



MRI – ~~Nuclear~~ Magnetic Resonance Imaging – Produces a 3-d image inside the body.

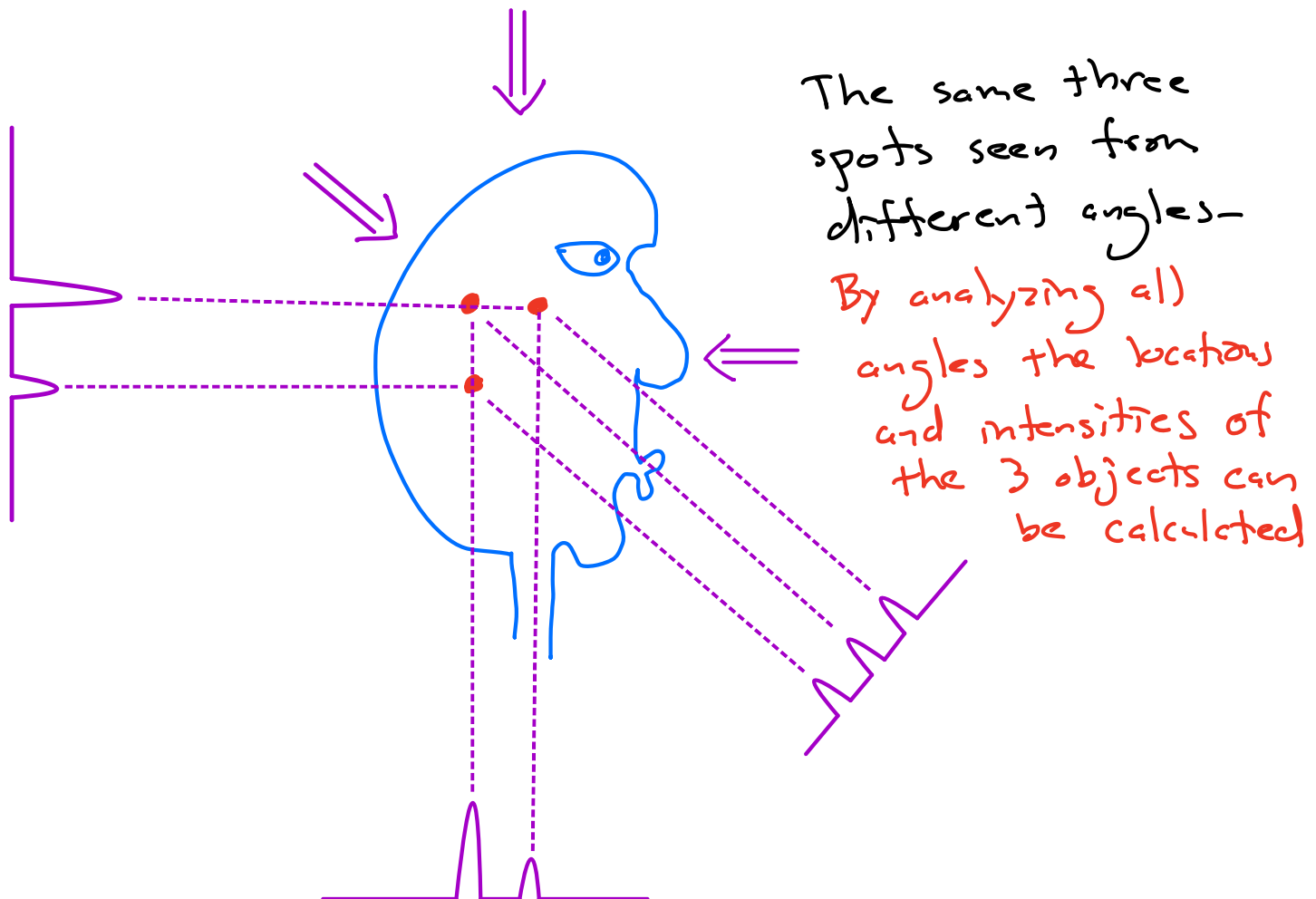
MRI is similar in approach, but complementary to, a CAT scan, which uses X-rays for imaging.

MRI is therefore safer than a CAT scan (no X-rays or other damaging radiation is used). Radiofrequency electromagnetic radiation does not cause DNA damage or any other kind of damage.

MRI primarily visualizes soft-tissue and especially cancer tumors while a CAT scan primarily visualizes bones or Calcium based dyes drunk to visualize the digestive tract.

MRI uses the same principles and NMR.

- 1) The patient is placed in a very strong magnetic field. Creating this very strong magnetic field is technically very demanding, explaining MRI machines are so expensive (~ 0.5 – 1.5 \$ million)
- 2) The patient is irradiated with radiofrequency electromagnetic radiation.
- 3) The flipping (resonance) of ^1H nuclear spins is monitored – Actually emitted photons are measured using the FT method.
- 4) Magnetic field gradients are used to gain imaging information. The magnetic field gradients are rotated around a central point and measurements are taken at each angle around 360° to gain 2-dimensional information. This technique is called **tomography**.



The overall **MRI** imaging approach involves looking at each 2-dimensional slice. ✓

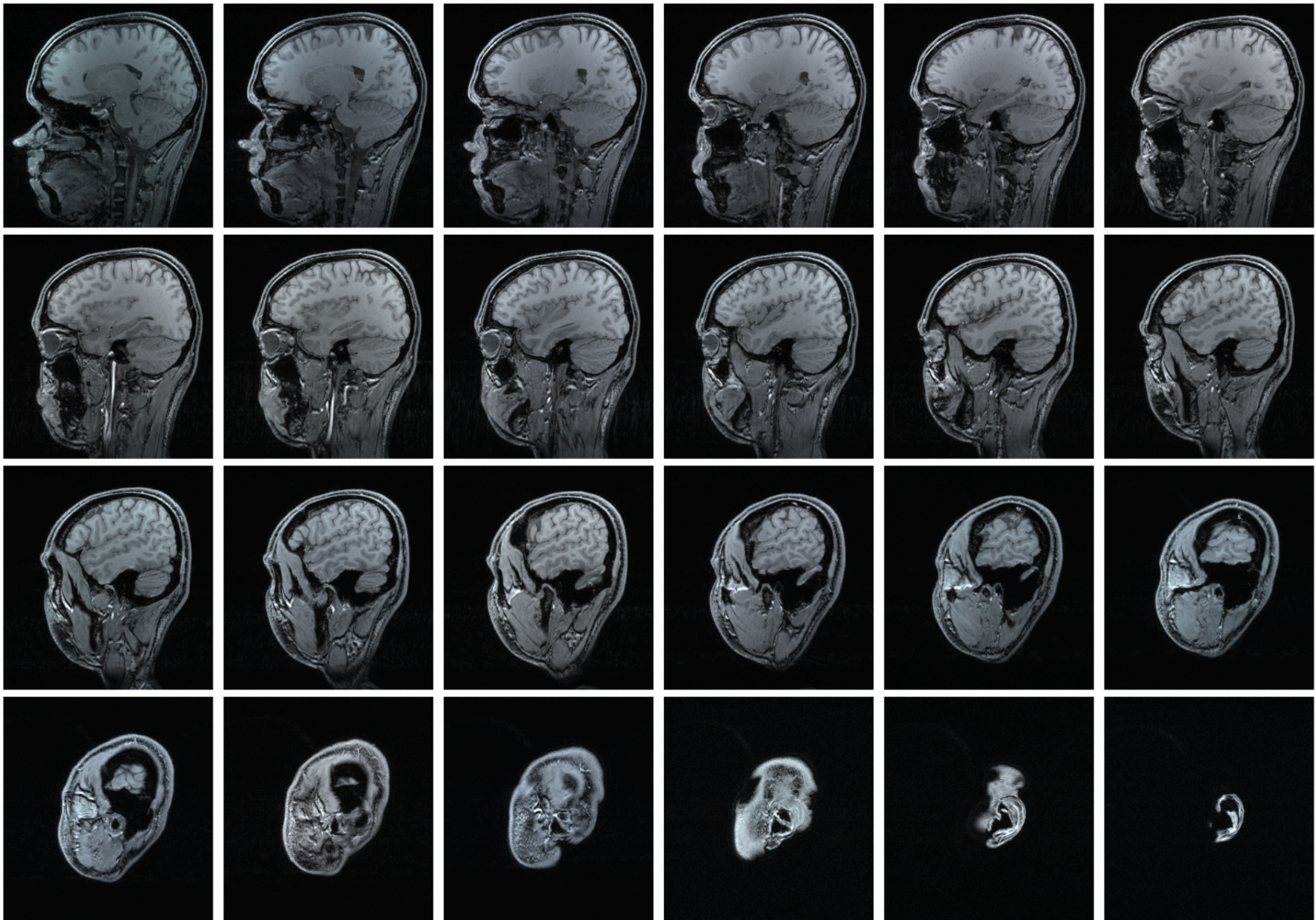
Each slice is added to give a 3-dimensional stack (analogous to stacking DVD's or CD's). ✓

Each slice is shaded to indicate differences in the amount of ^1H atoms in different areas/tissues.

Water and fat have the highest density of ^1H atoms, so these are primarily being monitored in an **MRI** image.

The popular medical diagnostic technique of **magnetic resonance imaging (MRI)** is based on **the same principles as NMR**, namely the **flipping (i.e. resonance) of nuclear spins of H atoms by radio frequency irradiation** when a patient is placed in **a strong magnetic field**. **Magnetic field gradients are used to gain imaging information, and rotation of the gradient around the center of the object gives imaging in an entire plane (i.e. slice inside patient)**. In an MRI image, you are looking at **individual slices that when stacked make up the three-dimensional image of relative amounts of H atoms**, especially the H atoms from **water and fat, in the different tissues** [Memorize the preceding passage, as it will be worth 14 points on the final. No I am not kidding, 14 points right there.]

↑
READ THIS



The Amide Bond Shows Up in Medical School
Monday, August 25

Hi Dr. Iverson,
I just wanted to share a little story from my first week of medical school:

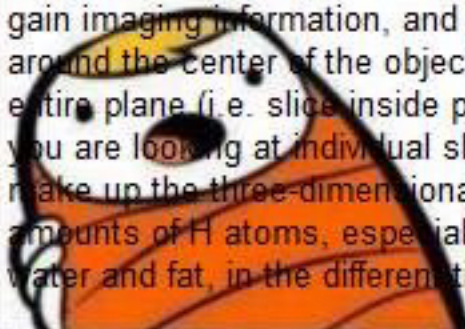
The other day in my biochemistry lecture, the professor mentioned factors that gave proteins stability, including the double bond nature of the peptide bonds. That idea sounded strangely familiar until I realized I'd already memorized it earlier when you made us write out all the contributing structures of amides on every exam!

In just the first week of med school I've had several moments like this where topics mentioned in your class have helped speed up my understanding (MRI, enzyme mechanisms, etc.). I never expected so many details of an organic chemistry course to play small but important roles in medical sciences.

Thank you for taking the time to make your class relevant for all the pre-health professions students in your class!



The popular medical diagnostic technique of magnetic resonance imaging (MRI) is based on the same principles as NMR, namely the flipping (i.e. resonance) of nuclear spins of H atoms by radio frequency irradiation when a patient is placed in a strong magnetic field. Magnetic field gradients are used to gain imaging information, and rotation of the gradient around the center of the object gives imaging in an entire plane (i.e. slice inside patient). In an MRI image, you are looking at individual slices that when stacked make up the three-dimensional image of relative amounts of H atoms, especially the H atoms from water and fat, in the different tissues.



HIV-1 protease: mechanism and drug discovery

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1 Introduction

It has now been two decades since acquired immunodeficiency syndrome (AIDS) was first reported by the US Center for Diseases Control (CDC). A few years later, it was found that a retrovirus called human immunodeficiency virus (HIV) is the causative agent in AIDS.¹ In a short time, AIDS increased to epidemic proportions throughout the world, affecting more than 40 million people today and killing so far more than 22 million (UNAIDS, 2001).

Since the outbreak of the AIDS epidemic, tremendous efforts have been directed towards development of antiretroviral therapies that target HIV type 1 in particular (HIV-1). The identification of the HIV retrovirus and the accumulated knowledge about the role of the different elements in its life cycle led researchers around the world to develop inhibitors that target different steps in the life cycle of the virus. One of these targets is HIV-1 protease (HIV PR), an essential enzyme needed in the proper assembly and maturation of infectious virions. Understanding the chemical mechanism of this enzyme has been a basic requirement in the development of efficient inhibitors. In this review, we summarize studies conducted in the last two decades on the mechanism of HIV PR and the impact of their conclusions on the drug discovery processes.

2 The life cycle of HIV

HIV belongs to the class of viruses called retroviruses, which carry genetic information in the form of RNA. HIV infects T cells that carry the CD4 antigen on their surface. The infection of the virus requires fusion of the viral and cellular membranes, a process that is mediated by the viral envelope glycoprotein (gp120, gp41) and receptors (CD4 and coreceptors, such as CCR5 or CXCR4) on the target cell. As the virus enters a cell, its RNA is reverse-transcribed to DNA by a virally encoded enzyme, the reverse transcriptase (RT). The viral DNA enters the cell nucleus, where it is integrated into the genetic material of the cell by a second virally encoded enzyme, the integrase. Activation of the host cell results in the transcription of the viral DNA into messenger RNA, which is then translated into viral proteins. HIV protease, the third virally encoded enzyme, is required in this step to cleave a viral polyprotein precursor into individual mature proteins. The viral RNA and viral proteins assemble at the cell surface into new virions, which then bud from the cell and are released to infect another cell. The extensive cell damage from the destruction of the host's genetic system to the budding and release of virions leads to the death of the infected cells.

3 HIV protease

3.1 HIV protease: a logical target for AIDS therapy

Unless the HIV life cycle is interrupted by specific treatment, the virus infection spreads rapidly throughout the body, which results in the weakness and destruction of the body's immune system. From the analysis of the HIV life cycle, one could conclude that there are several steps that might be interfered with,

thus stopping the replication of the virus. For example, there are several commercially available drugs that inhibit the enzyme reverse transcriptase (RT). The first class of RT inhibitors is the nucleoside analogs such as AZT, ddI, ddC and d4T. These dideoxy compounds lack the 3'-hydroxy, causing DNA chain termination when they are incorporated into the growing DNA strand. The second class of inhibitors is the non-nucleoside inhibitors (NNIs); these inhibitors are known to bind in a pocket away from the polymerase active site, and are believed to cause a conformational change of the enzyme active site, and thus inhibit its action. Currently, there are three available non-nucleoside reverse transcriptase inhibitors (nevirapine, delavirdine, and efavirenz) for the treatment of AIDS.

Another critical step in the life cycle of HIV is the proteolytic cleavage of the polypeptide precursors into mature enzymes and structural proteins catalyzed by HIV PR. It has been shown that budded immature viral particles that contain catalytically inactive protease cannot undergo maturation to an infectious form.² The necessity of this enzyme in the virus life cycle makes it a promising target for therapy of the HIV infection.³

3.2 Structure of HIV protease

Navia *et al.* from Merck laboratories were the first group to obtain a crystal structure of HIV PR;⁴ a more accurate structure was reported subsequently by Kent and coworker.⁵ HIV PR is a 99 amino acid aspartyl protease which functions as a homodimer with only one active site which is C₂-symmetric in the free form. More than 140 structures of the HIV-1 PR, its mutants and enzymes complexed with various inhibitors have been reported so far. A database dedicated to providing structural information about HIV PR has been created at the National Cancer Institute (<http://www-fbnc.ncifcrf.gov/HIVdb/>). The enzyme homodimer complexed with TL-3⁶ is shown in Fig. 1 (PDB ID: 3TLH). Each monomer contains an extended β -sheet region (a glycine-rich loop) known as the flap, that constitutes in part the substrate-binding site and plays an important role in substrate binding, and one of the two essential aspartyl residues, Asp-25 and Asp-25' which lie on the bottom of the cavity. The substrate binds in its extended conformation, in which its interactions with the different amino

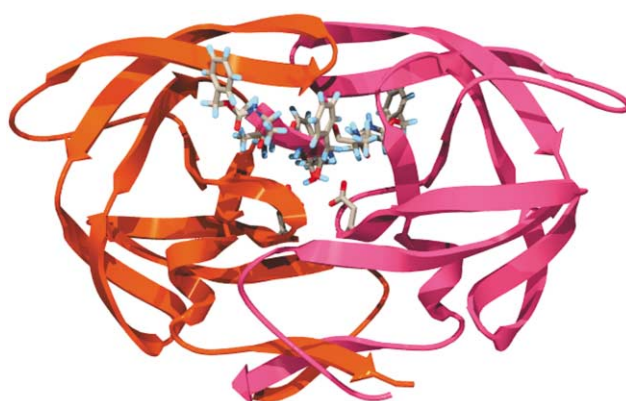


Fig. 1 Structure of HIV PR complexed with TL-3 (PDB: 3TLH).

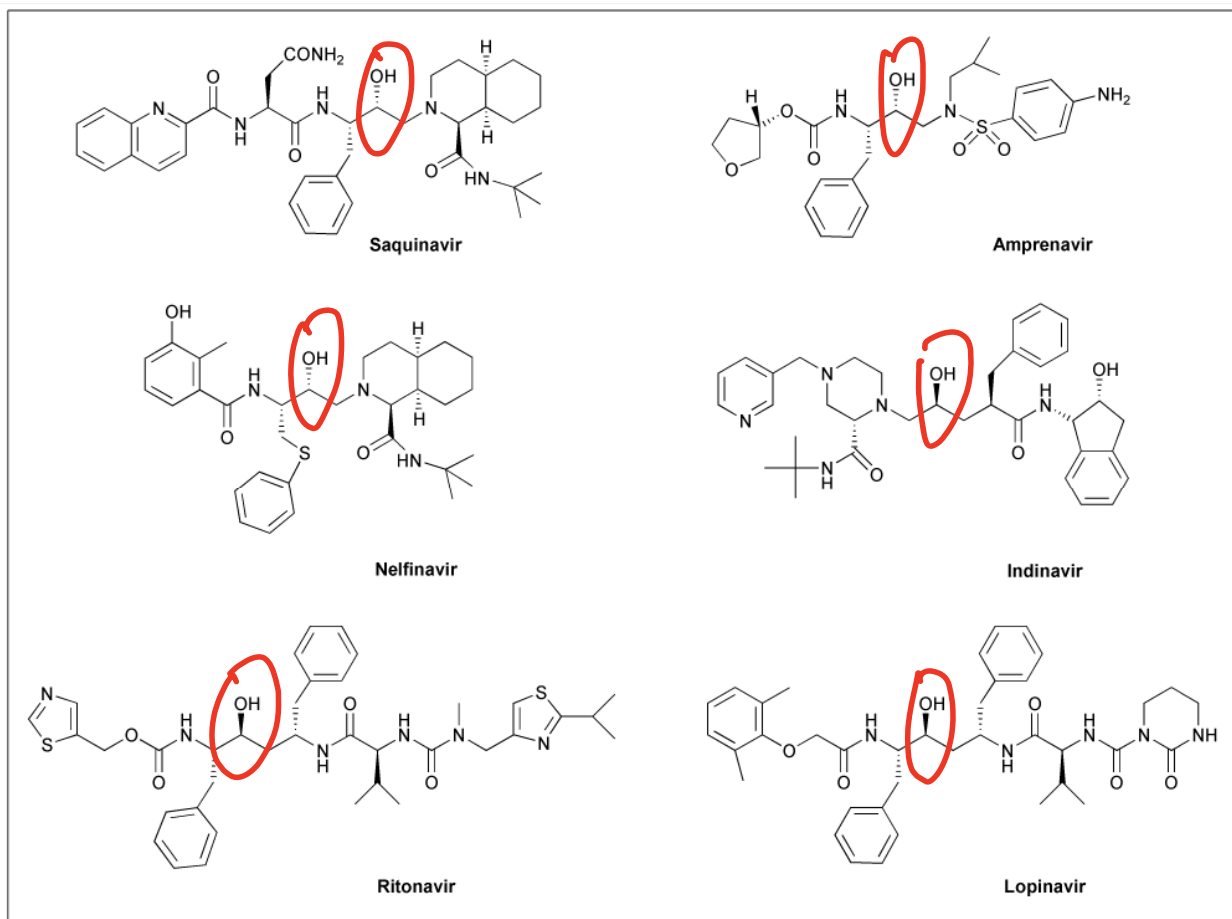


Fig. 10 FDA approved HIV-1 protease inhibitors.

These all have alcohol groups that resemble the $-OH$ of the key tetrahedral intermediate seen in the enzyme reaction.

 SOUTHWEST

'Operation Spider Web': 13 arrested in drug trafficking ring that sold to Texas college students



Elyse Russo, Billy Gates/KXAN

Posted: DEC 4, 2020 / 03:36 PM CST | Updated: DEC 5, 2020 / 07:50 PM CST

AUSTIN (NewsNation Now/KXAN) — Federal and local officials in Texas arrested 13 individuals in connection with drug trafficking operation that sold to students at University of Texas at Austin.

During the investigation, dubbed "Operation Spider Web," authorities discovered more than \$1 million worth of counterfeit prescription drugs laced with LSD, fentanyl and methamphetamine were sold and distributed to residents in Austin, Texas, including UT students.

CRIME

13 plead guilty in DOJ West Campus drug ring case, remaining 4 face jury trial

by: Cora Neas, Alex Capriello, Billy Gates

Posted: Nov 4, 2022 / 12:11 PM CDT

Updated: Nov 4, 2022 / 12:35 PM CDT



Drugs and money confiscated by law enforcement in recent drug trafficking arrests. (KXAN photo/Alex Capriello)

SHARE    

AUSTIN (KXAN) — Thirteen defendants plead guilty in 2022 to charges from the 2020 DOJ drug sting “Operation Spyder Web,” which uncovered a million-dollar drug operation near the University of Texas at Austin.

Defendants include eight former UT students, as well as individuals not associated with the school. All 17 defendants were charged with conspiracy to possess with intent to distribute a controlled substance.

PREVIOUS: 13 people including current and former UT students arrested on drug trafficking charges, DOJ says ▶

Those who pled guilty are: Charles Zenker, Varun Prasad, Drew Zarate, Christopher Edwards, Jakob Schelling, Madison Scott, Adrian Andreescu, Nikit Shingari, Nolan Fogleman, Samuel Parry, Brandon Carpenter, Robert Byabato and Ashley LaRue.

These defendants are scheduled for sentencing Jan.13, 2023.



OBITUARY

Jacob McAdams Ehlinger

DECEMBER 13, 2000 - MAY 6, 2021



IN THE CARE OF

Weed Corley Fish Funeral Homes and Cremation Services

University of Texas football player, McCombs School of Business second-year finance student, Phi Gamma Delta fraternity member, and a member of the Texas Silver Spurs, Jake was known for his larger-than-life joyful personality, grit, selflessness, and generous spirit of enthusiasm and encouragement.

A series of horizontal lines for writing, starting from the first line below the paragraph and extending to the bottom of the page.

Opiates (heroin, morphine, etc.)

The human body naturally produces its own opiate-like substances and uses them as neurotransmitters. These substances include endorphins, enkephalins, and dynorphin, often collectively known as endogenous opioids. Endogenous opioids modulate our reactions to painful stimuli. They also regulate vital functions such as hunger and thirst and are involved in mood control, immune response, and other processes.

The reason that opiates such as heroin and morphine affect us so powerfully is that these exogenous substances bind to the same receptors as our endogenous opioids. There are three kinds of receptors widely distributed throughout the brain: mu, delta, and kappa receptors.

These receptors, through second messengers, influence the likelihood that ion channels will open, which in certain cases reduces the excitability of neurons. This reduced excitability is the likely source of the euphoric effect of opiates and appears to be mediated by the mu and delta receptors.

This euphoric effect also appears to involve another mechanism in which the GABA-inhibitory interneurons of the ventral tegmental area come into play. By attaching to their mu receptors, exogenous opioids reduce the amount of GABA released (see animation). Normally, GABA reduces the amount of dopamine released in the nucleus accumbens. By inhibiting this inhibitor, the opiates ultimately increase the amount of dopamine produced and the amount of pleasure felt.

Chronic consumption of opiates inhibits the production of cAMP, but this inhibition is offset in the long run by other cAMP production mechanisms. When no opiates are available, this increased cAMP production capacity comes to the fore and results in neural hyperactivity and the sensation of craving the drug.

Opioid receptors

John McDonald BSc
DG Lambert PhD

Key points

The opioid system comprises four subtypes of receptor: MOP, DOP, KOP and NOP.

Opioid receptors all have selective endogenous peptides.

Analgesia elicited by opioids used clinically act predominately via the MOP receptor.

NOP receptor antagonists have been shown to cause analgesia in animals.

Tolerance to classical opioids may be attenuated by NOP receptor antagonism.

Opium and its derivatives have been used for centuries, both in a medicinal and 'recreational' manner. Indeed, findings of fossilized opium poppy seeds dating as far back as 30 000 yr ago suggest the use of opium by Neanderthal man. In 1799, Friedrich Serturmer discovered the major active ingredient of opium, which he named morphine and opioid pharmacology was born. Morphine and its derivatives are used today for the treatment of acute and chronic pain. It is now understood that morphine and other opioid drugs act on an endogenous opioidergic system, which is not only involved in setting pain (nociceptive) threshold and controlling nociceptive processing but also participates in modulation of gastrointestinal, endocrine and autonomic function, as well as a possible role in cognition.

Evidence for the existence of multiple opioid receptor subtypes arose from work identifying the different anatomical location and pharmacological profiles of compounds that were eventually used to name them, i.e.

morphine (*mu*), ketocyclazocine (*kappa*) and *vas deferens* (*delta*). Recently, a fourth opioid-like receptor has been included in the opioid receptor family and is termed the nociceptin orphanin FQ peptide receptor. Receptor nomenclature has changed numerous times but current International Union of Pharmacology (IUPHAR) opinion is MOP (*mu*), KOP (*kappa*), DOP (*delta*) and NOP for the nociceptin orphanin FQ peptide receptor. All four are G-protein-coupled receptors sharing the similar seven transmembrane topology (Fig. 1). Other receptor subtypes have been suggested (e.g. sigma receptor) but have been dismissed based on a lack of naloxone sensitivity.

Cellular mechanisms of action

G-protein-coupled receptors, such as those for opioids, have no direct link with effector proteins; instead the message is relayed via a G-protein. Both classical opioid receptors (MOP/KOP/DOP) and the non-classical NOP opioid

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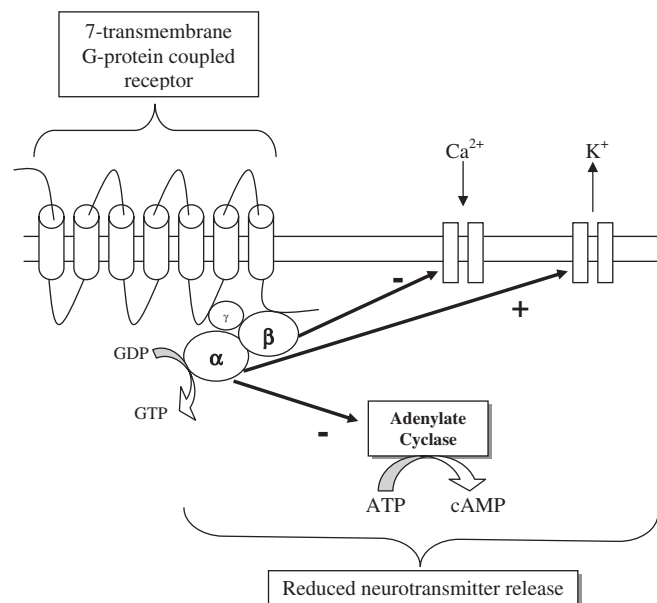


Fig. 1 Seven transmembrane structure of opioid G-protein-coupled receptor. Receptor activation by opioid receptor ligands leads to initiation of intracellular transduction pathways that include stimulation of potassium efflux, inhibition of VSCCs and inhibition of adenyl cyclase. In this diagram the G-protein is denoted α , β , γ but the α -subunit interacts with K^+/Ca^{2+} channel and adenylate cyclase.

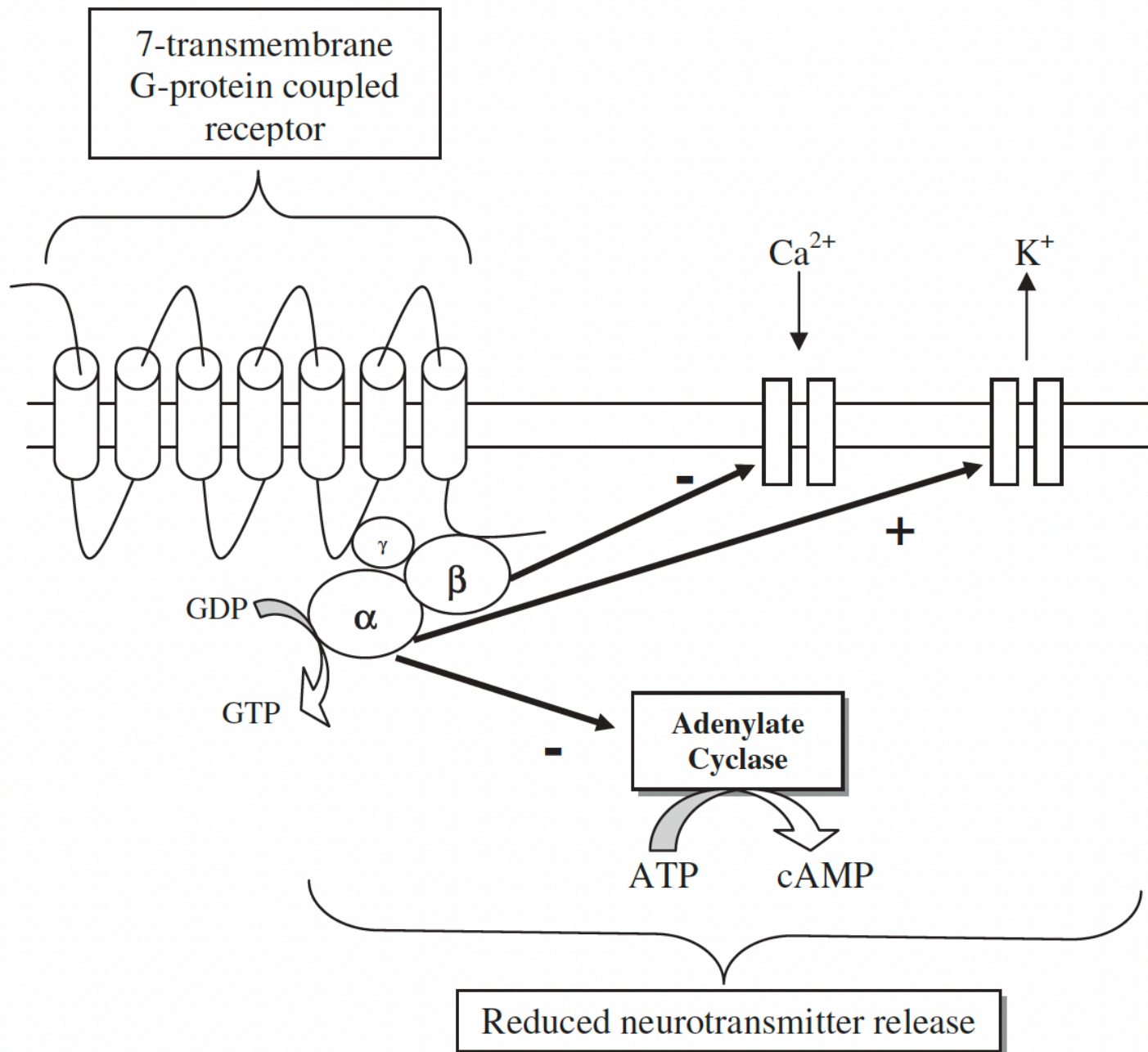


Fig. 1 Seven transmembrane structure of opioid G-protein-coupled receptor. Receptor activation by opioid receptor ligands leads to initiation of intracellular transduction pathways that include stimulation of potassium efflux, inhibition of VSCCs and inhibition of adenyl cyclase. In this diagram the G-protein is denoted α , β , γ but the α -subunit interacts with $\text{K}^+/\text{Ca}^{2+}$ channel and adenylate cyclase.

receptor couple to inhibitory G-proteins. Activation of opioid receptors, for example MOP with morphine leads to: (i) closing of voltage sensitive calcium channels (VSCC); (ii) stimulation of potassium efflux leading to hyperpolarization; and (iii) reduced cyclic adenosine monophosphate (cAMP) production via inhibition of adenylyl cyclase. Overall, this results in reduced neuronal cell excitability leading to a reduction in transmission of nerve impulses along with inhibition of neurotransmitter release (Fig. 1).

Endogenous and exogenous ligands

The endogenous opioid peptides are cleaved from four prohormone precursors: (i) pro-enkephalin, (ii) pro-opiomelanocortin, (iii) pro-dynorphin, and (iv) pre-pro-N/OFQ (pp-noc). The endogenous DOP receptor peptides are met-enkephalin and leu-enkephalin, cleaved from proenkephalin.¹ Pro-dynorphin gives rise to the KOP receptor agonists dynorphin A and B whilst N/OFQ is from the polypeptide precursor pre-pro-N/OFQ. Pro-opiomelanocortin encodes the peptide β -endorphin, which has agonist activity at all three classical opioid receptors. Presently, the precursor protein(s) for the endogenous MOP receptor peptides endomorphin 1 and 2 is unknown.²

The prototypical MOP agonist is the alkaloid morphine, extracted and purified from opium. Of the synthetic opioid agonists, whose structures bear no resemblance to morphine, fentanyl and remifentanyl are the more potent compounds. Pentazocine and buprenorphine are partial agonists. Owing to the reduced efficacy of partial agonists, they are able to antagonize or reduce the responsiveness of a full agonist such as morphine when acting at the same receptor. This may result in an increase in the dose of full agonist required in order to compete against the partial agonist and restore the full agonists maximal response. Differences in the pharmacokinetics of a partial and full agonist could lead to overdose if the partial agonist were to be metabolized more rapidly than the full agonist.

A number of the opioid ligands lack specificity for a particular subtype of opioid receptor. For example, the endogenous peptide β -endorphin has agonist activity at all three classical opioid receptors. Buprenorphine has partial agonist activity at MOP and NOP receptors and, as a result, has a bell shaped response curve for its analgesic activity *in vivo* (i.e. at low and intermediate doses an analgesic response results), at higher doses the analgesic response may be decreased. The complex pharmacology of buprenorphine is explained by its agonist activity at both MOP, resulting in analgesia at low and intermediate doses, and NOP, resulting in an anti-opioid/anti-analgesic action at higher doses (see later). Some other opioid drugs have mixed actions at different opioid receptors. For example, pentazocine behaves as an antagonist at MOP receptors but a partial agonist at DOP and KOP receptors. Currently, there are no clinically selective drugs available that work via DOP, KOP or NOP receptors (see Table 1 for a list of exogenous and endogenous classical opioid receptor ligands).

Table 1 Endogenous opioid peptides, synthetic and semi-synthetic opioid agonists and antagonists, along with their selectivity for the different subtypes of opioid receptor. N/OFQ = nociceptin orphanin FQ; × = no affinity; ✓ = low affinity; ✓✓ = intermediate affinity; ✓✓✓ = high affinity. (Modified from Rang, Dale and Ritter³)

Endogenous ligand	Receptor subtype			
	MOP	KOP	DOP	NOP
β -endorphin	✓✓✓	✓✓✓	✓✓✓	×
Endomorphin 1/2	✓✓✓	×	×	×
Leu-enkephalin	✓	×	✓✓✓	×
Met-enkephalin	✓✓	×	✓✓✓	×
Dynorphin A/B	✓✓	✓✓✓	✓✓	✓
N/OFQ	×	×	×	✓✓✓
Clinical Drugs				
Agonists				
Morphine	✓✓✓	✓	✓	×
Meperidine	✓✓✓	✓	✓	×
Diamorphine	✓✓✓	✓	✓	×
Fentanyl/remifentanyl	✓✓✓	✓	×	×
Antagonist				
Naloxone	✓✓✓	✓✓	✓✓	×

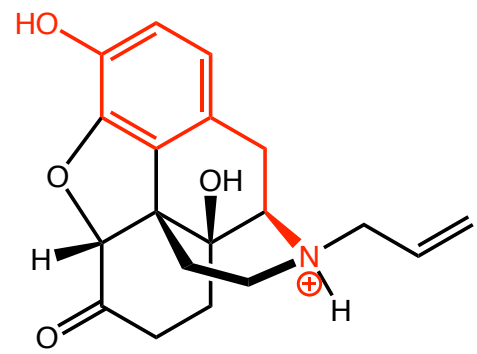
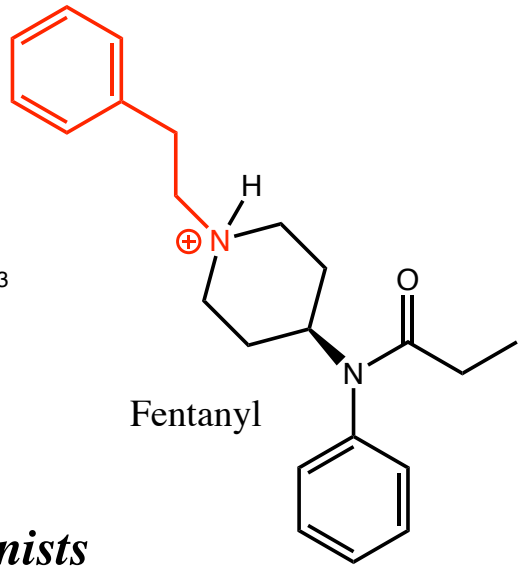
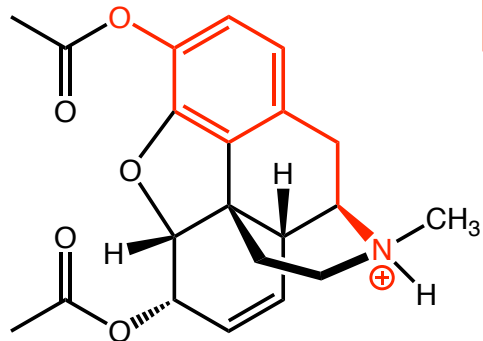
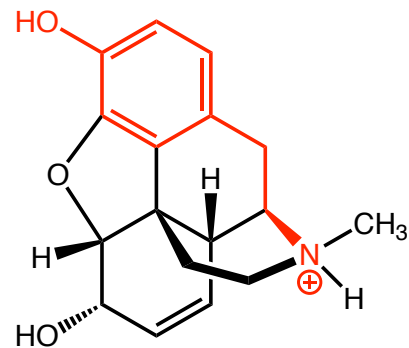
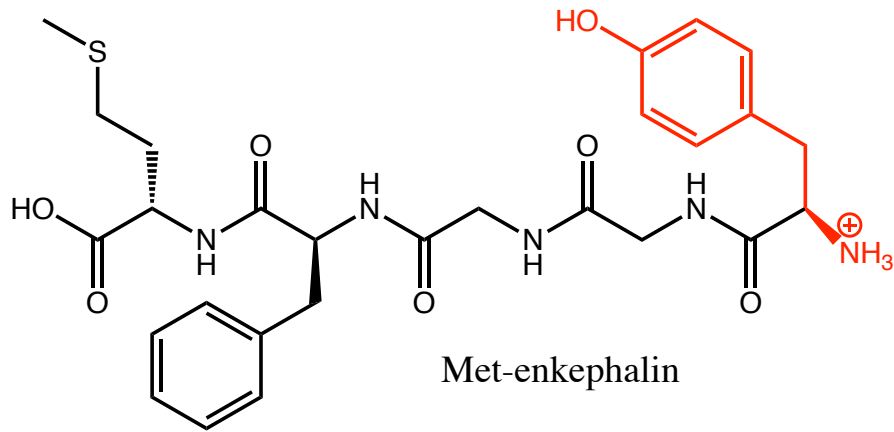
Opioid receptor subtypes

MOP

The MOP receptor was the last of the classical opioid receptors to be cloned and is located throughout the central nervous system in areas involved in sensory and motor function including regions concerned with the integration and perception of these senses, for example cerebral cortex, amygdala (of the limbic system). High density of MOP receptors is found in the caudate putamen (of the basal ganglia). MOP receptors are located presynaptically on primary afferent neurons within the dorsal horn of the spinal cord where they inhibit glutamate release and hence transmission of nociceptive stimuli from C and A δ fibres. The periaqueductal grey (PAG) is an area of the midbrain involved in the central control of nociceptive transmission. Efferent outflow from the PAG descends to the spinal cord where it acts to inhibit nociceptive transmission in afferent fibres, this pathway is known as the descending inhibitory control pathway. High densities of MOP receptor are found in the PAG and the analgesia of some opioids is proposed to come about from removal of an inhibitory γ -amino butyric acid (GABA)-ergic tone in this region of the brain. GABA is the main inhibitory transmitter in the brain and acts to reduce or prevent antinociceptive outflow from the PAG.

Major side-effects associated with MOP agonists include respiratory depression through a reduction in the sensitivity of central and peripheral chemoreceptors to hypercapnia. MOP agonists further inhibit gastrointestinal tract secretions and peristalsis, often causing constipation. MOP opioids also have effects on the cardiovascular system, thermoregulation, hormone secretion and immune function.

Studies using MOP receptor knockout mice have defined the role MOP plays tonically and when stimulated by exogenously applied ligands. MOP receptor knockout mice show increased sensitivity to thermal pain, implicating the receptor in this



Mu-Receptor Agonists

Antagonist

NEVER buy black market

or other pharmaceuticals → it is
not black market it is counterfeit

and contains **Fentanyl** ⇒ **Could be fatal !!**

↓
Xanax

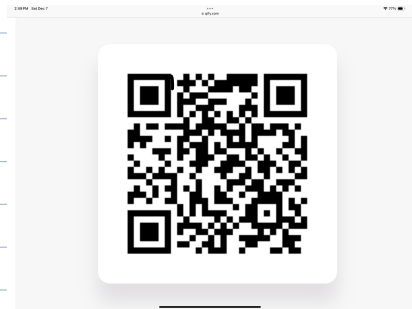
or **methamphetamine** ⇒ **Highly
addictive**

↓
Adderall

You should know where to find **Naloxone (Narcan)**
in case you need it to save a life!

⇒ RA's in UT dorms

⇒ Contact SHIFT



Naloxone (Narcan) is an **antagonist** to
opioid receptors



Does not cause
a reaction but blocks drugs like
fentanyl from binding and activating
the receptor

Organic Chemistry is the study of carbon-containing molecules.

This class has two points.

The first point of the class is to understand the organic chemistry of living systems. We will teach you how to think about and understand the most amazing things on the planet!!

Water is essential for life, you will learn why water has such special properties. 8/28/24

You will learn the secret structural reason proteins, the most important molecular machines in our bodies, can support the chemistry of life. 9/11/24

You will learn why when you take Advil for pain, exactly half of what you take works, and the other half does nothing. 9/25/24

You will learn how toothpaste works. 10/7/24

You will learn how a single chlorofluorocarbon refrigerant molecule released into the atmosphere can destroy many, many ozone molecules, leading to an enlargement of the ozone hole. 10/30/24

You will learn how medicines like Benadryl, Seldane, and Lipitor work. 11/13/24

You will learn how Naloxone is an antidote for an opioid overdose. 12/9/24

You will learn why Magic Johnson is still alive, decades after contracting HIV. 12/9/24

You will learn how MRI scans work. 12/4/24

The second point of organic chemistry is the synthesis of complex molecules from simpler ones by making and breaking specific bonds.

You will learn how to understand movies of reaction mechanisms like alkene hydration. 10/9/24

You will learn reactions that once begun, will continue reacting such that each product molecule created starts a new reaction until all the starting material is used up. 10/30/24

You will learn reactions that can make antifreeze from vodka. 11/13/24

You will learn a reaction that can make nail polish remover from rubbing alcohol. 11/16/24

You will learn how to look at a molecule and accurately predict which atoms will react to make new bonds, and which bonds will break during reactions. 11/20/24

You will learn how to analyze a complex molecule's structure so that you can predict ways to make it via multiple reactions starting with less complex starting molecules. 11/20/24

GWH

(Good Will Hunting)

Vladimir Markovnikov

Alkenes are the very best, their reactions are insane.
Alcohols are no match, and neither are alkanes
Electrophiles hit alkenes, without a lot of pain
Use HBr and HCl to make haloalkanes

But then I get confused as to what we're going to see
Which carbon gets which atom? Call it regiochemistry
I hear there's a brilliant man who gave us clarity
What's his name? What's his name? I have to find the key

Well the word got around, they said "this guys insane, man!"
From the 1800's, a Russian dude they say, man.
Got his education and didn't forget from whence he came, man
The world's got to know his name. What's his name, man?

Vladimir Markovnikov
My name is Vladimir Markovnikov
And there's just one thing I did explain
Which carbon gets, the halogen?

The key to understanding products of hydrohalogenation
Is that a carbocation is formed by the HX protonation
Stabilize the plus charge with overlapping orbitalization
The official term of course, is hyperconjugation

The more stable cation forms of lower energy
The halide ion comes in and reacts there naturally
It's the one with more carbons, that's maybe two or three
So X is on more substituted carbons don't you see

Vladimir Markovnikov
My name is Vladimir Markovnikov
And there's just one thing that I did explain
Which carbon gets, the halogen?

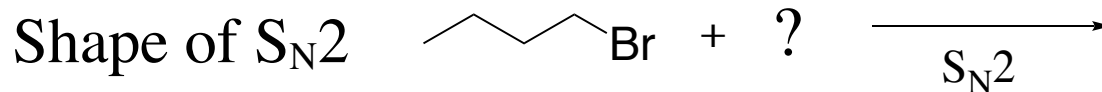
So don't be throwing away my rule
Don't be throwing away my rule
Hey yo, its not that convoluted
Halogen on the carbon that's more substituted
So don't be throwing away my rule

Vladimir Markovnikov
My name is Vladimir Markovnikov
And there's just one thing I did explain
Which carbon gets, the halogen.

So don't be throwing away my rule
Don't be throwing away my rule
Hey yo, its not that convoluted
Halogen on the carbon that's more substituted
So don't be throwing away my rule

Vladimir Vasilyevich Markovnikov





The club ain't the best place to find nucleophiles
So the lab is where I go
Me and my friends in solvent
Moving fast but reacting slow
Come over and start up a reaction with just me
And trust me I'll give it a chance now
Let's react, stop, put your charge over here
And then we start to react, and now I'm singing like
You know I want to make a bond
Your charge was handmade for a reagent like me
Come on now, follow my lead
I'm an electrophile, don't mind me
Say now let's not talk too much
Get through solvent and put your electrons on me
Come on now, follow my lead,
Come, come on now, follow my lead

I'm attracted to the charge of you
We push and pull like charges do
Although my orbitals are reacting too
Come on let's get bonding
Transition state is coming true
Time to finish the S_N2
Creating a bond brand new
Come on let's get bonding
Oh-I-oh-I-oh-I-oh-I
Come on let's get bonding
Oh-I-oh-I-oh-I-oh-I
Come on let's get bonding
Oh-I-oh-I-oh-I-oh-I
Come on let's get bonding
We need to make a bond brand new
Come on now let's S_N2 .

" Don't Stop, Believin'

Just a Houston girl,
Living in a Longhorn world.
She took the premed train
Straight to OChem 1

Just a Plano boy,
Born and raised in full burnt orange,
He took the premed train
Straight to OChem 1

They study in a smoky room,
Smell of vapes and Mountain Dew
For an A they study all night
It goes on and on and on and on

Alkanes to alkynes
Up and down the I-35
Two students studying in the night
Roadmaps, reactions
Living just to pass the final
Will they ever get it right

Working hard to get my grade
Everybody wants an "A"
Trying anything to ace this class
Just one more exam
Some will win, some will lose
I don't want to sing the blues
OChem never seems to end
It goes on and on and on and on

Alkanes to alkynes
Up and down the I-35
Two students studying in the night
Roadmaps, reactions
Living just to pass the final
Will they ever get it right

Don't stop believin'
Hold on to that "A" feelin'
Roadmaps, reactions
Ohohohohohoh

Don't stop believin'
Hold on
Roadmaps, reactions
Ohohohohohoh

Don't stop believin'
Hold on to that "A" feelin'
Roadmaps, reactions
Ohohohohohoh

We All Love Organic Chemistry

**In the town where I was born,
Lived a man of chemistry.
And he told us of his life
In the organic laboratory.**

**Making molecules to fight disease
Coming up with their syntheses.
So we sit in 320M
Learning organic chemistry.**

Refrain:
We all love organic chemistry
Synthetic chemistry
Molecules with "C"
We all love organic chemistry
Synthetic chemistry
Molecules with "C"

**All our friends think we're a bore
Our grade point averages begin with
4.
But we await graduation day
To work in lab for meager pay.**

**But its OK, who else can say
They're curing cancer or fighting
AIDS.**

**We hope that you in 320M
Respect organic chemistry**

Refrain:
We all love organic chemistry
Synthetic chemistry
Molecules with "C"
We all love organic chemistry
Synthetic chemistry
Molecules with "C"