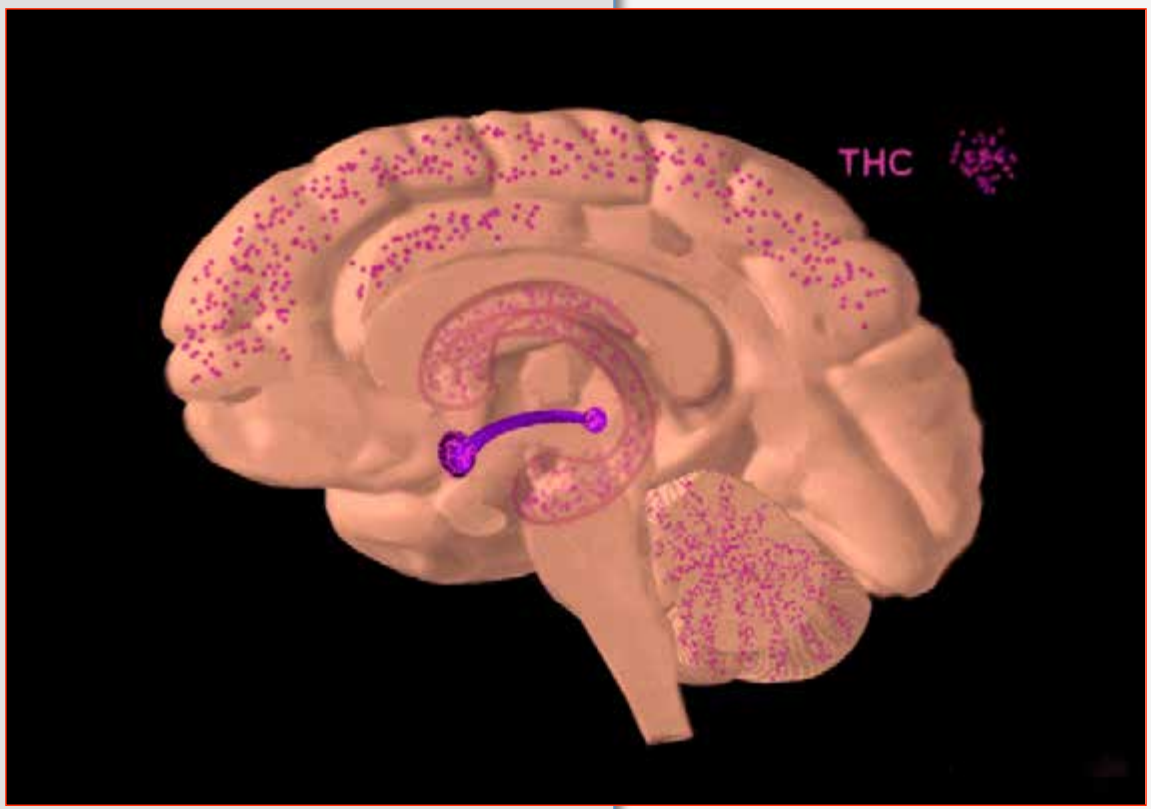
MARIJUANA



- VTA
- Nucleus accumbens
- Naudate nucleus
- Hippocampus - interference with memory
- Cerebellum - coordination and loss of balance

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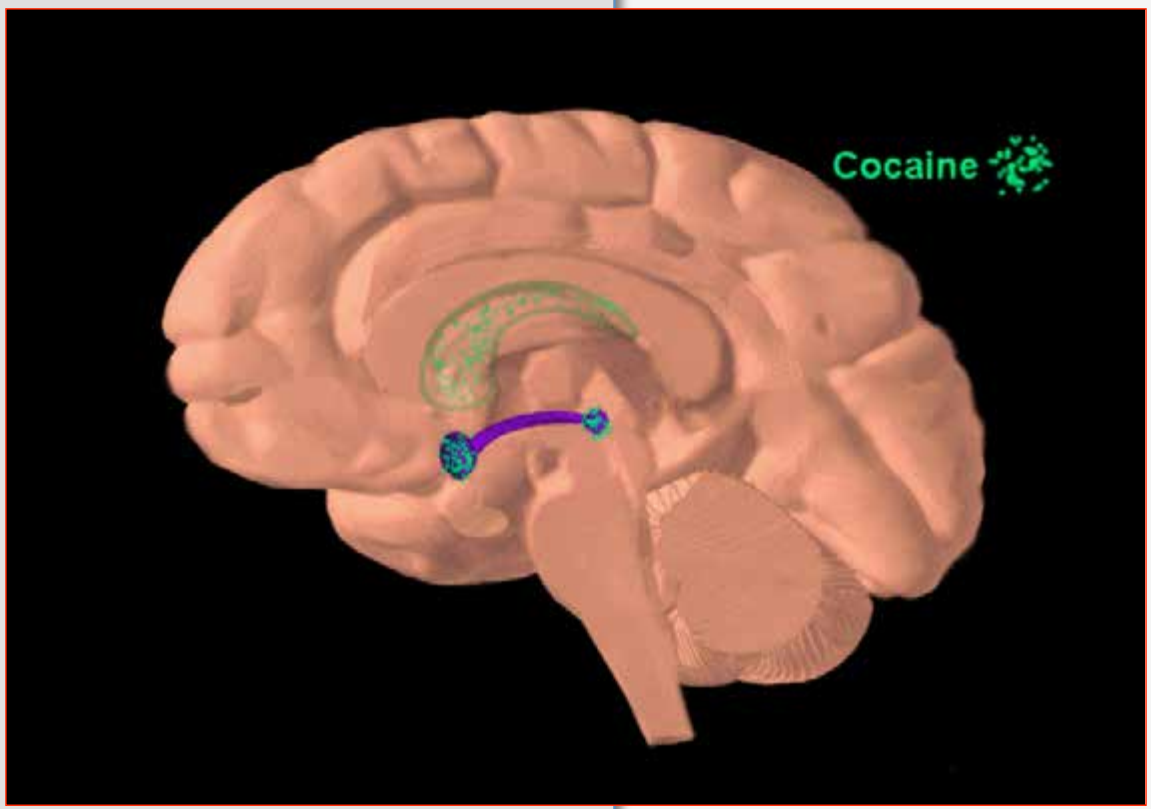
COCAINE



- VTA
- Nucleus accumbens
- The caudate nucleus
- Concentrates in areas rich in dopamine synapses

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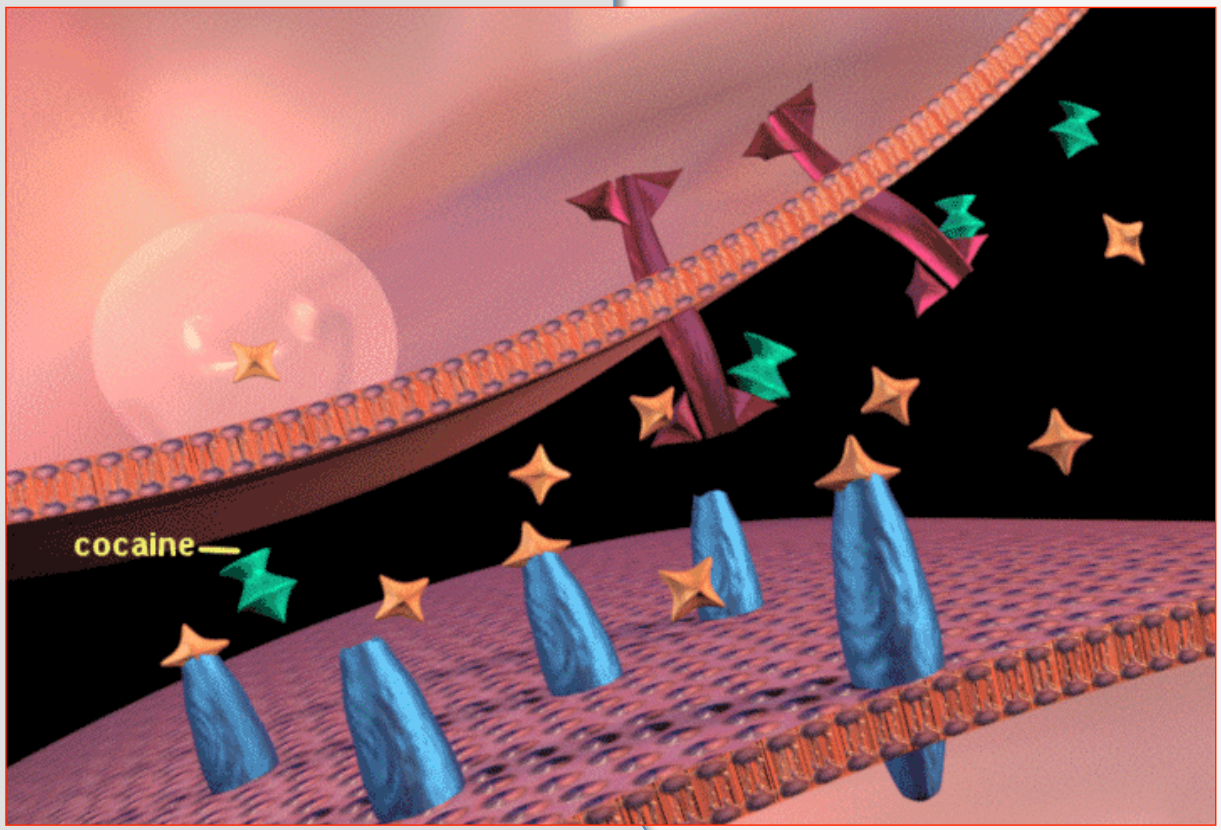
DOPAMINE TRANSMISSION IN A SYNAPSE IN THE NUCLEUS ACCUMBENS



- ORANGE
  Dopamine
- BLUE
  Dopamine receptors
- RED
  Reuptake pumps on the terminal
- Cocaine binds to the reuptake pumps
- Prevents removing dopamine from the synapse
- More dopamine in the synapse, more activated dopamine receptors

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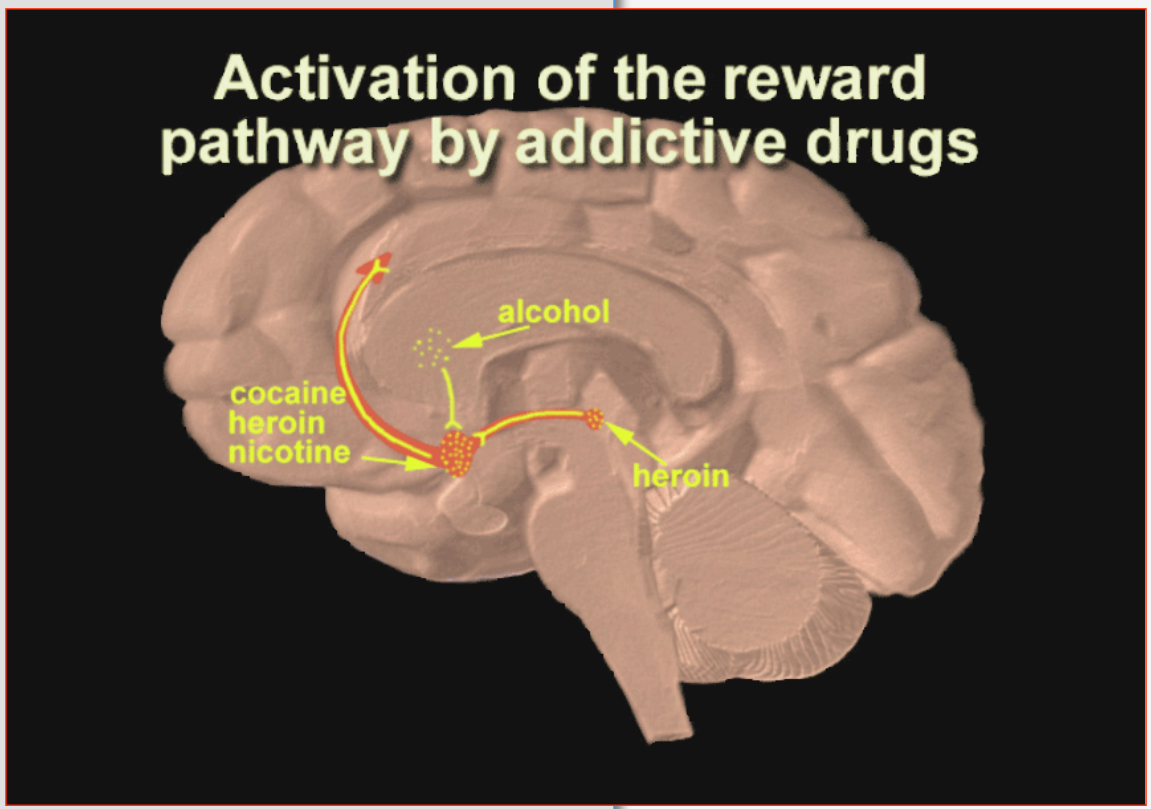
RESULTS OF COCAINE'S ACTIONS IN THE NUCLEUS ACCUMBENS

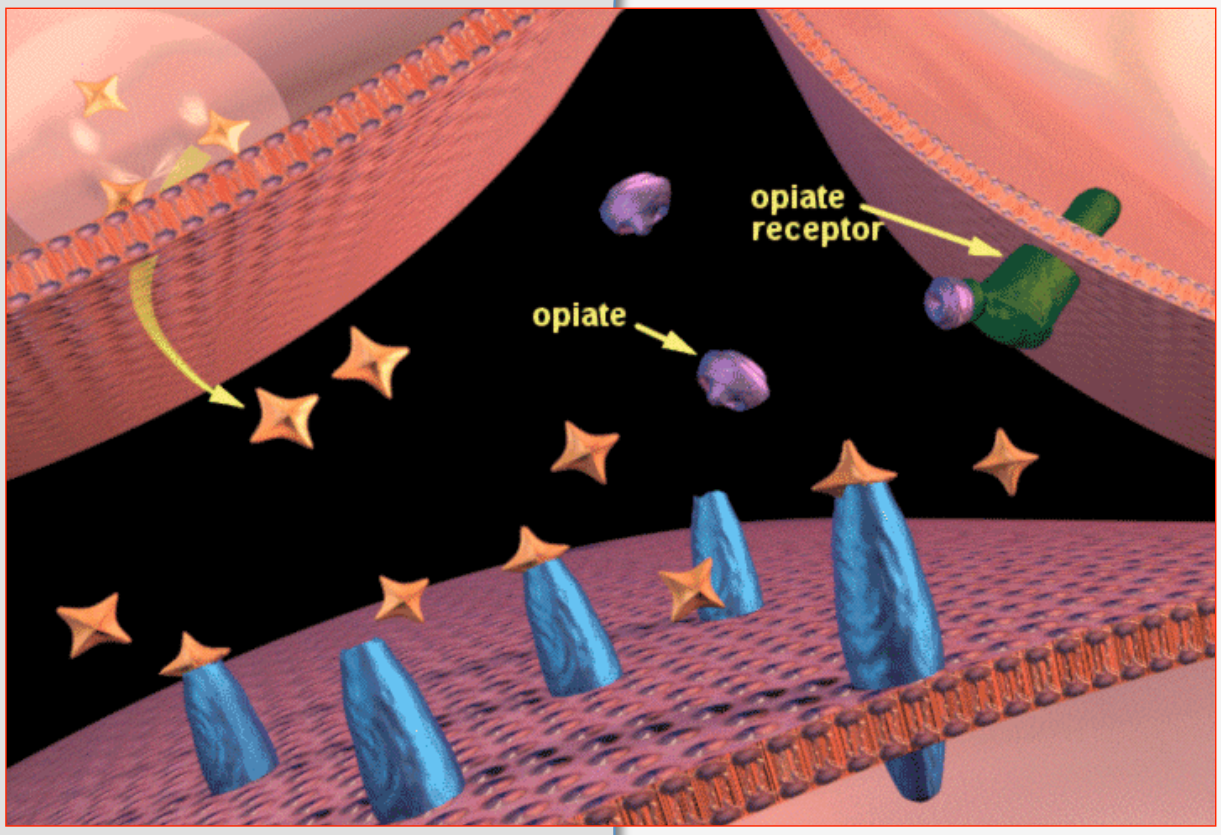
- ↑ impulses leaving the nucleus accumbens to activate the reward system
- with continued use of cocaine, the body relies on it to maintain rewarding feelings
- The person is no longer able to feel the positive reinforcement or pleasurable feelings of natural rewards (food, water, sex)

OPIATES



- VTA
- Nucleus accumbens
- The caudate nucleus
- Thalamus - contributes to their ability to produce analgesia

OPIATE



DA terminal

Another terminal (on the right)

- different neurotransmitter (GABA)

The post-synaptic cell - DA receptors

Opiates bind to opiate receptor:

- signal to the DA terminal for more DA
- ↓ GABA release which normally inhibits DA release - so DA release ↑

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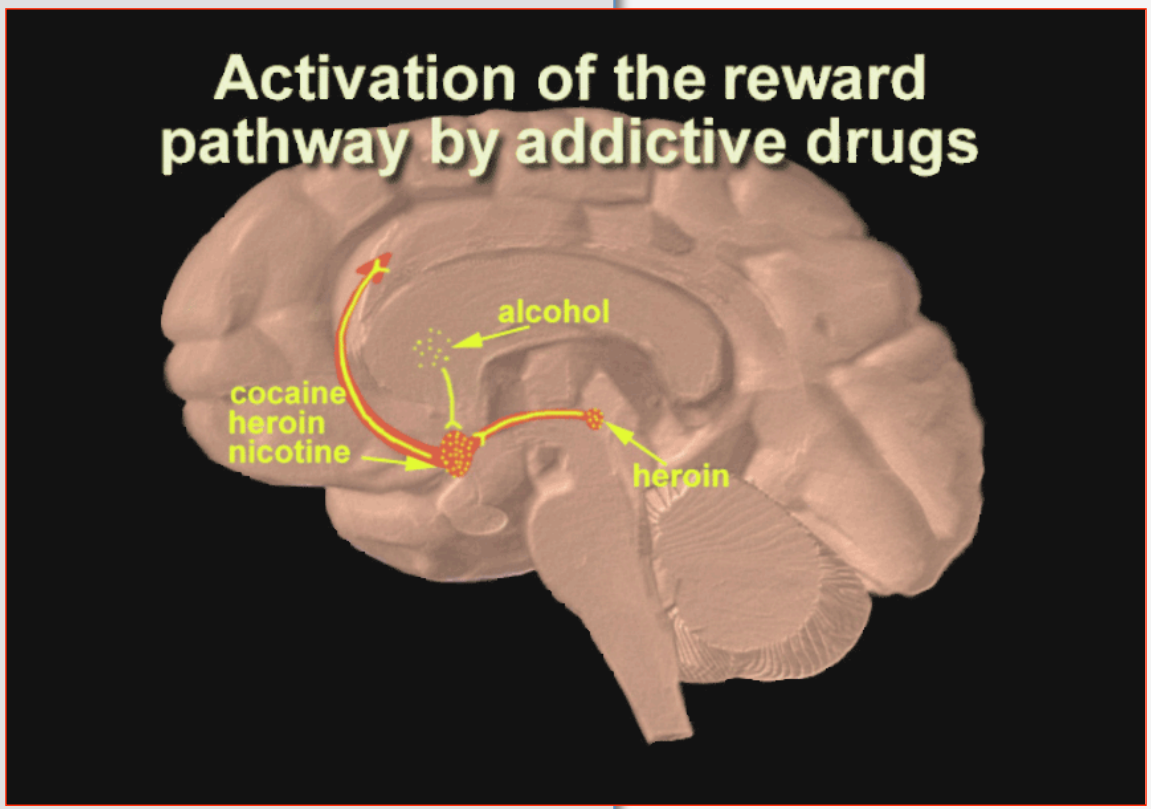
AS A RESULT OF OPIATE ACTIONS IN THE NUCLEUS ACCUMBENS

- ↑ impulses leaving the nucleus accumbens to activate the reward system
- Continued use of opiates makes the body rely on the presence of the drug to maintain rewarding feelings
- The person is no longer able to feel the benefits of natural rewards (food, water, sex) and can't function normally without the drug present



IN SUMMARY

- Binding of all three drugs is one of the reward areas, the nucleus accumbens
- Each drug ↑ the activity of the reward pathway by ↑ DA transmission
- Because of the way our brains are designed, and because these drugs activate a particular brain pathway for reward→ ability to be abused

MECHANISMS OF NEUROADAPTATION

WITHIN-SYSTEMS

- Mediated by reward pathway (mesolimbic DA system)

BETWEEN-SYSTEMS

- Corti-cotropin-releasing factor (CRF)

CRF SYSTEM

- Hormone released by hypothalamus and amygdala in response to stress
- CRF → stress hormones into blood stream from pituitary gland and adrenal cortex
- HPA axis-hypothalamic-pituitary adrenal axis

AMYGDALA → behavioral response to stress

VARIETY OF STRESSORS → sensitization

GLUTAMATE – major excitatory NT

GLU antagonist ≠ sensitization

COUNTERADAPTATION

- Both systems
- Within: ↓ DA in nucleus accumbens during withdrawal from cocaine, opiates, alcohol
- Between: CRF and HPA axis
  - rats are stressed after termination of drugs

CONCLUSIONS

- Further investigations in NT and reward pathway
- Genetic and environmental factors influence on reward pathway of individuals

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QUESTIONS FOR DISCUSSION

- Present treatment of addiction uses talk therapy (AA, group therapy, etc.), suggesting that addiction is a behavioral problem.
- What is then the evidence of the addiction being a medical disease?

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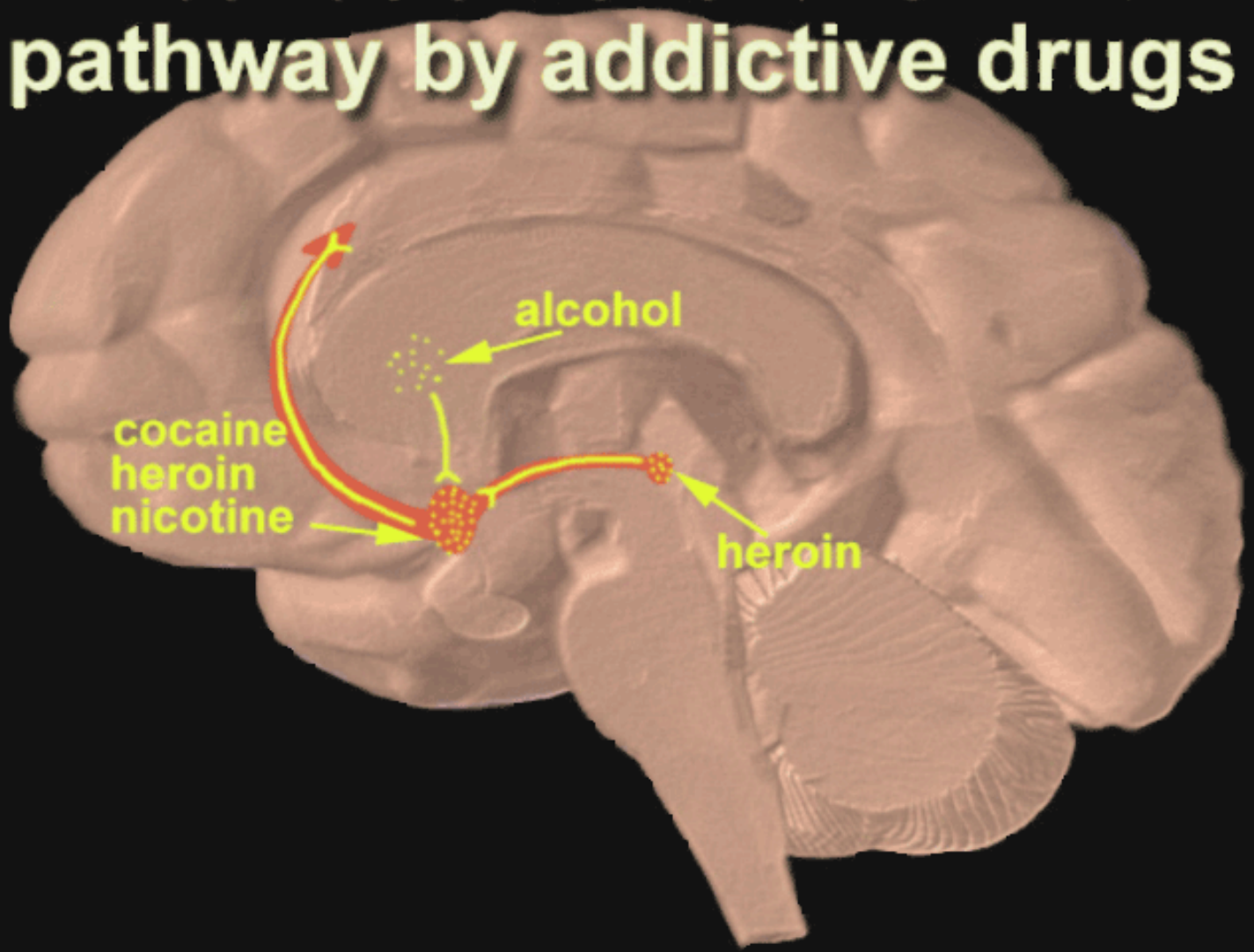
Activation of the reward pathway by addictive drugs



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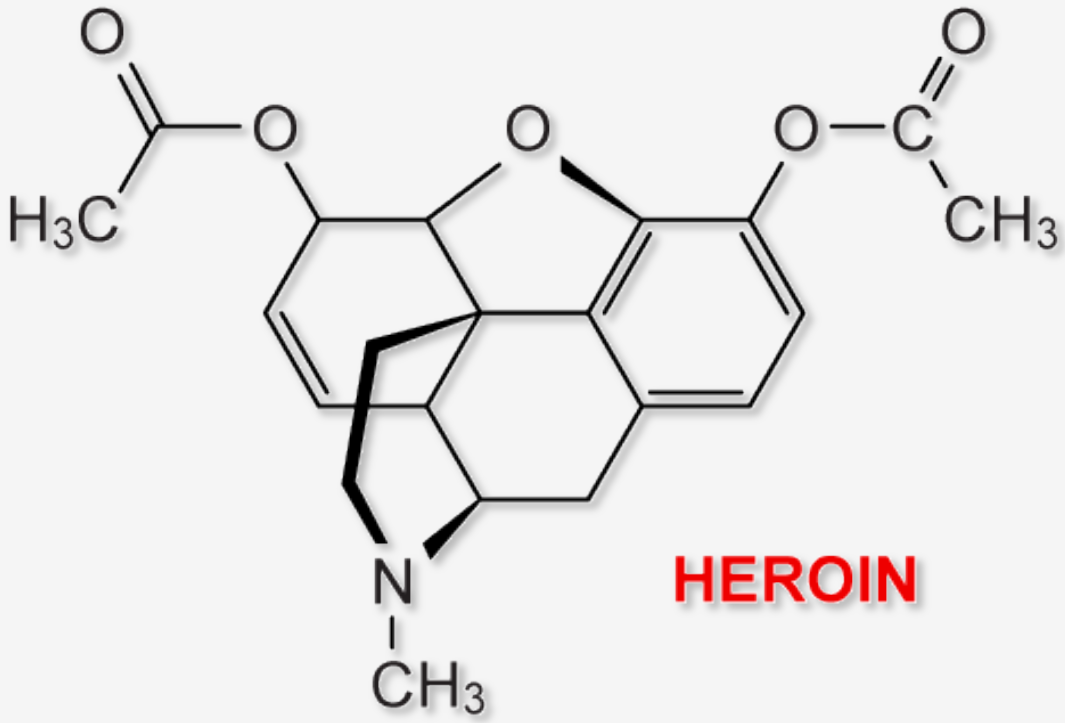
OPIATES

- OxyContin
  - Long acting oral
- Propoxyphene
  - (Darvon)
- Hydrocodone
  - (Vicodin)
- Hydromorphone
  - (Dilaudid)
- Meperidine
  - (Demerol),
- Diphenoxylate
  - (Lomotil)
- Codeine



HEROIN

HEROIN

- Heroin is processed from morphine (diacetylmorphine)
- Morphine is a naturally occurring *substance extracted from the seedpod of the Asian poppy plant.*
- *Heroin usually appears as a white or brown powder.*
- *Street names*
  - *“Smack," "H," “Horse,” “Skag," and ”Junk." "Mexican Black Tar,” and “China White.”*

Originally produced by Bayer as a “non addictive” analgesic

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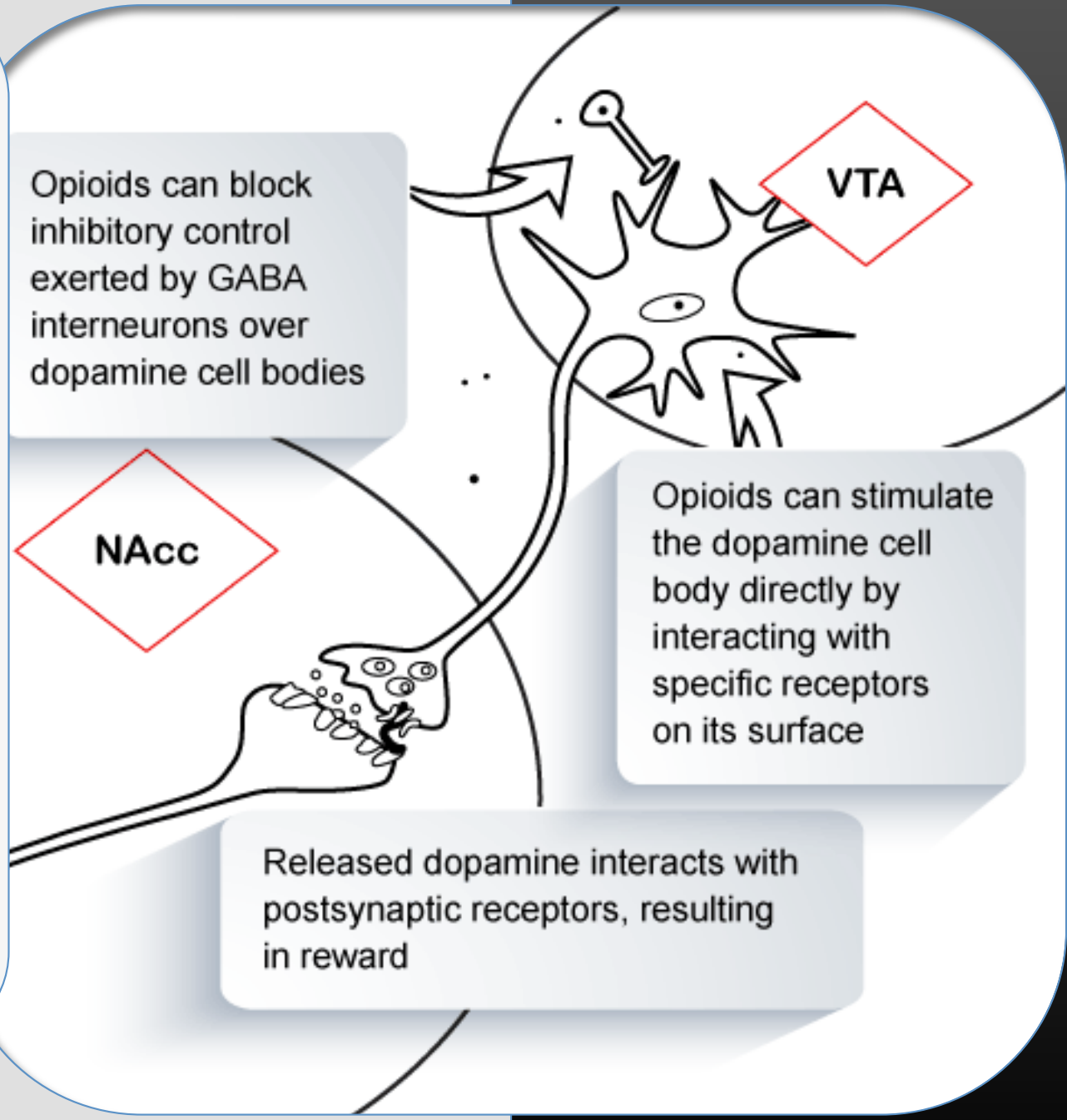
OPIATE EFFECTS

- DESIRABLE **THE GOOD**
  - Euphoria - heroin produces greater 'rush' than morphine due to ↑ lipophilicity
  - Prolonged sense of contentment and well-being
- UNDESIRABLE **THE BAD**
  - Nausea and vomiting
  - Respiratory depression – ↓ in sensitivity of respiratory centre to PCO_2
  - Constipation - ↑ tone + ↓ motility in GI tract
    - DON'T RX OPIATES WITHOUT CONSIDERING THIS
  - Pupillary constriction - stimulation of oculomotor nucleus

HOW IT WORKS

- Heroine metabolites act on mu receptors on GABA neurons to uninhibit the firing of dopinergic neurons in VTA
- This results in increase of DE release



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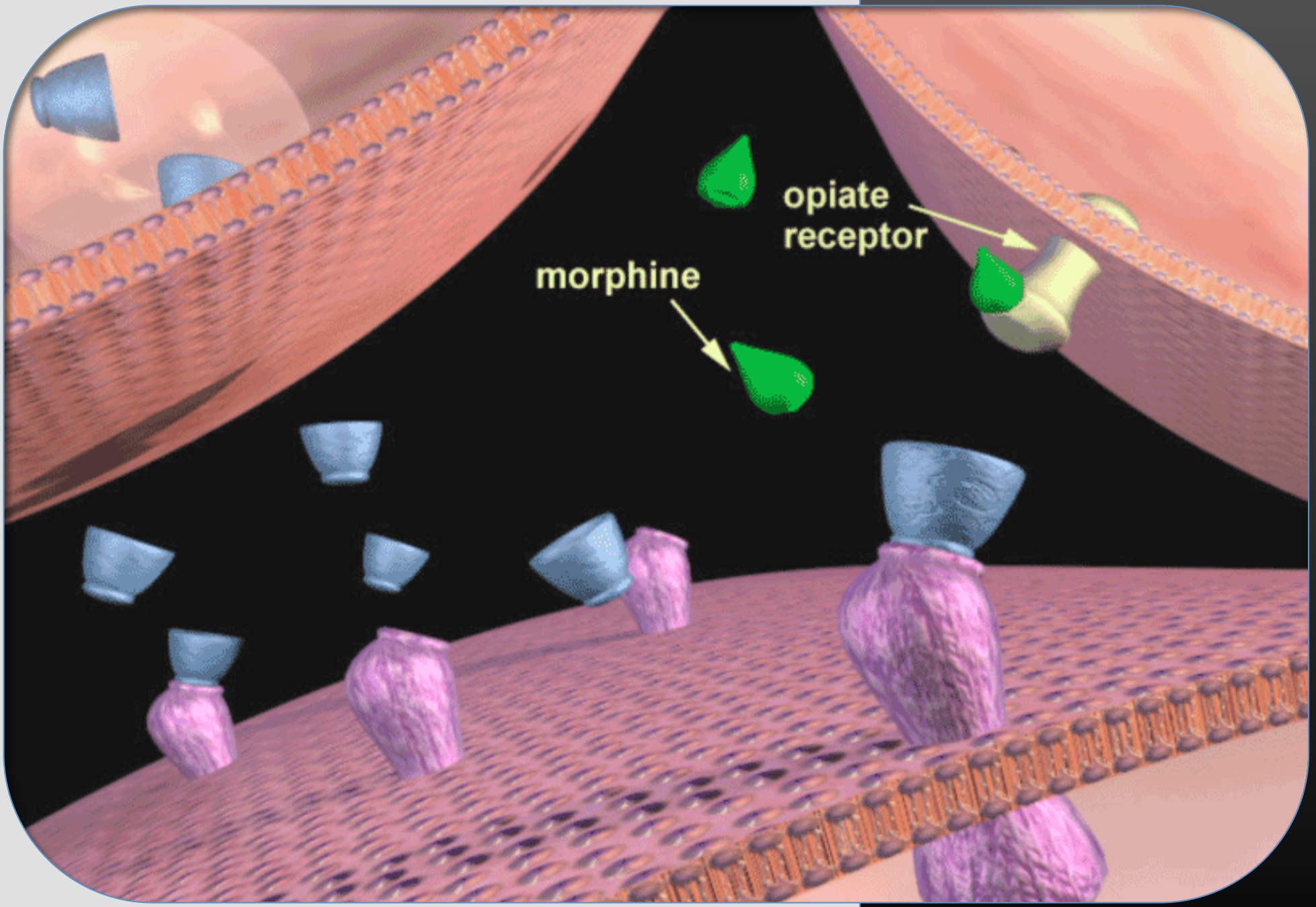

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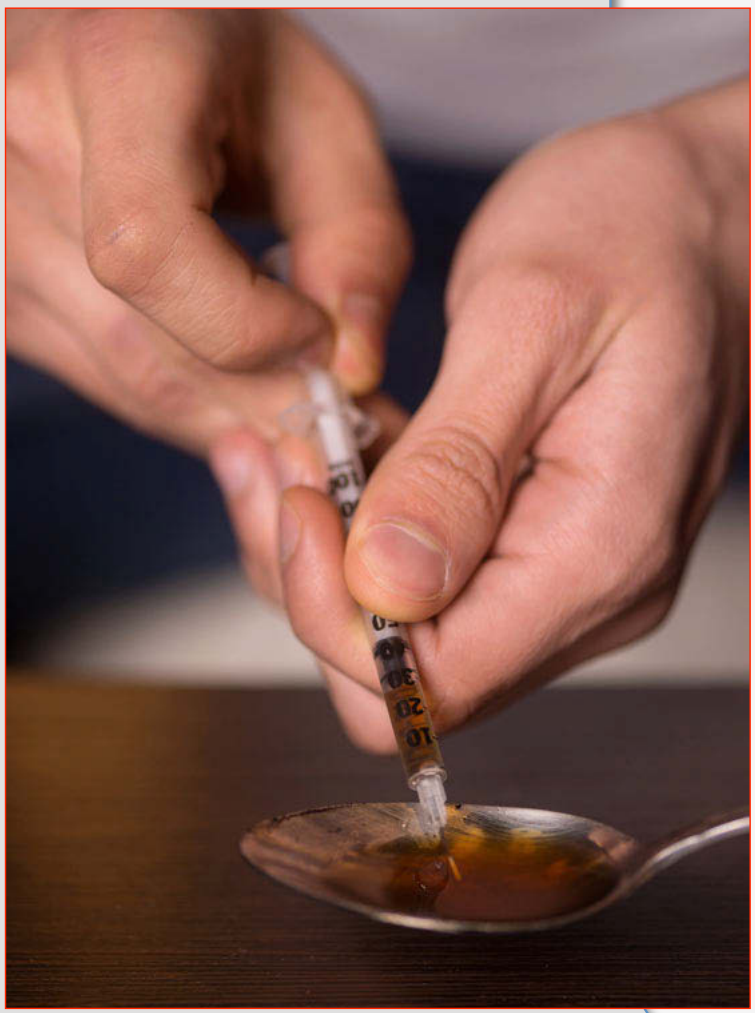
THE BAD



TOLERANCE, ADDICTION & WITHDRAWAL

- With regular opiate use, tolerance develops.
- As higher doses are used over time, physical dependence develops.
- Withdrawal, which in regular abusers may occur as early as a few hours after the last administration
- Drug craving, restlessness, muscle and bone pain, insomnia, diarrhea and vomiting, cold flashes with goose bumps ("cold turkey"), kicking movements ("kicking the habit"), etc.

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OPIATE WITHDRAWAL

- Withdrawal symptoms peak between 48 and 72 hours after the last dose
- Duration and intensity dependent on quantity and half live of opiates being used
- Heroin WD usually subsides after about a week.
- Methadone WD can last weeks
- RX OPIATES CAUSE WITHDRAWAL TOO

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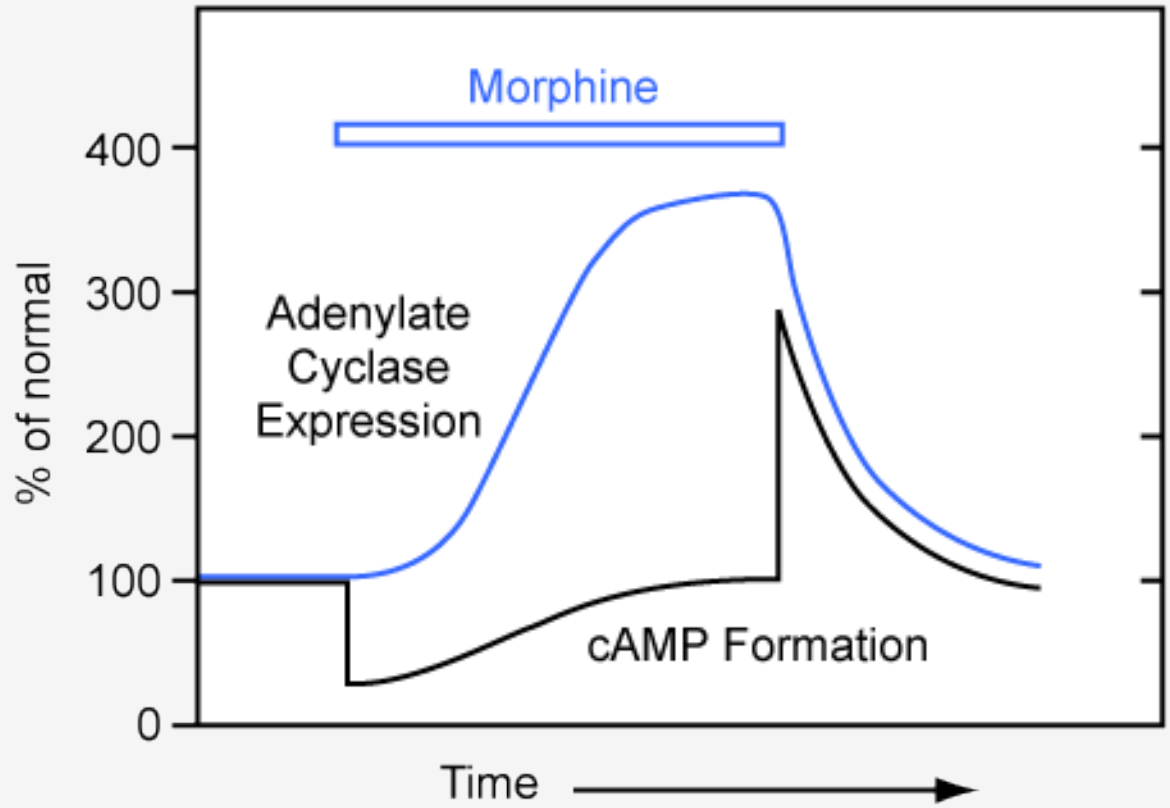
ON CESSATION EXCESSIVE cAMP PRODUCTION

- Heroine metabolites act on mu receptors on GABA neurons to uninhibit the firing of dopinergic neurons in VTA
- This results in increase of DE release



OPIATE OVERDOSE TX

- Respiratory depression, CNS depression, Myosis, signs of drug abuse, history
- R/O hypoglycemia, acidemia, fluid and electrolyte abnormalities
- Support: airway, ventilation, cardiac function
- Naloxone HCL 0.4-0.8mg initially
- Repeat PRN

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TX OF OPIATE DEPENDENCE

Comprehensive treatment gives best chance of long lasting remission

- Opiate replacement or pharmacologic support of withdraw symptoms
- Cognitive Behavioral Treatment: matrix, counseling, etc.
- 12 step work
- CAN NOT RX OPIATES FOR OPIATE WD

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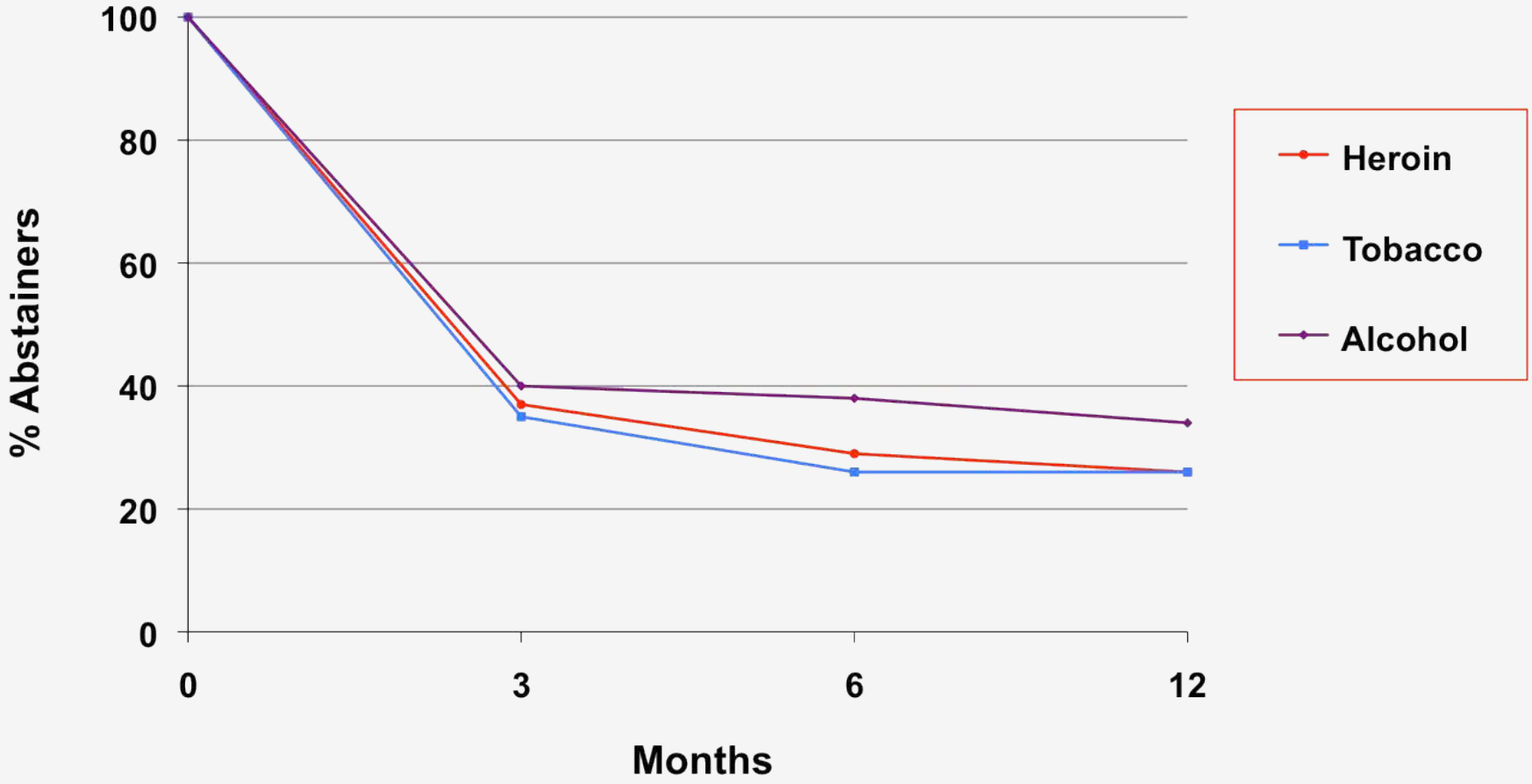
RELAPSE CURVE FOR HEROINE, TOBACCO & ALCHOL ADDICTION



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EFFECT OF COMMON OPIATES AT Mu RECEPTOR

FULL AGONIST	○ Heroin, morphine, methadone
PARTIAL AGONIST	○ Buprenorphine ○ Tramadol
ANTAGONIST	○ Naltrexone (Revia, Vixo) ○ Nalmefene ○ Naloxone

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RECEPTOR BINDING AT Mu RECEPTOR

AGONIST Opens Door	Morphine like effects
PARTIAL AGONIST Opens Door With Safety Chain	Weak morphine like effects with strong receptor affinity
ANTAGONIST Dummy Key	No effect in absence of an opiate or opiate dependence

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AGONIST THERAPY

- tMethadone is the gold standard
  - Must be administered in setting of OTP, Opiate Treatment Program
  - Highly regulated
  - Can be used for pain
- Legislation prevents the use of agonists specifically for the treatment of opiate dependence outside the setting of OTP

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FOUR QUESTIONS PATIENTS ASK

- HOW IS METHADONE BETTER FOR ME THAN HEROIN?
- What is the right dose of methadone for me?
- How long should I stay on methadone?
- What are the side effects of methadone?

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HOW IS METHADONE BETTER FOR ME THAN HEROIN?

HOW IS METHADONE BETTER FOR ME THAN HEROIN?

- Legal
- Avoids needles
- Know amount ingested
- Slow onset no “rush”
- Long acting: can maintain “comfort” or normal brain functioning.
- Stabilized physiology, hormones, tolerance

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FOUR QUESTIONS PATIENTS ASK

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WHAT IS THE RIGHT DOSE OF METHADONE FOR ME?

WHAT IS THE RIGHT DOES OF METHADONE FOR ME?

- Eliminate physical withdrawal
- Eliminate ‘craving’
- Comfort/function: usually trough is 400-600 ng/ml, peak no more than twice the trough.
- Not over-sedated
- Blocking dose

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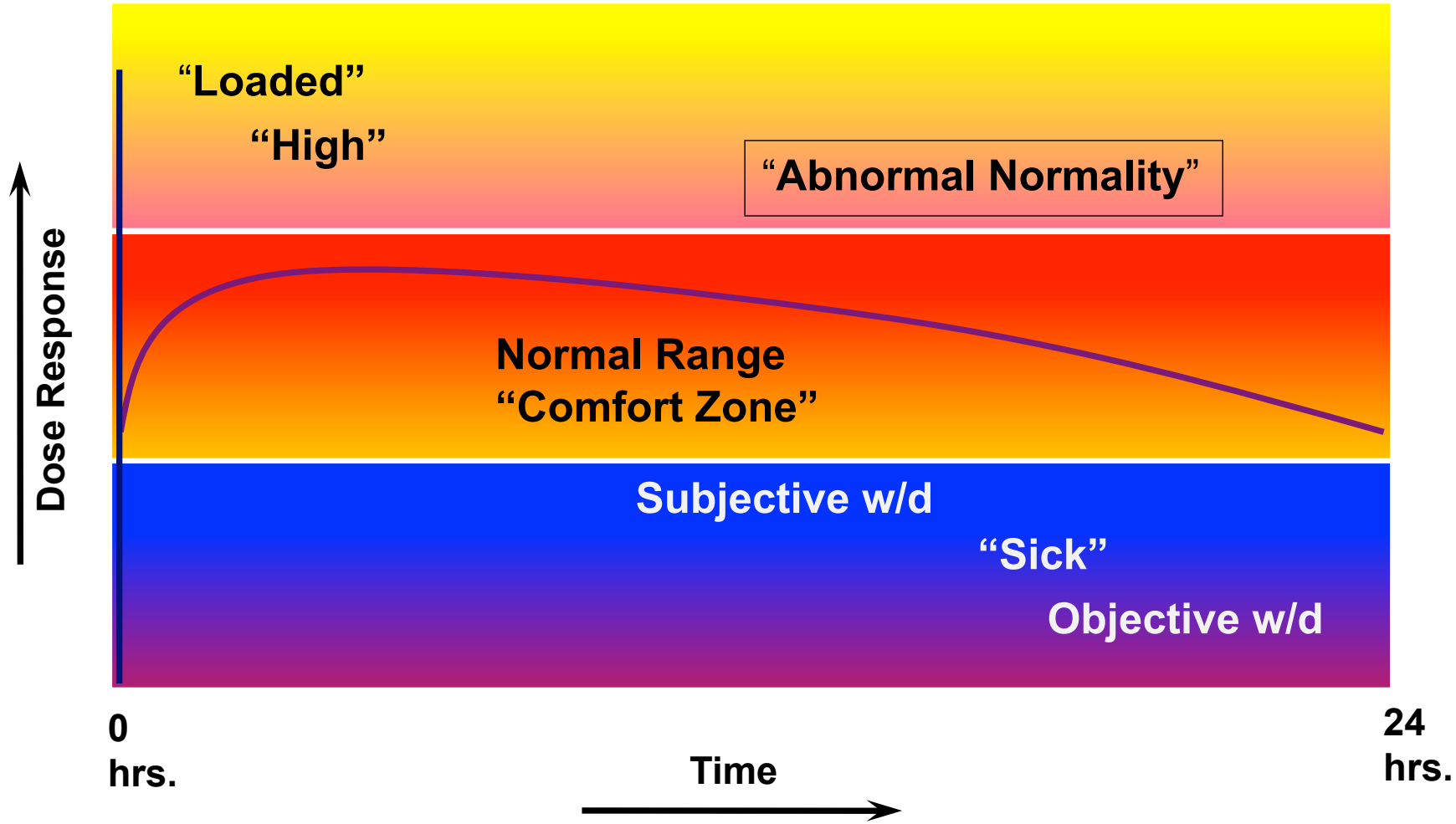
METHADONE SIMULATED 24 HR. DOSE/RESPONSE

At Steady-State in Tolerant Patient



Opioid Agonist Treatment of Addiction - Payte - 1998

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FOUR QUESTIONS PATIENTS ASK

- How is methadone better for me than heroin?
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HOW LONG SHOULD I
STAY ON METHADONE?

HOW LONG SHOULD I
STAY ON METHADONE?

HOW LONG?

AS LONG
AS REQUIRED!!
LONG ENOUGH!!

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FOUR QUESTIONS PATIENTS ASK

- How is methadone better for me than heroin?
- What is the right dose of methadone for me?
- How long should I stay on methadone?
- WHAT ARE THE SIDE EFFECTS OF METHADONE?

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WHAT ARE THE SIDE EFFECTS OF METHADONE?

WHAT ARE THE SIDE EFFECTS OF METHADONE?

- General opiate effects:
  - Sedation/stimulation
  - Maintained phys. dependence (stable)
  - Hypogonadism (not as severe as with heroin, may be dose dependent)
- Constipation
- Slight QTc prolongation on ECG
- Sweating
- Methadone treatment tied to regulated clinic

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WHAT ARE THE SIDE EFFECTS OF METHADONE?

OPIATE PHYSICAL EFFECTS?

- Predictable physical effects of administering opiates:
  - TOLERANCE: the body becomes efficient in processing the drug and requires ever higher doses to produce the desired effect.
  - DEPENDENCE: when the drug is discontinued there are typical withdrawal signs and symptoms.

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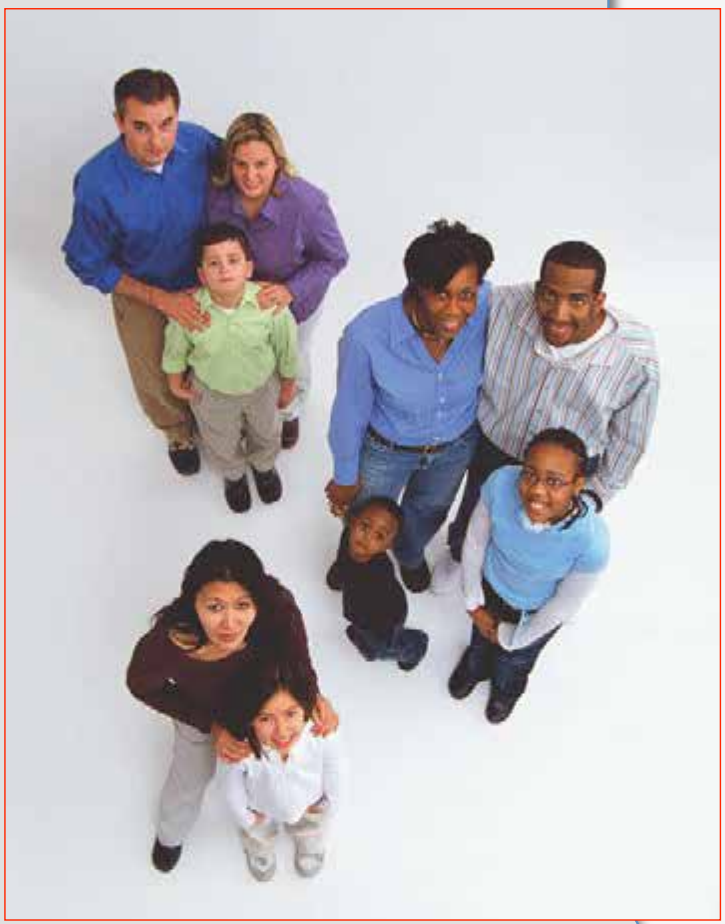
TREATMENT OUTCOME DATA



- 4-5 fold reduction in death rate
- Reduction of drug use
- Reduction of criminal activity
- Engagement in socially productive roles
- Reduced spread of HIV
- Excellent retention

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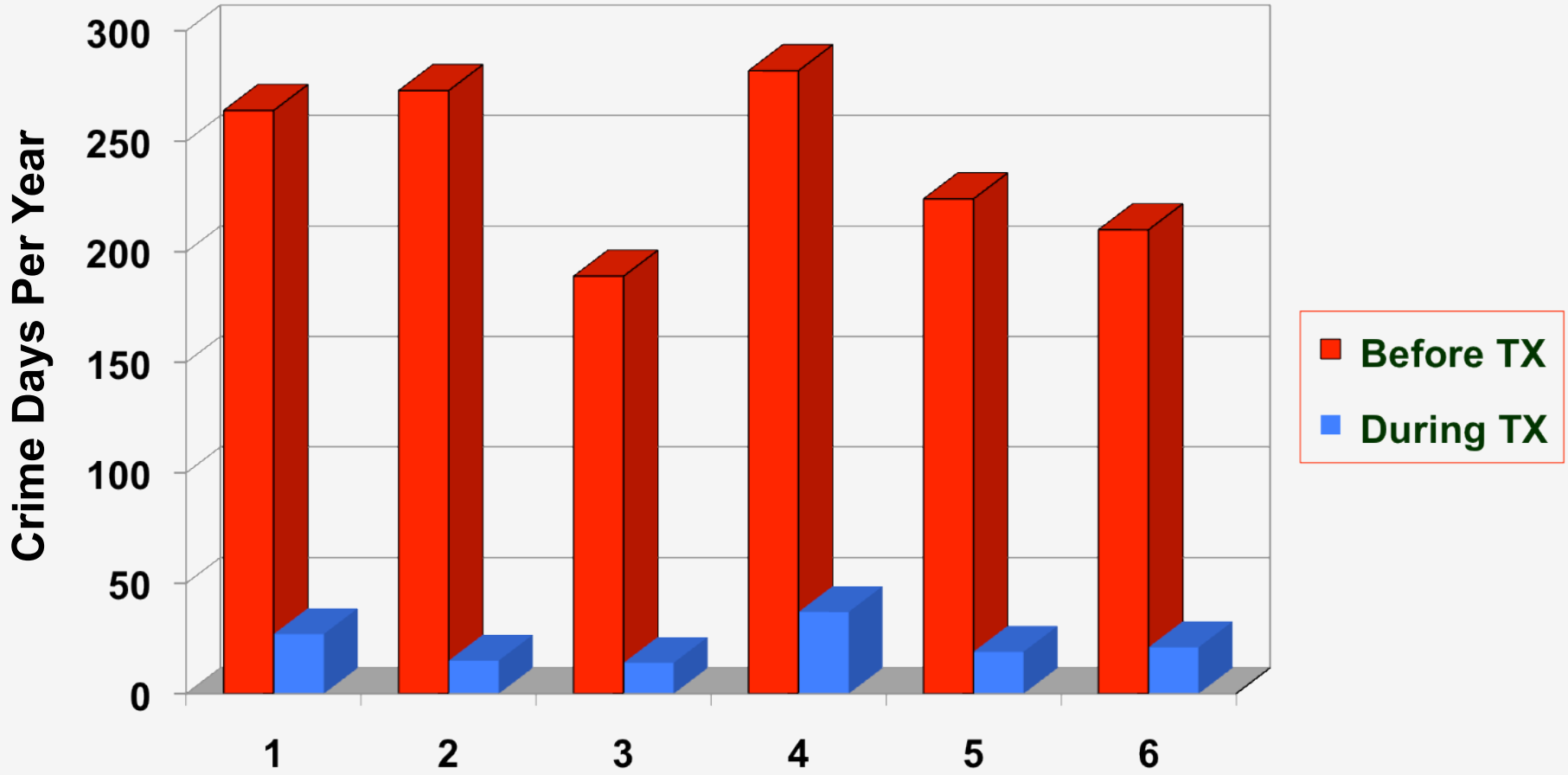
CRIME AMONG 491 PATIENTS BEFORE & DURING MMT AT 6 PROGRAMS



Adapted from Ball & Ross - The Effectiveness of Methadone Maintenance Treatment, 1991

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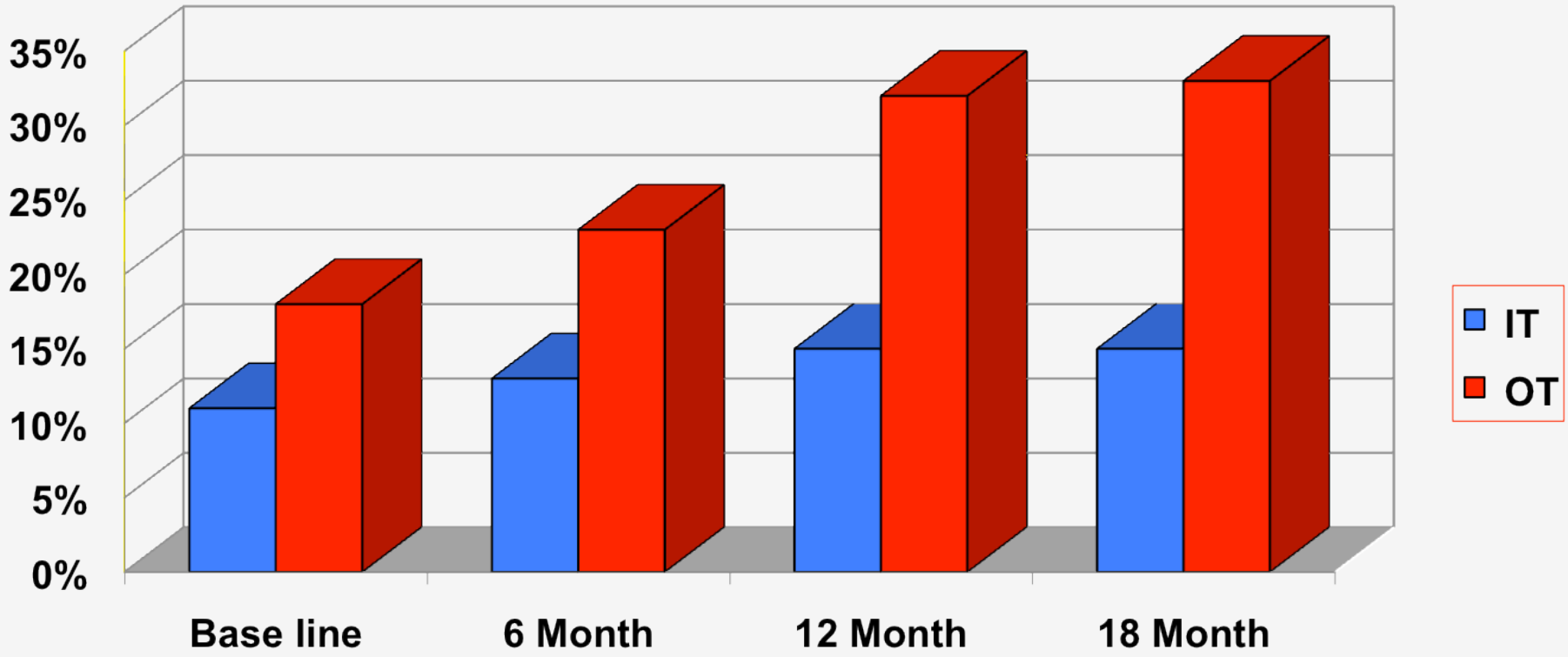
HIV CONVERSION IN TREATMENT



HIV infection rates by baseline treatment status. In treatment (IT) n=138, not in treatment (OT) n=88
Source: Metzger, D. et. al. J of AIDS 6:1993. p.1052

Opioid Maintenance Pharmacotherapy - A Course for Clinicians - 1997

BUPRENORPHINE

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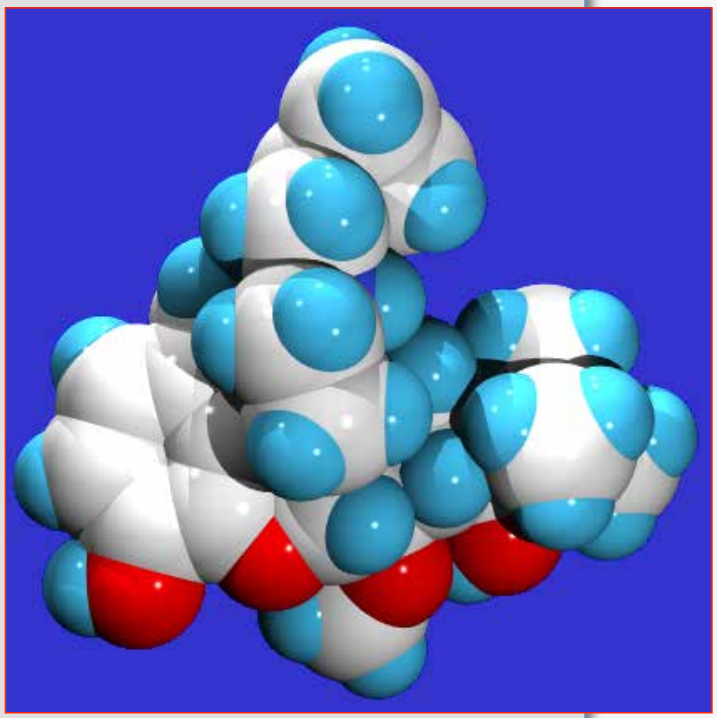
BUPRENORPHINE FOR OPIATE DEPENDENCE



- Suppresses withdrawal
- Substitutes for street opiates
- Blocks subsequently administered opiates
- Safety in long term use

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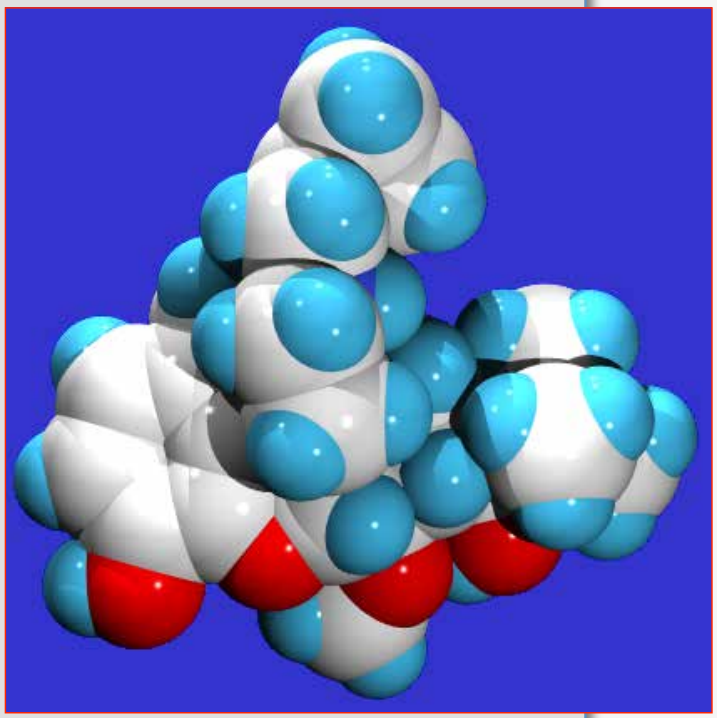
BUPRENORPHINE PHARMACOLOGY



- Less bounce to the ounce
- Ceiling effect on respiratory depression
- Less physical dependence capacity
- Blunts effect of subsequently administered full agonists
- Precipitates withdrawal in moderate to severely dependent people

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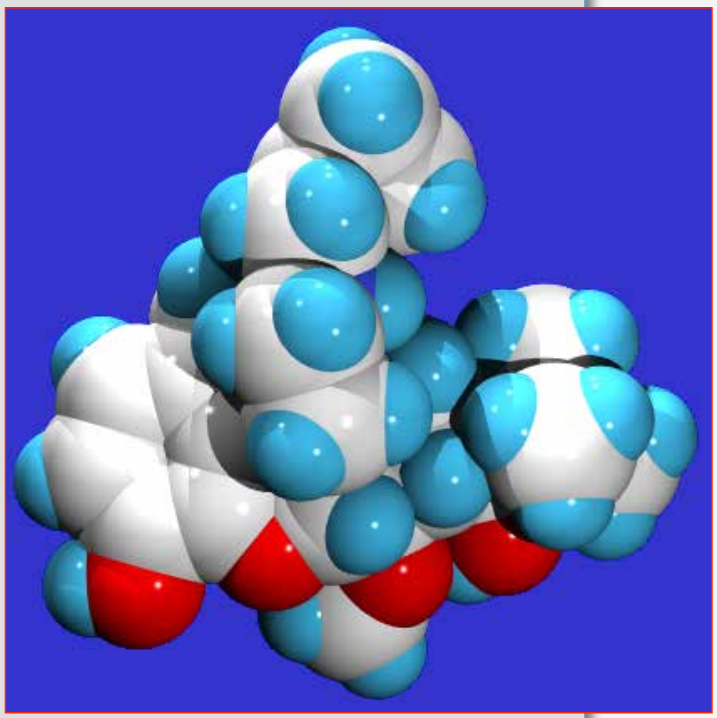
BUPRENORPHINE



CLINICAL PHARMACOLOGY

TIGHT RECEPTOR BINDING

- Long duration of action
- Slow onset mild abstinence
- Long t 1/2 for tx of opiate dependence
  - 37.5 hours
- Shorter t 1/2 for analgesia
  - 3-6 hours

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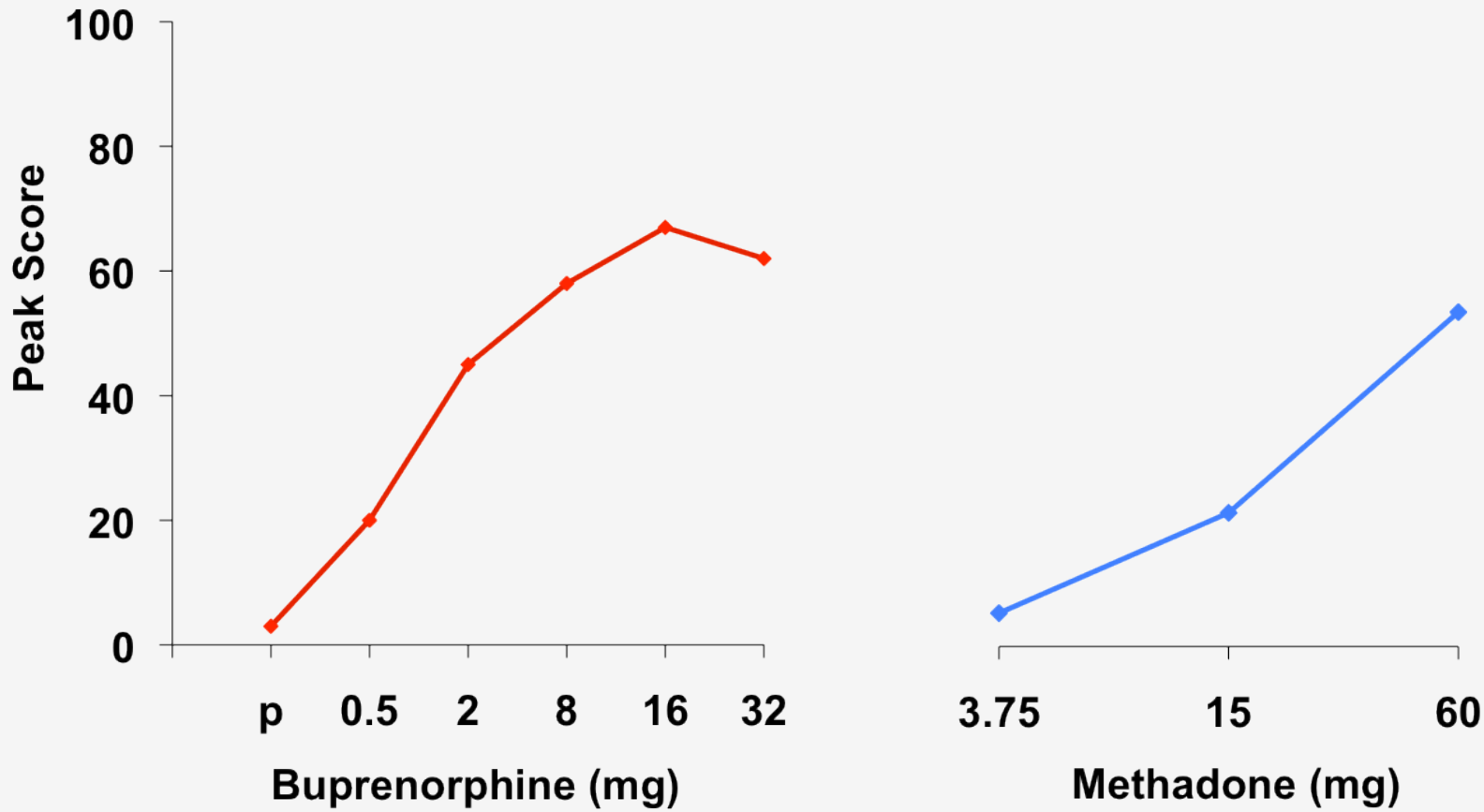
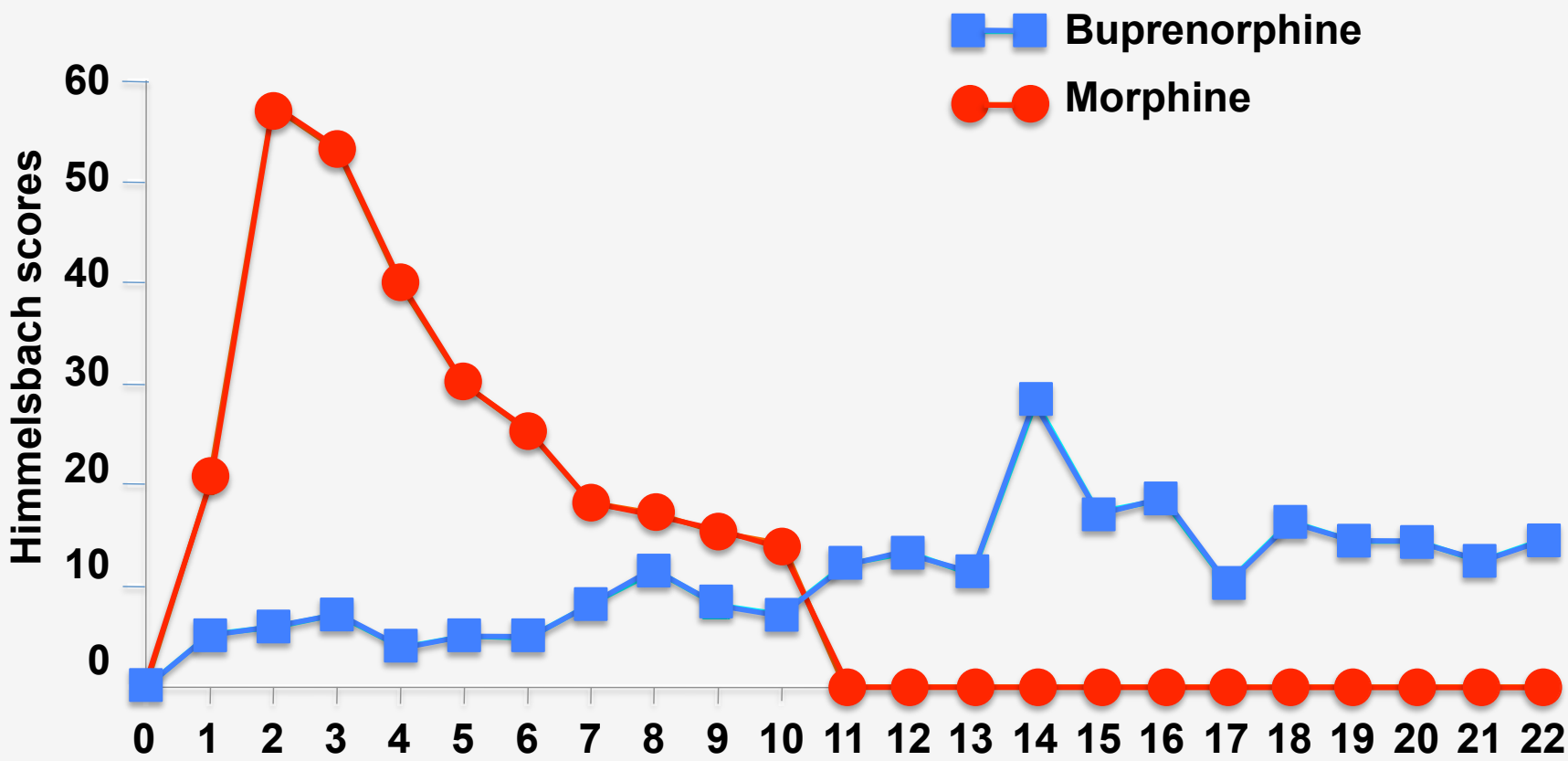
GOOD EFFECT



INTENSITY OF ABSTINENCE



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BUPRENORPHINE/NALOXONE COMBO

4 PART BUPRENORPHINE: 1 PART NALOXONE

SUBLINGUAL: Opiate agonist effect from buprenorphine

INTRAVENOUS: Opiate antagonist effect from naloxone

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NALOXONE REDUCES ABUSE POTENTIAL

- Naloxone will block buprenorphine's effects by the IV but not the sublingual route
- Sublingual absorption of buprenorphine @ 70%; naloxone @ 10%
- If injected, BUP/NX will precipitate withdrawal in a moderately to severely dependent addict

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