

Welcome!



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Pain Management

*Pain is Inevitable.
Suffering is Optional.*

CareOregon Pharmacy



Today's Agenda

- Welcome and Introduction – 8:00
- Chronic Pain 101– 8:05
- Medication Review – 8:55
- **Break – 9:30**
- Difficult Conversations– 9:45
- New in 2016 – 10:45
- Questions – 11:00
- Closing – 11:20

Hot Topic



TV RADIO NEWS

Local Nation World Economy

Health | Local | News

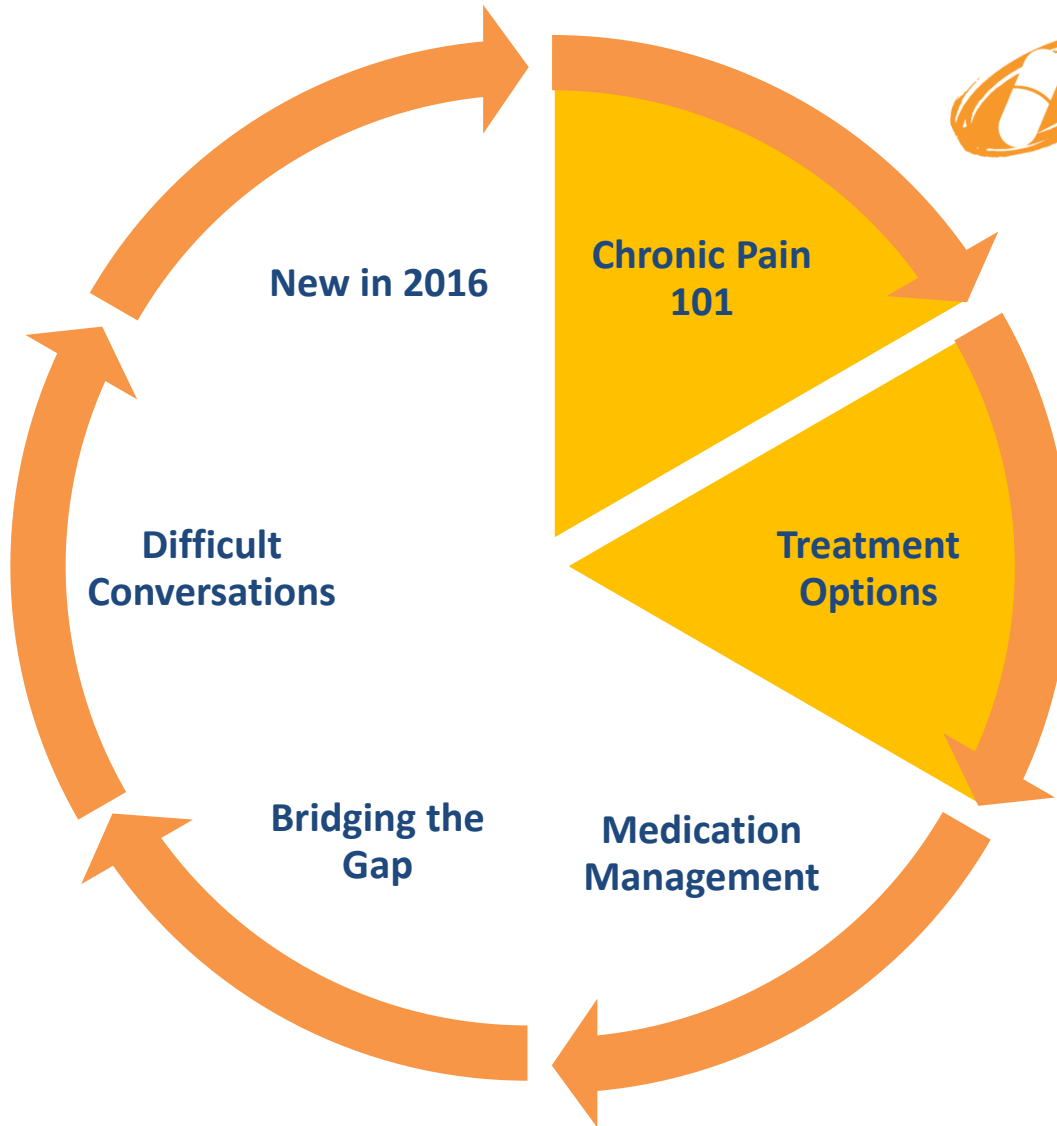
Oregon Health Providers Make It Harder To Get Opioids

by Kristian Foden-Vencil OPB | Dec. 1, 2015 2:43 p.m. | Updated: Dec. 1, 2015 4:35 p.m. | Portland



Objectives

1. Understand the pathophysiology of pain and neurobiologic pain mechanisms.
2. Describe the impact of environment and life experiences on the patient's pain management.
3. Summarize non-pharmacologic, behavioral, and pharmacologic treatments for chronic pain.
4. Identify key medication problems with a patient's pain management regimen.
5. Gain concrete tools to address difficult conversations with patients about pain management.



Chronic Pain 101: Sensation, Emotion and Reward

Rachel Solotaroff, MD, MCR

Central City Concern

Chair, CareOregon Metro

Chronic Pain/Chemical Dependency Task Force



Objectives

- Understand neurobiology and underlying mechanisms of sensory aspects of pain
- Provide examples of pharmacologic, behavioral and activity-based therapies to address pain
- Present a new paradigm for understanding pain based on emotion, reward, and interaction with addiction

Disclosures

- I have no financial disclosures

- I believe in embracing the complexity of chronic pain

Understanding Sensory Pain

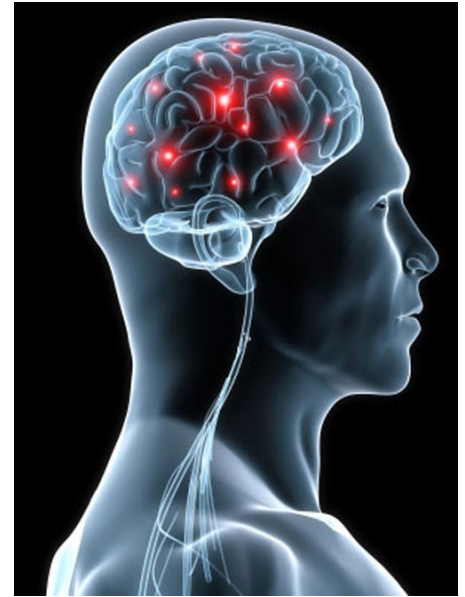
Understanding Pain in Less than 5 minutes!



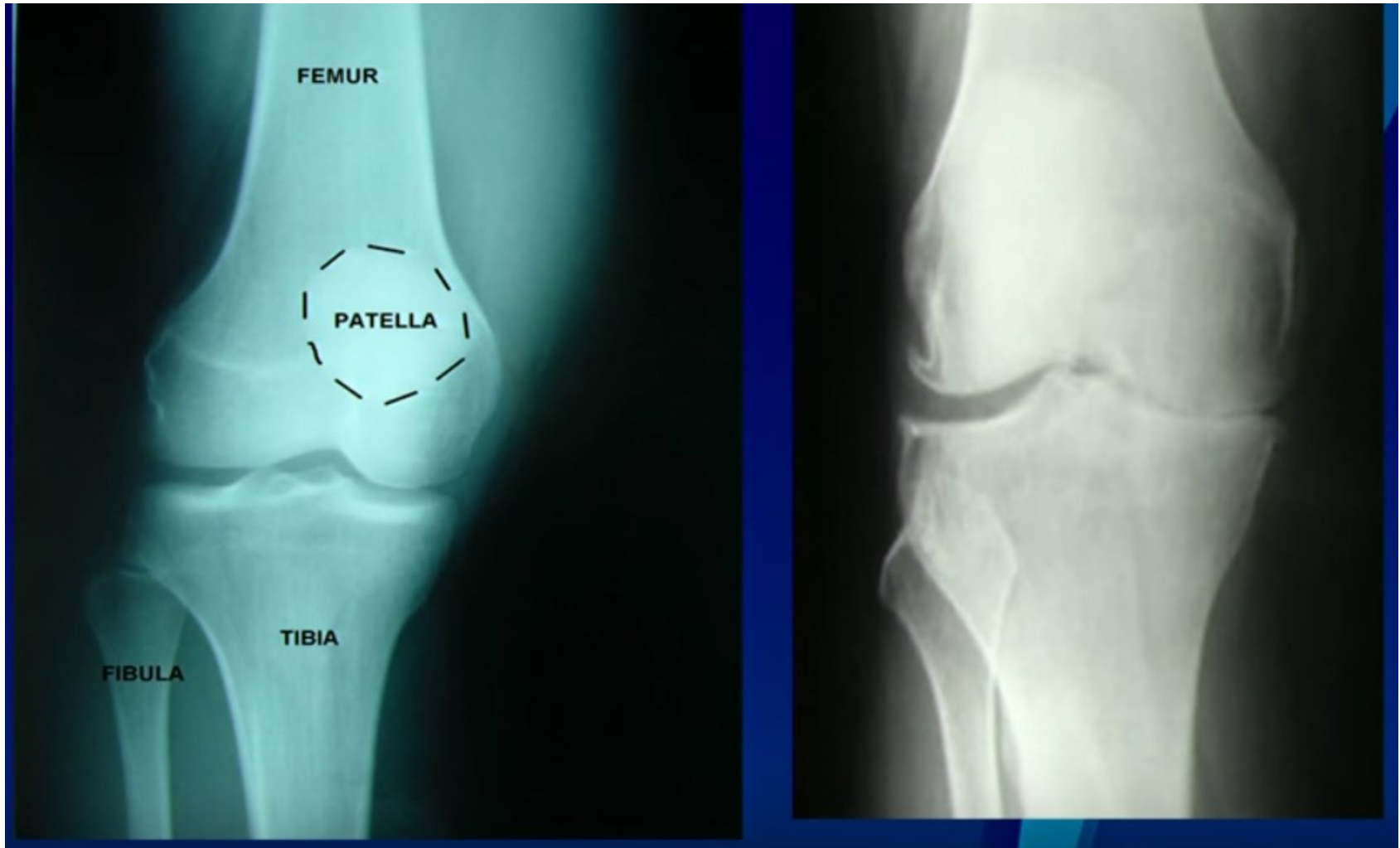
<https://www.youtube.com/watch?v=gy5yKbduGkc>

What is “Brain Pain?”

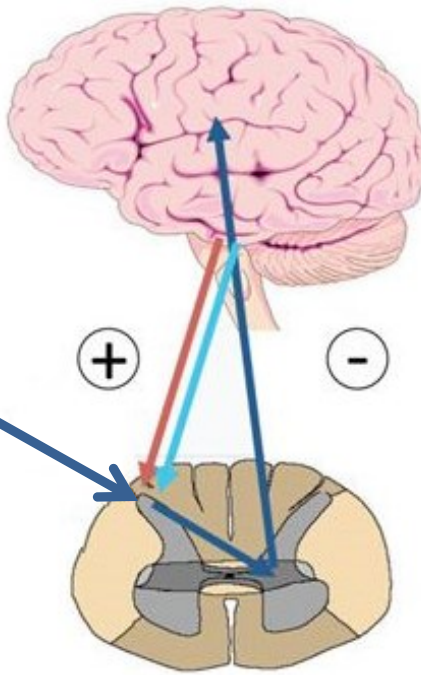
- Pain **not** from a hip, a knee, an abdomen
- Evolution of terms:
 - “Spastic colitis” → Irritable Bowel Syndrome
 - “Fibrositis” → Fibromyalgia
 - “Interstitial Cystitis” → Painful Bladder Syndrome
- Different treatments for Brain Pain than acute pain or true peripheral pain
- Namely, opioids, injections, surgical procedures do not work in brain amplification syndromes



Which Person Has Pain?







Mechanistic Characterization of Pain

Any combination may be present in a given individual

Peripheral (nociceptive)	Peripheral Neuropathic	Central Neuropathic or Centralized Pain
Inflammation or mechanical damage in tissues	Damage or dysfunction of peripheral nerves	Characterized by central disturbance in pain processing
NSAID, opioid-responsive	Responds to both peripheral and central drugs (TCA's, neuroactive compounds)	Responsive to neuroactive compounds altering levels of neurotransmitters involved in pain transmission and rehab
Responds to procedures	May respond if pain is localized to specific nerve	No response
Classic examples: <ul style="list-style-type: none"> • Acute pain due to injury • Osteoarthritis • Rheumatoid arthritis • Cancer pain 	Classic examples: <ul style="list-style-type: none"> • Diabetic neuropathic pain • Post-herpetic neuralgia 	Classic examples: <ul style="list-style-type: none"> • Fibromyalgia • IBS • TMJD • Tension headache

How Do You Identify Brain Pain?

- Involves more areas of the body, “multi-focal”
- Flares are not predictable
- Use different descriptors, hard to localize, sound “nerve” like
- Higher current and lifetime history of pain
- Accompanied by:
 - Fatigue
 - Memory problems
 - Sleep disturbances
 - Mood disturbances
 - Same neurotransmitters as “volume control”
- Sensitive to multiple sensory stimuli

Other Clinical Characteristics of Central Pain

- 1.5 – 2x more common in women
- Strong genetic predisposition (take a family hx)
- Can be triggered or exacerbated by stressors:
 - Early life stressors
 - War (but not natural disasters)
 - Infections (Lyme, Epstein Barr)
 - Environmental (mold)
 - Major surgical procedures
 - Peripheral pain syndromes (eg. RA, SLE OA)
 - Psychological stress/distress
- Generally normal physical exam except for diffuse tenderness

Central Pain on a Continuum

- Not “yes” or “no” – occurs over a wide distribution on a bell-shaped curve
- Diagnostic labels largely historical and irrelevant
 - Fibromyalgia (FM)
 - Irritable Bowel Syndrome (IBS)
- “Fibromyalgia-ness” predicts pain intensity, symptoms and disability over a wide range of rheumatic disorders
 - RA, OA, regional musculoskeletal pain, FM

Central Pain on a Continuum

Proportion of individuals in chronic pain states that have centralized their pain

Peripheral

Centralized



Acute Pain

Osteoarthritis
Rheumatoid
Arthritis

SC Disease
Ehler's Danlos
Low Back Pain

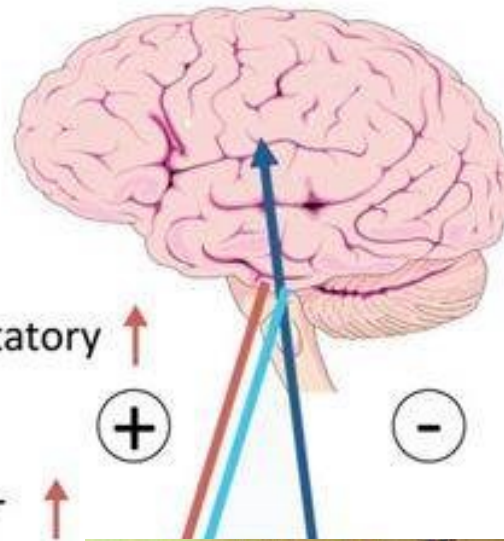
Fibromyalgia
Tension HA
IBS

Treating Sensory Pain

Neurotransmitter Targets in Central Pain

Facilitation

- Substance P ↑
- Glutamate and excitatory amino acids ↑
- Serotonin (5HT_{2a,3a}) ↑
- Nerve growth factor ↑



Inhibition

- Descending anti-nociceptive pathways
 - Norepinephrine-serotonin (5HT_{1a,b}), dopamine ↓
 - Opioids ↑
- GABA ↓
- Cannabinoids ↓



Drugs to Treat Chronic Pain

“Rule of 30% and 50%”:

- Our best drugs work marginally well in **50%** of people, fairly well in **30%** of people
- **50% of people will fail to respond at all**, or have side effects

Strong Evidence

- Dual reuptake inhibitors such as Tricyclic compounds (amitriptyline, cyclobenzaprine) and SNRI's and NSRI's (minicpran, duloxetine, venlafaxine)
- Anticonvulsants (eg. pregabalin, gabapentin)

Modest Evidence

- Tramadol
- Other less selective SSRI's or NRIs
- Low dose naltrexone
- Cannabinoids (neuropathic pain, MS Spasticity)

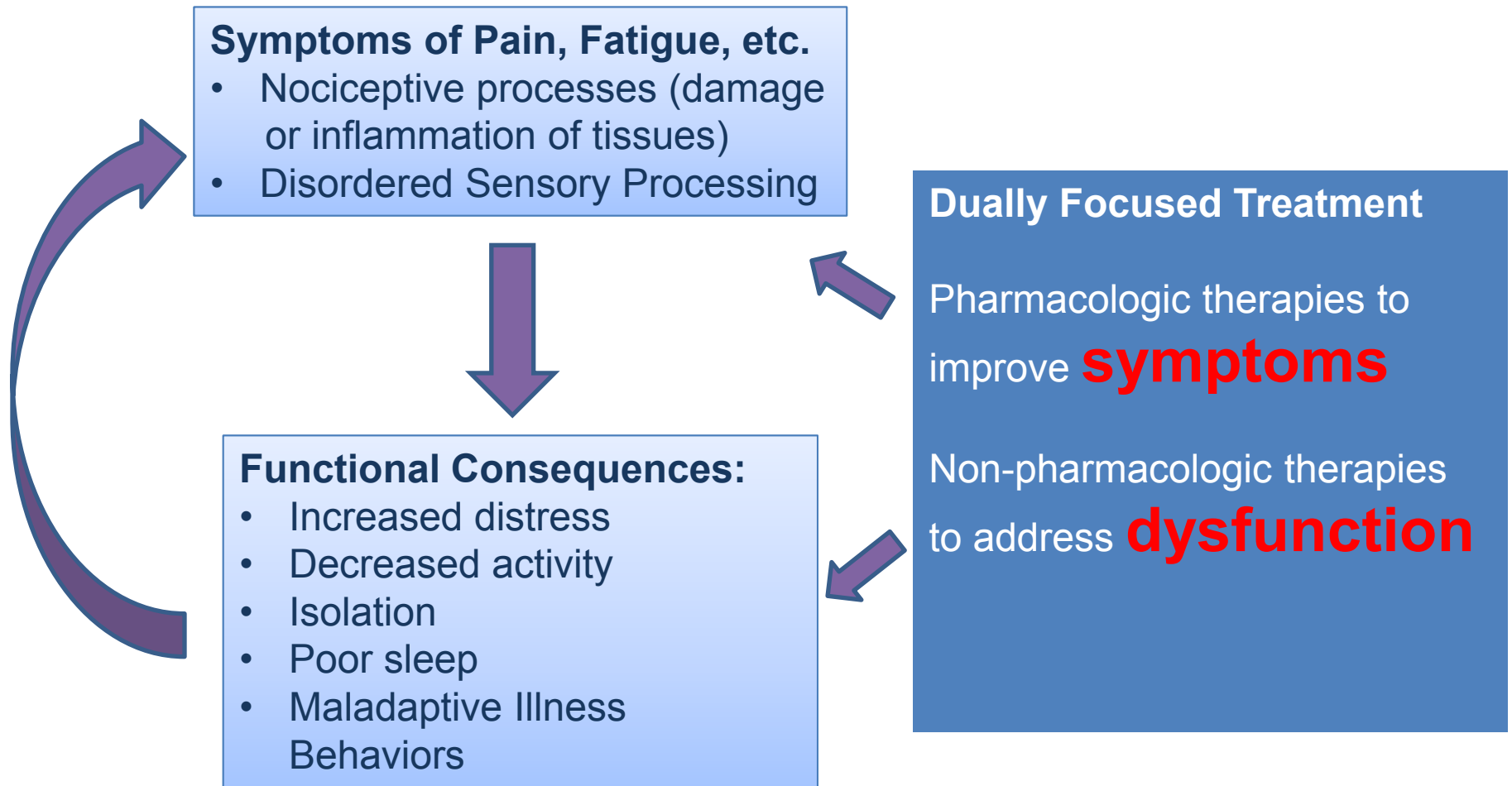
Weak Evidence

- Cannabinoids for non-neuropathic pain
- Growth hormone
- SAMe

No Evidence

- Opioids, corticosteroids, NSAIDS, benzodiazepine and non-benzodiazepine hypnotics

Downstream Consequences of Pain



Psychotherapeutic Approaches

- **Cognitive Behavioral Therapy:**
 - What we choose to think about has an impact on our behaviors
 - Thoughts, behaviors and mood interact
 - Can use skills like reality testing, examining thoughts to change behaviors
 - Challenges negative thoughts
- **Acceptance and Commitment Therapy:**
 - Doesn't challenge negative thoughts, as much as produces awareness of present
 - Avoid thoughts of what has happened /what will happen to me
 - Become more centered on what we have right now
 - Aware of what you have without judgement
 - Strategies/commitments of what to do with the present

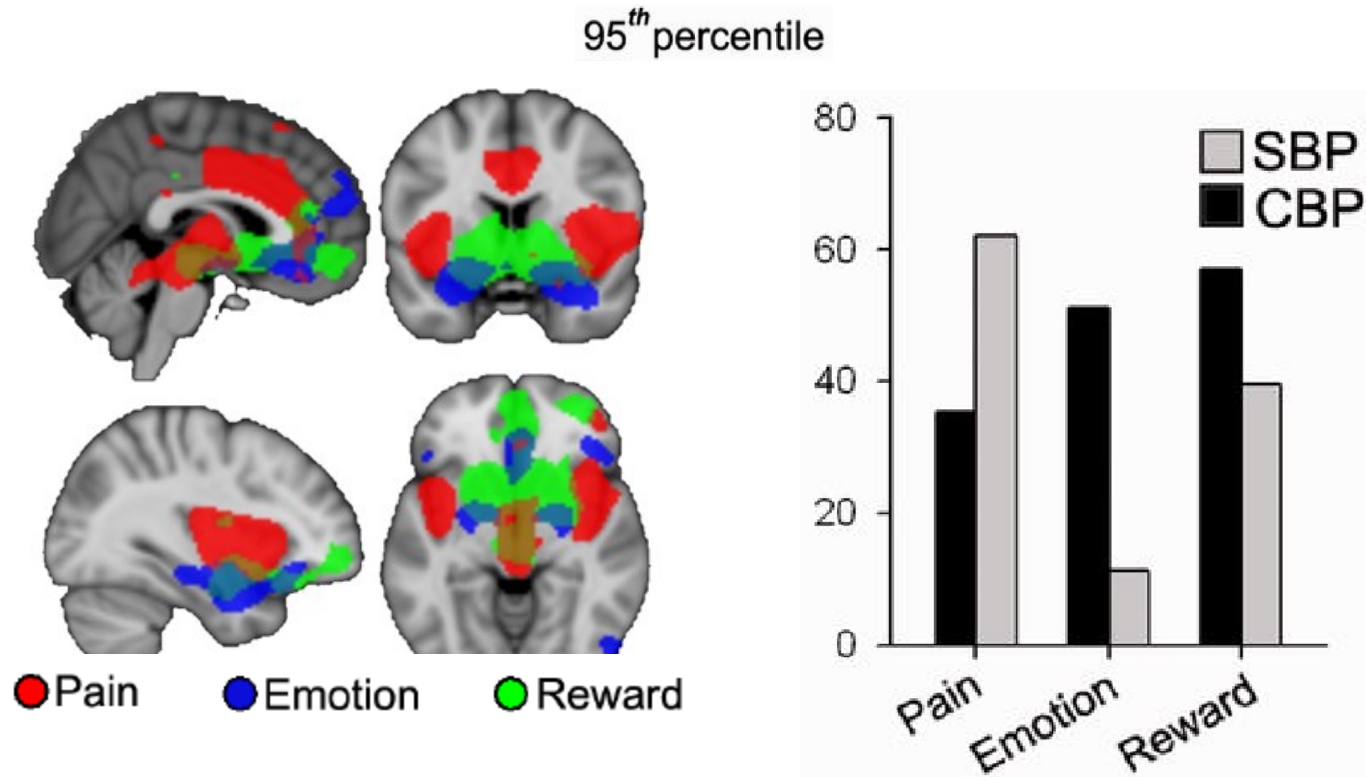
Behavioral Approaches

- Energy Conservation
- Pacing
- Pleasurable Activity Scheduling
- Activity → Exercise
- Sleep Hygiene

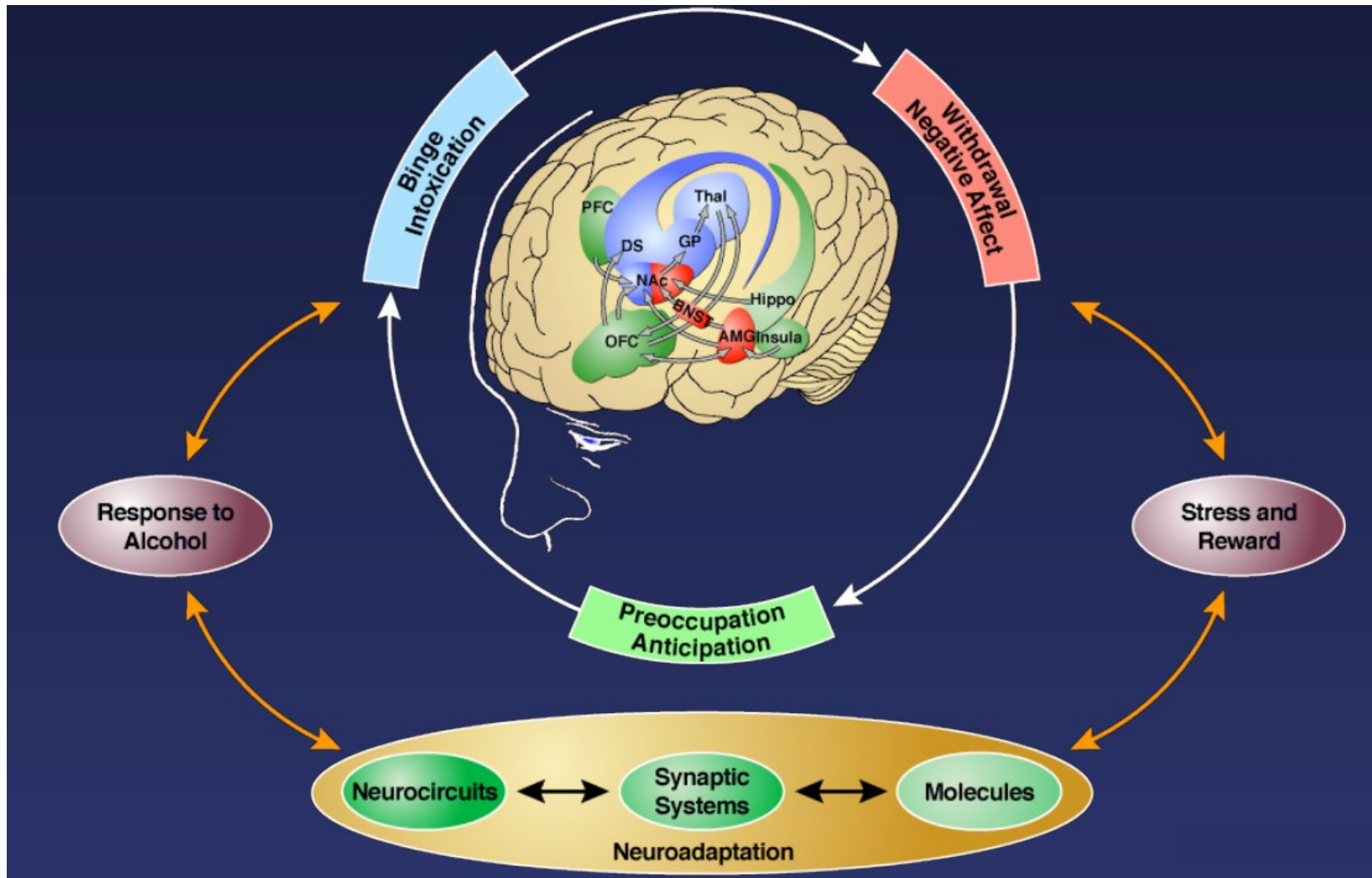
Understanding Emotion and Reward in Chronic Pain (and Addiction)

Understanding Reward and Emotion in Chronic Pain

Reward learning processes may contribute to persistence and amplification of pain

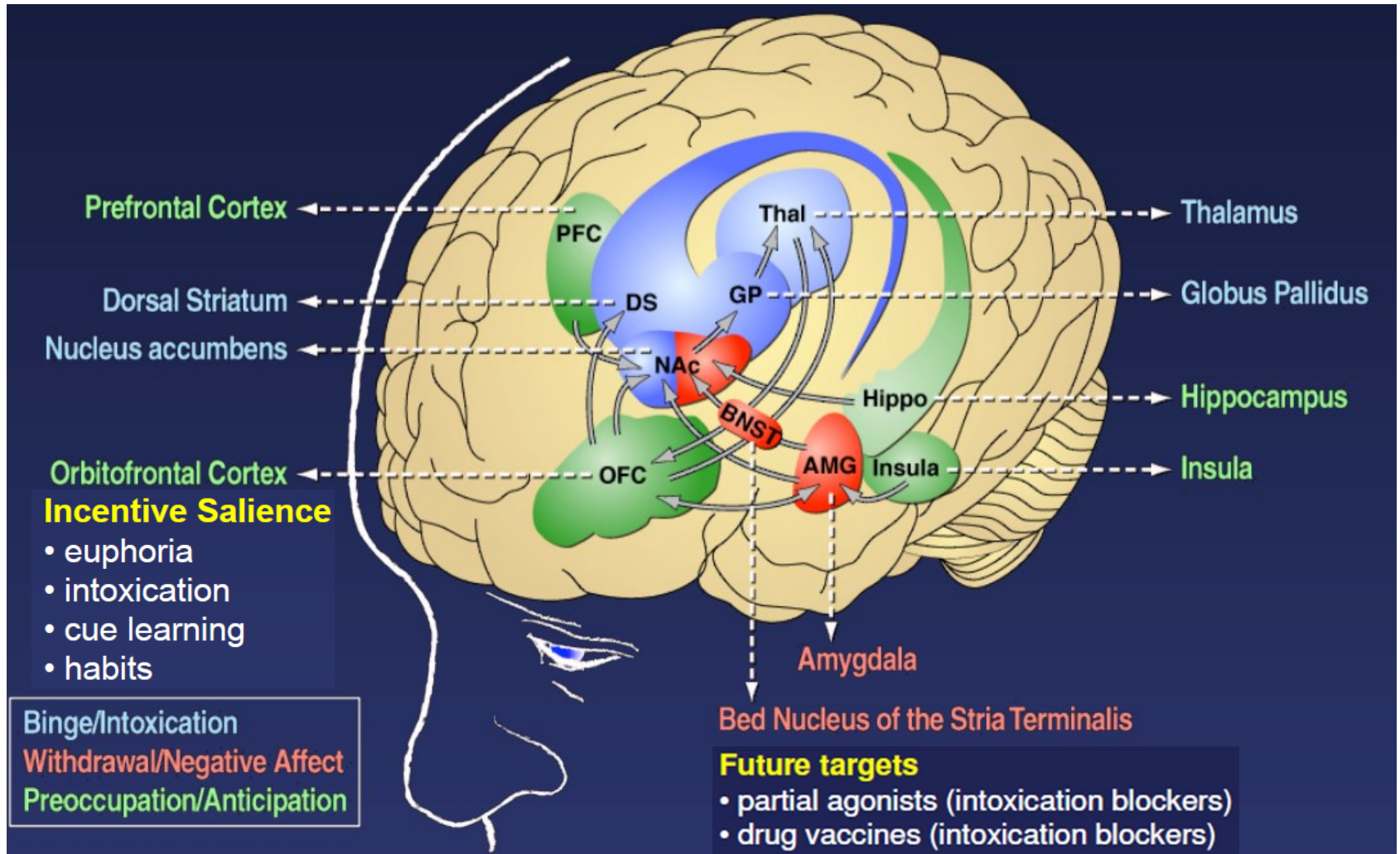


Review of the Neural Circuits of Addiction



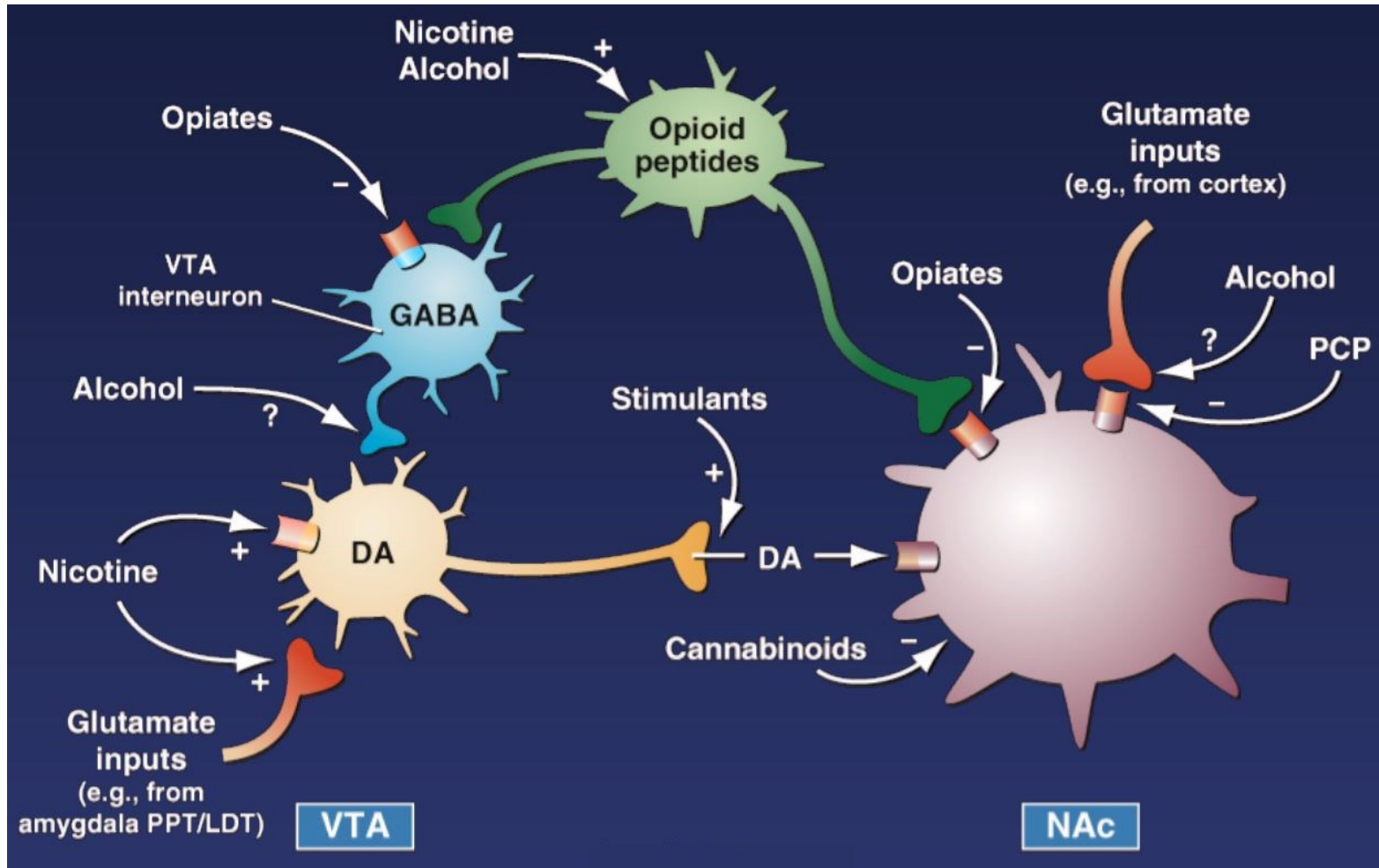
Koob, CSAM Addiction Medicine Review Course, 2014

Neural Circuits of the Binge/Intoxication Stage



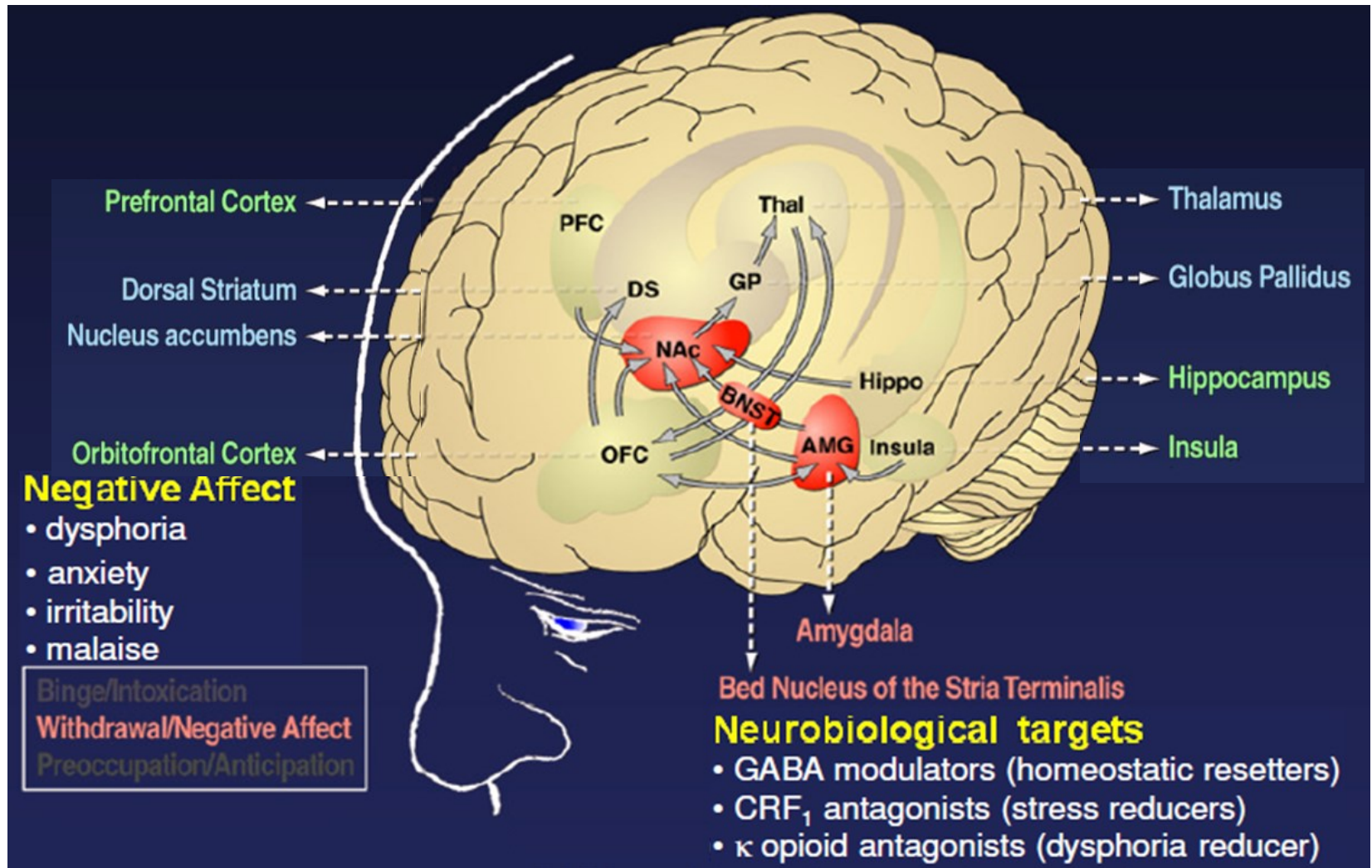
Koob GF, Volkow ND. *Neuropsychopharmacol REV*, 2010, 35:217-238

Converging Acute Actions of Drugs of Abuse on the Ventral Tegmental Area and Nucleus Accumbens



Nestler EJ *Nat Neurosci*, 2005, 8:1445-1449.

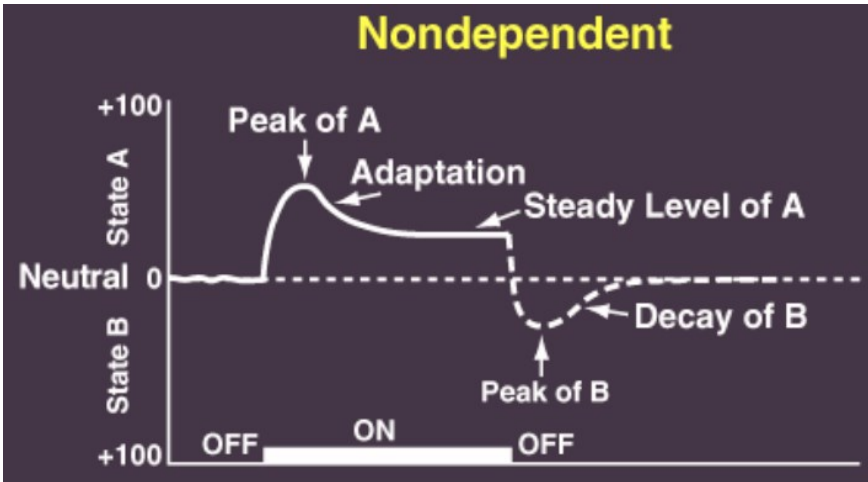
Neural Circuits of the Withdrawal/Negative Affect Stage



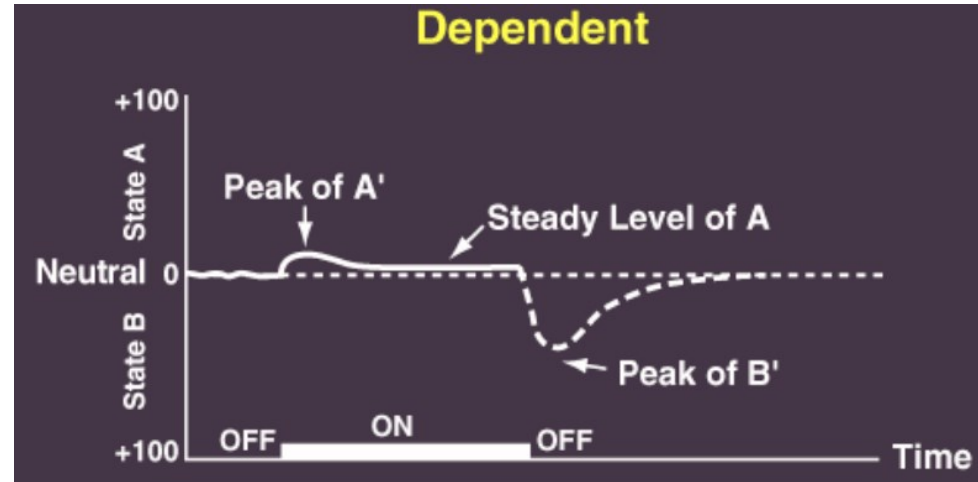
Koob, CSAM Addiction Medicine Review Course, 2014

Conceptual Model of Alcohol/Drug Dependence

Nondependent











Dependent



From Solomon RL, American Psychologist, 1980, 35: 691-712

Reward Transmitters Implicated in the Motivational Effects of Drugs of Abuse

Positive Hedonic Effects	Negative Hedonic Effects of Withdrawal
 Dopamine	 Dopamine – “dysphoria”
 Opioid Peptides	 Opioid Peptides – pain
 Serotonin	 Serotonin – “dysphoria”
 GABA	 GABA – anxiety, panic attacks



Koob, CSAM Addiction Medicine Review Course, 2012

Anti-Reward Transmitters Implicated in the Motivation Effects of Drugs of Abuse

Positive Hedonic Effects



Dynorphin – “dysphoria”



Corticotropin-Releasing Factor (CRF) – stress



Norepinephrine – stress

These are **ACTIVATED** in amygdale and ventral striatum during withdrawal

Koob, CSAM Addiction Medicine Review Course, 2012

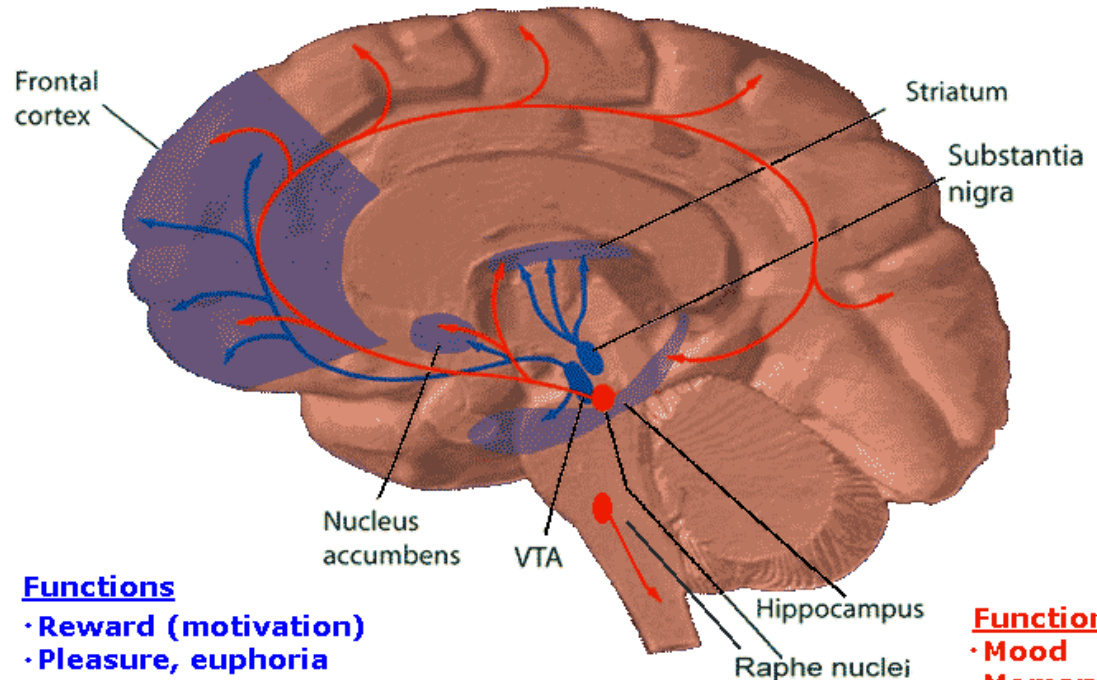
The Reward System in Pain

What does the reward system do? A Quick Decision-Making Process:

- Dopamine neurons from Ventral Tegmental Area estimate value of reward/relief-seeking
- Nucleus Accumbens (NAc) neurons decide whether to initiate reward/relief-seeking behavior then initiate reward/relief-seeking habits based on decision
- Frontal cortex also receives information, can inhibit NAc, but slowly

Dopamine Pathways

Serotonin Pathways



Functions

- Reward (motivation)
- Pleasure, euphoria
- Motor function (fine tuning)
- Compulsion
- Perseveration

Functions

- Mood
- Memory processing
- Sleep
- Cognition

So What's the Problem?

- Addictive drugs and search for pain relief can dump tons of dopamine into these circuits
- Addictive drugs increase activity in these neurons, or prolong actions of neurotransmitters they release
- New research show pain relief activates these neurons to drive habitual relief seeking

An Example:

“I’m going to lie down on the couch.”

- Back pain gets better, and your brain listens: “I got reward!”
- Your brain will refer that relief back to the laying down, and learn lying down as new context – as new opportunity to get relief
- When pain relief was present, your reward learning circuit turned on



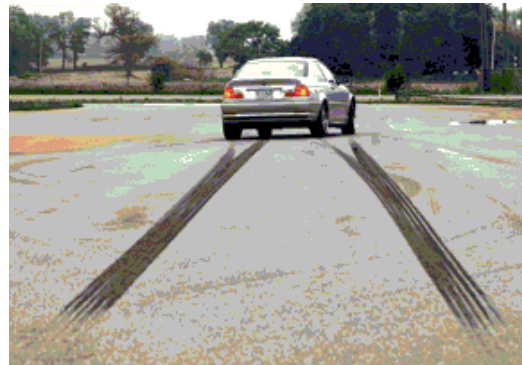
What Happens Over Time?

Accelerator:
D1 receptor



D1 Receptors:
Dopamine in the receptors tells Nucleus Accumbens to say “Yes!”

Brakes:
D2 receptor



D2 Receptors:
Activation of these receptors slows decision-making; allows frontal cortex time to step in

Too Much Accelerator is a Bad Thing

- When DA neurons are chronically over-active, they activate D1 receptors to:
 - Activate anti-reward circuits that increase stress response and worsen mood – both amplify pain signals
 - Desensitize and down-regulate D2, eliminating the brakes
 - Sensitize D1 pathways, speeding up decisions to seek relief
 - Pain severity increases and relief-seeking behaviors become compulsive

What Happened to the Brakes?

- What happens to D2?
- If you hit them hard with a lot of dopamine at once, they are desensitized
- D2 Receptor can't work again until it is recycled (takes an hour) or until a new receptor is synthesized
- You may ultimately wind up with a system that has no brakes



Inflammatory Injury Model

- All about elevated dopamine
- Patients with elevated dopamine are more likely to develop chronic pain
 - Acute injury → chronic pain
 - Drugs that increase dopamine → chronic pain
 - Smokers and people given opioids for an acute injury → chronic pain even after injury heals



How to Get off the Couch

- Get your brakes back on
- Need just enough DA to activate D2 receptors, get some inhibition but not knock them out
- Consistent low level DA input to build back inhibition
- Lots of tiny opportunities for little reward: make someone smile, enjoy sunshine
- The tiny things in life are what make life good, and allow D2 receptors give your brain time to make a choice.



Implications for Treatment

In both chronic pain and addiction, same healing process:

- Reduce exposure to huge dopamine signals:
 - Limit use of addictive drugs or medications, junk food, fast-acting analgesics, tobacco
 - Prevent desensitization of D2 pathway
- Increase exposure to small rewards:
 - Social reinforcement, problem-solving, effective emotional coping, small goal achievement
 - Increase activity of D2 pathway

Take Home Points

- Central pain:
 - Affects ~**30% population**
 - May be treated with behavioral and pharmacologic approaches
 - Opioids generally make this condition worse
- Treating behavioral health conditions in people with chronic pain:
 - Is important – keep treating them!
 - Won't solve the underlying pain problem

Take Home Points (continued)

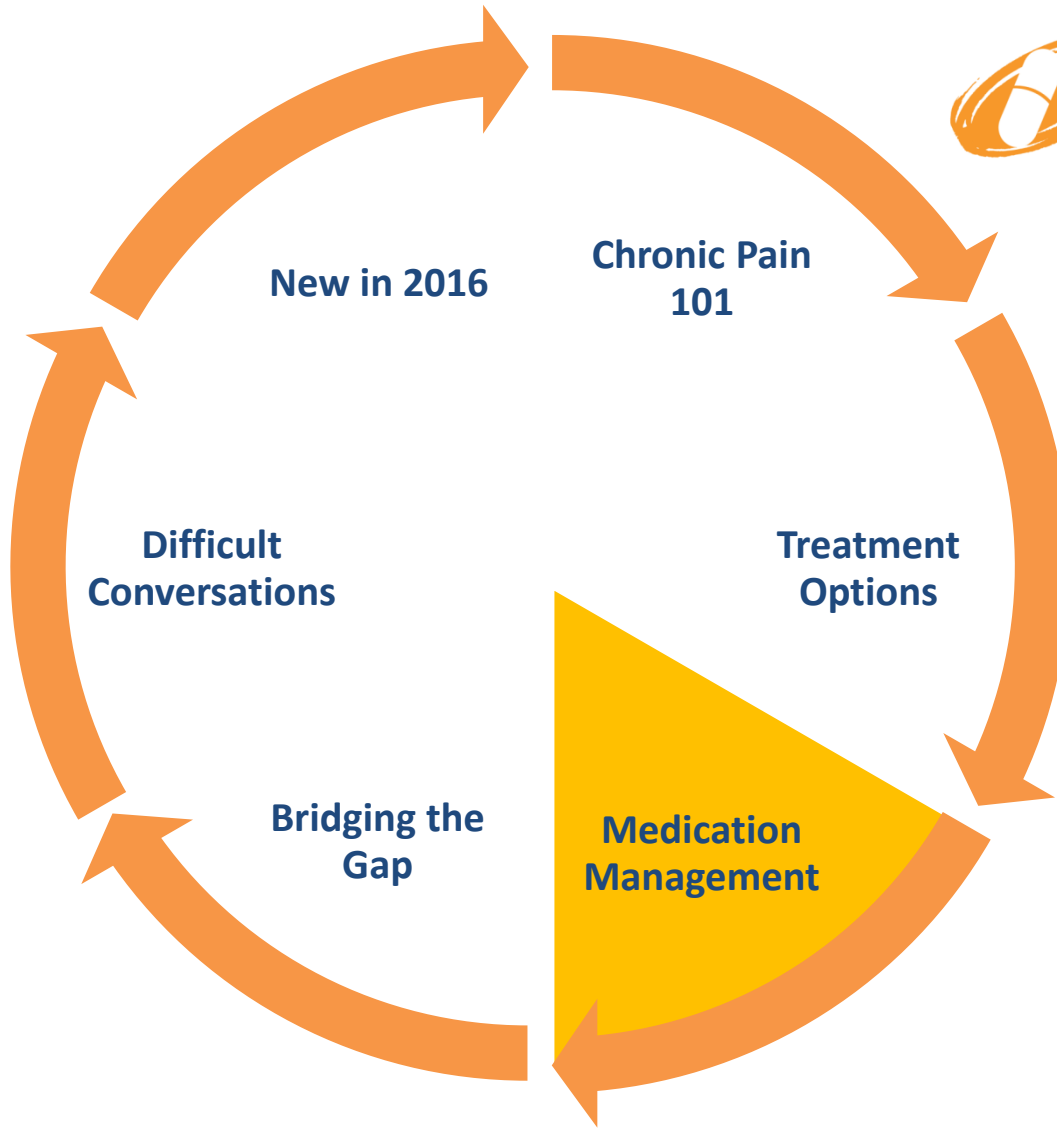
- Be thoughtful in assessment and diagnosis to identify conditions that are opioid-responsive
- Addiction and chronic pain have common neural circuitry → treatments are similar

Acknowledgements

- Daniel Clauw, MD
- Jodie Trafton, PhD

Please hold questions – thanks!



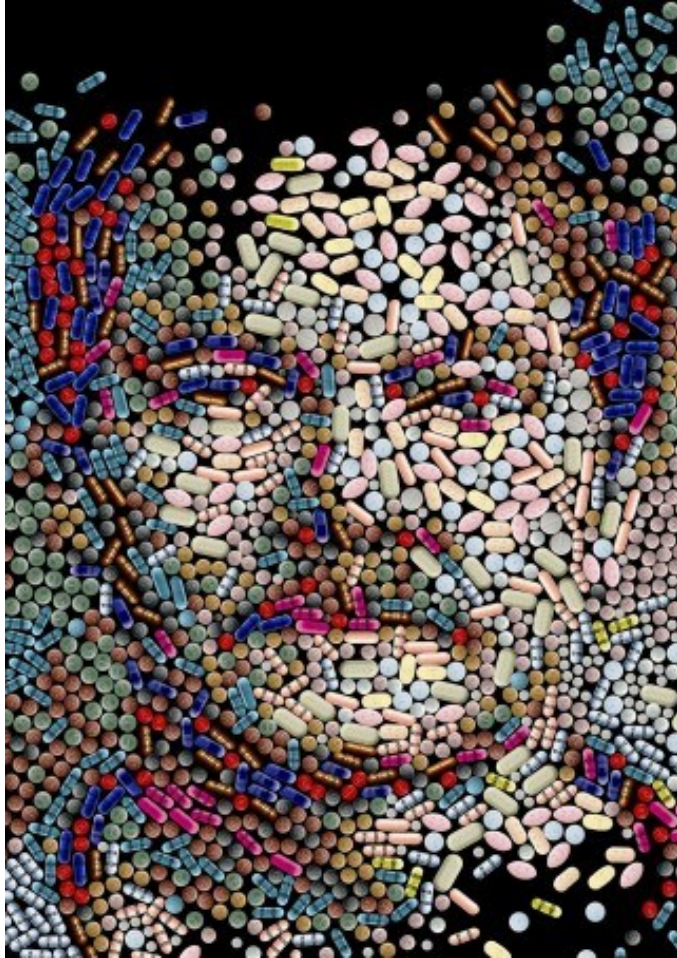


Pain Medications: Dr. Feelgood or Bad Medicine?

Tara Berkson, PharmD
MEDS Ed Coordinator
CareOregon



Dr. Feelgood or Bad Medicine?



THE NEW YORKER

LETTER FROM WICHITA | MAY 5, 2014 ISSUE

PRESCRIPTION FOR
DISASTER

The heartland's pain-pills problem.

Take Home Points

- What are our patients learning about pain medications from the media?
- What evidence exists about the benefits of pain medications?
- What are the top risks of chronic pain medication use?



Treatment Recommendations

	Osteoarthritis	Somatic Pain
1 st line	Acetaminophen (APAP) NSAIDs	APAP NSAIDs
2 nd line	Topical analgesics Intra-articular analgesics	Anticonvulsants TCAs
3 rd line	Opioids	Cyclobenzaprine Opioids: short-term

Treatment Recommendations cont

	Neuropathic Pain	Fibromyalgia
1 st line	TCAs SNRIs Gabapentin, pregabalin Topical analgesics	APAP TCAs SNRIs Cyclobenzaprine
2 nd line	Carbamazepine Oxcarbazepine Topiramate	Gabapentin, Pregabalin Older SSRIs Tramadol
3 rd line	Opioids	
Not Recommended		Opioids NSAIDs

Acetaminophen (Tylenol)



The Good

- OTC pain relief
- Fever reduction
- Appropriate for children
- Few drug interactions
- Minimal GI upset
- Helps opioid effectiveness

The Bad

- Not an anti-inflammatory
- Hard to keep track of doses- in multiple combination products

The Ugly

- Only 2-3 extra tablets per day may cause liver injury
- Boxed warning

Maximum dose: 4g per day or 12 regular strength tablets

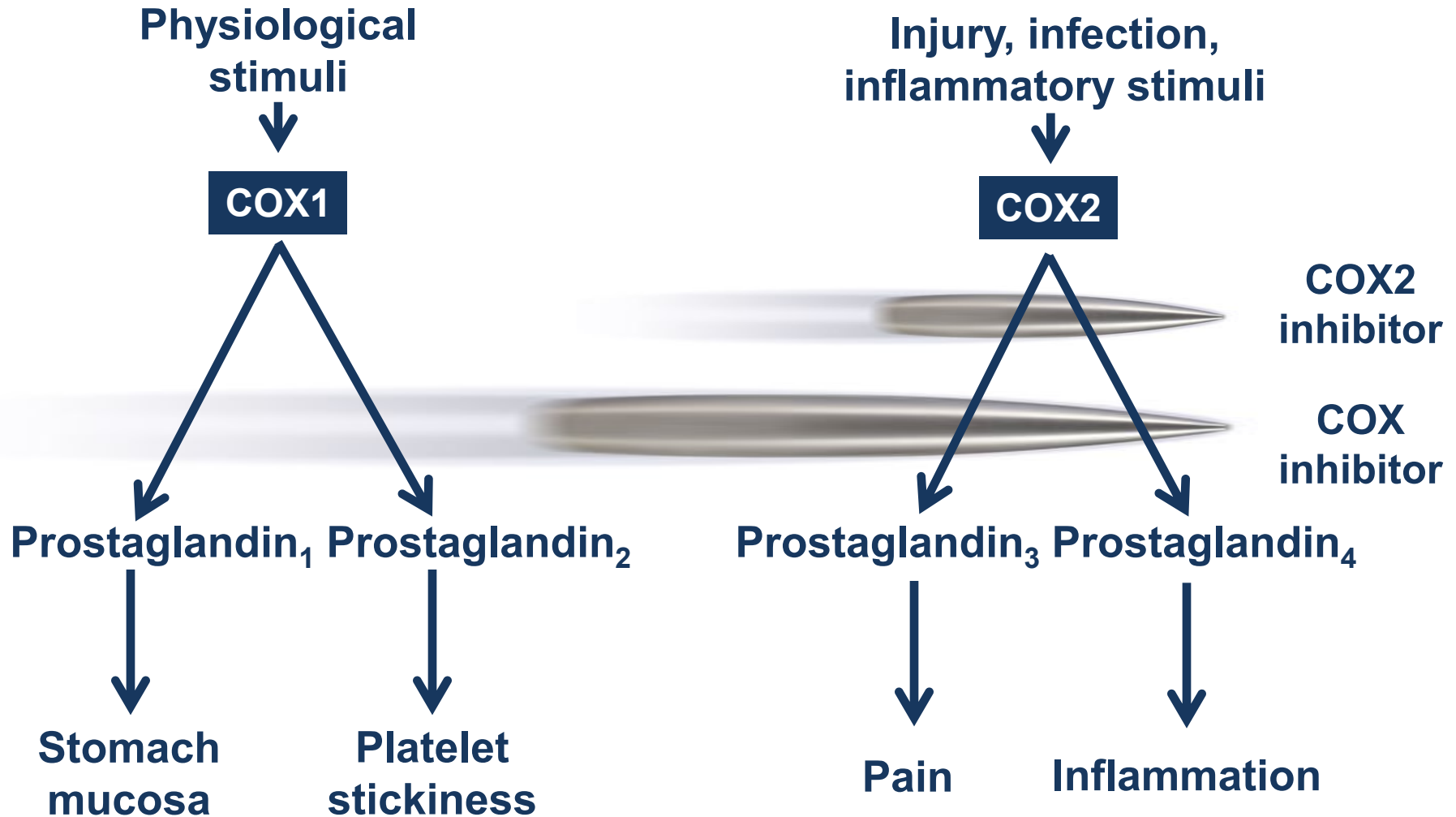
Nonsteroidal Anti-Inflammatory Drugs

First line for arthritis, inflammation

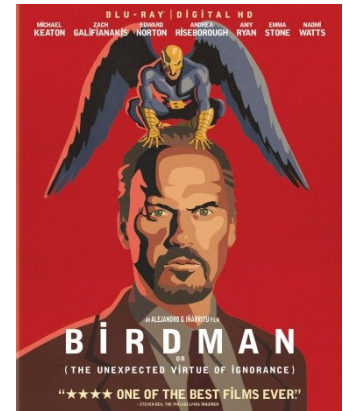
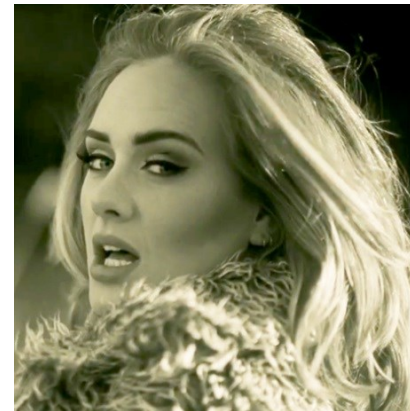
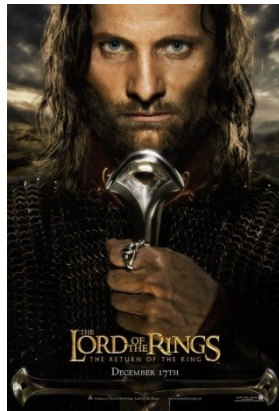
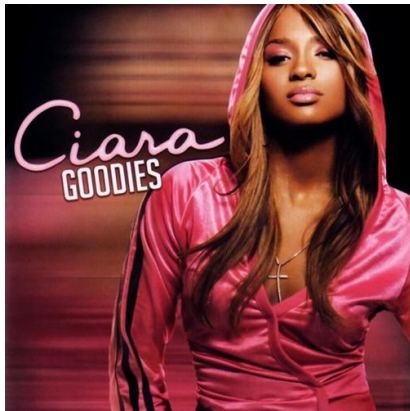
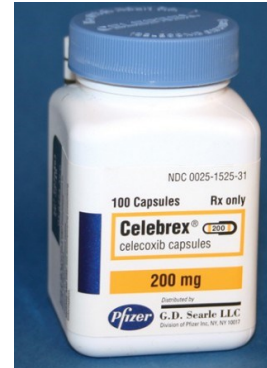
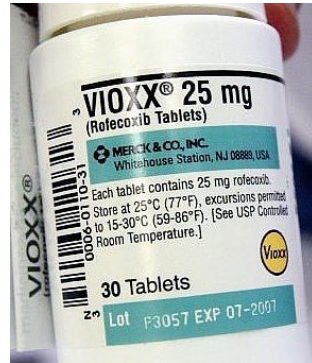
- Side Effects: GI upset
dizziness
- Caution:
renal dysfunction
CV disease, heart failure

Generic	Brand
Celecoxib	Celebrex
Diclofenac	Cataflam, Voltaren (topical)
Etodolac	Lodine
Ibuprofen	Motrin, Advil
Indomethacin	Indocin
Ketorolac	Toradol
Meloxicam	Mobic
Naproxen	Naprosyn

NSAID Selectivity



Are We Still Talking About CV Risk?



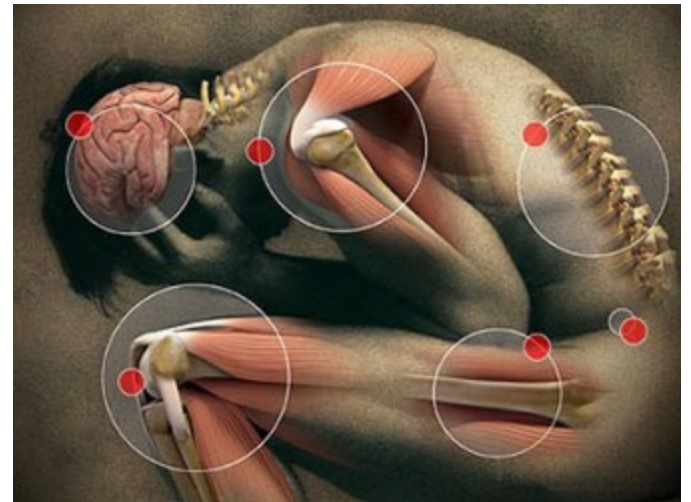
Antidepressants

Drugs that affect serotonin (5-HT) and norepinephrine (NE) receptors

Older SSRIs	SNRIs	TCAs
Fluoxetine (Prozac)	Duloxetine (Cymbalta)	Amitriptyline (Elavil)
Sertraline (Zoloft)	Milnacipran (Savella)	Desipramine
Paroxetine (Paxil)	Venlafaxine (Effexor XR)	Nortriptyline (Pamelor)
	Desvenlafaxine (Pristiq)	

FDA Indications

- Duloxetine: diabetic neuropathy, fibromyalgia, chronic pain
- Milnacipran: fibromyalgia
- Venlafaxine: diabetic neuropathy, cancer pain, migraine/HA prophylaxis



Side Effects



- SSRIs: nausea, vomiting, diarrhea, sexual dysfunction, headache
- SNRIs: nausea, sexual dysfunction, increase in diastolic blood pressure, sweating
- TCAs: sedation, dry mouth, weight gain, constipation, blurred vision



Dr. Feelgood

FDA indications = more evidence

May increase dose of older SSRIs instead of switching drugs

Bad Medicine

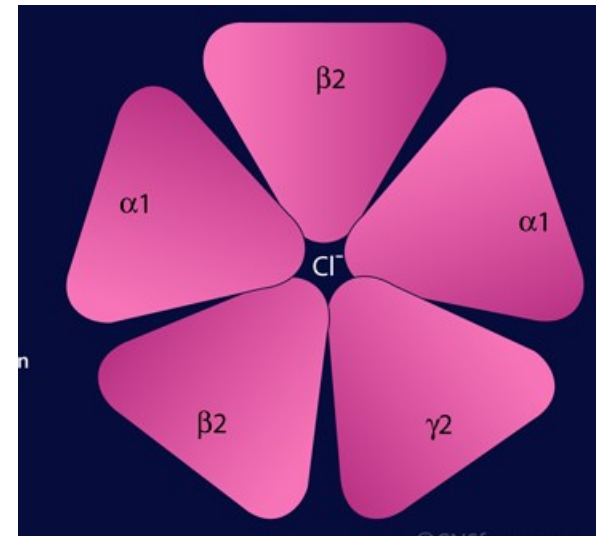
**Placebo-controlled,
Short term trials**

SNRIs/TCAs: higher doses = more side effects

Brain Break

True or False

Gabapentin and pregabalin work on the GABA receptors in the brain.



Anticonvulsants for Pain

- Gabapentin (Neurontin, Gralise) & pregabalin (Lyrica)
- FDA indications:
 - Gabapentin: postherpetic neuralgia
 - Pregabalin: neuropathic pain, fibromyalgia, postherpetic neuralgia
- Side Effects: nausea, vomiting, fatigue, dizziness, dry mouth
- Titrate slowly
- Do not stop suddenly



Other Anticonvulsants

- Carbamazepine, oxcarbazepine: trigeminal neuralgia
 - Side Effects: nausea, constipation, dry mouth, rash
- Topiramate: migraine prophylaxis
 - Side Effects: weight loss, dizziness, memory impairment (“Dope-amax”)

Generic	Brand
Carbamazepine	Tegretol
Oxcarbazepine	Trileptal
Topiramate	Topamax

“I Don’t Have Seizures”

- Patient education is key for drugs with multiple indications
- “What did your doctor tell you this was for?”



OTC Topicals



- Capsaicin: desensitizes sensory nerves over time
 - Caution when applying- wear gloves, wash hands
 - Use 3-4 times per day on affected area
 - May cause burning sensation which fades over time
- Menthol/methyl salicylate (Bengay cream, Salonpas patches)
 - Don't apply heat to the area
 - Menthol causes cooling sensation

Prescription Topicals

- Lidocaine patch:
 - Wear for 12 hours, remove for 12 hours
 - May cut patches
- Diclofenac (Voltaren):
 - Apply up to 4 times daily
 - Do not apply heat or cover area with bandages
 - Carries same Black Box warning as oral NSAIDs



Skeletal Muscle Relaxants

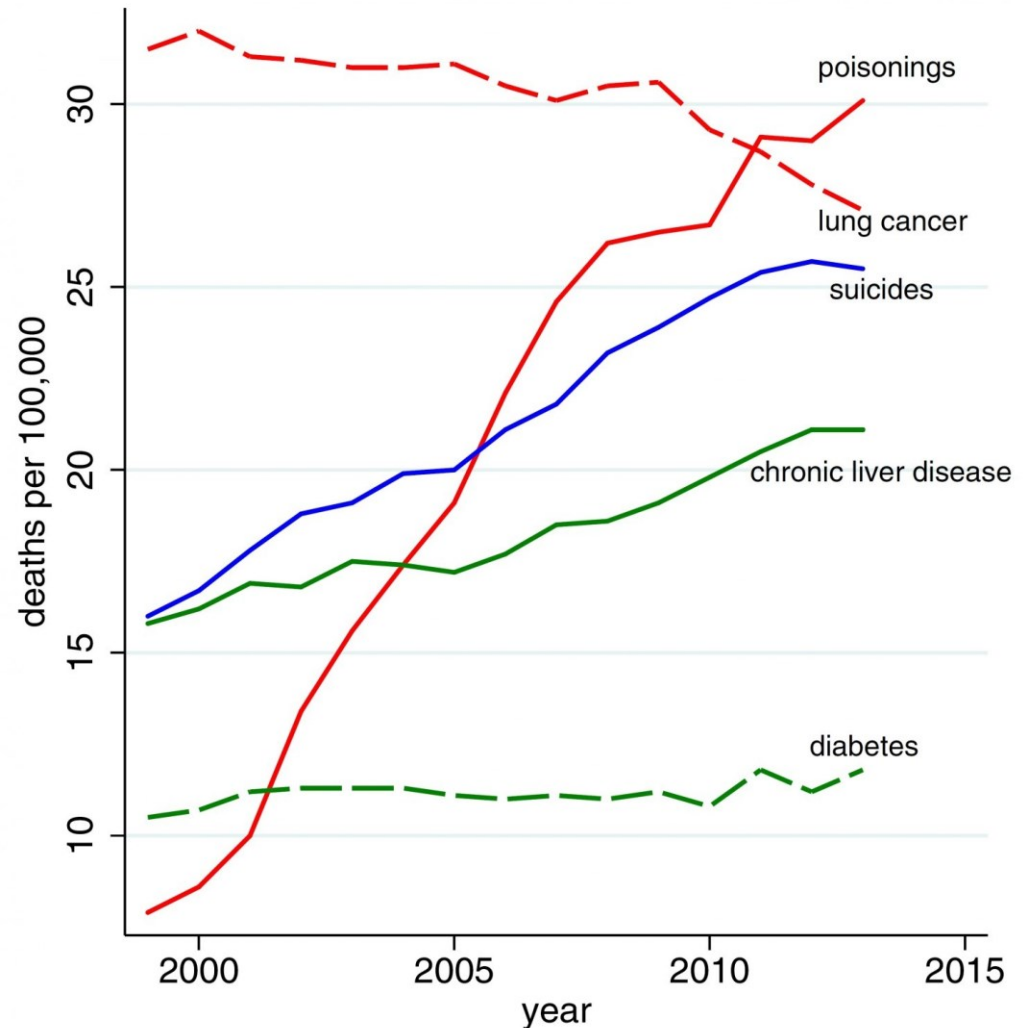
- Work in CNS to relax muscles
- Side Effects: dizziness, drowsiness, dry mouth, confusion, GI
- Cyclobenzaprine: evidence in fibromyalgia

Generic	Brand
Baclofen	Lioresal
Carisoprodol	Soma
Cyclobenzaprine	Flexeril
Methocarbamol	Robaxin
Tizanidine	Zanaflex

Opioids, Opiates, Oh My!

“A group of middle-aged whites in the U.S. is dying at a startling rate”

-Washington Post, 11/2/15



Opioid Receptor Subtypes

Receptor Subtype	Effects
Mu1	Euphoria, supraspinal analgesia, confusion, dizziness, nausea
Mu2	Respiratory depression, CV effects, GI effects, miosis, urinary retention
Delta	Spinal analgesia, CV depression, decreased brain and myocardial oxygen demand
Kappa	Spinal analgesia, dysphoria, psychomimetic effects, feedback inhibition of endorphin system

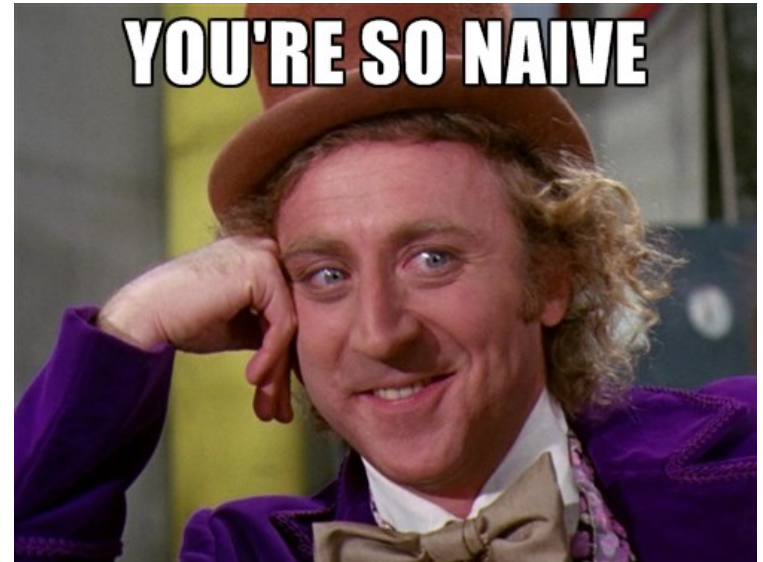
Opioid Analgesics

- All Schedule II except:
 - Tramadol (Sched IV)
 - < 90mg codeine
- Limit 30 day supply with no refills

Generic	Brand
Codeine	
Fentanyl	Duragesic
Hydrocodone	Vicodin, Norco, Lortab
Hydromorphone	Dilaudid
Meperidine	Demerol
Methadone	Dolophine
Morphine	MS Contin, Kadian, Oramorph
Oxycodone	Oxycontin, Percocet
Oxymorphone	Opana
Tramadol	Ultram

Don't Be “Naïve”

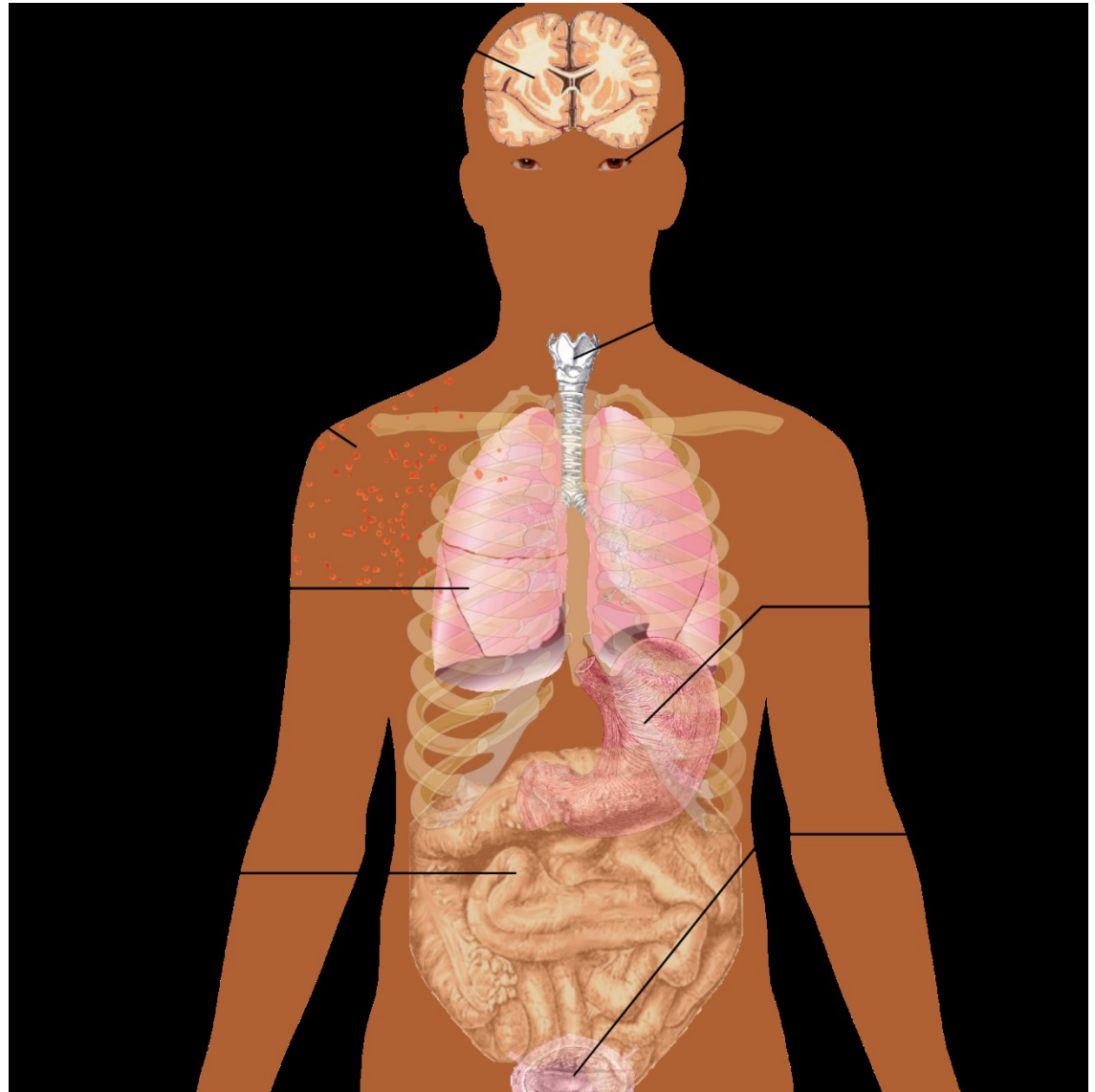
- Opioid-naïve:
 - < 60mg daily morphine equivalent dose (MED)
 - < 1 week therapy
- Start low and go slow
- Reserve long-acting opioids for around the clock pain management
 - Oxycodone CR, MS Contin, Methadone, Fentanyl Patch
- Watch total APAP daily dose
 - Vicodin, Percocet: 325mg APAP



Onset and Duration of Action

- Short-acting or immediate release
 - Hydrocodone, hydromorphone, morphine, oxycodone, oxymorphone
 - Onset: ~15 to 60 min
 - Duration: 4 to 6 hours
- Long-acting or extended/controlled release
 - Oxycodone CR, MS Contin, Methadone, Fentanyl Patch
 - Onset: ~30 to 90 min
 - Duration: 8 to 12 hours (oral), 72 hours (Fentanyl)

Side Effects



How to Manage Constipation

- Most common side effect
- Does not fade over time
- “Push and Mush” treatment
 - Laxative (senna) + stool softener (docusate)
- Naloxegol (Movantik): new Mu receptor antagonist
 - Does not cross blood-brain barrier
 - Placebo-controlled trials
 - No evidence with conventional treatment



Tolerance to Side Effects

- Nausea/vomiting
 - Most patients develop tolerance
 - Treat with ondansetron or prochlorperazine PRN
- Itching
 - Most patients develop tolerance
 - Cautiously consider diphenhydramine PRN
- Sedation
 - Most patients develop tolerance
 - Decrease opioid dose or change opioids



Pop Quiz

What is the source of the following information?

“Prescriptions for [opioid] drugs have climbed 300 percent in the last decade or so. In fact, Vicodin and other hydrocodone-combination painkillers are the most commonly prescribed drugs in the U.S.”



Patient Education

- Don't drive or operate heavy machinery
- Don't drink alcohol
- Difficulty breathing
→ call doctor or go to ER
- Fentanyl patch
 - Remove old patch before applying new one
 - Apply patch intact (do not cut)
 - May use tape around the edges of patch to hold in place
 - Do not apply heat
 - To discard, fold in half and flush down the toilet



**Take Control
of Your Pain**
Become Educated
and Empowered

Higher Risk Patients

- Sleep apnea
- Kidney dysfunction
- Use of CNS depressants → benzodiazepines, sedatives
- History of alcohol or drug abuse



Opioid Conversion

- General method
 1. Calculate 24-hr opioid requirement (ER + IR)
 2. Convert to equianalgesic oral dose using opioid conversion chart or calculator
 3. Consider 50% dose decrease for cross-tolerance
- Methadone and Fentanyl have separate conversion tools



New Formulations

- Zohydro ER: long-acting hydrocodone
 - No abuse deterrent
- Hysingla ER: long-acting hydrocodone
 - Forms thick gel if crushed
- Targiniq ER: oxycodone + naloxone



Dependence vs Addiction

- **Dependence:** stopping the drug will cause physical withdrawal; body has adapted to having drug around
 - Physiologic response
 - Anxiety, hypertension, intense pain, diarrhea
- **Addiction:** cravings or compulsive drug use despite harm or negative effect on life



PDMP

- Prescription Drug Monitoring Program
- Oregon pharmacies submit prescription data for controlled substances within 72 hours
- Healthcare providers may apply for access limited to patients under their care

Patient Case

SR is a **27-year old female** who recently underwent ORIF surgery for a fractured left ankle during a roller derby bout.

Allergies: NKDA PMH: not significant

Medications: APAP 650mg PRN for pain after roller derby practices

Which of the following is an appropriate post-op pain management regimen for SR?

- A. Fentanyl patch 50mcg every 72 hours
- B. Oxycontin 30mg every 12 hours + Oxycodone IR 15mg every 4 hours PRN
- C. Hydrocodone/APAP 5mg/325mg every 4 hours PRN
- D. None of the above

Tapering Opioids

- Slower taper for long-acting opioids & stable patient
- Faster taper for short-acting opioids & less stable patient
- Short-acting opioids → 10% every 3 days
- Long-acting opioids:
 - 10% of initial total dose until at 30% of initial dose
 - Then 10% of current dose
- Manage symptoms with antidepressants, NSAIDs, clonidine, anti-nausea, anti-diarrhea

Take Home Points

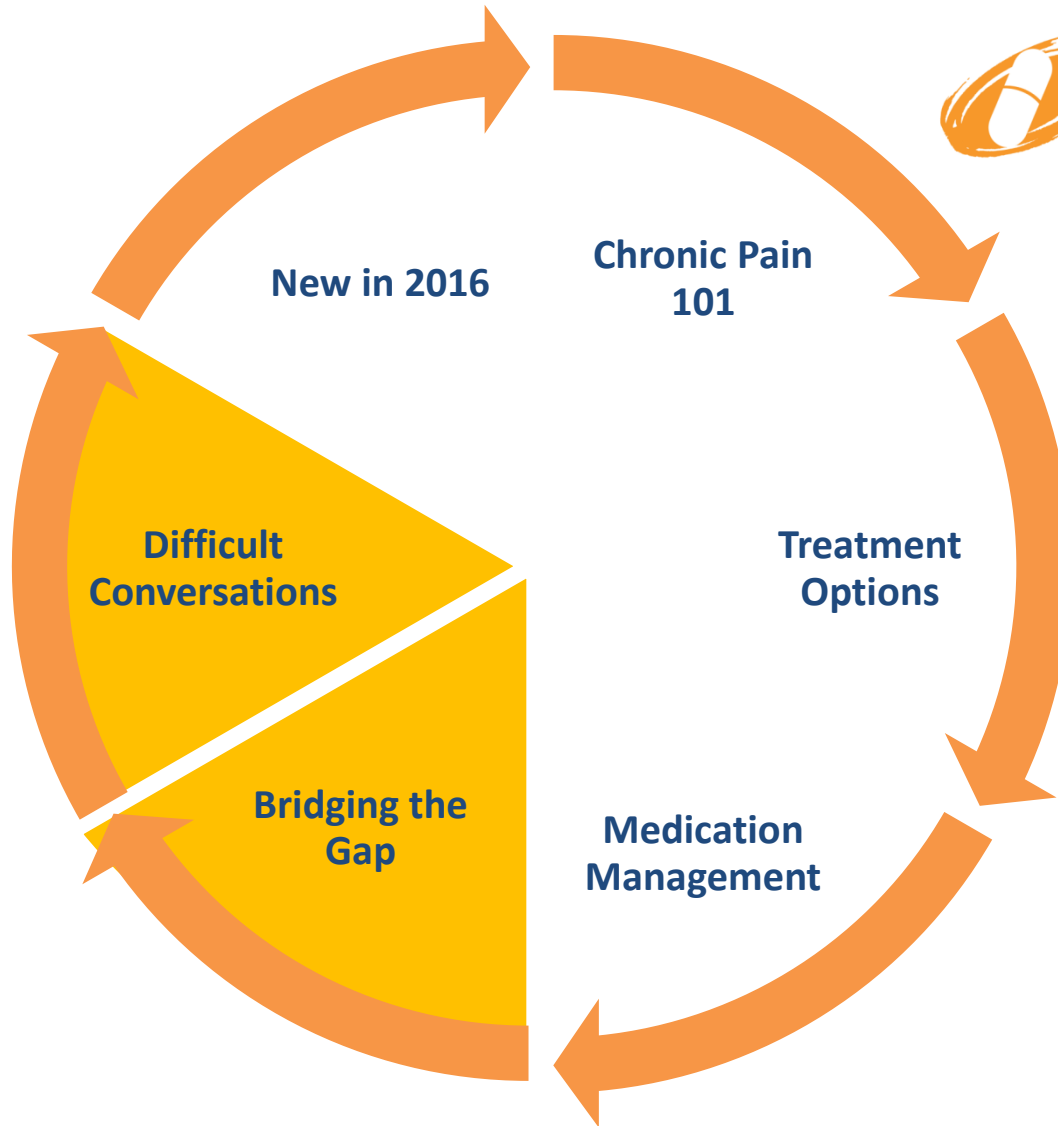
- Opioids are last resort options for chronic, non-cancer pain
- Low doses, short courses
- “Don’t be naïve about opioid-naïve patients”
- Ensure side effects are appropriately managed

Please hold questions – thanks!





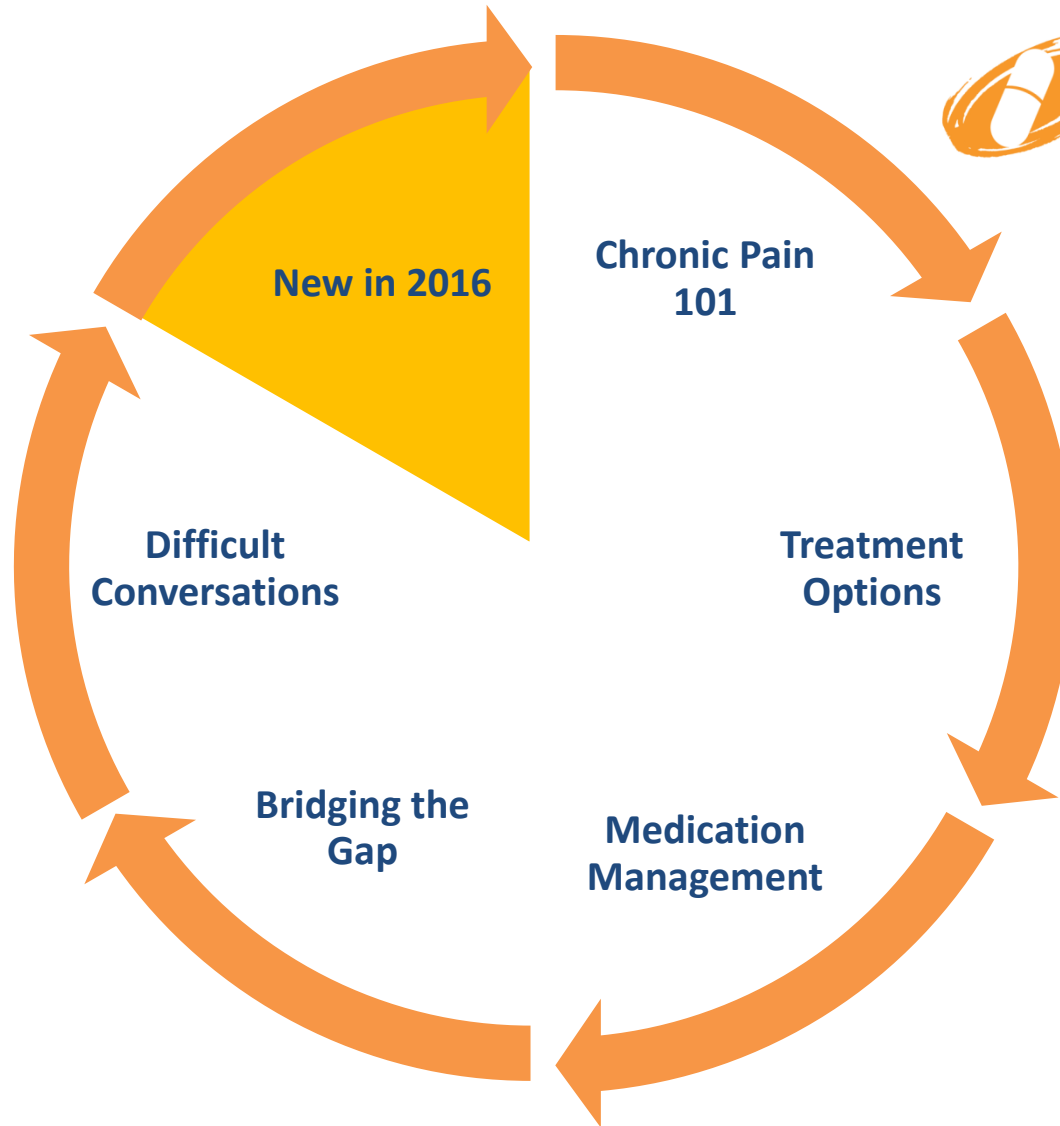
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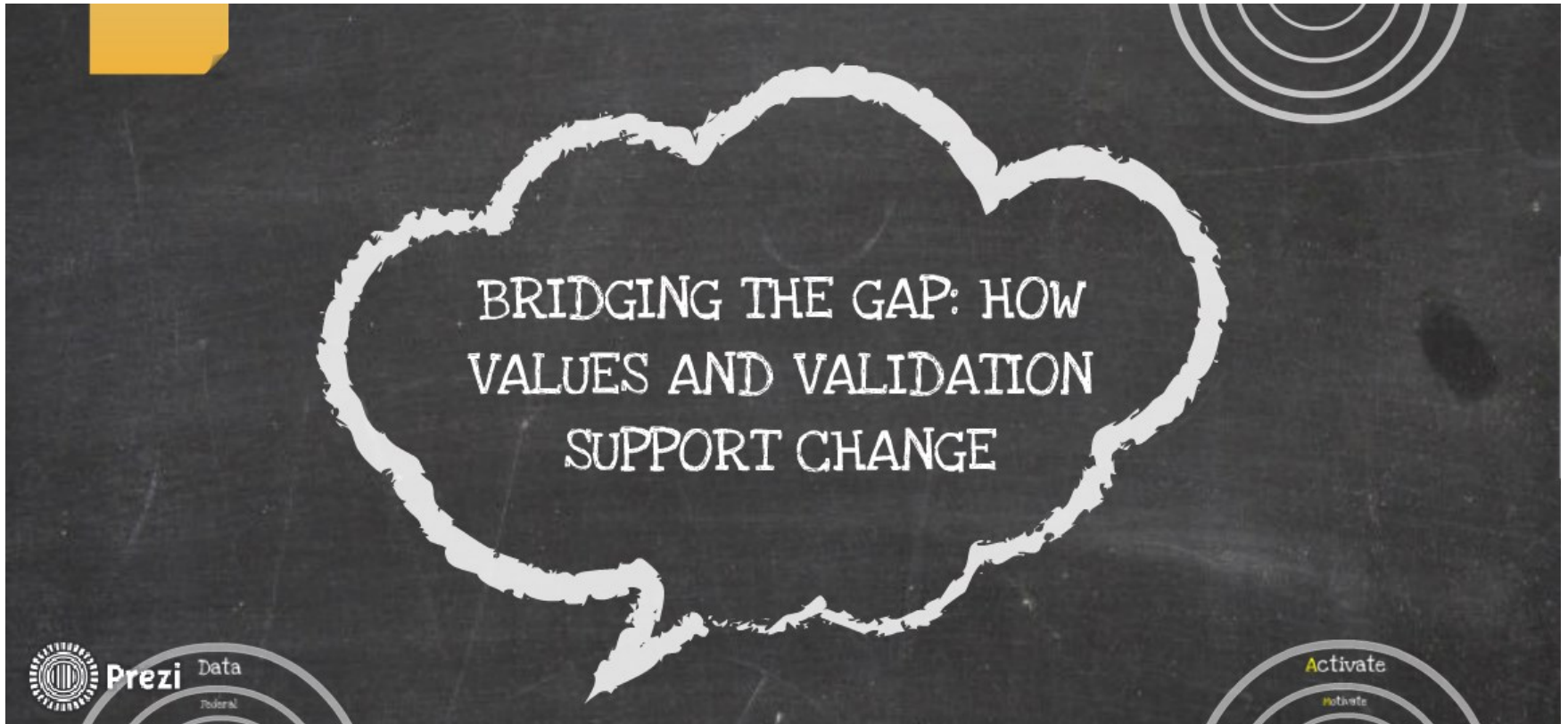
Bridging the Gap: How Values and Validation Support Change

Nadejda Razi-Robertson, LCSW
Behavioral Health Consultant





https://prezi.com/ynnibxh8nneq/bridging-the-gap-2/?utm_campaign=share&utm_medium=copy



CareOregon Metro's Approach to Pain Management and Substance Use Disorders

Rachel Solotaroff, MD, MCR

Central City Concern

Chair, CareOregon Metro

Chronic Pain/Chemical Dependency Task Force



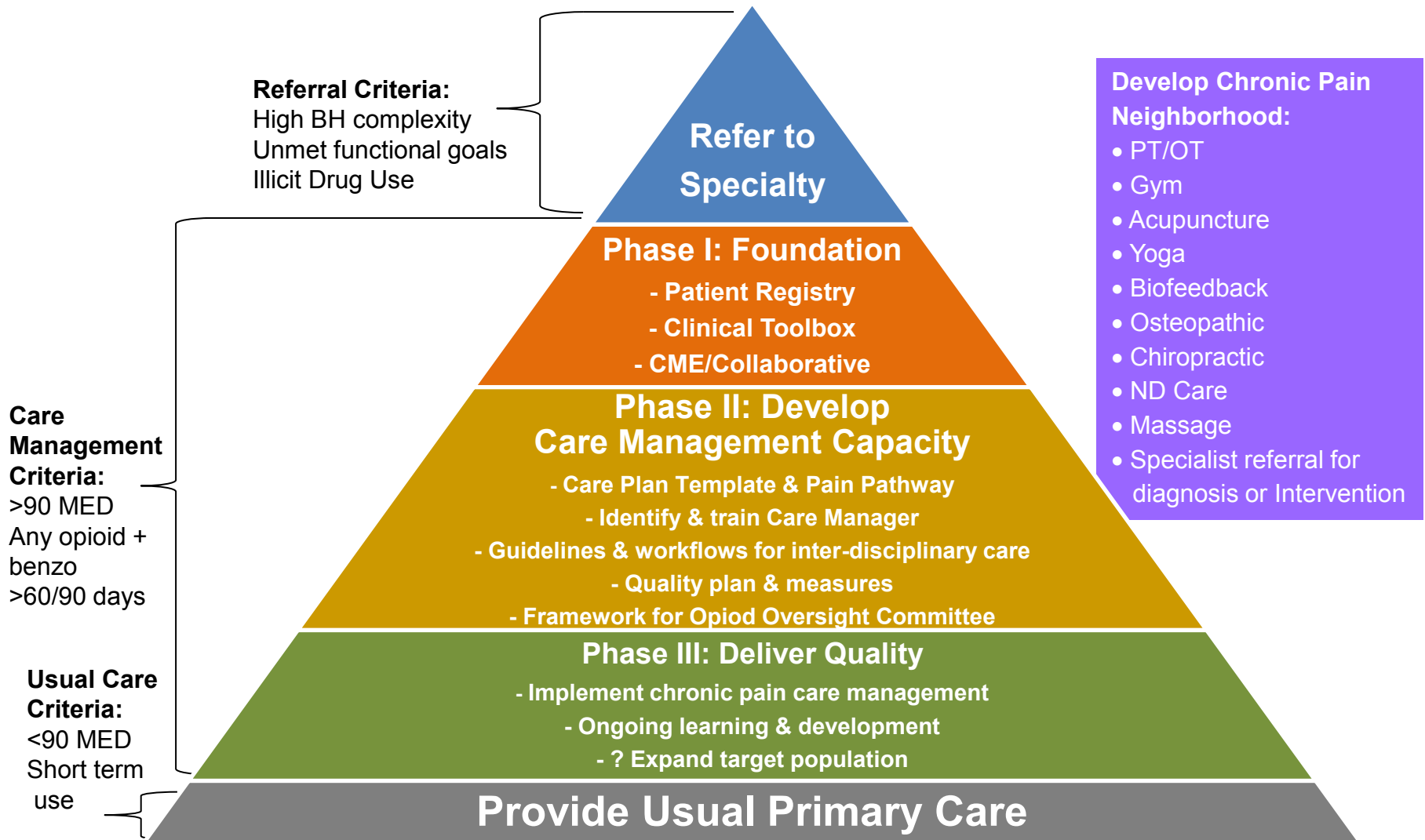
Overview

- Goal of CPCD Task Force is to formulate interventions to support populations with chronic pain, substance use disorders, or both.
- Theme: no single intervention could be called upon as “the solution”; instead, focus on engaging patients at their own stage of change and readiness.
- Focus on patient- and population-centered care.
- Spirit of the recommendations is that positive patient experiences and improved clinical outcomes will be accomplished by providing low barrier, compassionate, relationship-based interventions that exist in a seamless care continuum.

4 Components of Recommendations

1. Design and implement chronic pain care management initiative for primary care clinics
2. Expand access to Medication Assisted Treatment (buprenorphine and Naltrexone XR) in primary care and specialty addictions settings.
3. Expand access to chronic pain supportive and specialty services, including acupuncture
4. Partner with Health Share of Oregon to continue and enhance CME offerings for chronic pain and addiction.

Chronic Pain Care Management Model



Team Members

- **Care Manager (1 FTE)** – focuses on identifying patients via registry, outreach and engagement with patient, co-development of care plan in partnership with patient and team, and ongoing coaching/support to patient. Strong emphasis on building patient's motivation to engage in program.
- **Primary Care Champion (0.1 FTE)** – expands skill level in knowledge of chronic pain neurobiology, treatments, evidence of opioid harms. Serves as champion and thought leader of these concepts in primary care clinic. Staffs inter-disciplinary care team meetings, communicating with other providers regarding patients and care plans
- **Behavioral health Clinician (0.25 FTE)** – provides evidence-based therapies to support patient in psychosocial management of pain, shift in focus from pain to function, readiness for other treatments (such as physical therapy or acupuncture), and crisis/anxiety management regarding issues such as pain flares and fear of decreased opioid dosages
- **Pharmacist (0.1 FTE)** – assists care team with difficult questions surrounding medication tapers, conversions, and interactions

Next Session



February 25th, 2016

Questions?



Summary

Charmian Casteel, RN

Primary Care Innovations Specialist

CareOregon



Thank you!