Addiction 101:
what neurobiology teaches us about long-term opioids on the brain, and how it changes our approach to treatment

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Disclosures

- Camden Coalition-Salary
- RWJF- funding for website development
- Nothing else to disclose

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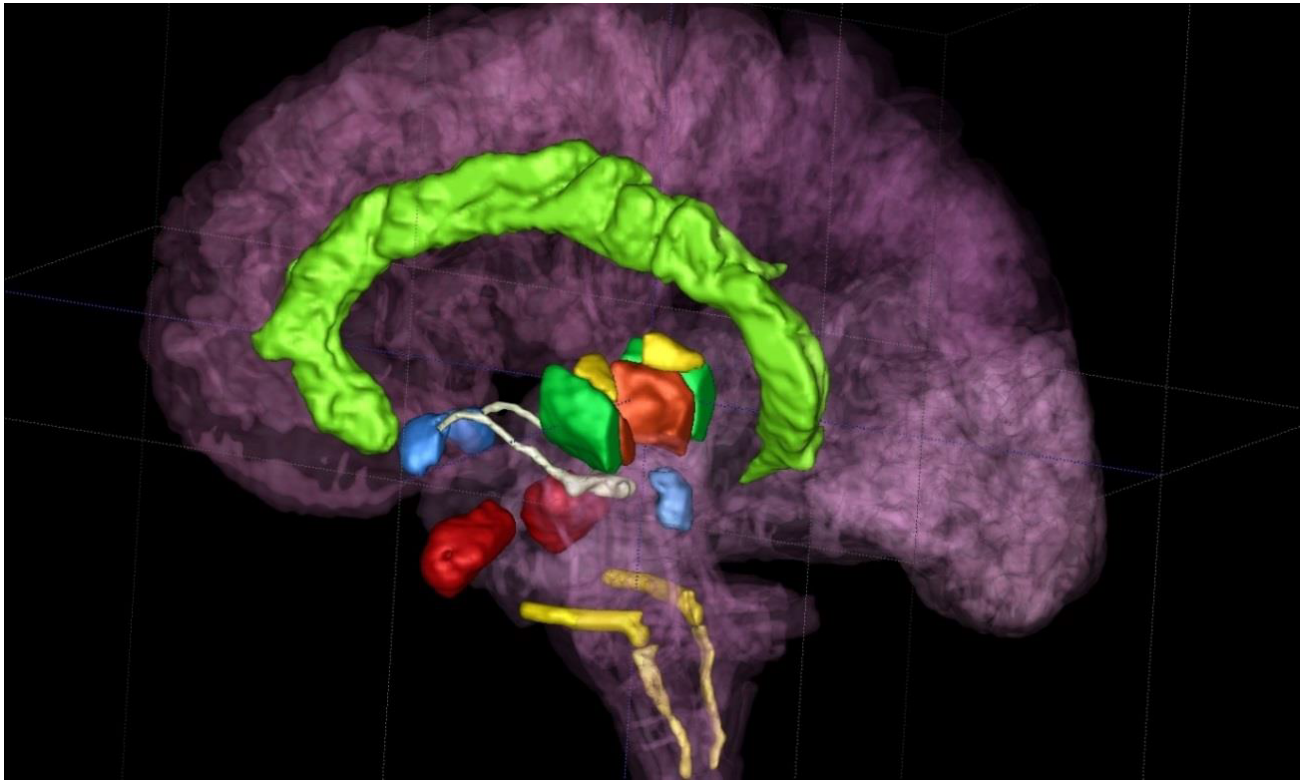
Objectives

By the end of the lecture the learner will:

- Understand the brain's approach to pain
- Understand how opioids affect this approach
- Understand how chronic opioids can lead to addiction
- Understand the neurobiological overlap between pain and addiction

Neurobiology of Pain



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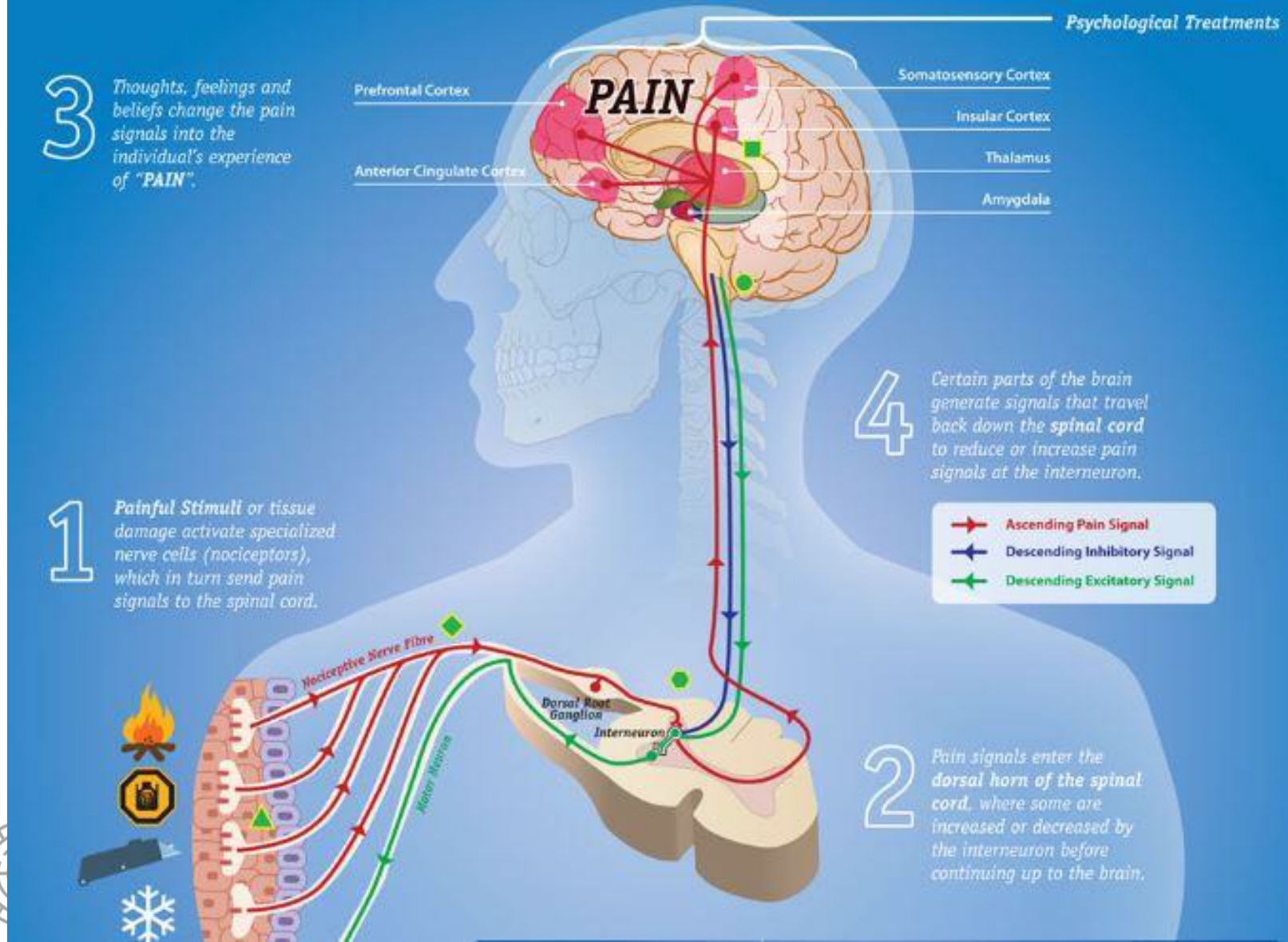
painexplained

Pain Pathways and Medications



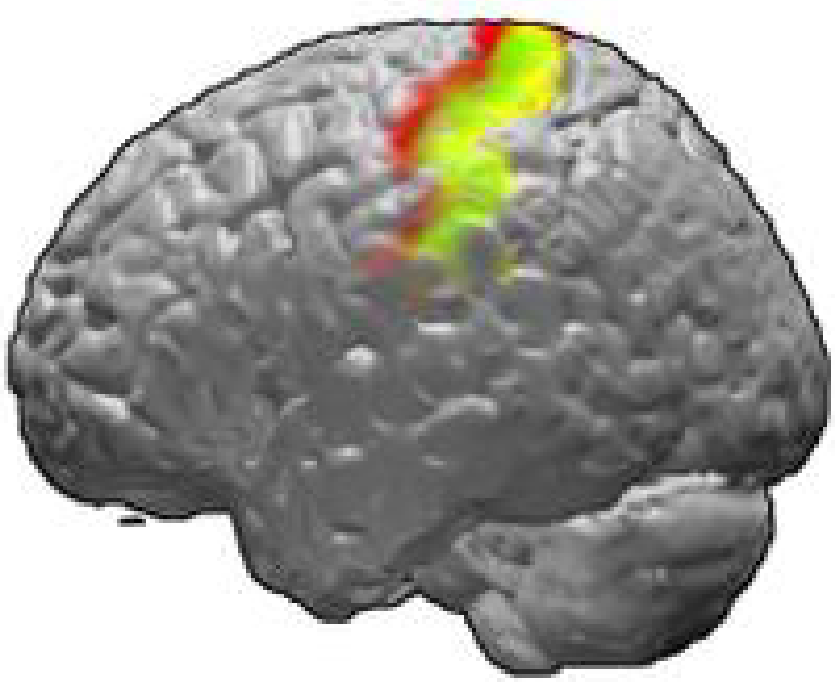
3 *Thoughts, feelings and beliefs change the pain signals into the individual's experience of "**PAIN**".*

4 *Certain parts of the brain generate signals that travel back down the **spinal cord** to reduce or increase pain signals at the interneuron.*

1 ***Painful Stimuli** or tissue damage activate specialized nerve cells (nociceptors), which in turn send pain signals to the spinal cord.*

2 *Pain signals enter the **dorsal horn of the spinal cord**, where some are increased or decreased by the interneuron before continuing up to the brain.*

Somatosensory Cortex



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Normal response with “high/average pain tolerance”

- Acute on chronic pain (twisting a chronically painful back)
- Emotionally assess, and if all good, then,
- Increase descending inhibition
- Thus decreasing the ascending pain signal
- All happening while we produce our own endorphins from the dorsal horn and the periaqueductal grey (PAG)

- This equals less pain and greater function

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Normal response with “low pain tolerance”

- Acute on chronic pain (twisting a chronically painful back)
- Emotionally assess -- and not all good
- Increase in descending excitatory pathway
- Decrease in inhibitory pathway
- Increase in perceived pain followed by hysteria and tachypnea
- This changes the pH in the serum and thus increases the amount of endorphin released in response from PAG and Zone II and III of the dorsal horn
- Then after the panic-like state, pain normalizes

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When opioids are added

- Decreased production of endogenous opioids
- Body “ramps up” pain signal frequency
- Thus greater signal from ascending tracks (spinothalamic, Spinoreticular and Spinomesencephalic)
- More pain in widened area
- Decreased endorphin production from PAG and Dorsal Horn
- Worsened sleep patterns
- More emotional lability from opioid effects in limbic system

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So...

- Emotionally assess: and all good or not all good?
- Increase in descending excitatory pathway to overcome outside opioids
- Decrease in inhibitory pathway given presence of opioid
- Increase in perceived pain followed by hysteria (tachypnea blocked by opioids)
- So no change in the pH in the serum and thus no increase in the amount of endorphin released from PAG and Zone II and III of the dorsal horn
- Then after the panic like state, pain continues and in many cases widens in area and intensity from increased c-fiber signal

Common Pain Behaviors

- Behavior changes
- Medication “issues”
- Physical characteristics

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Pain Behaviors

- Rapid escalation or changes in mood
  - -Emotions going from 0 to 100 quickly, without much awareness or control.
  - -This may be anger, crying, anxiety etc.
  - -Low emotional distress tolerance
  - -Irrational thinking or behaviors
  - -Excuses Excuses Excuses
- Remember behavior is a symptom. Do not take it personally

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When opioids are stopped

- Return to baseline
- But first, everything hurts worse
- Emotions are more distinct
- Fear of pain creates significant anxiety

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Medication Issues:

- Needing early refills on prescriptions
- Seeing several doctors or going to the ED for meds
- Taking more than prescribed dose
- Abnormal attachment to medications
- Buying meds “on the street”

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Setting Expectations?

- Get rid of all your pain?
- Make you forget you have pain?
- Decrease your pain and improve your function!

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Pain vs Suffering *(ie. pain behaviors)*

- Intense emotional reaction to pain
- “This is the worst pain ever, 10/10 pain”
- Rapidly escalating behaviors

- *Not everyone with pain has pain behaviors or suffering, why is that?*

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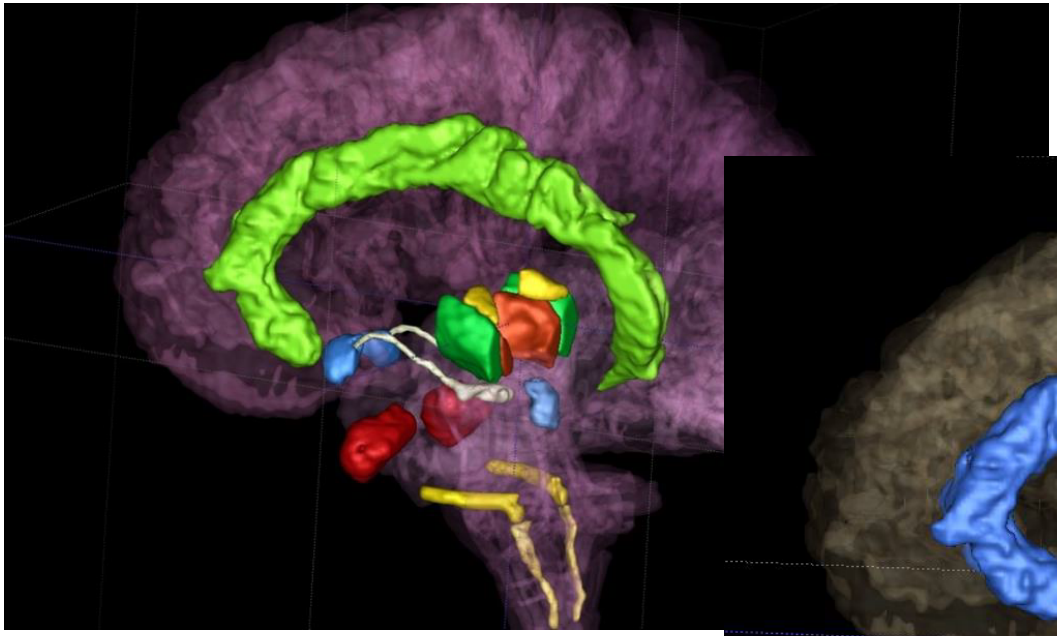
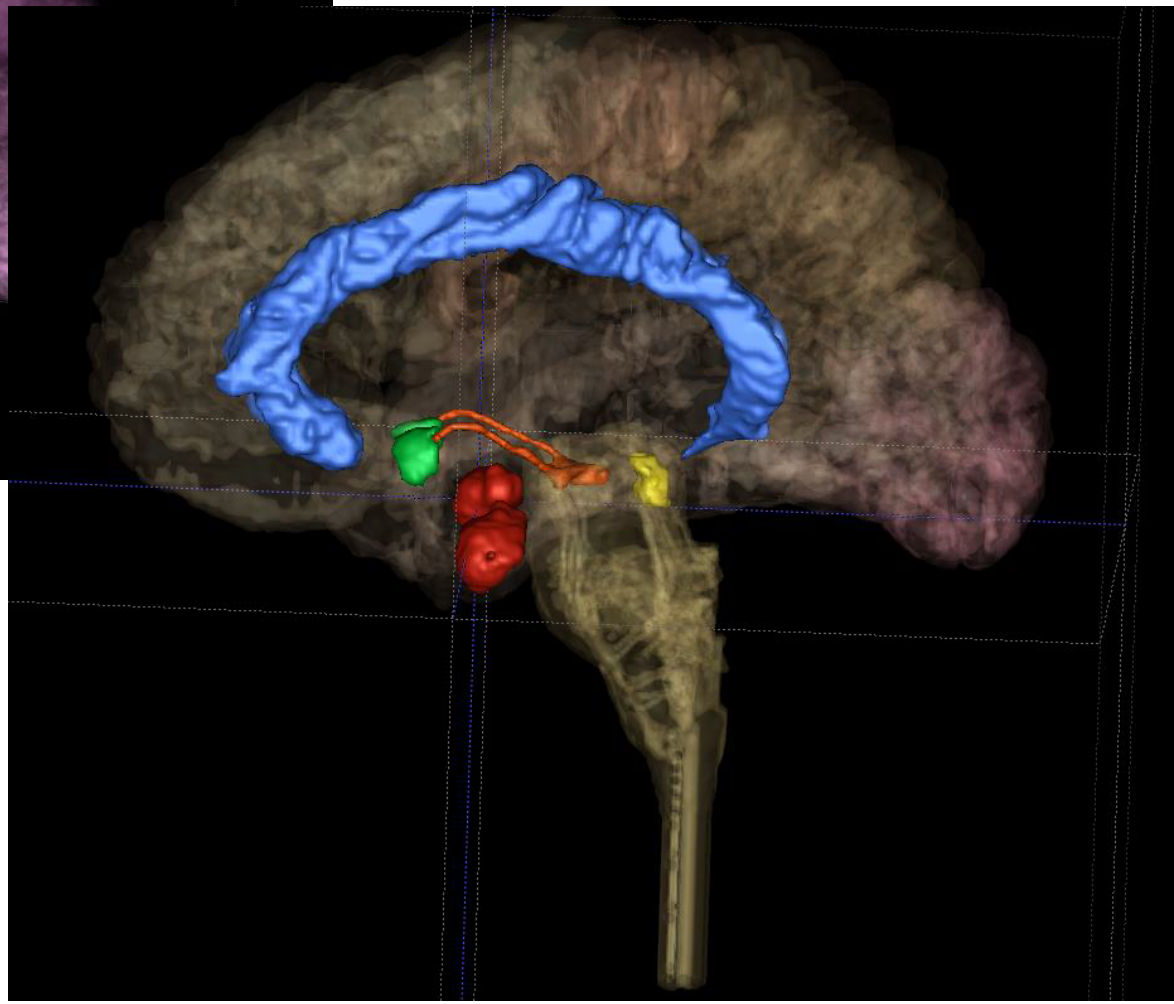
Neurobiology of Addiction



Pain

Addiction



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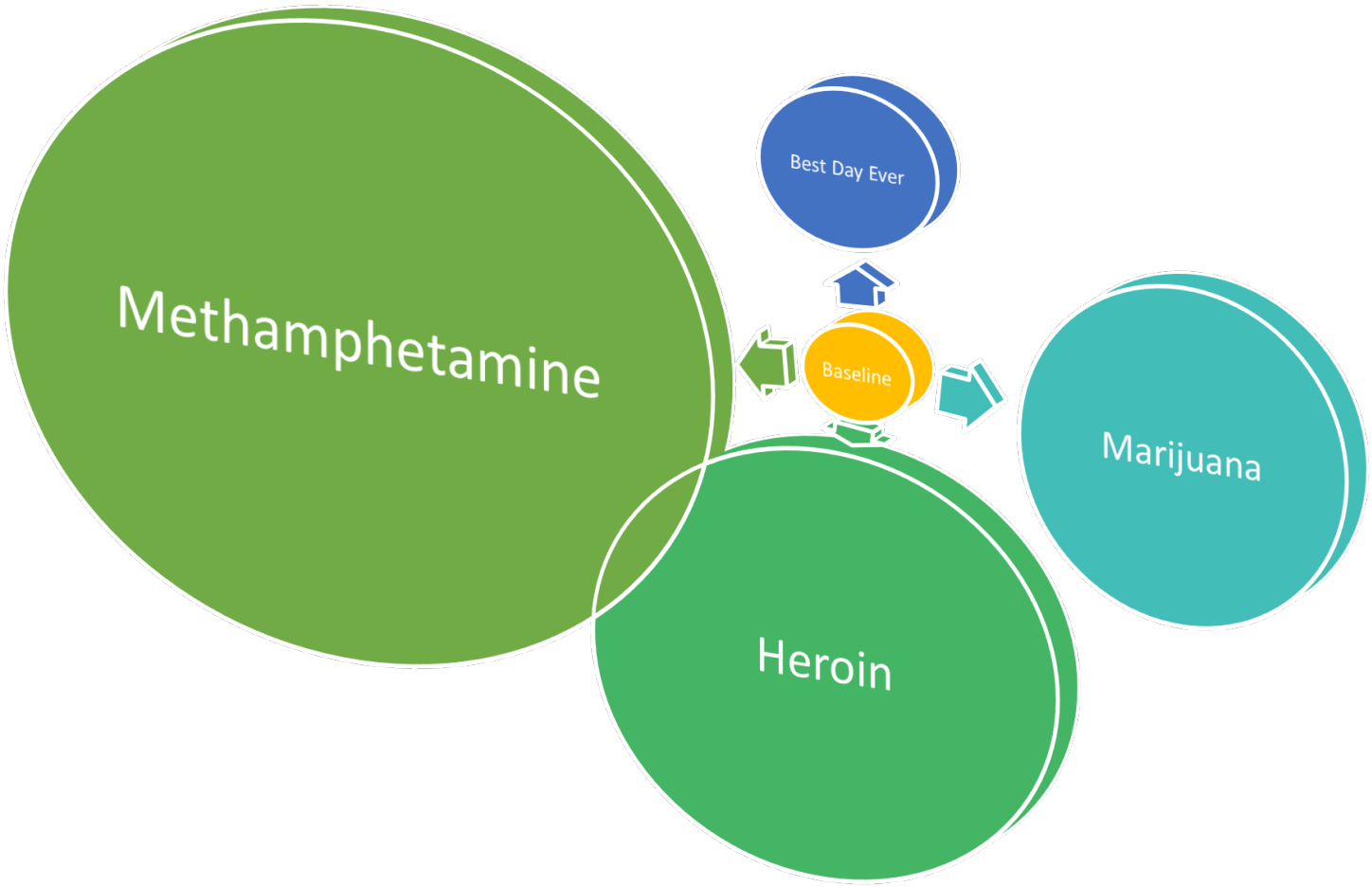
Pain to Addiction

- When there is no pain the PAG doesn't "filter" the dopamine bump at the nucleus accumbens and the ventral tegmental area
- This starts to effect the reward axis
- Leads to typical addiction neurobiology



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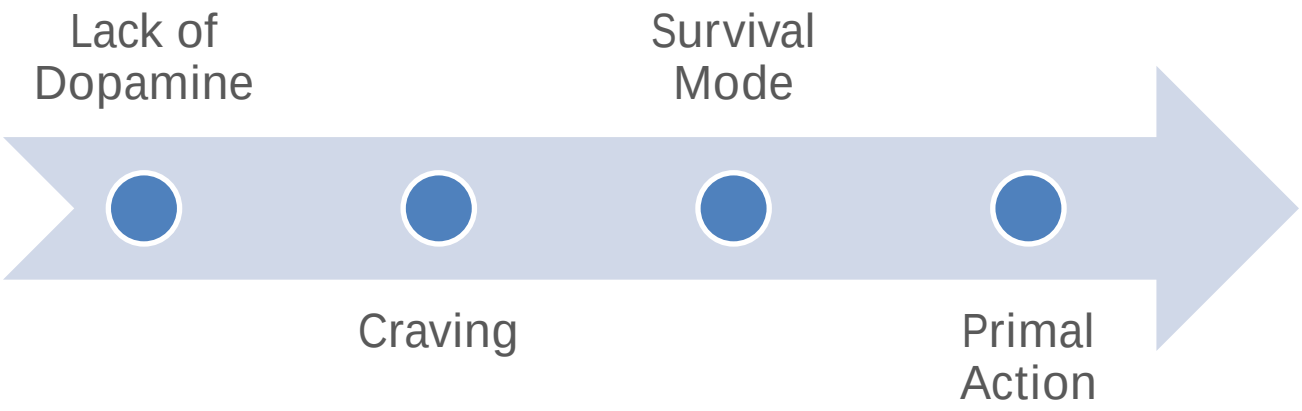
Behavior



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- Notice any similarities?

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Conclusions

- The overlap between pain and addiction is a huge grey area
- Many ways they are exactly the same
- Chronic opioids exacerbate the risk even in “real” chronic pain states
- Thus low-to-no opioids are now becoming the best practice

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References

- Principles of neuroscience, 5th edition Hudspeth, Kandel, Jessell and Siegelbaum (2013)
- The Neuron, cell and molecular biology, 3rd edition; Levital and Kaczmarek (2012)
- Principles and Practice of Pain Medicine 2nd edition, Warfield and Bajwa

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