Saturated/Unsaturated/Trans/Cis Fats

What is fat? - George Zaidan

This video goes over saturated and unsaturated fats, as well as trans and cis configurations. It does a great job breaking them down while keeping it interesting.

If you are struggling with telling the difference between saturated and unsaturated fats, here is how I think about it…

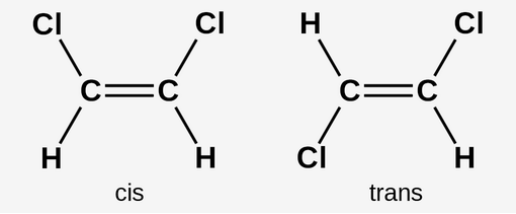
When I think of saturated fats, I imagine they just had a big meal of hydrogens and they are saturated or "satiated" with hydrogens.

When I think of unsaturated fats, I imagine them being very hungry and "unsatiated" (unsaturated) meaning they could have more hydrogens!

We can also use this to remember which would be SOLID at room temp, and which would be LIQUID at room temp.

- In my mind, saturated fats are solid at room temperature because they are super full!
- Conversely, unsaturated fats are liquid at room temperature because they do not have anything "filling them up" and they are super hungry!

Additionally, if you struggle between trans and cis fats… remember that "cis" means "same" so the carbon chains will be coming off the same side of the double bond. Conversely, "trans" means "opposite" so the carbon chains will be coming off of opposite sides of the bonds.



If you are still struggling to understand, here is another video that goes through different triglycerides to explain all of these.

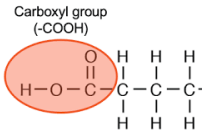
Essential Fatty Acids (+ Clinical Relevance)

What are essential fatty acids? Essential fatty acids are termed "essential" because we cannot synthesize them on our own and need to get them from an outside source.

Fun fact: we cannot make double bonds past the 9th carbon from the carboxyl end! (The video uses Omegas, so do not get confused!)

Clinical relevance: Linoleate (Omega 6) and a-Linoleic Acid (Omega 3)
These essential fatty acids are talked about in this video as well as what an essential acid is!
~I would make sure to watch this whole video- she talks about their importance in membrane fluidity which is good to know!

Naming
MCAT Bio/Biochem Review: Fatty Acid Nomenclature
This video goes over the naming of fatty acids. Unfortunately, she goes over 4 methods, but for this class you only need to know two! She describes the omega system (#3 in the video), but the systemic naming system is considered to be a slight mix of both the IUPAC and Delta naming system as she describes them. (These are #1 and #4 in the video). See below for how they come together.
Remember…



Systemic naming begins at the CAYBOXYLIC end (so any time you see this guy) →
This naming system also uses Δ (delta) in front of each *double bond* and uses this number as a superscript.
Here is an example:

16	1	Palmitoleate	*cis*-Δ^9-Hexadecenoate
18	1	Oleate	*cis*-Δ^9-Octadecenoate
18	2	Linoleate	*cis, cis*-Δ^9, Δ^{12}- Octadecadienoate
18	3	Linolenate	all-*cis*-Δ^9, Δ^{12}, Δ^{15}- Octadecatrienoate
20	4	Arachidonate	all-*cis* Δ^5, Δ^8, Δ^{11}, Δ^{14}- Eicosatetraenoate

(# of carbons, # of double bonds, common name, systemic name)
If you notice, both of the bonds contain the delta and superscript, while including the IUPAC "cis" and "trans" nomenclature.

Triacylglycerols
Molecular structure of triglycerides (fats) | Biology | Khan Academy
This video does a great job explaining triacylglycerols. He goes over how they get their name, how they are formed, and what their structure looks like.

To remember why they are great energy sources, I like to think about triacylglycerols in their components.

1.) Highly reduced→ more electrons for oxidation (saturated with hydrogens)
    a.) When you need energy (lets think of all of the hydrogens as "potential energy" for now) wouldn't you prefer to have the ability to squeeze LOTS of energy out of the molecule, rather than just a little bit? That is what the hydrogens are doing. The more saturated with hydrogens, the more capacity for energy you have!
        i.) Now, do not get confused, triacylglycerols can be considered both "saturated" and "unsaturated". This just refers to the bonds we were talking about earlier→ cis and trans double bonds. However, these triacylglycerols are still generally considered saturated with hydrogens since they contain a bunch of them, especially relative to the other types of lipids we will be talking about.
2.) Anhydrous
    a.) This just means it has no water weight.
        i.) Unlike glucose, which is stored in the body with about 3-4 grams of water per gram of glucose, triacylglycerols do not interact with water or store any weight with it in the body! → This is what allows humming birds to fly as long as they do (500m+) as they have a great energy source but very low weight.

Membrane Lipids

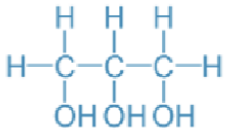
Phospholipids

Phospholipid structure | Cells | MCAT | Khan Academy (Until 2:40)

This video goes over the structure of phospholipids if you would like a review. It also shows the individual components of a phospholipid separately.

Remember, the two types of phospholipids:

1. Glycerol (the most common)
    a. These guys are described in the video, 3 carbon backbone (that then attaches the 2 fatty acid chains and a phosphate group)
        i. Ester bonds!!
2. Sphingosine (sphingolipids)
    a. These are long, 18 carbon backbones.
        i. Found in nerve cells



Glycerol

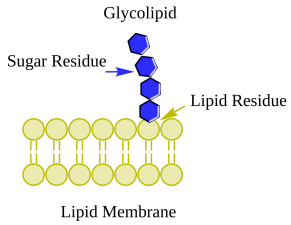
Sphingosine

I remember the differences between these two because glycerol is the "short" word→ short backbone, and vice versa!

Glycolipids

What are Glycolipids? Difference between Cerebroside and Ganglioside (Stop 1:44)

This video goes over glycolipids and explains their components. It also explains what cerebroside is.



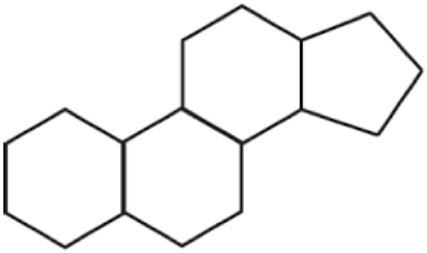
I like to think of glycolipids as a flag hanging outside the membrane. They are there to help other cells recognize them and communicate, help with immune system identification, and aid in tissue specific interactions!

Steroids

Lipids Part 2: Steroids

This video by Prof. Dave does a great job summarizing steroids!

To remember steroids, I like to think of the 4 carbon rings as being "really buff".



If you look at the structure of the 4 carbon rings, it almost makes the shape of someone flexing their muscles…
Since steroids are typically known for their use with body builders, it helps me remember that if I see a four carbon ring, it's a steroid!

Protein-Lipid Interaction

Lipid anchors: Lipids that allow proteins to localize to the cell membrane.
Case study:
During folding, Lamin A is bound to the nuclear membrane by farnesyl A (lipid anchor). When this association does not degrade and allow Lamin A to the nuclear lamina, it causes Hutchinson-Gilford progeria syndrome.
Understanding Hutchinson-Gilford progeria syndrome

Chapter 12: Membranes

In Da Club - Membranes & Transport: Crash Course Biology #5 (Stop 9:00)

This video goes over general properties of membranes and transport. If you are struggling to conceptually understand active transport, this video would be super helpful!

So to start, lets go over some *general characteristics*

1.) Selective barrier

So, to think about this conceptually, I like to think about holding water in my own hands. When you cup your hands together, they do not close all the way, meaning there are small gaps where water can pass through. This is what happens with membranes! Further, if I were to place substances that maybe took up more space than water, or were thicker than water, they may have a harder time getting through. This is also the same for membranes! Bigger, more polar molecules have a more difficult time passing through the barrier, which makes it selective.

2.) Flexible structure
   a.) We want membranes to be malleable to a certain extent. Our cells bump around, reshape, and move a lot, etc so it is important they are able to "go with the flow" so they do not get easily damaged.

3.) Self-assembling
   a.) Lipids, as we learned from the last section, really do not love being around water. Due to this, when in aqueous solution (our bodies!) membranes will assemble quickly to "protect" the fatty acid legs of the membrane and keep the phosphate heads out to interact with the water.
   b.) This video shows a depiction of self assembly to avoid the fatty acid tail (red) from interacting with water.

4.) Asymmetric
   a.) This may seem counterintuitive as we think of the lipid bilayer as being two layers of identical molecules that come together. However, the inner and outer sides have different compositions due to the differences in what they are interacting with.

5.) Dynamic
   a.) This just tells us that proteins etc found in the membrane are able to move around, and are not stuck in one specific location.

The Hydrophobic Effect

If you are unfamiliar with this concept, check out this video

For this concept, I like to think of the water molecules and the lipid tail as magnets of the same pole. When the water has to be pushed up against the lipid tale, it is going to want to push away, much like a magnet would. In trying to push them together, you will feel a great deal of resistance which shows how the water will be at a "high energy state".

Then, when you let them go, they will want to move away from each other, and causatively push the magnets back. If you have a magnet then on the opposite pole, it will want to move towards it. The water molecules behave very similarly, the water does not want to be around the hydrophobic tails of the lipid, but would rather interact with the hydrophilic head of the molecule.

This is energetically favorable, as you no longer have to input all of that "force" to keep the magnets of the opposite pole together.

(Magnets: This tik tok video shows the magnet phenomenon I am referring to!)

The Goldilocks Principle

The membrane should not be too fluid or too ridgid… it should be justttt right.

How to control membrane fluidity:

1.) Saturation
    a.) If the fatty acid tails are saturated, they will be densely packed together. Remember, if something is unsaturated, it means it contains double bonds which "kink" the linearity of the molecule.
        i.) Increased saturation, higher melting temperature since they are densely packed.
2.) Chain length
    a.) If it is longer, it will be more rigid, shorter will be more fluid.
3.) Cholesterol: this is ambiguous and acts as a fluidity buffer!
    a.) In high temperatures→ it stops phospholipids from moving around, meaning it decreases fluidity
    b.) In low temperatures→ it stops membranes from becoming too rigid, meaning it increases fluidity!
        i.) Remember, hopanoids are bacterias version of cholesterol, and sterols are for plants!

Integral/Peripheral Proteins

Integral proteins: *integrated* into the membrane. (in→ inside the membrane)
For them to even be able to do this, they need to be able to interact with the inside of the membrane→ the lipids!
This means these proteins must have hydrophobic regions that can interact with the fatty acid tails, and have nonhydrophobic regions that can interact with the extracellular surface.
Peripheral proteins: on the periphery of the cell (outside!)
Associated with the membrane *surface*
These do not span the membrane, which means they only need to have the ability to interact with the polar headgroups→ NO hydrophobic regions needed!

Transport proteins:

Active transport: When you need energy to move something against its gradient
→ Think… if you are being active, you are going to need an energy input, right? This is the same for active transport! I also sometimes need a nudge to start exercising… so I think of active transport as moving something *against* what they want to do.

1) Primary active transport:
    a) Na+/K+ pump. Video to Watch
        i) This pumps 3 Na+ molecules in, and 2 K+ molecules out.
            (1) To do this, ATP gets dephosphorylated (gives us our energy input!)
            (2) *Why is it important?* It allows for the maintenance of the membrane potential
            (3) Watch this video if you want some more information on that.
2) Secondary active transport: Both in same direction

Use one gradient to drive transport of another molecule!
-Symporter: sym (same)→ move same direction
-Antiporter: anti (different) → opposite directions

    a) Na+ glucose symporter (same direction!)
        i) Na+ gradient wants to move Na+ to the other side, so, glucose, which does not want to be transported to the other side, "buddies up" with him, and passes over.
            (1) In this scenario, imagine that you are taking public transport that requires you to scan a card and pass through a gate (like the T in Boston). If the person in front of you scans their card, and you move fast enough, you can squeeze in as one person. This is true for secondary active transport. The first person (Na+) scans their card (energy from gradient) and you (the glucose) squeeze in on their dime to save money (energy).

Passive transport: To move something along a gradient, no energy barrier.

- For passive transport, I like to think of my older brother. He is super "passive", and will just go wherever you take him as long as he doesn't have to be in big crowds. Passive transport is the same, as long as they are going somewhere they wouldn't mind being (energetically moving down a gradient) they will do it, and it takes no energy input for them to do so.

Ion Channels:
Ion Channels Explained With Fun Animations!
Clinical relevance:
Tetrodotoxin
→ blocks voltage gated Na+ channels
This makes it so that Na+ cannot flood the cells for neuron activation which results in paralysis and lung failure.
-To remember this, I think of tetris ("tetro-"…"tetris"…I make it work), and that tetris uses blocks… → tetrodotoxin *blocks* Na+

Similarly, when you get enough "blocks" in a row they fizzle and disappear. If you can track my metaphor, congratulations, but if not, this is exemplary of how the Na+ gradient can no longer generate neuron activation! (Trust me, the more ridiculous the comparison, the more you'll remember it…).

CHAPTER 13
Receptors: Signal Transduction and Phosphorylation Cascade This video goes over signaling and general topics that we will go into when we go through specific examples.
Do not worry about endocytosis

"Release glucose NOW!" pathways
Glucagon and Epinipherine

- The name Glucagon to me sounds a lot like the name of a big scary monster. To remember this pathway is used to release glucose, I picture something like this. (Just replace cookie with "glucose")
* It's a bit cringe, I know, but it really makes it stick in my head…

For epinephrine, I remember that this is the same term as "adrelenine", which allows me to conceptually think about the fact that we need energy if we're going to be running away from something really big (or fighting if that's your prerogative), we will need to release a bunch of glucose to make ATP.

G-Protein Coupled Receptors (GPCR)
This video does a great job of going over this! You can stop at 2:00.
-7 transmembrane receptor (meaning it spans the membrane 7 times)
-Has a G-protein with alpha, beta, and gamma subunits.

Glucagon/Epinephrine #1 Beta Adrenergic:
Glucagon Signalling Pathway | GPCR Signalling this video quickly goes over the process. You do not need to know any more than the activation of pka, but make sure you know the result!

To review:

1.) Glucagon or Epinephrine bind to the receptor
    a.) You can think of these as the "switch" that activates the whole cascade. They want something to happen, so they bind and turn the process on!
2.) The G-protein subunit undergoes a conformational change that causes it to RELEASE GDP and bind GTP.
    a.) Remember, GDP is *not* being phosphorylated here! It is letting go of the GDP in favor of the GTP
3.) Gas (alpha subunit and GTP) is formed by the dissociation of the beta and gamma subunits→ this just leaves the alpha subunit and GTP
    a.) I love when the names of molecules just *make sense*. The reaction needs "gas" to go forward.
4.) Gas binds to adenylate cyclase
    a.) The name of this molecule gives you a helpful hint on what it does→ it catalyzes the *cyclization* of ATP to cAMP (cyclic AMP)
5.) cAMP is cyclized
    a.) SECOND MESSAGER!! This is what amplifies the signal
        i.) If you are struggling to remember, you can think of camp, and the use of those loud megaphones to call all the campers; Or, if it's easier, I see the lowercase c to uppercase AMP as a sort of "amplification" which helps me to remember. (Low→ high). It's a bit of a stretch, but it works for me!
6.) cAMP then binds protein kinase A (PKA)
    a.) This results in PKA going around and phosphorylating things. For this class, you do not have to know what exactly.

RESULT: Glycogen breakdown→ get a release of glucose!

PKA:
PKA has two subunits→ catalytic and regulatory.
As their names suggest, the catalytic subunits want to be "active" but are regulated by the regulatory subunit.

When cAMP binds to the regulatory subunits, they disassociate from the catalytic subunits, exposing their active site! This means they can now go catalyze reactions!!

For this, imagine you had an outlet with the babyproof outlet covers on. In this scenario, the regulatory subunit would be the two covers blocking the current, while the catalytic subunits are the two outlets.

So, cAMP in this scenario comes and pulls out the outlet covers, which allows the outlet to now be used. Aka. cAMP binds to the regulatory subunits which disassociate from the catalytic subunits, allowing them to do their job!

<u>Turning off the signal:</u>

To stop the signal from going on indefinitely, the signal must be terminated.

1. Ligand dissociates from the receptor. If the ligand is no longer bound, it is not in the "active" state anymore
2. However, the alpha subunit is still bound to adenylate cyclase. Therefore, to turn it off, the GTP must be converted back to GDP, and the other two subunits must come back and bind to the alpha subunit.
3. Next, cAMP needs to be decyclized to stop amplifying the message. This is done by *phosphodiesterases.*
    a. I think about how I know cAMP is made from ATP which has a lot of *phosphate* groups, and since it wants cAMP to stop working its going to make it <u>die</u>. → *phospho*<u>die</u>sterases
        i. It's not scientific, but if it helps you remember, it can't hurt.
4. Lastly, phosphatases remove phosphate groups from target proteins that were added by PKA.

Case Study:

-Cholera causes Gas to be stuck "on" → therefore, it causes the loss of mass amounts of water and NaCl into the intestine.

This leads to excessive diarrhea and it can be fatal due to dehydration.

<u>Epinephrine #2 Alpha Agernergic</u>:

1) Instead, the alpha subunit (Gaq now, not Gas) activates phospholipase C!

~How I remember that the *alpha* agernergic pathway is Gaq and not Gas, is that similar to "alpha" Q comes before S in the alphabet.

2) Phospholipase C cleaves PIP2 to become TWO second messengers→ IP3 and DAG
3) IP3 is no longer stuck in the membrane, and it travels to and binds the receptor on a calcium ion channel which floods calcium into the cell.

4) DAG is still stuck in the membrane, so it travels along it and binds to protein kinase C, which is also waiting for the calcium ion

→ Once they both bind, it results in smooth muscle contraction, limiting blood flow to these organs.
Conceptually: if we are in fight or flight, we want to stop focusing energy on our digestive system, which is part of why blood flow gets limited. Also, it allows us to oxygenate more blood to provide more energy!

Insulin Receptor:
No longer a GPCR→ now a receptor tyrosine kinase (RTK)

0.55-2:15 The Insulin Signaling Pathway
The Insulin receptor is a dimer, meaning it is two identical subunits that come together.
When insulin binds, either side of the dimer phosphorylates the other.
→ Next, this complex phosphorylates the tail of IRS
→ The phosphorylation of IRS is now a binding site for phosphoinositide 3 kinase
→ This then phosphorylates PIP2 to PIP3
→ PDK1 binds to PIP3
→ PDK1 phosphorylates Akt
More signaling → Increase glucose transporters of GLUT4

Remember.. Since PIP3 is NOT a protein, we need to use a lipid phosphatase as well to stop signaling!

Case Study:
Type 2 diabetes: signaling impaired→ less GLUT4 and less glucose uptake in cells
→ hyperglycemia!

CHAPTER 15
Why does your body need energy??
1. Mechanical work: moving your muscles or or cells moving around.
2. Active transport
3. Biosynthesis!

Metabolism & Nutrition, Part 1: Crash Course Anatomy & Physiology #36 You can stop at 4:45.
This video summarizes metabolism very well!

Catabolism: The break down of molecules
→ *Cata*bolism… *Cata*strophic
Anabolism: The building of molecules

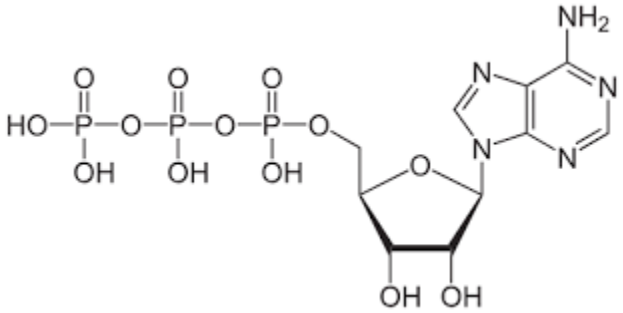
→ I do not have a fun trick to remember this one, just know it is *not* catabolism…
Metabolism: sum of all chemical reactions
→ *Met* in the middle ; It is both catabolism and anabolism.

Similarly, a pathway that is amphibolic has both anabolism and catabolism involved!
→ In greek, amphi- means "both"

Types of Pathways:

1. Linear: these reactions start somewhere, and end somewhere else.
    a. A→B→C
        i. You can think of linear pathways as walking on a straight path. Every step forward you take is a new reaction, and where you started is not where you are going to end up.
2. Cyclic: for this reaction type, the final step restarts the process again.
    a. A→B→C→A (which goes to B..C..A..B..C..A..B..C..)
        i. Cyclic→ you are going in a circle! You can walk around and around in a circle, and you will always end up back where you started.
3. Branched: This reaction proceeds like a linear pathway, but has one or more steps that can begin a "new" pathway.
    a. A→B→C but it branches at B such that this is also true A→B→C'
        i. Think of the branches of a tree, and how they all come from the same trunk, but do not all end up in the same place.

ATP
Metabolism and ATP Video



-Adenosine group on the right

-Sugar in the middle

-Phosphate groups (3→ tri) on the left

All of the oxygens surrounding the phosphate give it a negative charge→ this tells us that ATP wants to interact with things that are *positively* charged!!

Fun Fact: Mg2+ is used in reactions that utilize ATP to stabilize its negative charge.

- Also, ATP does not have a phosphodiester bond→ it is a phosphoanhydride bond!

Why ATP?
ATP is used due to the fact that it is *kinetically stable* and *thermodynamically unstable*.

Let's look into this further…

Kinetically stable:
So when we say something is kinetically stable, we are essentially saying that it is not going to easily undergo a reaction.
This matters because when we leave ATP alone, we do not want it to get randomly broken down. ATP gets stored, so it is important it has a good "shelf life".

Thermodynamically unstable:
ATP has a lot of energy stored in its bonds. In fact, ATP has a ΔG°' of -30.5kJ/mol. So, when it undergoes a reaction, it will release this energy.

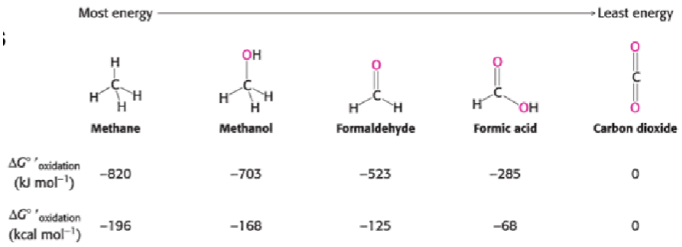
ATP Coupling
Energetic Coupling | Adenosine Triphosphate (ATP) | Metabolism Biochemistry | V-Learning™
This video goes over the concept through a quick example.

To summarize, ATP coupling uses ATP hydrolysis to "power" a nonspontaneous reaction (positive delta G).
To figure out the new delta G for the reaction, add the two values together.

For example:
Glucose → P-Glucose Delta G=15kJ/mol
ATP→ ADP + Pi Delta G= -30.5kJ/mol
If you couple these two together, there will be a final Delta G of -14.5kJ/mol, indicating it will now proceed spontaneously.

Energy from Carbon Fuels

C-H bonds + $O_2 \rightarrow CO_2 + H_2O$ + Energy (ATP)



The energy available here correlates to the amount of "free" hydrogens there are. As you can see CO2 has none, so it has no energy.

Conversely, methane has a *bunch* and they are not associated with oxygen, so it has the most energy.

TABLE 15.3 The B vitamins

Vitamin	Coenzyme
Thiamine (B_1)	Thiamine pyrophosphate
Riboflavin (B_2)	Flavin adenine dinucleotide (FAD)
Pyridoxine (B_6)	Pyridoxal phosphate
Nicotinic acid (niacin, B_3)	Nicotinamide adenine dinucleotide (NAD^+)
Pantothenic acid (B_5)	Coenzyme A
Biotin (B_7)	Biotin–lysine adducts
Folic acid (B_9)	Tetrahydrofolate
Cobalamin (B_{12})	5′-Deoxyadenosylcobalamin

I do not really have any fun tricks for this one (sorry!) but make sure to memorize B1, B2, B3, and B5 for the exam.

~You finished reviewing for this exam! Good luck studying!! :)