MODULE 2

MACRONUTRIENTS

2. MODULE 2: MACRONUTRIENTS

2.1. Module aims

- To introduce the macronutrients and explain their function in nutrition
- To describe the benefits of elevated protein
- To give the benefits of carbohydrates and to explain sugars and starches
- To give the benefits of fats such as monounsaturated, polyunsaturated and saturated
- To explain the way that you can determine macronutrient needs, both in athletic and sedentary populations
- To detail why the macronutrient intake required for recreational athletes is different to that of the general population

2.2. Key principles from module 1

In the last module, we discussed the importance of maintaining proper weight management, and gave you a solid understanding of the role of calorie balance in implementing that. As a very brief recap, we discussed the following key concepts:

- Nutrition plays a role in health, as defined by the WHO, not only by improving disease risk, but in terms of improving overall holistic wellbeing
- Nutrition can be considered hierarchically, with different levels playing dis-proportionate roles in maintaining optimal health and body composition, therefore deserving disproportionate amounts of your attention
- Lifelong dietary adherence is the single most important factor for long-term success
- Bodyweight management is crucial for reducing disease risk
- Calorie balance over prolonged rather than acute periods, is key to managing your bodyweight
- Eating a calorie surplus/deficit over a set period of time will lead to weight gain or loss
- There are several physiological factors that play into your calorie needs which can be roughly estimated to give an idea of how much to eat to gain, maintain or lose weight
- Your body will make efforts to keep your weight somewhat stable, meaning that giving specific numerical recommendations for intakes which are likely to lead to a given outcome is difficult, but some overall guidelines can be given
- Your calorie intake doesn't need to be the same every day, especially for fat loss
- Once weight loss has been achieved, you need to be able to transition to a method of eating which can maintain that weight loss
- **Everyone** doesn't necessarily need to count calories

- Calorie intake isn't everything

2.3. Introduction to macronutrients

In this module and the next we will be mostly expanding upon the last point listed above. As we mentioned in the last module it is very possible for an individual to be of healthy weight but have a waist circumference or other health marker which places them in a higher risk category. We all know someone who isn't overweight, doesn't have an obviously oversized waist but who would like to carry less bodyfat or be more muscular – the reason for both of these is at least partially the term we discussed in the last module, body composition.

While 'weight loss' per se is at least initially the goal for a lot of people, and while it can indeed reduce the risk that overweight populations have in relation to long-term disease, it is by far not the whole picture. An individual with more muscle mass and less bodyfat at a given total bodyweight will in general have a far better hormonal profile, blood lipid profile and immune system alongside a lower risk of various diseases. Moreover, a leaner, more muscular individual will be more capable of engaging in exercise which has almost innumerable benefits. Additionally, speaking only about calories doesn't give any information about the nutrient content of a food – and it is therefore to this subject that we turn over the next three modules.

In module 2, we will be discussing the macronutrients protein, carbohydrate and fat, which we are sure you have heard of. The macronutrients provide the calories which foods contain (we'll explain this in a second) but also have specific functions within the body. Without consuming the right amount of each within a certain range, we run the risk of having poor health independent of our total bodyweight. To make a more basic point, your macronutrient intake can play a large role in your health at a given bodyweight as well as the composition of that weight. If you have desires of carrying a relatively large amount of muscle mass, whatever that means to you, and having a lower bodyfat percentage, it is this module to which you should direct a lot of your attention.

Note: Alcohol is also, strictly speaking, a macronutrient. It will be briefly mentioned a few times throughout this module for the sake of completeness and to give you something of a working knowledge of its impacts but will not be delved into in great detail as it is a complex topic in and of itself and covered on the BTN Practical Academy.

The most important thing to note for now, outside of what is mentioned here, is that alcohol has profound effects on your health independent of its calorie value. Excessive consumption is associated with liver and cardiovascular health problems, while drunken behaviour is one of the leading causes of accidents, injuries, traffic incidents and violence (domestic and public). Moderate alcohol consumption is associated with improved health in comparison to being teetotal, but the amount of alcohol that constitutes 'moderate' is relatively low. The Chief Medical Officer (CMO) recommended in 2016 that you drink no more than 14 units of alcohol over 7 days, a value which goes for both men and women.

It is also heavily advised that everyone includes alcohol free days. Check www.drinkaware.co.uk for more information and to see how many drinks that actually constitutes, and join us on the Practical Academy to learn the biological mechanisms involved with alcohol metabolism.

Here and in the next two modules we will be focusing on constituent parts of foods because this gives you a grounded understanding of how things 'work'. You can consider this as looking at nutrition with a high level of magnification. In later modules, we will then zoom out and talk about the way you can actually implement the knowledge you will gain here by altering your food choices and building meals differently. While macronutrients and micronutrients make up nutrition, our diet is made up of food and we believe you need to understand both.

As a final introductory note, this module and the next two give in-depth theoretical overviews of nutrition, but we will not go into much detail about how to actually apply this to your diet – that's the role of the remaining modules. Here we aim to provide you with the theory: the detailed understanding of exactly what it is your body needs to thrive, and then we will arm you with the tools to give it what it wants every single day. Please don't be overwhelmed by the numbers, the terminology, the maths and the science, because it's all necessary and you have as much time as you need in order to understand it all. Take your time to read and assess, use the calculation flowchart in the summary along with a pen and paper (and calculator!) to write down and understand exactly where you need to aim for.

2.4. The problem with focusing ONLY on calories

Calories are an energy unit and the first law of thermodynamics states that within a closed system energy cannot be created or destroyed, only converted from one form to another. That means that the calories you consume and the calories you expend are a reliable predictor of the total amount of energy you will have stored within your body and as such, your weight. This is true and undeniable, but as we all know there is a lot more to consider than simply how much someone weighs.

The calories we consume are packaged into macronutrients which we refer to as protein, carbohydrate, fat and alcohol. The macronutrients are named macronutrients to differentiate them from micronutrients (which are the vitamins and minerals). From a molecular standpoint they are physically larger, and we consume them in gram amounts per day (as opposed to micronutrients which we may consume in mg amounts or less).

Each macronutrient has a given calorie value, usually expressed as the amount of calories that it provides per gram. The values are:

- **Protein** = 4kcal per gram
- **Carbohydrates** = 4kcal per gram
- **Fats** = 9kcal per gram
- **Alcohol** = 7kcal per gram

These are approximate measures because, as you will see, each macronutrient (especially fat and protein) is not a single thing, but rather a heterogeneous group of smaller things which may differ in their energy value. Some grams of protein that you eat may have slightly more than 4kcal and some might have slightly less, but these values are workable averages which are used in almost all calculations for practical purposes and are more than close and accurate enough to base your food choices from.

As a form of explanation, you will learn that protein is made up of amino acids. Each amino acid can have as many as 7 and as few as 3kcal per gram, but 4 is the average.

For example, if a portion of Greek yoghurt contains 8g fat, 7g carbohydrate and 15g protein, we can deduce that it provides (8x9) + (7x4) + (15x4) = 160kcal.

While all of these provide energy, this is not necessarily the prime role that they play nutritionally. Each can also be considered a substrate which has a given function within our body, for example:

- Dietary protein is used to build and maintain skeletal muscle, synthesise enzymes needed in various cellular actions, to build white cells used within your immune system and a ton of other things
- Dietary fat is used to give every cell within your body an outer membrane, to improve the absorption of some key vitamins, and after storage it can be used to keep you warm and to provide a store of usable energy to prevent starvation, amongst other functions
- Carbohydrates are the macronutrient which we can indeed consider to be primarily an energy source, but that is not to downplay their importance for optimal health and body composition – after all, you need a lot of energy every day just to stay alive and considering that alongside the fact that some cells can **only** use carbohydrates to function, you should be able to deduce that carbohydrates are something worth considering. After all that energy has to come from somewhere!

One problem with focusing only upon calories is that we then run the risk of skewing our macronutrient intake too far in any one direction. Having more of one nutrient within a given calorie amount necessarily means having less of another, and consuming insufficient amounts of any one of the macronutrients (granted, carbohydrates less so) will have negative consequences eventually, either in terms of your health, your energy levels or your adherence to your approach.

When you eat a meal, it will likely contain all of the macronutrients in some ratio or other, packaged into a different form. Carbohydrate may be consumed as starch or sucrose (table sugar) amongst others, fats covers a huge range of different molecules which may come as plant oils or animal fat, and protein will typically come in the form of either animal contractile tissue, albumen, the proteins found in milk such as beta-lactoglobulin, alpha-lactalbumin, bovine serum albumin and alpha s1 and s2 caseins, or the proteins found within plant cells. These raw materials all have corresponding enzymes which are secreted at some point in the

digestive system and are therefore broken down into smaller parts for use. In their larger forms, we cannot use them.

Note: Please bear in mind that from here we will talk quite a lot about biology and biochemistry. Though some of the content in this module may seem complex, understanding it will allow you to truly understand the topics at hand. Please don't be intimidated by the more in-depth looking content, as we will fully explain everything in the simplest terms possible.

A brief tangent: enzymes

Enzymes are proteins found in almost all life forms. They are catalysts, which means they speed up certain reactions by reducing the amount of energy that is required for the reaction to take place. As you will discover later, proteins are comparatively large molecules which adopt a specific 3D shape that in turn gives the protein it's function, much like the shape of a Lego brick or cog determines how well it works in the environment in which it is found.

Each enzyme has a certain shape which can be considered like a lock, into which specific keys will fit. The key is the specific thing that the enzyme interacts with – for example, the enzymes collectively known as DNA Helicase are produced within your cells, and act **only** upon your DNA, where they function to break it up to allow your cells to read the code. Without DNA helicase, your cell couldn't work properly because DNA couldn't be temporarily broken up, but if you applied these enzymes to any other structure, nothing would happen.

It's worth remembering that after an enzyme has performed its function by making a reaction happen it dissociates and often that enzyme can be used again. What's also really important here is that enzymes are proteins made within the body, so we don't need to consume them and we never 'run out'. Enzymes are made by almost every life form known right down to the bacterial level which shows just how critical they are.

When it comes to digestion, enzymes have a very simple function: they facilitate the breaking down of macronutrients into smaller molecules so that they can be absorbed from your digestive system into your body. It may seem strange to think of things in your digestive system as being 'not in your body' but it's a useful perspective to adopt. In effect, the digestive system acts like a hose which separates the things inside it from the surrounding environment, selectively allowing certain things to pass through its walls. This is why food you consume can make its way to your bloodstream, but a metal pellet for example, would pass out unchanged rather than making its way to your organs. This function is vital because we consume things that could kill us every day if they ended up in our bloodstream – your digestive system provides a barrier which keeps you alive.

Proteins are enzymatically broken down into their constituent parts, amino acids, via enzymes called proteases which are found mostly in the stomach. Starches become glucose when they come into contact with amylase which is found in the mouth and intestines (this is why bread will start to taste sweet if you chew it for a long time as the starch is being broken down into glucose which is a sugar) and fats are digested from the release of bile from the gall bladder

into the intestines, becoming fatty acids and glycerol. Don't worry, you don't need to remember all of this just yet.

As you can see, enzymes play a crucial role. If food particles were not broken down into their simplest forms via digestion, we could not survive and that's to say nothing of all the other functions enzymes play in more or less everything that goes on in your body on a chemical level! Now, back to the topic.

We will now detail the three macronutrients, their digestive fate, some roles they play in your body and the key reasons that you should pay attention to them nutritionally, before giving some guidelines for determining your ideal intake. Note that we will be giving guidelines which are somewhat specific and numerical, but that is not to say that weighing and measuring food is the only way to go. These recommendations are there because it is, according to the best information we can gather, what you should aim for in an ideal world, but that doesn't mean you can't be healthy unless you now the precise protein and fat gram content of your steak.

In the final section of this module, the recommendations are summarised and a flowchart is provided to outline the steps simply, so don't worry if you don't quite see how it all fits together as we proceed.

2.5. Protein

The first macronutrient that we will discuss here is protein. 'Protein' is named from the ancient Greek word meaning 'the most important one', and we tend to agree with that.

Protein is contained in most foods in some amount or other, but the kind of foods which would ideally be emphasised in a diet are those containing 'high quality protein' which denotes foods such as meat, fish, poultry, dairy, eggs, offal, tofu, some meat substitutes and some protein powders of both animal and plant varieties. Where possible, animal products should be used (including dairy and eggs) as your main protein sources, preferably in forms which are 'lean' (which simply denotes a lower fat content) but plant sources like tofu and powders can be used also.

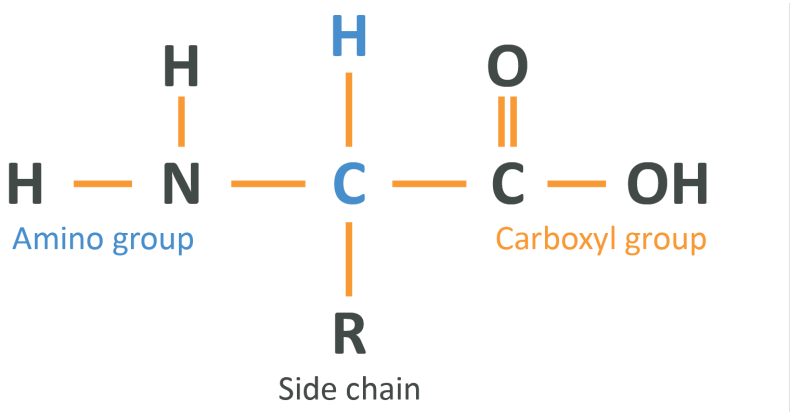
To be clear, this is of course not to say that vegetarian or vegan diets cannot provide adequate protein intake. This simply takes a little more planning.

While protein is the nutritional term, it is far more accurate to think in terms of 'proteins'. Animal bodies make millions of proteins every day in order to serve a huge range of functions. The process of making proteins occurs within most cells of your body and is one of the main things which keeps you alive day-to-day.

To give examples, collagen and elastin are 2 proteins which make up your skin, actin and myosin are 2 contractile proteins that allow your skeletal muscle to make you move, and we've already discussed enzymes, which account for the vast majority of proteins synthesised on any given day (to synthesise means to make out of smaller parts). In order to show you just how important it is to consume sufficient dietary protein, we'll talk about what proteins are, how they are made and the way which the food that you eat plays into the whole cycle.

Proteins are made of smaller units called amino acids which are arranged in a linear chain, like a string of beads. Every time a cell makes a protein, what it is doing is linking together a bunch of amino acids in a particular way. Amino acids, structurally, look like this:

Fig. 6



This can be viewed from left to right as such:

- The N-H-H (NH_2) is referred to as the Amino Group or N Terminal. It is made up of 2 hydrogens and 1 nitrogen
- The central molecule is carbon, and it attaches to a hydrogen and 1 variable side chain
- The right-hand side is the carboxyl group or C Terminal, which is made up of a carbon attached to 2 oxygens. 1 oxygen is bound in a 'double bond' denoted by 2 lines, which means that the 2 atoms share 4 electrons rather than just 2. The other oxygen is attached via a single bond and also attached to a hydrogen which, as we'll mention in a minute, sometimes separates from the structure

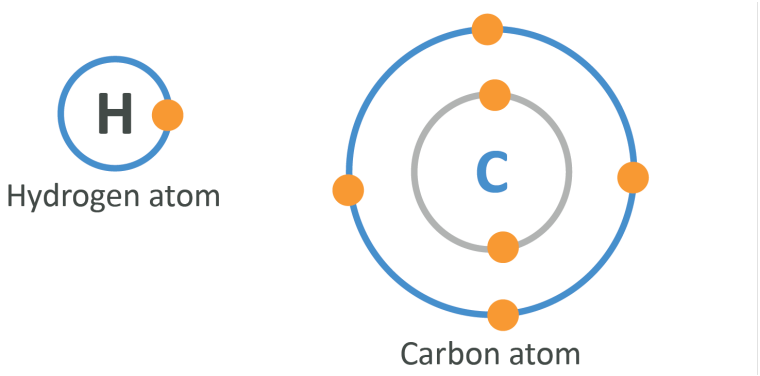
A quick chemistry reminder

Atoms are made up of a nucleus which is itself made up of protons and neutrons, surrounded by electrons which are located in the 'rings' of orbit. The outer ring of electrons is what determines how reactive an atom is. To join 2 atoms together, one of two things can happen:

- Either a metal atom and 1 or more non-metal atoms meet, in which case the metal 'loses' electrons and becomes positively charged, magnetically, and the non-metal(s) 'gain(s)' electrons to become negatively charged and they all become attracted magnetically – a process called ionic bonding, or
- As happens here, 2 atoms 'share' a pair of electrons, 1 from each original atom (single bond) or 4 electrons, 2 from each original atom (double bond) from their outer ring. This is called a covalent bond

One thing you need to know for the rest of this module is that carbon has 4 electrons on its outer ring meaning it can make up to 4 bonds, and hydrogen has only 1 outer electron so it can only bond to one thing at a time. Both are non-metals, and as such can only bond through the second process.

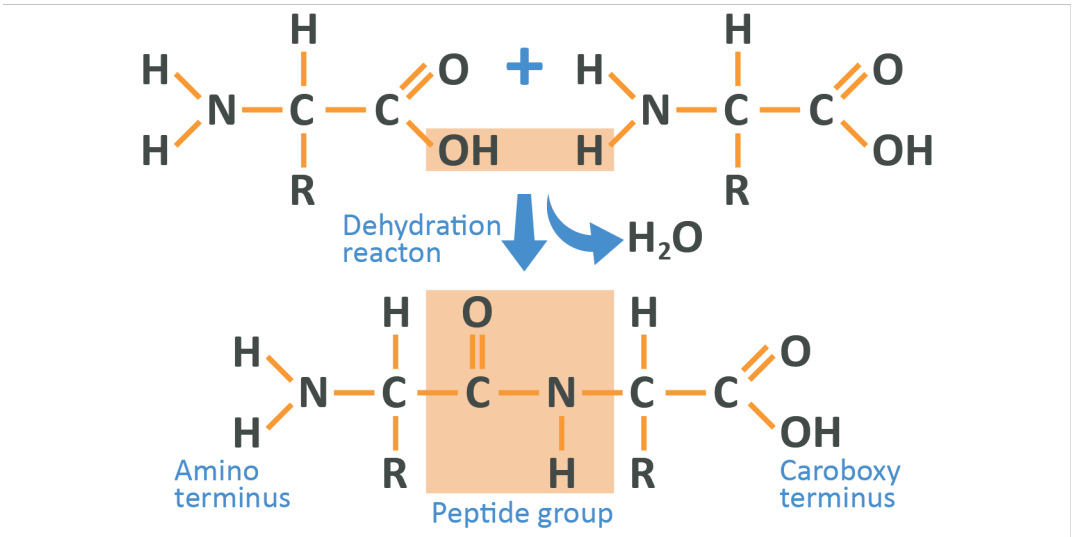
Fig. 7



When 2 amino acids are linked together, the hydrogen and oxygen from the carboxyl group of 1 amino acid combine with 1 hydrogen from the N Terminal of the next amino acid and are released (as H_20 – water – oxygen with 2 hydrogens), leaving behind 2 amino acids linked in what is known as a dipeptide, joined via a peptide bond. As such, a protein chain could be considered to start at the N Terminal of the first amino acid, and then continue until reaching the final C Terminal which is also 'free'. When a number of dipeptides are linked in a chain the resultant structure is known as a polypeptide.

Diagrammatically, the linking of 2 amino acids looks like this:

Fig. 8



See above that the OH of the left-hand amino acid and the H of the right-hand one have combined to make water (which is therefore 'lost'). This is the reason this is known as a dehydration reaction, and the remaining molecules have joined together to form the start of a chain. The main thing to remember from this section is that amino acids have a specific common structure, but their side-chains vary. Then when they are linked together they lose a molecule of water and start to form a chain.

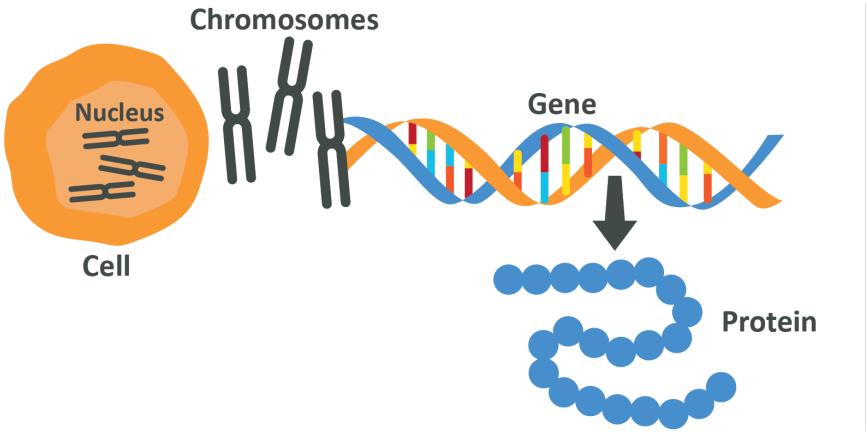
Each amino acid, as we've discussed, has a side chain – it is this side chain which determines which amino acid it is, and it is this side chain which gives it it's function. There are 20 different amino acids relevant to humans, each having a unique side chain. They are as follows:

Essential amino acids:	Non-essential amino acids:
Histidine	Glutamine
Isoleucine	Alanine
Leucine	Arginine
Lysine	Aspartate
Methionine	Cysteine
Phenylalanine	Glutamate
Threonine	Glycine
Tryptophan	Proline
Valine	Serine
	Tyrosine
	Asparagine

Your cells use these amino acids to make proteins in perhaps one of the most fascinating aspects of biology – gene expression.

DNA is formed out of 2 long strings which form a double helix, which then bundle together into chromosomes. Along each string are single units which follow a particular order – this is your genetic code, and various sections of that code can be thought of as being separated into specific genes. To conceptualise this, think of DNA being a library filled with genes which are books, written in words which are single units. If you want to make something you could go into a library, pick the requisite manual and read the words in order to know what to do. Your cells do the same.

Fig. 9



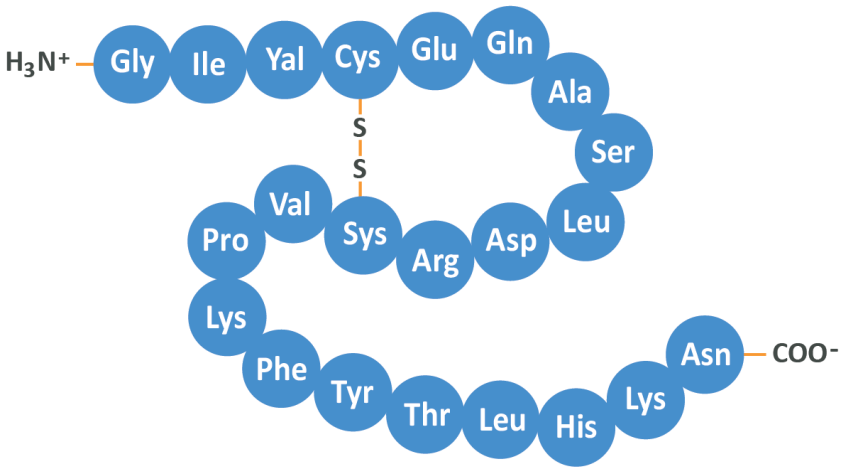
To make a protein, a gene is 'expressed' meaning that it's individual units are read and translated by cell substructures which then start to link together a precise set of amino acids in a specific order. Going along the gene (which is a DNA chain section), each 3 units of DNA information correlate to a specific amino acid. Because of this a gene can be considered to be a set of instructions for making a protein.

Once the cell has read the code and linked together the string of amino acids which it's been 'told to' use, the string will begin to fold in on itself in a specific way depending on the amino acids which it contains. Each amino acid, as you know, has a certain side chain and that side chain, because it's made up of atoms, will have a specific magnetic field and set of places where binding can occur. All of this means that there is only one shape which that chain can stably adopt.

This means that the folding of the string is precisely predictable from the constituent amino acids, which are in turn predictable from a given section of DNA (a gene). Every time that particular chain of amino acids is formed in that particular order from that particular section of DNA, the final 'string' folds in on itself in exactly the same way.

The order of the amino acids in the chain therefore determines the final shape of the protein and it is this 3D shape which ultimately gives a protein it's function. In fact, many ailments can be chalked up to this process happening ineffectively, resulting in proteins being formed which are misshapen and do not 'work' as they should. The diagram below illustrates how a protein forms.

Fig. 10



So, DNA codes for amino acids, which link together and then fold up into proteins. The shape of those proteins, along with the chemical properties their surface amino acids have, then allow them to perform a given job, be that to speed up cellular reactions, to help a muscle contract or to make your hair brown. The above is a little complex, but this is what protein 'is'. When we eat a rump steak, what we are doing is consuming the resultant matter from countless millions of proteins being formed according to the cells present in the butt of a cow. We can then use the amino acids formed therein to perform cellular processes of our own.

Forgetting about nutrition for a second

At all times, as we discussed in the last module very briefly, certain proteins within your body are being broken down as they become damaged or obsolete and are then being replaced by newer proteins. As you can now probably deduce, the proteins are broken up into their amino acids, most of those amino acids are then recycled (some are broken down further and the individual atoms are used elsewhere), and they can be recombined into either the same or different end-products. Say, for example, your body breaks up a slightly damaged collagen protein which was a part of a tendon. The amino acids therein could be used to synthesise another collagen to use elsewhere, they could be used to make a completely different protein that might be needed at the time or they could be sent to your liver to be broken down and made into something else entirely. Your body really is incredible.

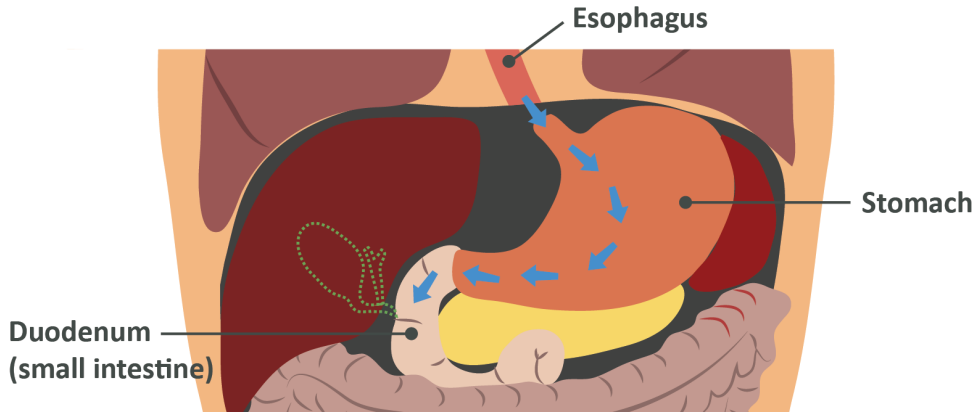
If your aim is to build muscle mass, what that means is that the cells in your muscles need to have the amino acids on-hand that they need to synthesise new muscle proteins.

Now, look back at the list of amino acids and notice that they are grouped into two categories – essential amino acids and non-essential amino acids. Don't let the names lead you to think that non-essential is not important. Essential, in terms of nutrition, simply means that it must be consumed as it cannot be synthesised. As you have seen, an amino acid is just some nitrogen, carbon, hydrogen and oxygen atoms smashed together in a particular way with a variable side chain, and as such we are able to break down amino acids, sugars and other things to 'build' amino acids that we need. We unfortunately lack the necessary mechanisms to be able to build certain amino acids though, and as such, we need to get them from food.

When we talk about foods with 'complete' protein profiles or proteins that are high 'quality' we are referring to them containing the 9 essential amino acids in ratios which make them useful for building proteins in our body. A complete protein source is one which allows our cells to perform the above routine when required, because they have all of the necessary 'building blocks'… and **that** is why dietary protein is important. Without it, you would not be able to synthesise the proteins which make up your skin, organs, muscles, enzymes or blood, nor the proteins which are needed so that your cells can communicate with each other. Protein deficiency would lead to an effective shutting down of most of your metabolic processes and ultimately death. This is illustrated when scientists feed animals an artificial diet containing only collagen protein which is very low in isoleucine, threonine and methionine while being completely void of tryptophan, which are essential amino acids.

When you eat a food containing protein, for the sake of this example we'll say a steak, first you chew it to break it down into smaller chunks with a greater surface area, and then you swallow it into the oesophagus and eventually the stomach. When the steak reaches the stomach, it comes into contact with the inactive enzyme precursor pepsinogen which is rapidly activated to become the active enzyme pepsin. Pepsin breaks the bonds in between sections of amino acids, leaving slightly shorter chains or polypeptides which then pass through the pyloric sphincter and into the first part of the small intestine known as the duodenum. The duodenum is the main site for nutrient absorption and digestion.

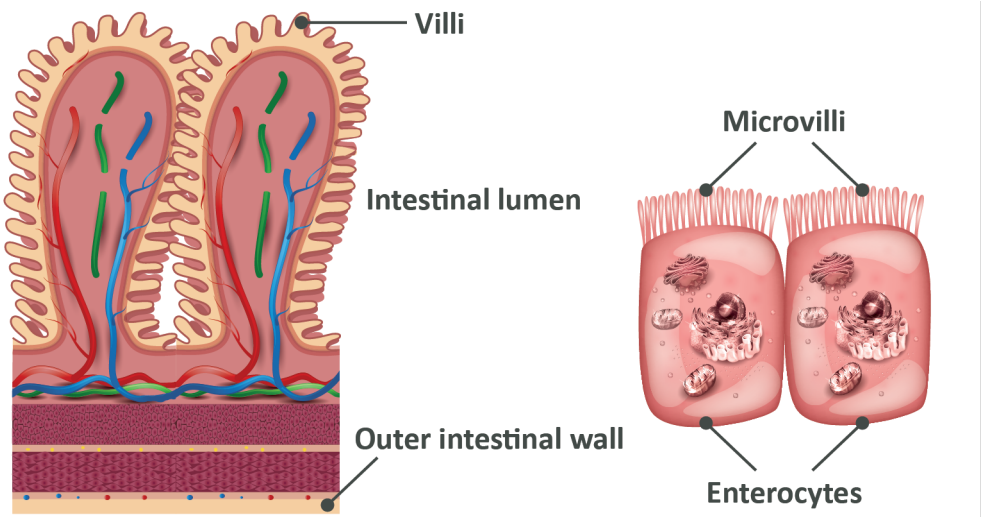
Fig. 11



Once the polypeptides are in the duodenum, the pancreas introduces 3 more enzymes called trypsin, chymotrypsin and carboxypeptidase (car-boxy-pep-tie-daze) along with bicarbonate ions which are alkali to neutralise the stomach acid – if this did not happen the proteases here would be damaged. These break down the peptide bonds further, leaving us with very small polypeptides only a few amino acids in length.

The cells on the walls of the small intestine are called enterocytes and they look like the below. What you see here is a surface which has small protrusions on it known as villi, which act to increase surface area. This increased surface area allows for greater rates of absorption from the small intestine. On these small protrusions are even smaller 'hairs' called microvilli, and amongst these you will find special enzymes called brush-border enzymes.

Fig. 12



These brush border enzymes are responsible for the final stage of breaking proteins down into amino acids, di and tri peptides which can all be absorbed into the cells. Once inside the cells, the di and tri peptides are broken down into single amino acids and finally they pass through the other side and into the blood.

Once the amino acids are in the blood, they are taken first to the liver to either be used to make liver proteins, broken down into glucose or put back in the blood to be distributed around the body for cellular processes. The amount of protein which is turned into glucose via a complex series of reactions called gluconeogenesis (gluco-neo-genesis) varies depending on how many grams of protein you just ate as well as how many grams of carbohydrates and how many total calories you eat. All you need to remember here is that excess protein eaten above levels which you need to maintain bodily tissues and build muscle is converted to glucose and used as glucose, meaning that you can't **really** eat too much protein, and that excess protein eaten above need is not simply wasted but converted to glucose to use to make energy.

Similarly, in between meals when blood glucose starts to drop, your body is able to release amino acids from proteins and re-absorb them into the blood via the hormone glucagon, which we'll return to. These amino acids are then absorbed by the cells of the liver and converted into glucose, which can be used to fuel various cells around the body.

As a summary of protein in the body, consider that most structures within your body are made up of proteins, which are themselves made up of strings of amino acids. We are able to make some of these amino acids from other things, but a lot of them we need to get from the things we consume. When we consume them, proteins are broken down in to their constituent amino acids and these are then distributed via the blood to cells where they are required for protein synthesis. Without getting adequate dietary protein, our body must get the amino acids it needs from those which are already stored, which as you can probably deduce means increasing protein breakdown from immediately non-essential things (such as muscle mass beyond the bare minimum) and using this to facilitate protein synthesis in more vital places like your organs and blood. If you eat more protein than you need, it's converted to glucose and used in the same way as glucose is always used.

2.5.1. Protein in the diet

With the slightly more complex stuff out of the way, let's talk about the role which protein plays within your diet, and the reasons that it should be included (barring the above-mentioned idea that deficiency will kill you).

Perhaps the most commonly discussed role for protein is its ability to increase muscle protein synthesis, though it may not always be spoken about using that terminology. Protein turnover which we have already outlined happens in every tissue, but the tissue we will focus on for the rest of this module is specifically skeletal muscle, because it is chiefly muscle protein synthesis which you should really concern yourself with for practical purposes. The amount of protein required on a daily basis to cover your needs for organ tissue maintenance and enzyme production is comparatively low, and provided you are eating sufficient calories and a relatively balanced diet you aren't likely to become deficient.

The main reason we need to talk about protein is due to its impact on skeletal muscle tissue.

Muscle protein synthesis is the process by which amino acids are used to build or repair skeletal muscle specifically. As already discussed, a certain amount of protein is broken down

within muscle tissue during a given day, and therefore at various points you will be in fact losing muscle. This is not something to be concerned about, however.

Much like it is the case that, calorie balance works over an extended period of time (meaning that you could overeat some days and under-eat others and still lose fat if the net result is a negative), muscle protein balance is cumulative and best thought of over the course of a day (it's not advisable to consider your protein needs over the course of a week, as muscle protein synthesis can only work 'so quickly', and if you severely limit it for 4-5 days, 2 days of high protein intake won't compensate for it).

Over 24-hours certain things that you do will increase the rate at which protein is broken down – you could be stressed, you could fast for extended periods, you could exercise – and from the beginning of that stressful process to the end you could end up with less skeletal muscle protein. But all is not lost!

Ingesting protein initiates muscle protein synthesis. Assuming your overall protein intake is adequate and you are not in an extreme calorie deficit (this can also be skewed if you have certain health issues or are extremely lean), all of the lost protein is then replaced, and you carry on your life without suffering muscle wastage.

When you perform resistance training, your muscles become somewhat more receptive to dietary protein, and the ceiling for the rate of muscle protein synthesis is increased. Now muscle proteins that are broken down are not only replaced but super-compensated for, resulting in a greater amount of stored protein, and therefore a larger muscle over time.

It's not just about having available building blocks, though.

Muscle protein synthesis must be 'activated' and the key signaller for this response is the consumption of the amino acid Leucine. In research where subjects consumed the same amount of protein, with one group consuming more leucine and the others less, the former group got a greater muscle protein synthetic response, and when two different proteins are compared but the leucine content is matched, the response is equal. It's a widely accepted fact that leucine is the amino acid mostly needed to activate muscle protein synthesis, and as such we should wherever possible aim to choose proteins with a reasonable leucine content if we want to maximise muscle gain/retention. This is why animal-based protein sources are preferable – while some are better than others, with whey protein and eggs being richer in leucine than pork. For practical purposes this is much of a muchness, and so long as the bulk of your protein intake is from animal-based sources or well-balanced vegan ones (again, something we'll discuss in later modules) then you have no need to start searching for the leucine content of your foods – this is **not** something you'll find on the label!

This isn't just about making gains, though. If you are losing fat at the minute then that means your overall energy intake will be less than your 'maintenance' level. This reduction in calorie intake has the ability to increase muscle protein breakdown and reduce muscle protein synthesis and as such, the ability of dietary protein, especially leucine rich protein, to increase muscle protein synthesis upon ingestion becomes of greater importance. Making sure that we consume enough protein during a dieting phase allows us to safely lose weight without

running the risk of excessive muscle loss (of course, as you might guess, resistance training augments this protective effect, too).

So, eating sufficient protein while in a calorie surplus maximises the amount of muscle you can gain from that given surplus with a given training stimulus, and conversely eating sufficient protein while in a calorie deficit allows for minimised muscle tissue loss (which would lead to losses in strength and a comparatively unfavourable body composition relative to the same leanness with more muscle mass).

That isn't all protein does, though. Some other key roles for dietary protein are:

- Dietary protein has a higher TEF than other nutrients. Because of this, it means that you could theoretically eat the same amount of total calories with a high and low protein intake, but the net result would be lower for the higher protein diet. This is relevant, but not really ground breaking – seeing as the TEF for protein is around 20% and the TEF for carbohydrates is around 10, it probably wouldn't make a difference to the point that your body composition would likely change to a noticeable degree
- Dietary protein is the nutrient which, calorie for calorie, reduces hunger the most in between meals. In one study, just 20g of whey protein reduced the amount of food participants consumed in a subsequent meal. It does this by increasing the circulation of appetite reducing hormones including GLP-1, peptide YY and cholecystokinin while reducing grehlin, the hormone which makes you hungry. One study found that simply by giving participants a protein intake equal to 30% of calories they were able to reduce their calorie intake by 411 calories without effort!
- Foods which provide protein also come with a ton of important micronutrients, some of which (like Iron) are especially necessary for some individuals to pay attention to
- Increasing your protein intake seems to reduce cravings for other foods, especially late at night which we all know is the time of day where dietary adherence is the hardest. In one study, cravings were reduced by 60% throughout the day by just increasing protein intake to 25% of calories, which could make a massive difference
- Protein can be used to make glucose if needed

2.5.2. How much should we aim for?

According to the UK Eatwell Guide, the current recommended nutritional intake (RNI) of protein for adults aged 19-64 is 0.75g per kilogram of bodyweight per day, calculated to approximately 55.5g per day for the average man and 45g per day for the average woman. This is the amount of protein on average to ensure you do not end up with a negative nitrogen balance (remember that there's nitrogen in amino acids? If you are wasting more protein than you are building, you end up with increased nitrogen excretion, which is a surrogate measure for protein balance used because measuring protein balance is comparatively difficult). The RNI is therefore enough protein to maintain health for the vast majority of the population. The RNI, however, is calculated to the average, mostly sedentary individual and of course the amount needed to avoid deficiency is not the same as that needed to optimise your diet.

The amount of protein that you personally require is largely dependent on your activity levels, exercise modality and calorie balance. Protein synthesis is energy dependent and an increased energy expenditure also increases protein breakdown as amino acids can theoretically be used as fuel. Finally, contracting muscles with large amounts of force or against resistance causes small amounts of damage which further augments the muscle protein breakdown process. As such, those who exercise regularly require more protein to maintain a level protein balance, and slightly more to actually develop larger muscles. At the same time, a given individual would benefit from slightly more protein, the fewer total calories they consume.

Protein requirements are best expressed as a gram amount per kilogram of bodyweight, but there's a caveat here. We would highly recommend that if you are quite overweight, you consider your protein intake in terms of your 'goal weight' or ideal weight according to BMI. Protein requirements are met when you have provided your body with enough amino acids to do what it needs to do, and that's dictated by your activity levels, food intake and the amount of muscle/fat free bodyweight you have. Additional weight caused by additional fat mass doesn't increase protein needs but it would skew grams/bodyweight calculations. If your bodyfat is outside of the healthy range, you need to account for that, and 'goal weight' is as good a surrogate measure as any.

The protein amount which seems to be beneficial for the general population according to Laymen et al who did a large review on the metabolic functions of protein is 1-1.2g per kilogram of bodyweight, though an overview on the impact of dietary protein on weight loss and weight loss maintenance by Leidy et al suggests that this could increase to 1.2-1.6g per kilogram for individuals looking to lose fat. This was agreed upon at the Protein Summit 2.0 which was a large conference of over 40 nutrition researchers held in 2007, indicating that the numbers recommended by the SACN (UK nutrition body) may be too low to optimise health, although they are certainly enough to avoid becoming deficient. The evidence on this is pretty clear, in isoenergetic diets (multiple diets with the same calorie amount), higher protein diets reliably cause improved body composition and health results even in those who are not performing regular progressive resistance training.

If you are a highly active individual partaking in sport then we need to look at sport specific research. The protein requirements recommended by the International Society for Sports Nutrition, and therefore the ones we recommend are 1.4-2g protein per day per kilo of bodyweight, the higher figure for individuals taking part in resistance training and the lower figure for endurance athletes. If you are looking to maximise hypertrophy (gain the most muscle possible) then this could potentially go a little higher, to 1.8-2.2g per kg or even higher if the individual chooses, but there would be no additional benefit under most circumstances.

It's worthy of note that intakes higher than this have been cited as potentially useful for muscle mass retention when fat loss is the aim for someone who is already very lean, but this is beyond this course, and is indeed theoretical. It's unlikely that anyone but the most advanced of athletes would benefit from anything above 2.2g per kilogram.

2.5.3. Is it just about total daily amount?

You will recall that one of the higher levels of our nutritional pyramid is meal frequency, timing and distribution and we will now cover some aspects of that.

In order to maximise muscle protein synthesis, it appears that one should consume protein approximately every 3-5 hours, totalling 3-5 protein feedings per day spread out relatively evenly. This is because once muscle protein synthesis is maximally stimulated it seems that it cannot be stimulated again until it has returned to baseline, which takes a certain amount of time. These meals should contain at least 20g of protein to maximise the satiety response, but around 2-3g leucine is needed to maximise the muscle synthetic response in the majority of the literature (this number can go as high as 3.5g in some studies). Of course, going back to the fact that leucine isn't something you'd ever find on a food label, this would be easier to consider in terms of 'quality protein', which should be consumed at around 0.25-0.4g per kilo of bodyweight, per meal. Finally, a dose of protein within 2 hours pre and post exercise seems beneficial for creating adaptations to that exercise.

Relating to the above, we recommend the following as a 'perfect protein distribution':

- 3 main meals containing 0.4-0.5g protein per kilogram (or 2.5-3.5g leucine), 1 post-workout feeding containing the same, and a small snack somewhere (potentially before bed) containing somewhere around 0.3g per kilo. This would give us an ideal total protein intake of around 2g per kg spread out over 5 feedings which maximise protein synthetic response, the maximal amount of protein induced satiety possible and pre/post-workout protein covered. Now for some context

As we keep mentioning, the key to dietary success is dietary adherence, and one of the big factors for this adherence is a reduction in dietary complexity. Looking at the above, you can see that what is scientifically valid and recommended may not be something which you could actually apply in the real world (at least not for your entire life) because it would require an awful lot of preparation and planning, and that isn't going to happen if you are on holiday, if you're facing a stressful time at work or if you are generally really busy. In order to simplify things, we need to consider the impact which having 'the perfect protein distribution' would actually have. Would it make much of a difference if you got it perfect in comparison to getting it 'pretty close'?

Firstly, it's not entirely clear from the research that the proper distribution which in theory maximises muscle hypertrophy, is the same distribution which minimises muscle loss, so if we are talking about someone who is losing fat it gets a lot easier.

In fact, in studies where participants went almost without any food at all on alternative days (alternative caloric restriction, a form of intermittent fasting) they had no problems maintaining muscle mass. It's pretty clear that if you are resistance training and eating enough protein overall, muscle loss isn't really a concern (and even if you aren't resistance training, this probably isn't going to be too much of a concern for you as you will more than likely not have a huge amount of muscle mass to begin with – the less muscle you have the easier it is to maintain). Then when looking at muscle **gain**, there is very little conclusive evidence that 4

meals is significantly better than 3, or that 5 is significantly better than 4 for health, body composition or much of anything over the long-term – in theory there would be a difference, but intuitively it won't be a huge one.

Because of this, meal frequency shouldn't really be something you worry too much about if you aren't aiming to be 'the best of the best', though breaking your protein out evenly isn't too taxing and could likely have at least a modest beneficial impact.

Similarly, it's a huge mistake to think that per meal protein dosing necessarily needs to meet the 'roughly 3g leucine' threshold. It's not the case that it's all or nothing, and a meal which contains only 1-2g leucine (contained in a relatively small dose of quality protein) isn't 'wasted', albeit just not optimal. Our position is, therefore, that people who are not high level athletes or highly dedicated recreational exercisers should primarily concentrate on consuming somewhere between 1.2 and 1.8g protein per kilogram of bodyweight throughout the day, ideally in doses of over 20g at a sitting to maximise satiety.

To quantify that, an 85kg male could aim for around 135g protein per day, which could be achieved with 3 meals containing 35g protein and a 30g protein snack.

As for pre and post-workout nutrition, again we need to consider the real-world magnitude of difference. While there does seem to be some benefit of consuming protein before and after training, you need to look at the bigger picture. Is it stressful to you or impacting on your life to worry about a pre/post-workout meal? If so, and you aren't a high level athlete, don't. It won't make a huge difference – though if pre-workout nutrition is skipped, it's not too much to ask to make sure you have something as soon as is convenient/possible after training. This is somewhere that a protein supplement could be used for the sake of convenience.

A highly dedicated recreational exerciser who wants to come a little closer to optimal should do something similar, but with protein at 1.8-2.2g, and maybe consider placing some emphasis on having **something** within an hour or so of either side of training.

The best way to introduce a greater protein focus into your diet is to consider it the 'backbone' of a meal. When deciding what to eat, first think about your lean protein source, and choose protein based snacks wherever possible because it is these which will have the greatest impact on your satiety.

While nutrition is complex, it's application doesn't have to be.

The means that you can adopt to realise these recommendations will be spoken about at length in later modules, so for now please just remember the roles and benefits of protein and the recommended intakes.

2.5.4. The safety of a higher protein intake than the RDA

To conclude, it's a good idea for us to turn our eyes to the possible health impacts of your protein intake as these can sometimes be a concern. There are two things to talk about here:

- Increasing your protein intake to the ones recommended here has never been shown to negatively impact health in any way. Your kidneys may need to do a little extra work in order to process the additional protein metabolites (waste materials from the

breakdown of proteins) but they are more than capable of doing this and it will cause no damage. Consider that having 5 cars on London Bridge is more stress than just one, but the bridge isn't any closer to collapse because of it. There have been a number of trials into the safety of elevated protein intakes, and intakes of up to 4.4g per kg (that's a **lot**) had no negative outcomes

- If you increase your protein intake, you will therefore increase your intake of protein-containing foods. It is possible, in this situation, to make better and worse choices. In general, you should opt for leaner, uncured protein sources – but really to discuss this we need to mention the impact of processed and red meats which we'll go through in turn. According to the WHO, red meats are a likely carcinogen which means that according to their population research, red meats are associated with a higher risk of cancer, but they can't pinpoint why. It is our view (and that of many) that this should be taken with a pinch of salt, because of certain flaws in the studies presented

Red meat intake is associated with higher risk of cancer, but it's also associated with lower exercise levels, lower vegetable intakes, more smoking, more alcohol and a number of other risk factors. It would be very difficult to draw a definitive conclusion, and as there is not currently a mechanistic theory as to why this is, we would suggest that consuming red meat 2-3 times per week will be harmless, and a higher amount (assuming a high intake of vegetables/fibre and general healthy lifestyle) **probably** would be too. Red meat is an excellent source of iron and B vitamins, and while the potential risks of large amounts of red meat consumption means that there more than likely is too much, the best intake for health is very unlikely to be none at all.

As for processed meat (sausage, bacon, pepperoni, ham, for example), the link is a little stronger. Research shows that your risk of colorectal cancer increases by about 18% for every 50g portion you eat, though this is not as worrying as it first appears. The total lifetime risk is your risk of getting a disease just because you are alive, and any increase in risk is expressed as a percentage of that risk. This means that rather than processed meat increasing your risk to 18% at 50g then 36% at 100g and so forth, it means that your lifetime risk which in the UK is about 7.14% as a male (1 in 14 people) will increase by 18% to just over 8%. This is a small increase which we need to consider alongside the above-mentioned lifestyle factors, but it is foolish to ignore it. As such, in order to attain optimal health, we recommend that you minimise your intake of processed and cured meats to as little as is feasible, while also remembering the context of the small impact this will have (for instance, smoking increases your lifetime risk by around 1900%). In the most basic of terms, a bacon sandwich once or twice per week is a very small risk, and it's really up to you whether you want to take it in the context of a diet which otherwise reduces that risk, but it's not really advisable to eat bacon every day.

Moving on, let's talk about dietary fat.

2.6. Fats

Dietary fats are contained in meats, eggs, nuts, seeds, dairy and fish and found in both solid and liquid (oil) form. They are a complicated topic owing to a great deal of cultural stigma

surrounding them, and what we will attempt to do here is to give you an impartial overview of what dietary fats are, what they do and how they can affect us. Again, we will go into a little detail about the structure of fats and their digestive fate, but we will do this as simply as possible and hope we make a great deal of sense while making the topic interesting.

Historically, dietary fats have been blamed for increases in obesity and ill health, but we hope that the previous module has shown you that the crucial aspect of weight maintenance is not macronutrients specifically but the calories they contain. In fact, the link between fat intake and fat gain is literally down to the fact that, as you saw in the introduction to this module, dietary fat has twice the calories per gram compared to carbohydrates and proteins, and therefore they are easy to over eat.

Hardly a convincing case against fats…

Rather than being something which should be cut as low as possible in an attempt to reduce your chances of ill health, fats are a nutrient which should be considered vital (indeed, some are essential in the same sense as the above amino acids) but we must again be wary of being too hasty and we should remember that just because eating fat doesn't cause fat gain directly, it doesn't mean we get a free ticket to eat as much dietary fat as we want.

Fats should be considered just as important as the other macronutrients, and after explaining their function and role we will give you some recommendations for determining just how much you should eat and in what forms.

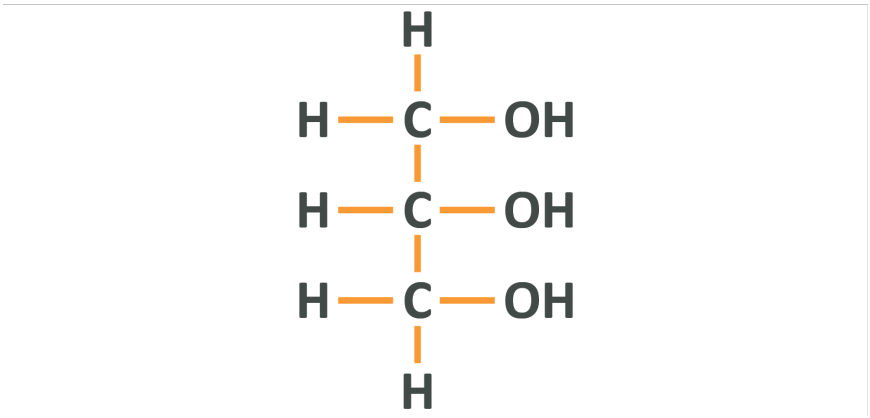
2.6.1. What is a fat?

Fats are an example of lipids (along with waxes and sterols) which are all organic molecules insoluble in water but soluble in organic solvents (in chemistry, 'organic' strictly denotes a molecule with a carbon base, it has nothing to do with whether something is alive, or whether something has been farmed in certain ways). The reason for this insolubility will be explained momentarily, but you may already be aware of it. If you have ever seen car oil sitting on top of a puddle or have ever seen an oil/vinegar salad dressing separate where the oil sits on top, then you have seen the hydrophobic (water hating) nature of fats at work.

What this insolubility gives fats in our body is a very unique niche. Fats are used in specific orientations to create the membranes which surround every cell in your body, for example. Because of the hydrophobic property of the membrane, the contents of a cell represent a different environment to the rest of the body, meaning that special chemical reactions can occur there. Make no mistake, the ability that fats have to separate inside a cell from outside is literally the fundamental factor by which every single life form is able to function. From an evolutionary standpoint, the formation of small groups of fatty acids into a membrane is more than likely what led to the formation of the first living cell, from which all life descended.

The fats which we consume in our diet are known as triglycerides which is a term you may have heard before, or the perhaps more accurate name tri-acyl-glycerol. The final term is more accurate because dietary fats are comprised of glycerol (sometimes called glycerine) attached to 3 fatty acids. Much like proteins, triglycerides are made up of hydrogen, carbon and oxygen, but they lack the nitrogen present in the former.

Fig. 13



What you are looking at here is a 3 carbon chain. Attached to the first and third carbons are 2 respective hydrogens and the central carbon has only one because it is bonded to the carbons next to it (remember, each carbon is limited to 4 bonds). Moving to the right, you will see that each carbon group has a hydroxyl group, which is simply the name for oxygen and hydrogen bound together. To make this a triacylglycerol or triglyceride, each hydroxyl group becomes fused to a fatty acid.

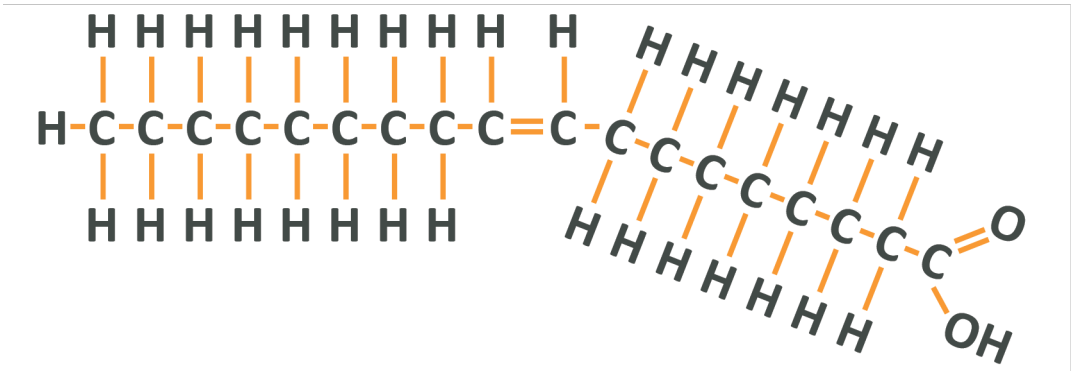
2.6.2. Structure of fatty acids and how we name them

Let's look at the chemical structure of Oleic acid as a means of giving an example of fatty acid structure. Oleic acid is the fatty acid found in high amounts in olive oil and is one of a few fatty acids which is associated with improved health.

Like all fatty acids, oleic acid has a certain structure. At one end, you have a carboxyl group, which is made up of a central carbon atom that has 1 oxygen double-bonded to it, and another oxygen which is single bonded to the central carbon and to an additional hydrogen atom (A C attached to an O with 2 lines, and also attached to an OH). After this group, there is a chain of carbon atoms which are attached to the maximum amount of hydrogen atoms possible according to their electron availability, and at the other end there is a methyl group which is a central carbon and 3 hydrogens. The amount of carbons in the chain varies between different fatty acids, but this carboxyl group – carbon chain – methyl group structure is how all fatty acids look. Sometimes the methyl group is referred to as the omega-end, which will become important momentarily.

Because each carbon has all of its outer ring electrons occupied the whole structure is extremely stable and as such will not dissolve in water, which would require some amount of bonding between the fatty acid and the free electrons on water molecules. It is this non-solubility in water that means fats can carry out the biological functions that they do and is likely the single most important fact about fats (it's also why they are referred to as 'fatty').

Fig. 14



As you can see, oleic acid has its carboxyl group (COOH) on the right followed by a chain of carbons in single bonds. In the middle of the chain, however, there is a double bond which means that, because each carbon effectively 'loses' 1 more electron than it usually would, each can only bond to 1 hydrogen. The 2 lone hydrogens appear on the same side of the chain and repel each other, thus creating a 'kink' in the shape of the final molecule. After this, there is another set of carbons and a final carbon which forms part of the methyl group. Note that if you count from the methyl or omega end of the fatty acid, the double bond is on the 9th carbon.

When we know the chemical makeup of fats, we can group them into their respective categories:

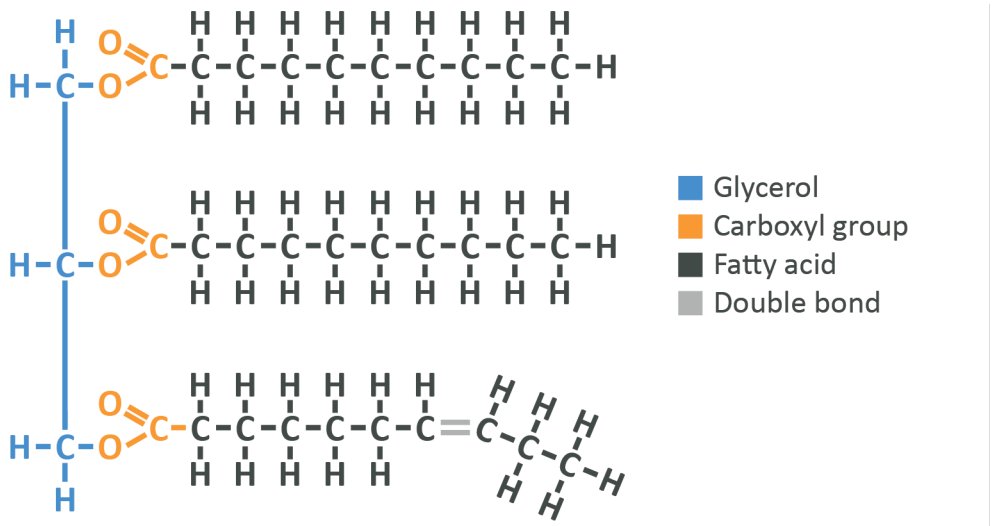
- **Short-chain fatty acids (SCFA)** are fatty acids with a tail of fewer than 6 carbons (i.e. butyric acid)
- **Medium-chain fatty acids (MCFA)** are fatty acids with tails of 6-12 carbons
- **Long-chain fatty acids (LCFA)** are fatty acids with tails 13-21 carbons long
- **Very long-chain fatty acids (VLCFA)** are fatty acids longer than 22 carbons

If a fatty acid has only single bonded carbons it is saturated with the maximum possible amount of hydrogen and is therefore a saturated fat. If it has 1 double bond it is monounsaturated and if it has multiple double bonds it is polyunsaturated. If the double bond in an unsaturated fat falls on the 3rd, 6th or 9th carbon away from the omega end, it is an Omega-3, 6 or 9 respectively.

Oleic acid has a total of 18 carbons, which makes it a long-chain fatty acid. Because it has a double bond and therefore fewer than the maximal amount of hydrogens it is an unsaturated fatty acid (it isn't saturated with hydrogen) and because it only has 1 double bond, it is a monounsaturated fatty acid. Because the double-bond occurs starting at the 9th carbon in the chain, it is a long-chain omega-9 monounsaturated fatty acid.

The amount of saturation and length affects the stability and structure of the fatty acid when exposed to cooking temperatures or the way they behave when used in the body. Generally speaking, saturation provides stability.

Fig. 15



As you can see here, the glycerol remains on the left-hand side, but now the OH group has lost a hydrogen and the fatty acid has lost an OH group. This means that in total we have lost 2 H's and an O. As you learned in the section on amino acids, this means that a water molecule has been created (not pictured), while the fatty acid has bonded to the glycerol.

Note: Each fatty acid bound to a given glycerol can be different. There is no need for all 3 to be the same molecule.

Now you have a broad idea of what a saturated and unsaturated fatty acid is, let's look a tiny bit closer and introduce a new form of fatty acids – trans fatty acids.

2.6.3. What is a trans fat?

As you've already discovered, saturated fatty acids have no double bonds and therefore they tend to form straight chains, and because of this shape they are easy to stack on top of each other in an orderly fashion. This simple act of physics is why saturated fats are usually solid at room temperature – animal fat and coconut fat are made up largely (though not entirely) of saturated fat.

If a fatty acid is unsaturated, it means that there is a double bond which tends to create a kink in the chain, resulting in a liquid makeup at room temperature. In the simplest terms, the kink in the chain makes the acids much harder to stack together into a solid shape. This is how things occur in nature, but nature can be altered.

A 'cis' unsaturated fatty acid is one where the missing hydrogens in an unsaturated fat occur on the same side. Because they occur on the same side this leaves a gap, and the corresponding hydrogens on the other side of the chain are allowed to repel each other, causing a bend in the fatty acid chain and a liquid nature of the resultant oil as per the above – this is the standard way for things to be. In the food industry, fats which are solid at room temperature are preferable because they have a longer shelf life and better mouthfeel, but plant oils which are liquid such as sunflower oil or soybean oil are far cheaper to manufacture.

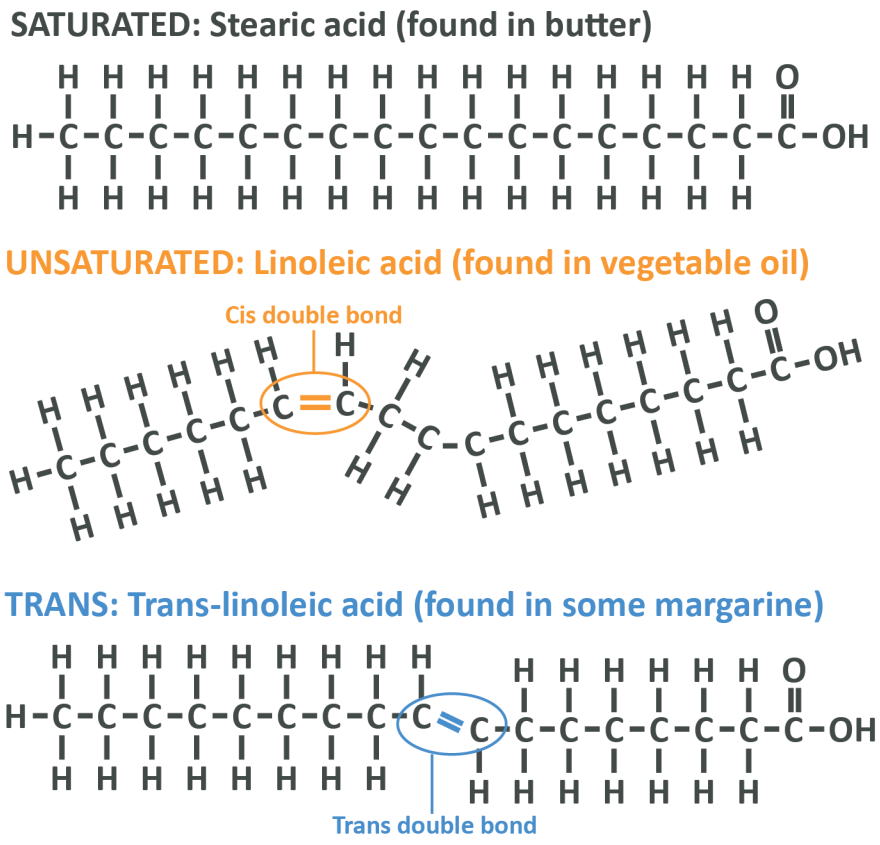
What food scientists discovered is that if you heat fats to a certain temperature in the presence of hydrogen, you can effectively break some of the double bonds and 'saturate' the fat with new hydrogens. This saturation removes the gap in the chain and straightens out the molecule, and as such, fully hydrogenated oils are solid at room temperature. This isn't ideal, though, because these fats are more or less inedible due to being **too** solid.

What we needed was partial hydrogenation, hydrogenation to the point that a fat was hard enough but still edible.

When an oil is partially hydrogenated, we turn cis fats into trans fats. What this means is that rather than the missing hydrogens occurring on the same side of the chain, they occur on opposite sites. This still straightens out the chain and creates a solid fat, but it's not so solid that you can't spread it – success! Well, that is success in the eyes of food manufacturers. The impact these fatty acids can have on your health is another story. We will come to that shortly

Notice in the image below that the 3 fats have the same number of carbons but their shape is different. In stearic acid the carbons are all fully hydrogen saturated, in linoleic acid there is a kink with 2 missing hydrogens on one side and in trans linoleic acid the hydrogens appear on opposite sides to each other, still resulting in a kink, but in a different direction and to a lesser degree. This tiny detail is very important because it changes the impact that a fatty acid has on your health. After all, we didn't evolve to handle these molecules.

Fig. 16



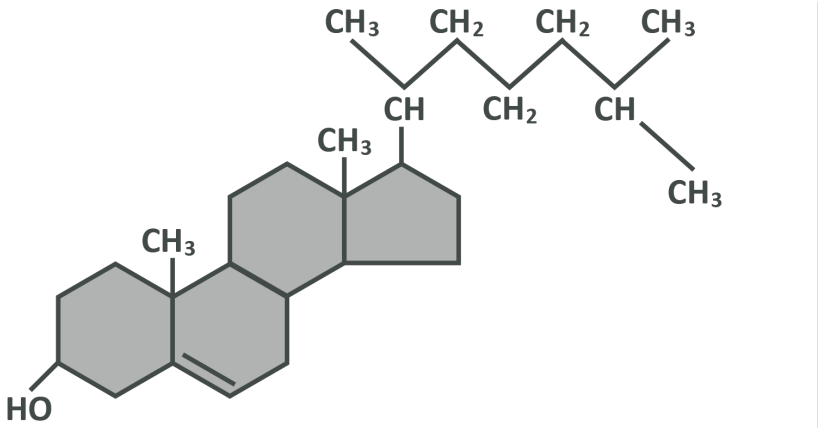
Before we move on, lets' talk about one final molecule – cholesterol.

2.6.4. Cholesterol

Cholesterol is, as the name suggests, a sterol (which is a lipid, just like fatty acids). Remember that a lipid is a compound of hydrogen, carbon and oxygen which does not mix in water.

We will not go into great depth about the structure of cholesterol, as all you really need to know is that it is made of three regions which you will see below – a hydroxyl group at one end which is soluble in water, 4 hydrocarbon rings (rings with carbon at each corner, single bonded to the carbons next to it and also bonded to 2 hydrogens which shoot off of it), and a hydrocarbon tail which is similar to the structure of a fatty acid. As you should now know, this chain is not soluble in water. Though cholesterol has parts which are and parts which are not soluble, for practical purposes here it is best considered to be a net insoluble molecule which will not mix into water (or blood for that matter).

Fig. 17



The vast majority of cholesterol you have in your system is produced endogenously, that is you make it yourself. It's produced primarily in the liver at a rate of about 1g per day, and you hold approximately 35g in your body at any one time. It's used for vital functions such as making up part of the membrane of every cell in your body, transferring information from cell to cell and helping to create hormones. One of the other roles cholesterol plays is as a precursor for the bile salts released from the liver and gall bladder to digest fat.

It is worth noting that while most of the bile salts are re-used after the triglycerides being digested are absorbed, some are excreted meaning that some cholesterol is lost – in fact, this is the way the body 'gets rid' of any cholesterol it doesn't need.

It helps us make cells, digest fat and produce hormones. It's also possible to regulate the amount of cholesterol your body has within it by reducing endogenous production and increasing secretion if needs be.

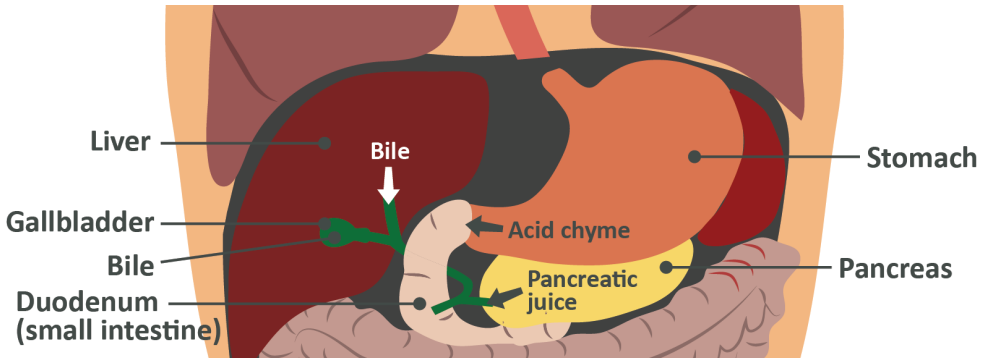
2.6.5. Fat digestion and absorption

Now you know what a saturated, unsaturated and trans fat is and have an understanding of cholesterol, let's zoom out a little and talk about how these are absorbed into your body.

Once you consume the steak we mentioned above, the fat within it (the triglycerides) make their way to the stomach and then to the small intestine – proper digestion of fats does not begin until this step. Once the triglycerides are in the small intestine they need to be absorbed, but because of the hydrophobic nature of these molecules, this is not easy to do. The bloodstream and the majority of your body is water based and so it is very difficult for triglycerides to enter them and they will generally 'clump' together as droplets of fat – in the same way that the 'lava' in a lava lamp forms blobs rather than mixing evenly.

This is why bile salts from the gall bladder are introduced along with enzymes called lipases, and the same neutralising bicarbonate ions mentioned earlier, to prevent your stomach acid destroying your intestinal walls.

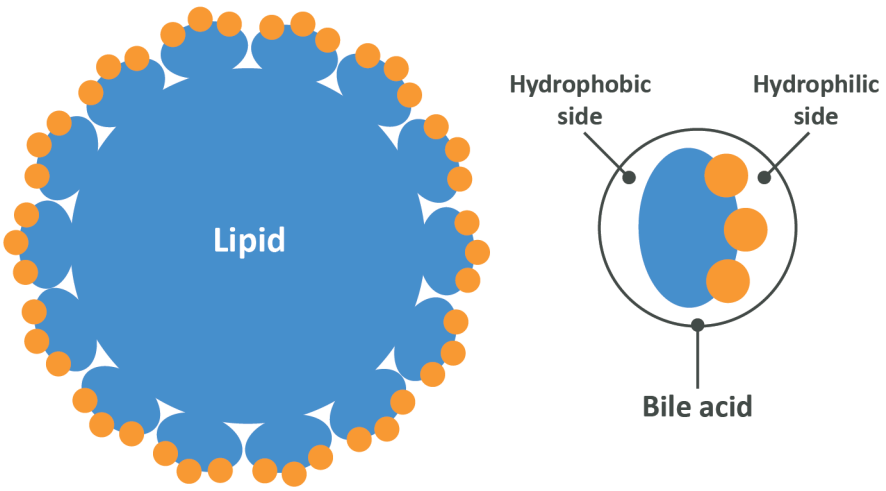
Fig. 18



Note: The gall bladder does not actually 'make' bile, rather that is synthesized in the liver. The gall bladder acts as a reservoir for larger amounts of bile to be stored for quick-release via the common bile duct that you can see in the image above, linking both the liver and the gall bladder to the duodenum. If an individual has no gall bladder they still produce bile, but they are unable to maintain this stored 'pool' and as such, their fat digestive processes may be inhibited in terms of having large amounts of dietary fat per meal. This is not to say they can't digest fat at all, rather, fat should be kept lower on a per-meal basis and eaten evenly though the day. This is a special case, though, and beyond the scope of this course.

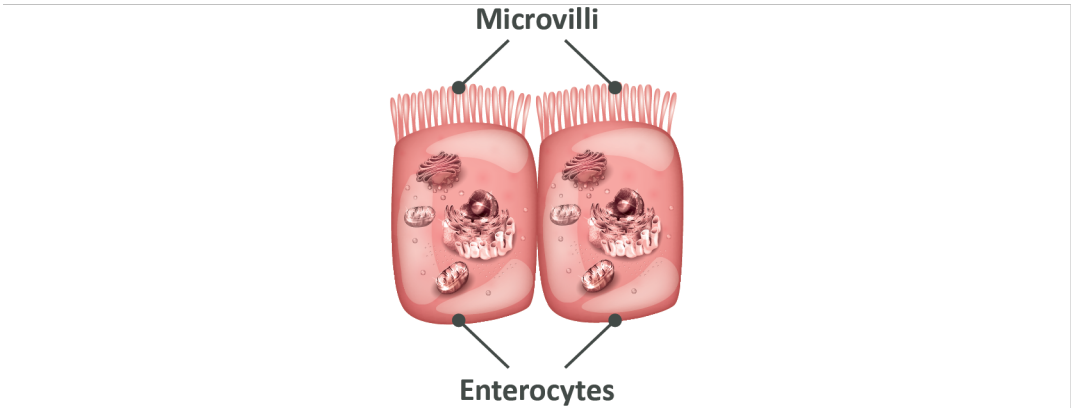
When bile is released into the small intestine, it acts as an emulsifier which means that it makes fats hydrophyllic (water loving). Soap is another example of an emulsifier, which is why applying soap to grease on a frying pan allows water to wash that grease away whereas it may have previously resisted rinsing. Bile does this very simply by virtue of the hydrophobic (water hating) side of a bile acid becoming attached to the hydrophobic surface of the triglyceride, while leaving it's hydrophyllic side exposed. This compound of bile and triglyceride is now able to be mixed into water easily. In the image below the bile salts have completely encased the lipid and only the hydrophyllic side is exposed.

Fig. 19



Now that this triglyceride is hydrophyllic, the lipase enzymes are able to get to work breaking the bonds between the fatty acids and the glycerol backbone, leaving two free fatty acids and one which is still bound to the glycerol, now known as a monoglyceride. These are now molecules which are small enough to be absorbed into the cells embedded in the intestinal wall. Any dietary cholesterol will be absorbed at this point, too, along with the 'fat-soluble vitamins' A,D,E and K. We will talk about these a little more in the module on micronutrients, but for now it's clear that without dietary fat you cannot absorb these very effectively, meaning that you are at risk of deficiency even if you are eating foods which contain a reasonable amount of them.

Fig. 20



In the image above, you are again looking at cells of the intestinal wall, the enterocytes.

As you already learned, the 'wavy' part at the top are known as villi and these serve to increase the surface area to allow for easier absorption. Once the free fatty acids and monoglycerides are absorbed into the cells of the intestinal wall, the next step is to absorb them into the blood.

The fatty acids and monoglyceride enter a cell structure (present in most cells, not just these ones) called the endoplasmic reticulum, which then re-packages them into triglycerides by

linking them back together and sends them on to another structure called the golgi apparatus. The golgi apparatus combines them with cholesterol and coats them in a protein to make them water-soluble. This final form is known as a chylomicron which is a lipoprotein (a term we'll return to later), and these water-soluble chylomicrons are then fed into the lymphatic system (a huge system of tubes carrying lymphatic fluid or lymph all around your body) which leads to the blood and finally to storage sites, such as muscles, your liver or most often, your bodyfat or adipose tissue.

Once the chylomicron (a protein-encased bundle of triglycerides and cholesterol) reaches the site in which it will be stored or used, enzymes called lipoprotein lipases break down the triglyceride once again, allowing them to be absorbed into the cell and used, or recombined a final time and stored. The chylomicron could now be considered 'empty', but this is not quite the case, as there will still be some remnants of triglycerides and cholesterol present which are useful to the body. This chylomicron remnant is delivered to the liver, and we can ignore it just for now.

Dietary fats are therefore eaten, emulsified by bile, broken down by enzymes into free fatty acids and monoglycerides, absorbed into intestinal cells, rebuilt into triglycerides, packaged as chylomicrons along with cholesterol, sent to lymph, sent to blood and finally transported around the body to be delivered to cells which will either use them for energy or store them. This storage is important, because although we can use fats for energy immediately after we eat them, we often go numerous hours between meals and for that we need to talk not only about the way our body handles fat in the 'fed state' after a meal, but also in the fasted state between meals.

Each gram of stored triglycerides is potentially worth 9kcal, and because fats are hydrophobic, they can be stored with no additional water (though in adipose tissue they are accompanied by some proteins and cellular structures). As such, 1kg of fat tissue will be around 87% fat and therefore yield somewhere around 7800kcal for use when needed. As you can probably imagine, it is through this process that the human race and those before it was able to survive famines, and between meals this is partly what allows us to fuel our cells despite a lack of immediately available food. This potential for being a vital energy source can be considered one of the main functions of dietary fat, alongside the use of fatty acids for forming cell walls, aiding with cellular communication and other similar roles.

Back on topic, fat which has been stored can be used as fuel but for that to happen, we need to release fat from our fat cells again…

2.6.6. Once fat has been stored, how do we use it?

Most of the fat you have in your body is stored in bodyfat tissue or adipose tissue. This is a dense network of fat cells known as adipocytes which are best considered to be like a bunch of balloons packed tightly next to each other, each containing some fat. The contents of each increases or decreases over time as you gain or lose fat. This should help you understand that your fat cells (in adulthood) typically get fuller and emptier as you gain and lose stored bodyfat, rather than it being the case that you create new cells if you gain additional fat. You may remember this principle from the last module where we mentioned that the reduction

of fat in the fat cells throughout your body is one of the primary signallers for your body to start resisting fat loss.

Most of the fat stored in these cells is in the form of the triglycerides which we have just explained. Once they are packaged by the golgi apparatus and transported to the blood via the lymph, they are carried through the bloodstream to fat cells around your body and deposited (without the 'casing' that they were packaged with before 'travel').

Between meals, during exercise or a times of low overall energy intake (such as when you are eating in a deficit) your fat cells receive a signal to start releasing their stored energy. In order for your body to now use these fats, the first step is to remove the 3 fatty acids on a triglyceride molecule from the glycerol backbone – this is, as you can probably imagine, again performed by enzymes, in this case called lipases (you were introduced to these as the enzymes responsible for breaking down triglycerides in the intestine). You may recall that in order to link fatty acids to a glycerol it is necessary to waste a molecule of water (H_2O) as 2 hydrogens and 1 carbon were lost; knowing this, your logic should enable you to work out how the lipases remove the fatty acids, they add water or hydrate them in a reaction known as a hydrolysis reaction.

Once the triglycerides have been hydrolysed, we are left with 3 fatty acids and a unit of glycerol which makes its way to the liver (we will discuss the fate of this glycerol in the next macronutrient section). The fatty acids are packaged into protein and delivered around the body for use as an energy source.

So far, we have explained that fat is eaten, absorbed into your intestines and then sent, via the lymph, around the body in special protein cases. It's used by cells for energy, or stored in specialised cells to be used later. We have explained two ways which you might end up with fatty acids in the blood – either you have just eaten and are in the 'fed state' during which time fatty acids in the form of triglycerides packaged as chylomicrons are being transported in your blood for use or storage, or you may be in the 'fasted state' between meals at which point fatty acids are being released and transported via the blood in albumin for use as energy – but there is another way that we can have fat in our blood, and in order to explain that we need to talk more about your liver.

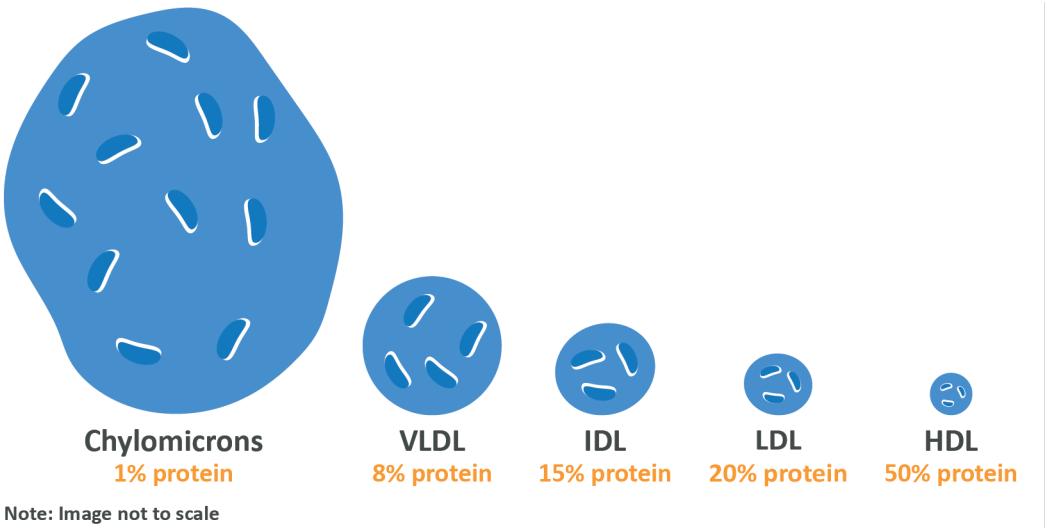
2.6.7. Lipoproteins

To explain this issue, we must explain a little more about lipoproteins. As you may be able to tell from the name, lipoproteins are made up of fat or lipids (lipo-) and proteins. Lipoproteins are fats (which you remember are insoluble due to being largely or entirely hydrophobic) encased in a protein so that they can be transported around your blood.

There are five key versions, one of which you have already met. These are chylomicrons (the protein-encased triglycerides which are made within the cells lining the intestinal wall during the initial process of fat digestion), Very Low Density Lipoproteins (VDL), Intermediate Density Lipoproteins (IDL), Low Density Lipoproteins (LDL) and High Density Lipoproteins (HDL). Some of these terms may be familiar to you as it is these lipids which are measured in your blood to determine disease risk.

The density of each can be considered to refer to the amount of protein contained in each, in comparison to fat. Additionally, the fat:protein ratio scales with the size of the molecules, with the relatively highest fat molecules being physically larger than the relatively highest protein molecules.

Fig. 21



We've already discussed the life of a chylomicron but briefly, it is created in the cells of the intestinal wall by combining triglycerides, cholesterol which we have eaten and a few other lipids and encasing them in protein. This is then absorbed into the lymphatic system followed by entering the blood and transported around the blood depositing lipids into various cells. Any chylomicrons which are not completely emptied then return to the liver where they are detected by special receptor sites and absorbed.

Within your liver cells a number of reactions take place which are dependent on a ready supply of carbohydrate and a number of enzymes. These reactions result in the production of cholesterol, fatty acids and monoglycerides. These monoglycerides and fatty acids can then in turn be combined to create new triglycerides which are then packaged with the newly created cholesterol (and some dietary cholesterol which came 'on board' a chylomicron) to produce 2 new lipoproteins – either VLDL or empty HDL molecules.

Remember that VLDL has a lot more triglycerides than does HDL, and this is because it's role is to transport those triglycerides to your cells or tissues for use as an energy source in almost the exact same way as a chylomicron except rather than taking triglycerides which have been ingested very recently to cells, it takes triglycerides which have been synthesised in the liver either from old chylomicron remnants or from reactions within the liver itself. Of course, triglycerides not used for energy are subsequently stored.

As a VLDL travels around your blood, it will come across the same lipoprotein lipases which will break off some of the fatty acids stored within it and donate these to cells. When this happens, the VLDL loses some lipid density and becomes IDL which eventually becomes LDL. LDL's job is to transport cholesterol to cells which need it in order to allow them to repair their membranes, build hormones and so on. After LDL donates its cholesterol, it returns to

the liver to either be recycled or excreted (along with the cholesterol it still carries) through bile. You can conceptualise this as a VLDL leaving the liver 'full' and gradually 'emptying' before returning to the liver.

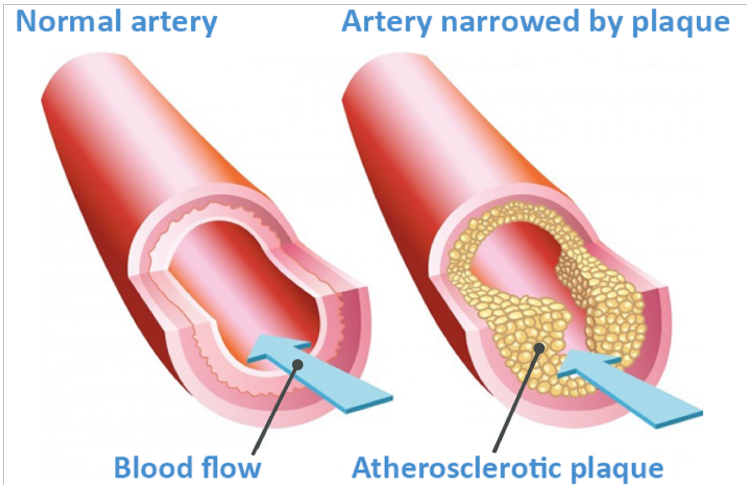
The HDL is considered to be a 'good' lipoprotein often simply referred to as 'good cholesterol'. It's role is to circulate in the blood and pick up excess cholesterol from cells to bring back to the liver for use or excretion. As such, it has the ability to lower your blood cholesterol levels.

As you can see, cholesterol per se isn't necessarily the big problem it is promoted to be. It's produced in the liver (and a small amount is eaten in the diet), the levels of cholesterol are pretty tightly controlled throughout your entire body through reduced production and an ability to excrete what isn't needed, and it serves a lot of key functions, so why do people worry about cholesterol in their blood?

2.6.8. Atherosclerosis

Atherosclerosis (coronary heart disease) is the narrowing of the blood vessels surrounding the heart which occurs due to a build-up of plaque. It is one of the most common cause of heart attacks, can lead to strokes, and is one of the biggest killers in the western world.

Fig. 22



Atherosclerosis occurs when either abnormal lipid profiles, irritants such as smoking or conditions like hypertension damage or irritate the inner wall of a blood vessel, allowing LDL (but also other non-HDL lipoproteins including chylomicron remnants) to pass through the thin inner layer of the artery and start to become oxidised. Oxidised lipoproteins are toxic and therefore initiate an inflammatory response, attracting white cells from your immune system.

The white cells 'eat' the lipoproteins present in the cell wall in an attempt to clear them out. Unfortunately, this is not as effective as we would like, and the white cells swell up (when collected they appear like foam under a microscope, hence the name 'foam cells'). When these cells die, they spill their lipid content into the wall of the artery, creating what is known as a lipid core. A fibrous cap forms over this core, attempting to heal this lesion, but as build-up continues the fibrous cap is unable to prevent the plaque from protruding into the artery.

As you can see, atherosclerosis is a major problem, but as you can also see it's not the molecule cholesterol which is causing the issue, but rather the lipoproteins which carry it. While an elevated cholesterol count was once thought to be a problem by itself, what we now understand is that the amount of cholesterol in your blood is only one potential risk factor, as the cholesterol in your blood may be in different places.

Recall that VLDL, IDL, LDL and chylomicron remnants (collectively known as non-HDL cholesterol, non-HDL lipoproteins or non-HDL-C) can carry cholesterol in your blood and deliver it to the body, while HDL is responsible for carrying cholesterol from your body back to your liver. Because of this, your non-HDL-C count and your HDL counts are likely to be a far more reliable measure of your heart disease risk than cholesterol alone. Total cholesterol measures do not account for HDL which is responsible for removing excess oxidised lipoproteins and cholesterol from the site of atherosclerotic plaque, and therefore they may not be reliable (which is absolutely not to say they should be discounted outright).

In fact, an abnormal lipid profile (elevated non-HDL and/or reduced HDL) is one of the biggest risk factors for atherosclerosis, and is so independent of changes in bodyweight, though this is not to say that bodyweight is not important.

Remember that VLDL and IDL as well as chylomicron remnants carry a significant amount of triglyceride, while LDL carries comparatively little, mostly carrying cholesterol? Well, high levels of blood triglycerides (hypertriglyceridemia) is also a major risk factor for atherosclerosis, and the primary cause of this is metabolic syndrome and diabetes, which results from over-nutrition, low physical activity and obesity. Because of this final factor, it's now widely considered that the two key markers for potential atherosclerotic issues are blood triglycerides and serum HDL, while serum LDL is of course still a very relevant issue.

In short, to avoid atherosclerosis the key things are to maintain a healthy weight as we mentioned in module 1, and maintain a healthy lipid profile. But how do we do that?

2.6.9. Fatty acids in the diet and your blood lipids

Hypertriglyceridemia is caused largely by your diet. There are familial traits which can influence your blood lipid levels but that is far beyond the scope of this course. What matters is, now that we know a healthy level of triglycerides in the blood (transported by all lipoproteins) and a healthy level of both HDL and LDL are required to improve health, we can ask the question – how can we make sure we do this in the context of our lifestyle choices?

Very briefly, before we move on, smoking, a high consumption of alcohol, extreme levels of stress and simply being overweight and/or inactive can cause both an impairment in HDL/LDL balance **and** an increase in triglycerides, and as such leading a lifestyle which avoids stress and the overconsumption of drugs is important. Also key, is maintaining a high activity level. The WHO recommends at least 30 minutes of moderate activity daily, with the ideal being 30 minutes intense or 60 minutes of moderate exercise. Just as crucially, it's important to maintain a healthy weight and avoid extended periods of time involving excessive food intake in general (while you do need to overeat if you choose to gain muscle, this is a moderate

increase and not the topic of this discussion. Here we are talking chronic overeating resulting in excessive nutrient intake and therefore fat gain). Now back to the focus on diet.

It's been long understood that aside from total calorie intake, one of the primary drivers for an impaired blood lipid profile is dietary fat intake.

Initially it was known that increased saturated or unsaturated fat caused an increase or decrease, respectively, in serum cholesterol levels (hence the abovementioned recommendations to cut out saturated fat and cholesterol). Later, when it became possible to separate lipoprotein fractions and study them, it was ascertained that LDL and VLDL were the primary corollaries with heart disease. Later, it was discovered that total cholesterol to HDL ratio (or triglyceride to HDL) was a better indicator of heart disease risk, meaning that it was possible to study the lifestyle and dietary factors which impacted upon these specific markers, and by the 1990's the mechanisms by which dietary fatty acids were impacting upon lipid levels was starting to be understood.

When your body's cells are starting to run low on cholesterol, a signal is sent from them which increases cholesterol synthesis in the liver. This signal can be regulated by consuming unsaturated fatty acids, and therefore these fatty acids can reduce serum cholesterol levels through their ability to reduce the rate at which cholesterol is synthesised in the liver. It's recently been discovered that short-chain fatty acids (we will return to these when we talk about dietary fibre) and medium-chain saturated fats seem to have a similar effect to this, too. On top of this, it's well established that a diet high in saturated fatty acids increases LDL.

This would seem to suggest that consuming low amount of saturated fat (or zero saturated fat) and higher amounts of unsaturated fat would be the most health-promoting approach, but this is perhaps hasty.

To begin with, this ignores the impact that dietary fats can have on HDL, instead focusing only on LDL. It also ignores the fact that all fatty acids within a group are not the same. For example, stearic acid and lauric acid may both be saturated fatty acids which increase total lipid levels in the blood, but stearic acid does not increase HDL whereas lauric acid does, significantly.

Finally, though risk factors and blood markers are crucial, they are not everything. We aren't really concerned about some particles in our blood, we are concerned with the impact that fatty acids actually have on our long-term risk of getting diseases, and when we think in these terms the waters get a lot more muddied. Two high quality reviews of the data which looked at the association between saturated fat and heart disease found no correlation at all. Although saturated fat can increase serum lipid levels, it may not always impact heart health directly due to mechanisms which we simply don't yet understand.

Here's what we know about the effects different fatty acids may have:

- It's known that polyunsaturated fatty acids can lower total blood lipids, but when they do this they lower HDL alongside LDL. Not only this, but it is the polyunsaturated fatty acids contained in LDL lipoproteins which can oxidise when the LDL enters the arterial walls, which is the reason that white cells arrive and the plaque build-up occurs –

clearly, overconsuming polyunsaturated fatty acids isn't a great thing, but at the same time they do indeed function to keep total blood lipid levels in check so they should be consumed in some amount, and certain polyunsaturated fats such as the Omega-3's we'll mention momentarily have beneficial properties all of their own

- Saturated fatty acids raise total blood lipid count, but they may also increase HDL depending on the specific acid. In some studies, high intakes of saturated fatty acids are associated with increased cardiovascular disease but when associating saturated fat with heart disease it's very difficult to isolate saturated fat as the only factor – saturated fat consumption is also associated with processed food consumption, smoking and a sedentary lifestyle which all serve to make this data less cut and dry
- Saturated fatty acids are vital for cell function, cell wall production and cellular signalling. Additionally, saturated fatty acids transported in LDL are not able to oxidise, and therefore cannot directly lead to atherosclerosis themselves (though an increased LDL concentration leads to an increase in all fatty acids being present in the blood)
- All of this adds up to say that while the idea that saturated fats are not a direct cause of heart disease by themselves holds water and they should not be excluded from the diet completely, it cannot be ignored that they **may** be harmful when consumed in excess. The 'ideal' intake for saturated fat may not be high, but it also cannot be considered to be zero
- Monounsaturated Fatty Acids (MUFA) seem to have a lowering or at least neutral effect on total cholesterol. They also raise HDL while lowering LDL, meaning that they tend to be considered 'the good guy' in all of this. MUFA are present in high amounts in Mediterranean style diets which are themselves considered to be extremely healthy approaches to nutrition, and it's a reasonable assumption that MUFA inclusion is at least one small factor in that
- Trans fatty acids will increase total cholesterol by increasing LDL while also decreasing HDL. Trans fatty acids produces in food manufacturing are generally considered to be a net negative, and their consumption could be considered a risk factor for heart disease by itself. Trans fatty acids, because of their unnatural shape, are very difficult for your body to deal with, and avoiding them as much as is reasonably possible (eating a doughnut once a month isn't going to kill you) is an exceptionally wise idea

Other than trans fatty acids, as we hope you can see, the research into fatty acid consumption is hugely nuanced and difficult to really pull together. It's generally considered that saturated fatty acids increase serum cholesterol levels which is a really strong predictor of heart disease, but at the same time the population data doesn't support the saturated fat/heart health link. Polyunsaturated fatty acids lower total cholesterol, but they also lower HDL and are susceptible to oxidation. Monounsaturated fats and trans fats appear to be a little more cut and dry as beneficial and harmful respectively, but it's still very early to draw strong conclusions about MUFA being a panacea of health due to limited strong long-term data.

2.6.10. What about Omega-3?

To throw yet another factor into the mix, let's talk about Omega-3's. Omega-3 fatty acids are polyunsaturated fatty acids where the first double-bond is found on the third carbon from the Omega-end. There are two broad 'kinds', namely DHA or docosahexaenoic acid, and EPA or eicosapentaenoic acid which are the long-chain forms and ALA or alpha-linolenic acid which is shorter chain. Long-chain Omega-3 fats are found in fish and shellfish (algae also contains DHA) whereas short-chain Omega-3 is found in plant sources of fat like nuts and seeds.

EPA and DHA are the most potent forms, and in fact ALA must be converted into DHA before it is useful and it takes a lot of ALA to make 'enough' as the conversion is highly inefficient – therefore animal sources of Omega-3 are preferable. Vegans may wish to supplement with an algae based product for this reason, though there should be no reason a vegan could not get the minimum required amount of Omega-3 to avoid deficiency, around 250mg combined per day of EPA and DHA, which can be consumed via plant sources in the form of ALA.

Long-chain Omega-3 fatty acids have a huge number of benefits for health, including a reduction in blood triglycerides, an increase in HDL and a very minor decrease in LDL. Omega-3 also seem to reduce inflammation which, if you recall, was a significant factor in atherosclerotic plaque formation due to irritation of the arterial walls. On top of this, EPA and DHA can potentially improve mood and even muscle protein synthesis!

2.6.11. So how much fat should I eat?

According to the Eatwell Guide, the British recommended intake of fat is less than 35% of total calories, which represents a moderate fat intake essential for a healthy, balanced diet. During times of high energy output the proportion of fat in your diet may reduce due to an increased carbohydrate intake, or you may reduce fat intake during times of caloric restriction in order to maintain a high carbohydrate intake for exercise performance purposes, but this should not drop below 20% as this is associated with impaired sex hormone production and a potential risk for certain vitamin deficiencies as explained in the next module. The Eatwell Guide also breaks this down to recommendations for each different group of fatty acids. Those recommendations are seen below.

Currently the British recommended intake of fat is less than 35% of total calories, and we do not seek to deviate from this. In previous years, it has been postulated that the ideal diet is a low-fat one, but it's worth noting that this 35% advice would represent a moderate fat intake. The lower-end recommendation is 20%, and this is again something which we fully support. Some dietary approaches may involve higher intakes than this, but for general purposes this recommendation holds water.

Saturated fatty acids should make up to 11% of total calories. As you now know, however, not all saturated fatty acids are the same, and there are some which appear to be more useful than others. The medium-chain saturated fats found in milk fat and exotic oils like palm oil or coconut products as well as those in animal products like beef, seem to be preferable to other saturated fatty acids which you might find in packaged goods, and realistically a good rule of thumb is to get saturated fat in your diet from unprocessed foods wherever possible. If you

do this, and don't over-emphasise one source over the other, it's unlikely that you will come to any harm.

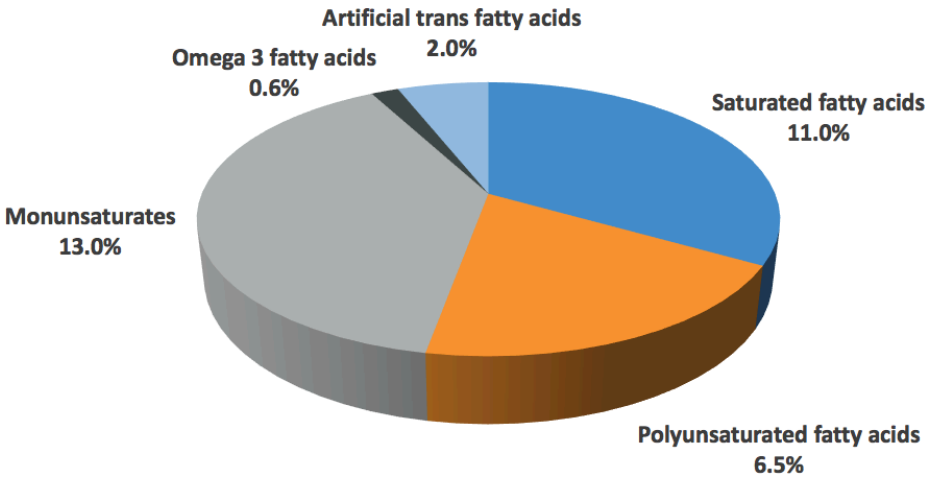
Polyunsaturated fatty acids should make up around 6.5% of your total calories. Poly-unsaturated fatty acids tend to be found in vegetable oils and some condiments like mayonnaise, but also in grains like oats.

Monounsaturates should make up around 13% of total calorie intake, coming from sources like avocado, olives and nuts and non-hydrogenated butter substitutes/oils made of them.

Omega-3 fatty acids from fish, algae or supplementation should make up at least 0.2% of your total calorie intake. The current literature appears to show that doses ranging from 1-6g of total EPA and DHA are ideal for improving health markers (though around 250mg is enough to avoid deficiency). Supplementation is a cheap and easy way to get this if you would like to experiment with higher intakes. As a warning, do not exceed the maximum studied beneficial dose of 6g, as after a certain amount (not 6g, but there is no need to go higher) your blood will be unable to coagulate properly, which is a major issue. 2-3g per day is a pretty safe, standard range to aim for to get additional benefits over and above deficiency avoidance.

Artificial trans fatty acids found in processed foods, margarines and other hydrogenated oils should make up no more than 2% of total fat intake, but the ideal intake here truly is as close to zero as you can get. Note that there is a very small amount of naturally occurring TFA in some meat products, but this isn't concerning with any normal level of intake.

Fig. 23



Applying this will be explained at the end of this module (because to track and measure that would be just about impossible), but as a last note on fats, remember that most foods contain a mixture of saturated, mono and polyunsaturated fats, so even foods which we might consider to be a source of one may in fact be a source of all three in differing amounts, for example:

- Butter which is considered a source of saturated fat actually contains 54g saturated fat, 19g monounsaturated fat and 2g polyunsaturated per 100g

- Coconut oil is considered a saturated fat source, and it largely is, but does contain 85.2g saturated, 6.6g monounsaturated and 1.7g of polyunsaturated fat per 100g
- Olive oil contains 14g of saturates, 70g mono and 11g polyunsaturates

With all of that covered, let's move on to our last macronutrient, carbohydrates.

2.7. Carbohydrates

A carbohydrate is an organic compound found in the foods we consume (remember, organic only means that they are carbon based at a molecular level). Much like fats they are a bone of contention amongst those in the nutrition field, with many believing that they are the root of all evil and the cause of all weight gain, while the government Eat Well Guide dictates that we should base every meal around them.

Though it is true that those who are overweight eat a lot of carbohydrate containing foods, the idea that carbohydrates cause weight gain per se is not strictly true, as we hope you understood from module 1 of this course. No amount of carbohydrate can cause weight gain if the second tier of the pyramid, calorie balance, is accounted for.

With that said we can state that carbohydrate intake is not essential for survival, and as such it is very easy to reduce carbohydrate intake when you are looking to create a calorie deficit without impairing muscle retention by reducing protein, or overall health by restricting fat by too much. It's also not to say that eating carbohydrate in place of adequate protein or fat is by any means a good idea.

Carbohydrate's main job within the body is to provide energy – in fact, when all macronutrients are available your body will 'burn' carbohydrates preferentially. You will always be turning over protein, you will always be using fats for cellular function and energy and your body will always be using carbohydrates for something, too, but the ratio of carbohydrates:fat:protein being used at any one time is not static. Exercise changes it dramatically, but at rest the key thing to remember is that your body will burn excess carbohydrate, then it will use excess protein to be converted into carbohydrate to use, and then it will use up fat.

This can often be seen misrepresented in phrases like "Eat fat and you burn fat, eat carbohydrates and you don't", but think carefully, do you want to burn fat, or do you want to burn bodyfat?

To burn bodyfat you must release it from fat stores during times of low energy, and the times of low energy are always going to be between meals and while you sleep. If you eat more fat, you'll be burning more fat, but you'll also be eating more fat to burn, leaving you at a net zero, and therefore only losing bodyfat if your calories are in line – logical, right?

If you are in a calorie deficit to lose weight, you are by definition eating less overall food. When you eat a meal, the nutrients within that meal must be stored, used for bodily functions or used as energy, and whether that meal was high-fat or high carbohydrate it doesn't really matter a huge deal for the discussion of weight management – you will either use that dietary

fat, use that dietary carbohydrate or use a mixture of the two for immediate energy needs, then you'll store some of it and later you'll start to mine into stored carbohydrate and fat.

Low carbohydrate or low-fat diets both work because calorie balance dictates that fat from adipose tissue will eventually be used as fuel. To assume dropping one nutrient will make a difference is a short-sighted approach and of course, above all, **we must remember that macronutrients govern almost everything, but calories are king.**

Let us repeat that again, so long as you're eating in a calorie deficit with enough protein to maintain your muscle mass and health, the ratio of carbohydrate to fat within your diet doesn't really dictate fat loss for a given calorie intake, and as such your macronutrient intake should be largely down to preference, with respect to the recommendations around the minimum protein and maximum fat intake which we have covered already.

So with that covered, let's get geeky and learn what a carbohydrate actually is.

2.7.1. Carbohydrate types and understanding the names

A carbohydrate is a sugar molecule or a chain of sugar molecules bonded together to form slightly different chemical structures. The sugars and sugar chains which we usually consume within our diet are part of the saccharide family of carbohydrates (from the Greek Sakkharon meaning 'sugar') and they contain carbon, hydrogen and oxygen.

For example, lactose is one example of a carbohydrate molecule, it is the main sugar found in milk and consists of a molecule each of the sugars galactose and glucose bound together.

Because Lactose is made from two simple sugar molecules, it is known as a disaccharide. There are four main categories of sugar, namely:

- **Mono:** Meaning one, or a simple sugar, such as glucose or fructose
- **Di:** Meaning two. Again a simple sugar, such as lactose or sucrose
- **Oligo:** Meaning a few, as they contain 2-9 monosaccharides. These tend to have a minimally sweet taste and are soluble in water while being indigestible to humans and therefore providing some benefits which we will discuss later in this course. One example is the collective fructo-oligosaccharides derived from onions or Jerusalem artichokes
- **Poly:** Meaning 'many', as polysaccharides have 10 to several thousand mono-saccharides within them. These are more complex sugars, often tasteless or slightly sweet and insoluble, such as starch from potatoes or cellulose, a form of fibre found in most vegetables. These are typically referred to as 'complex carbohydrates', though fibre is often taken as a separate topic

2.7.2. What is a sugar?

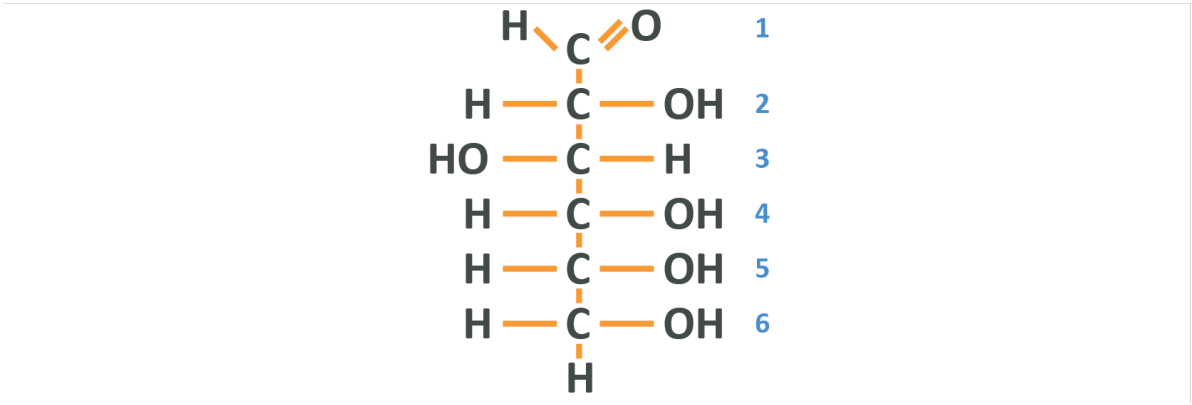
Glucose is the most important form of sugar/carbohydrate in nutrition as it is the main molecule used for energy transfer within your body.

Indeed, when we talk about blood sugar, we are referring to the glucose content within your blood. Photosynthesis in plants, which is the root way which all lifeforms get energy (plants convert sunlight to energy and store it, then get eaten by animals which store it, which then gets eaten by animals that store it) stores energy in the form of glucose, meaning this one sugar is the basis for all energy usage in all life forms that we know. While animals also use fat as fuel, most of that fat is created in animals after they consume glucose. Some plants (mostly the seeds) also contain fats, but again these are synthesised using glucose. In summary, glucose is important.

Glucose linked in long-chains forms glycogen, which is the name for the carbohydrates that get stored in your body. By linking it in another way we get starch which is the most widely consumed carbohydrate in our diets (found in rice and potatoes, amongst other things) and by linking it to another sugar, fructose, you get table sugar. But what **is** it?

The formula for glucose is C6 H12 O6 meaning 6 carbons, 12 hydrogens and 6 oxygens. You'll notice that this means we have carbon and 2 hydrogens for every oxygen, and this is where the name carbo-hydrate comes from. Drawn as a stick diagram it looks like this:

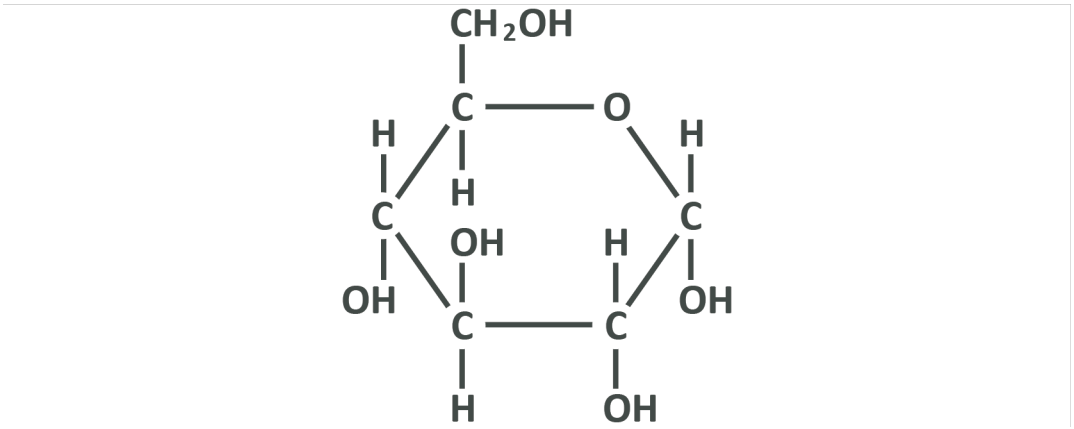
Fig. 24



Note: We have a familiar carbon chain where each carbon is 'full'. At one end, we have what is known as an aldehyde group which is simply a carbon, double bonded to an oxygen and also bonded to a hydrogen. We then have 4 carbons with a hydroxyl group which you will recall is an oxygen and a hydrogen, and another hydrogen, then a final carbon with a hydroxyl and 2 hydrogens. We can number the carbons from the top 1-6, which will be useful in a second.

Now, this is glucose in its simplified linear form which allows for analysis of the molecular components, but in nature glucose is not linear, it is hexagonal. The true shape of glucose more closely resembles the below:

Fig. 25

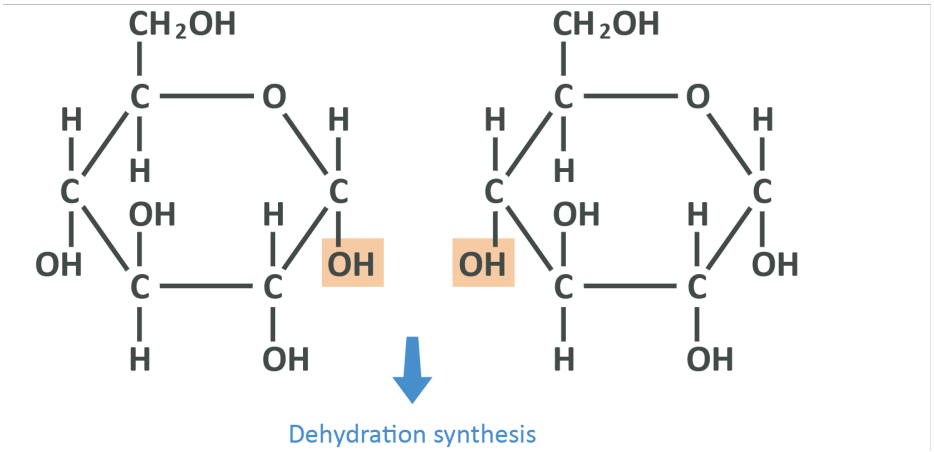


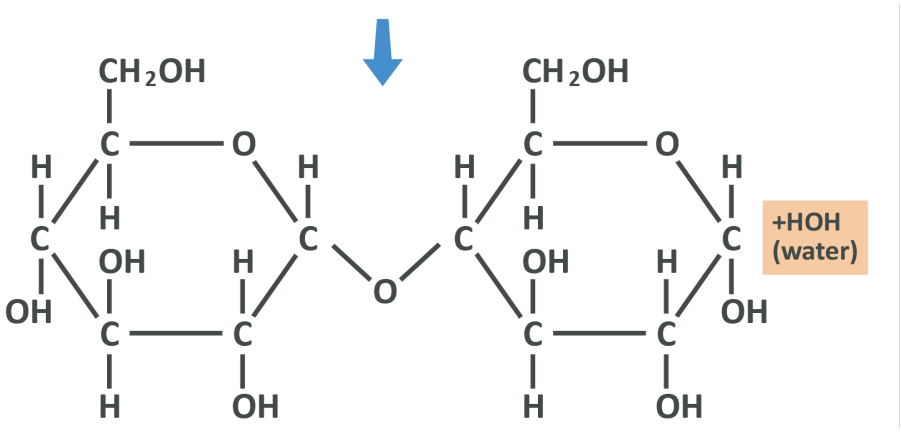
Here the carbon at 3-o'clock is the carbon 1 from the linear picture, and protruding from it you see the rest of the aldehyde group (OH and a separate H), and the top left protrusion is in fact the 6th carbon from the linear picture with the OH and 2 separate carbons. Here, too, the oxygen attached to carbon 5 in the linear picture has lost a hydrogen to bind to carbon 1, and the oxygen from the aldehyde group has lost one of its bonds (recall it was a double bond) and gained a hydrogen. All you **really** need to remember from this section, is that the carbons in glucose are in a chain and can be linked from 1-6, and in the body and in food the chain connects to itself end-to-end and makes a ring.

The next thing to see here is that the line closest to the bottom is thicker, and that is done simply to give perspective. This is not a 2d circle, the hexagon is lying horizontally and facing away from us, which is key as it shows that carbon 6 and some other atoms sit 'above' the glucose while the others sit underneath. This is important and we'll come back to it later, but just consider that you can look at the linear diagram as a side view.

So that's a single glucose – let's introduce another and bond them together in what is known as a 'condensation reaction' or 'dehydration synthesis' which you have already encountered a couple of times.

Fig. 26





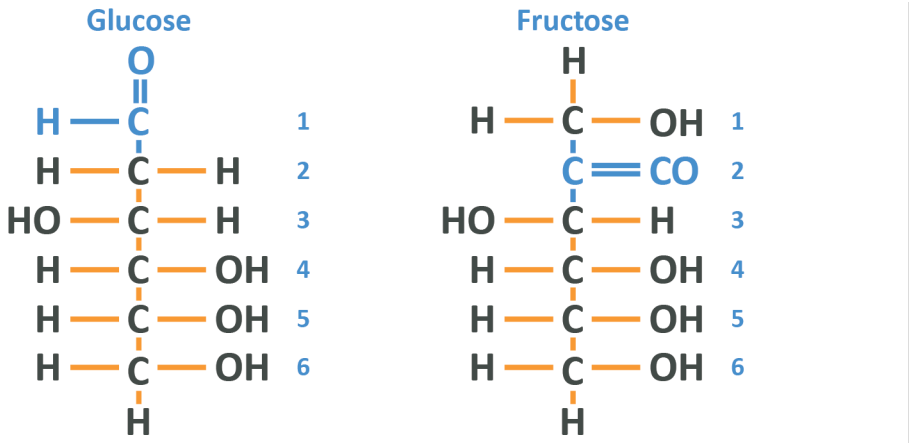
Here you see that the OH group from carbon 1 of the left-hand glucose and the hydrogen from the OH group on the right-hand glucose's 4 carbon have broken off, and formed a molecule of H_20 or water. This leaves the oxygen from the right glucose and the 1 carbon from the left both unstable, and so they bond together (in what is known as an alpha 1,4 glycosidic bond, 1-4 denoting the carbons that have been connected), linking the 2 molecules of glucose into the disaccharide known as maltose. These chains can continue lengthening to create oligo or polysaccharides. This 1,4 bond is the most common link between glucose molecules, though importantly it's not the only one – this will come up again later.

By linking together chains of monosaccharides we get a multitude of different poly-saccharides, with the type of saccharide being dictated by the chain length and 3D shape. Starch in potatoes as well as cellulose (plant fibre) which gives vegetables their shape and is the primary ingredient in paper are polysaccharides but interestingly so is chitin (kite-in) which forms the exoskeleton of some insects and the wings of flies, and naturally sourced cotton.

One other important monosaccharide is fructose, often referred to as "fruit sugar".

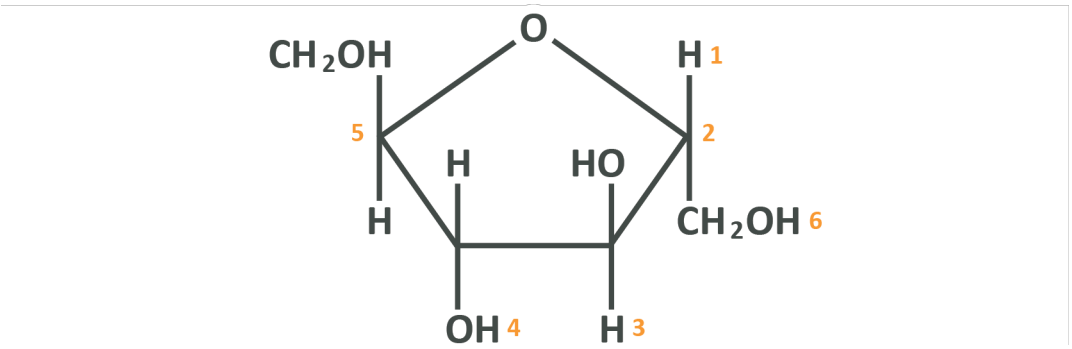
The chemical formula for fructose is C6 H12 O6 which should look familiar as it's exactly the same as glucose. In fact, side by side, they look very similar but have distinct differences.

Fig. 27



The difference here is that the glucose molecule has a carbonyl group (carbon double bonded to an oxygen) at the 1 carbon whereas the fructose has it on the 2 carbon. Other than that, the 2 are about the same in this format – the key difference comes in when we look at fructose in the cyclical formation which is, like glucose, how it actually appears in nature.

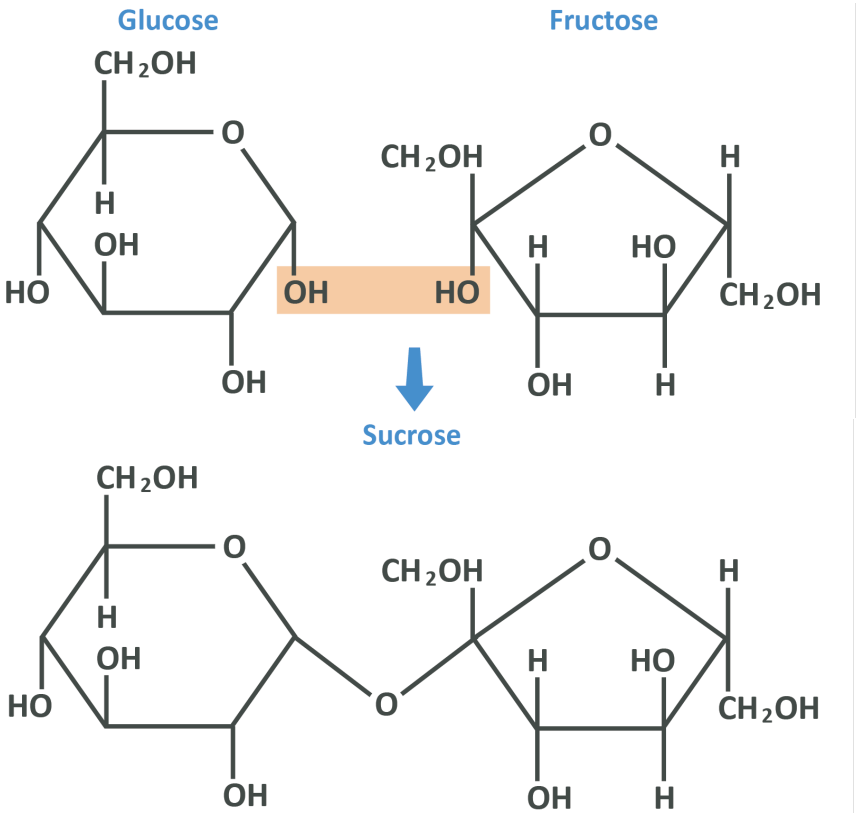
Fig. 28



Here the 1 carbon is the one on the far left, and exactly as in the glucose, the double-bonded oxygen becomes bound to the carbon/hydrogen molecule on the 5 carbon. The difference, of course, being that now the double-bonded oxygen is further down the chain compared to glucose. The 2 are very, very similar.

In fact, if the 2 carbon from the fructose bonds to the 1 carbon from the glucose, we get sucrose which is table sugar (this is an alpha 1,2 glycosidic bond).

Fig. 29



The key thing to remember here is that fructose and glucose are very similar from a molecular viewpoint but are structurally different and are capable of bonding to each other in more or less the same way as glucose can bond to itself.

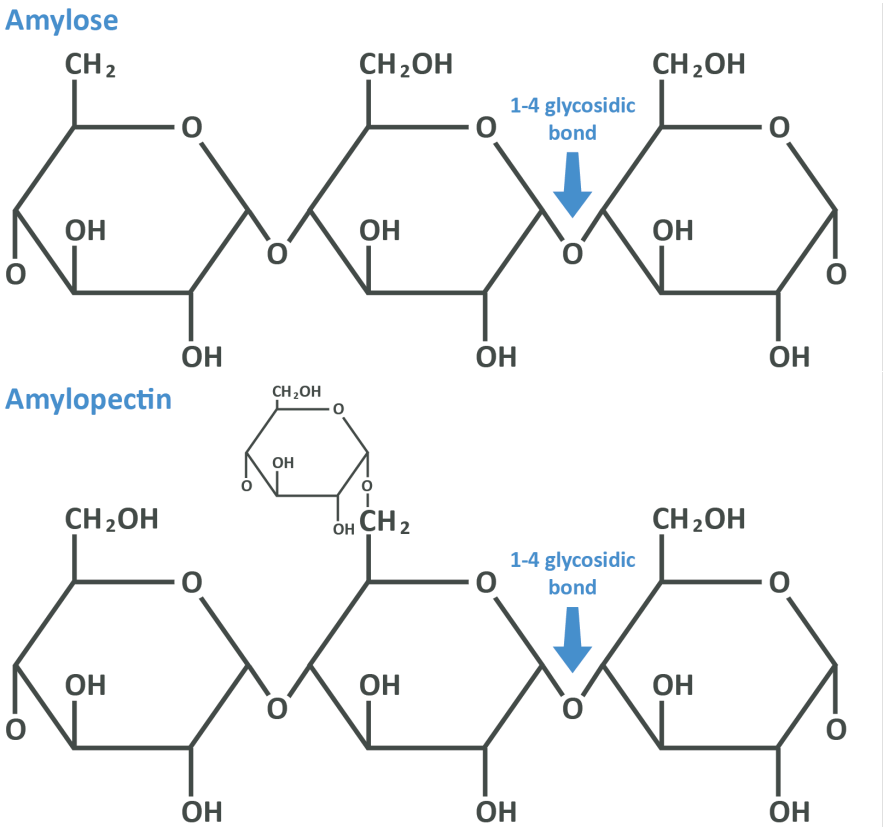
Now we know how di, oligo and polysaccharides are formed from monosaccharides, let's look at how we can digest and absorb them for use.

2.7.3. Starch digestion

To show how carbohydrates are absorbed, it's best to talk about one of the most commonly consumed carbohydrates in the modern diet – starch.

Starch is made up of 2 different polysaccharides: amylose and amylopectin. Amylose makes up 20-30% of starch and is a long-chain of 1,4 glycosidic bonds between glucose mono-saccharides, and amylopectin which makes up 70-80% has a similar configuration, but approximately every 24th to 30th glucose, a branch is formed when an additional glucose bonds in a 1,6 glycosidic bond as you see below.

Fig. 30



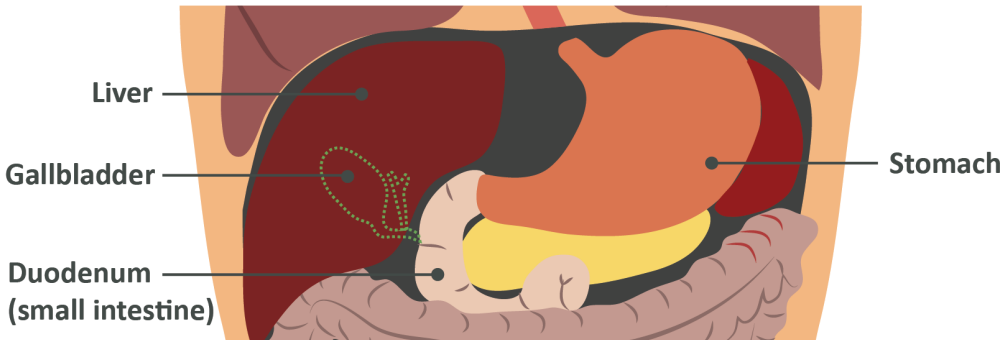
Now, each chain is extremely long. Amylopectin is comprised with between 2 and 20,000 glucose units and amylose tends to be around 300-3000 units. We therefore definitely need to process these before we can absorb them and use them for anything. Remember, it is the simple sugar glucose which we transport in our blood, and here that glucose has been combined into a larger form – by now you should be able to guess that this is a job for our digestive system and, mostly, for enzymes.

When you consume a food containing starch, as for any food the process of digestion begins in the mouth. For protein and fat this process simply involves physical digestion by chewing but carbohydrates have a slightly different fate – your salivary glands produce salivary amylase, which is one of the enzymes capable of chemically digesting the starch polysaccharides. Amylase is capable of hydrolysing (adding a molecule of water to) some of the 1,4 bonds within both polysaccharides. As you learned earlier, the reaction required to join 2 glucose molecules is dehydration synthesis and as such you can think of this like salivary amylase adding that molecule of water back and therefore separating the 2 units again. This is a hydration reaction or hydrolysis.

Salivary amylase is only capable of partial digestion, but its effects are profound nonetheless. As we said above you'll notice this by chewing some bread for a long time – as the starches are gradually reduced to their constituent glucoses, it will start to taste sweeter.

Next, you swallow and the food travels to the stomach. In the stomach, the acidic environment which was required for pepsinogen to activate and become pepsin denatures the amylase enzymes (as you'll remember, enzymes are proteins and if their shape is altered they no longer work) and here digestion of the starches pauses. When you are at this point, amylose and amylopectin have been broken down from very long polysaccharides into shorter polysaccharides, some oligosaccharides and a few disaccharides. Of course, as always, the next stop is the first section of the small intestine, the duodenum.

Fig. 31



When starch (which is now partially hydrolysed) reaches the intestine, the pancreas releases more amylase (specifically pancreatic amylase) into the area to continue breaking down the 1,4 glycosidic bonds. This breaks the starch down even further into disaccharides and a few very short oligosaccharides.

Recall the billions of small hairs on the walls of the small intestine cells, enterocytes, called microvilli? When the disaccharides and oligosaccharides reach these, they encounter specialised brush border enzymes called maltase and sucrose-isomaltase which finish off the job by respectively breaking down 1,4 bonds between maltose disaccharides left over, and the 1,6 glycosidic bonds located on the branched sections of the amylopectin chains.

What is important to remember here is that starch breakdown starts in the mouth, then is continued in the main part of the small intestine before being finished off by enzymes on the cells lining the intestine. After this entire process, the long-chains of polysaccharides which

we consumed are no different to how they would be if we had consumed straight glucose. By the time it reaches your stomach – glucose is glucose.

These glucose molecules are now absorbed through the intestinal wall and into the blood for transport to the liver.

Note: Quick note before we continue – some starch does not fully digest, and therefore is not absorbed. This is known as 'resistant starch'. Remember that amylopectin which makes up the majority of starch is branched? Well this branching creates a great deal more surface area for enzymes to attack, and therefore makes it easier to digest. Amylose is a little harder to break down and therefore it is hydrolysed more slowly in the small intestine, and sometimes some is missed.

Resistant starches cannot be absorbed and so they pass to the large intestine, the colon. We will return to these in detail in the module on fibre but consider for now that this is a food from which we cannot extract energy, and which will stay in the digestive system for longer, therefore keeping you better satiated.

2.7.4. Lactose digestion

One other common carbohydrate we consume is lactose, the disaccharide formed between glucose and galactose. Much like starch, it passes through the digestive system but until it reaches the enterocytes, it remains untouched. Amylase cannot hydrolyse bonds between glucose and galactose, which is a great example of the specific nature of enzyme action.

Once lactose reaches the enterocytes, the enzyme lactase breaks these bonds and the glucose and galactose monosaccharides can be absorbed. Galactose is mostly sent to the liver and converted into glucose to use just like any other glucose.

Some individuals, however, do not produce as much lactase resulting in the digestive abnormality known as lactose intolerance. Because the disaccharides are not digested properly, they make their way in-tact to the large intestine and ferment, causing GI distress and its associated symptoms. Note that this is very different to an allergy, which is an immune response rather than a gut-focused fermentation and is beyond the scope of this course.

It is estimated around 5-15% of people with white European descent have lactose intolerance, around 30% of those with Indian descent, around 70-75% of those with African descent, around 80% of those with central Asian descent and up to 100% of east Asian descent.

The reason for the differences by ethnicity is interesting and is more to do with the geographical location of your ancestral roots than anything else. In human beings, the ability to produce the lactase enzyme is almost always present at birth but diminishes over time. This is because, evolutionarily, mammals stop consuming milk after weaning. At the dawn of the agricultural revolution approximately 10,000 years ago, many humans began drinking bovine milk until later life, and over a very short period our ability to consume dairy evolved (this is a great example of the speed at which evolution can occur with intense environmental pressures) but the uptake of this practice greatly varied by region. In central European areas (especially Scandinavia), dairy consumption is huge, and therefore lactose intolerance in

those descended from these areas is very rare. In East Asia, soy-based milks were preferred and so the adaptation to this dietary practice did not occur.

While stomach flu and other stomach issues can temporarily cause lactose intolerance, the idea that you can cause lactose intolerance by abstaining from dairy for extended periods is unsupported in the literature, this can be mistakenly believed because lactase production may naturally decline through life meaning that those who could consume it in their 20's but do not, may indeed find themselves unable to in their 40's.

Lastly, lactose intolerance is not a black or white issue, and many who suffer are able to consume a certain amount of dairy before issues arise, and many who do not suffer can encounter problems if they consume truly vast amounts of dairy in one sitting. It is very rare that someone is unable to have any dairy at all, but this should be assessed on a person to person basis. If you do not experience symptoms with a given intake, it can be assumed that this intake is not harmful to you as either your lactase production is sufficient for that small amount, or the small amount of disaccharides which make it to your large intestine do not cause problems.

According to the British Nutrition Foundation many with lactose intolerance can tolerate some amount of milk, yoghurt and cheese (especially low lactose cheese like parmesan). This would also stretch to whey protein where the lactose content is extremely low. When in doubt, however, speak directly to a dietician. This course is not here to diagnose or help manage dietary allergies or intolerances.

2.7.5. Sucrose digestion

The final carbohydrate which we need to discuss is sucrose – table sugar. Sucrose is a disaccharide of glucose and fructose joined together as you have seen.

Sucrose is probably the most commonly consumed disaccharide in the Western World, largely due to its sweet taste. Glucose is not actually that sweet, despite being a 'sugar', but fructose binds to taste receptors very well and provides a very enjoyable flavour – leading to sucrose being added to packaged and processed foods, snacks, drinks and desserts the world over.

There are a number of issues surrounding table sugar which we'll discuss throughout this course, but for now all you need to know is that much like lactose, it reaches the walls of the small intestine intact, but once there it is broken down to its constituent parts by the same sucrose-isomaltase which breaks down isomaltose.

2.7.6. What happens once sugars are absorbed?

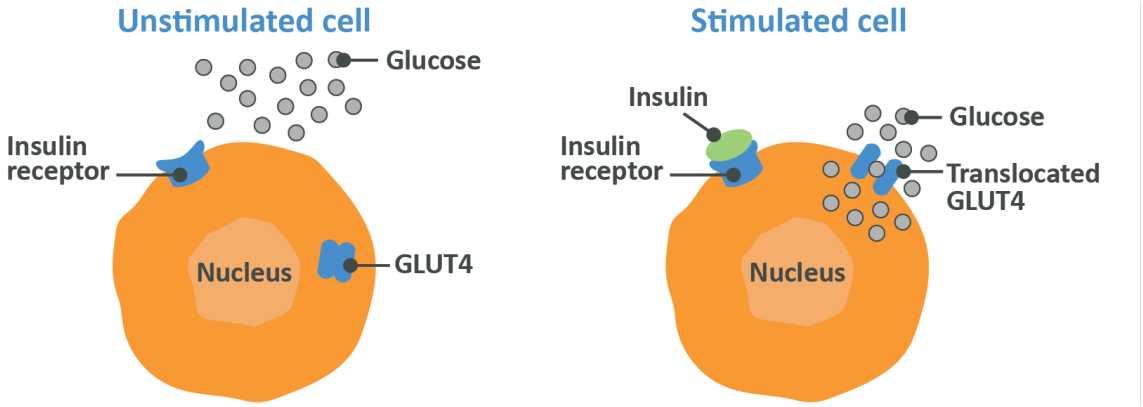
Whether you ate bread, sweets, yoghurt or an apple, what you have now are single sugars – mostly glucose and fructose but also others like galactose, ribose and mannose. These last three we will not explain in a huge amount of detail – they have functions and they are metabolically interesting, but to keep this module reasonably readable we will say only that galactose is used more or less in the same way as glucose, practically speaking (we've already explained that it is exported to the liver and converted to glucose there), and ribose and

mannose as well as a list of other monosaccharides we have not mentioned are used for very different, typically structural functions.

Let's instead focus on glucose and fructose – the **main** dietary monosaccharides. We'll start with how they get from the intestine to the blood:

- In order to reach the blood, monosaccharides are absorbed into the cells on the walls of your intestine, travel through these cells to the other side, and are then absorbed into the blood from these cells, rather than being absorbed into the blood directly from the intestinal cavity. They go across one membrane, across the cell and then across the other
- Sugar molecules are not able to diffuse across a cell membrane by themselves, they need to be helped out by transporter proteins
- The transporters we are talking about here are known as Glucose Transporter proteins (GLUTs), as well as Sodium-Glucose Transporters (SGLTs)
- There are 14 known GLUTs, often expressed at different tissues, for example GLUT1 is found in a great deal of places whereas GLUT4 is expressed near muscle tissues, fat tissues and your heart. You don't need to know them all, and only 4 will be mentioned in this course. They are incorporated into the cell membrane and allow glucose to cross into the cell but may not be here all of the time. GLUT4 transporters are usually kept inert inside the cell, and are transported to the membrane to start doing their job when certain signals reach the cell from elsewhere in the body
- After you eat a carbohydrate rich meal, GLUT2 transports glucose from the intestine and into the intestinal wall cells
- If there is fructose present, GLUT5 transports it into the cell
- GLUT2 is then responsible for transporting glucose and fructose into the blood from the intestinal wall

Fig. 32



So now the monosaccharides are in the blood. What next?

Following transport of glucose and fructose across the cells on the wall of the small intestine, they are deposited into the portal vein which carries them directly to the liver. The liver takes up most if not all fructose and metabolises it into glucose (along with galactose), meaning that from this point on they are identically managed. It also takes up some amount of glucose while the rest is deposited immediately into the blood.

Note: That because liver absorption of fructose is so effective, there is almost no trace of it in the rest of the body's tissues.

Some glucose which is absorbed into the liver at this step is used within the liver for energy, some is stored as liver glycogen (we'll return to what this means shortly) and the rest is returned to the blood as blood glucose or as it is typically referred to – blood sugar.

This is what happens when a person consumes a diet which is not in significant excess of their calorie requirements. We will return later to the fate of carbohydrates, more specifically fructose, if you are in a calorie surplus while consuming excessive amounts of it.

2.7.7. Maintenance of blood glucose levels

After glucose reaches the blood beyond the liver it immediately starts to raise blood glucose levels (not too surprisingly). Generally speaking, your body 'likes' to keep your blood sugar levels between 70-100mg/dl, so around 70-100mg of glucose per 10ml of blood. It's important to hold a certain amount of glucose in your blood because, as we said, glucose is an important energy source – your brain can only use glucose and it needs around 120g per day alone. Your red blood cells must use glucose, too while, as we will go in to momentarily, it's also an incredibly efficient energy source for almost all of your other cells, too.

If your blood glucose drops too low we call this hypoglycaemia. Due to your brain being literally starved of fuel, you will feel tired, lethargic and confused while potentially being prone to losing consciousness. If your blood glucose rises too high you enter hyperglycaemia and risk damaging your arterial walls (and you now know where that leads) along with your nerves, kidneys and eyes. If this is chronic then this is the condition referred to as diabetes. Homeostasis is the term used to describe the body's preferred equilibrium, with the maintenance of that equilibrium being a function of homeostatic feedback loops – when you are cold, you shiver and seek warmth to heat back up, and when you are low on energy you feel hungry. maintenance of healthy blood glucose levels is an incredibly important homeostatic function.

To keep your blood glucose levels in check your body obviously needs to be able to remove glucose after a meal and replenish it during periods of fasting, and it does this through virtue of the 'storage form' of glucose, glycogen. During periods of feeding and excess glucose, your body is able to store glucose as glycogen in skeletal muscle and the liver, and then during times of low energy availability it is then able to release this stored glucose again. Your liver is able to store around 100g of glycogen, while your muscles can store somewhere in the range of 350-450g.

Glycogen resembles a chain of glucose molecules linked together with 1,4 glycosidic bonds, with a branch every 8-10 glucoses caused by a 1,6 bond – this is very similar to amylopectin in starch, but with a greater regularity of branches. It's synthesised within cells of either the liver or the muscle tissue after these cells absorb glucose, but it's somewhat more complex than simply linking them together in basic dehydration reactions. It's managed by a series of enzymes and inter-linked reactions, which we will spare you the details of so long as you remember the following:

- The process of synthesising glycogen from glucose within cells differs between liver (hepatic) and muscle cells. In muscle cells, glycogen is produced relatively slowly, and once muscle glycogen is full, muscles stop absorbing glucose. In the liver, glycogen production and also glucose absorption speed up as your blood glucose rises in an attempt to keep levels stable even after muscle glycogen is full

Muscle can store around 450-500g of glucose as glycogen and your liver can store around 50g. On top of this, that stored glucose is constantly being used for something. What this should tell you is that you would need to eat a **lot** of carbohydrate, and total calories, throughout the day in order to maximise total glycogen storage.

Finally, it should also tell you that if you do indeed manage to consume glucose (and fructose) above your needs, and maintain a calorie surplus so that the excess sugars have nowhere to go, your muscle will stop absorbing them from the blood, but your liver won't, and your liver can't store it all. This is a problem, and we'll return to it at the end of this module.

In summation, under normal circumstances when glucose and fructose are eaten, fructose is converted to glucose and it largely ends up in the blood. The muscles and liver absorb the glucose that they need, and store it as glycogen for later use, with the liver taking priority. Then between meals this needs to be released from the liver to keep your blood sugar levels stable. This is mostly controlled by two hormones: insulin and glucagon, and we will explain everything you need to know about these momentarily.

2.7.8. Carbohydrate in the diet

Carbohydrate is the only macronutrient which is not considered essential, but as already mentioned it would be a mistake to think that this means carbohydrates aren't important.

In fact, it is because of the crucial nature of carbohydrate that it could be considered non-essential in the first place. Confused? Consider this – a non-essential nutrient is one which the body is capable of making if it needs to. If your body was not able to make glucose out of other things, that could spell disaster during times of famine, and therefore our body (and that of many animals) has developed this mechanism as a failsafe. Without sufficient glucose, your brain will die, your red blood cells will die, and therefore we are able to make enough each day for survival. Your body does this by breaking down amino acids and/or triglycerides in the liver in a process called glucogenesis – you already encountered this somewhat when we mentioned the fate of digested protein.

We don't just want to survive, we want to thrive, and carbohydrate has other benefits.

Glucose is the only substrate that can fuel anaerobic exercise, which is more or less any intense exercise lasting between around 4-240 seconds (there will of course be some variation). Sprinting, multiple-rep resistance training, uphill cycling, crossfit style conditioning and all other forms of 'short, hard' exercise will require carbohydrate to be performed optimally. You are of course able to do these things on an extremely low carbohydrate diet, but performance is impaired and fatigue accumulates much faster.

This means is that without carbohydrate, you are going to be unable to perform at your best in the gym, and we don't really need to tell you that performing some form of resistance training and/or conditioning work (hard cardio) is hugely important for body composition improvement and overall health.

Your carbohydrate need is going to be largely dictated by two things – your athletic endeavours and your overall calorie intake. Because carbohydrate is non-essential, dropping your total intake to quite a low level would not be directly harmful, and therefore carbohydrate is the macronutrient which can be considered the easiest to manipulate for fat loss. After you have determined the calorie intake you are going to be consuming, first work out your protein intake as this is key, then ensure you are eating enough fat to maintain health, and then your carbohydrate need makes up the remainder. This means that as your calories go up, generally speaking your carbohydrates will too – and vice versa. However, it cannot be stressed enough that carbohydrates are an integral part of a healthy balanced diet. For reference the UK Eatwell Guide in accordance with SACN dietary reference values recommends adults age 19-64 consume 50% of their total calorie intake from carbohydrates.

Additionally, as a final note, if you are inactive you do not strictly 'need' carbohydrate to fuel your activity in the same way as a hard exerciser does, you can feel free (and it might even be a good idea) to increase protein and fat to the top end of their ranges, therefore reducing your carbohydrate a little bit. This will likely make very little difference to physiological fat loss rates, but it might make some amount of difference to your hunger and satiety levels, as protein is more filling, and fats tend to offer a pleasant mouthfeel which can reduce cravings.

With carbohydrate discussed, we will now turn to the two hormones which control storage and release of the three macronutrients – insulin and glucagon.

2.7.9. Insulin: An overview

One hormone which you have probably heard of is insulin, often referred to as 'the storage hormone'. We have explained how glucose is handled within a cell and we may have given you the impression that glucose (and fructose and galactose) are simply 'soaked in' to the cells when local concentration is high, and this is kind of true, but not quite. As we mentioned earlier, monosaccharides are not able to cross cell membranes all by themselves and must be transported via special proteins known as GLUTs. A number of the different GLUTs (mostly GLUT1) allows glucose to slowly absorb into the cells in between meals, but then when blood sugar increases and must be absorbed rapidly, something needs to happen. In the same way, as amino acid levels in the blood begin to rise, your body needs to shuttle them to where they should be (protein manufacturing areas within cells). This is a job performed by insulin too.

The first thing that happens when blood glucose rises is that beta cells on your pancreas receive a signal to tell them that blood glucose is rising. When this happens, beta cells release the insulin they have stored within them, and simultaneously start producing more. This is a very similar process to how bile is released into the small intestine; there is an initial release of a product which has been stored in a reservoir and then a gradual continuation as that product is produced effectively 'to order'. Insulin is a hormone, which means that it is a signalling molecule (a protein) which is transported via the blood to cells in the body a long way from the original secretory glands, then binds to receptors and changes something about the cell. This is as opposed to other signals in the body which may travel via nerves, or travel between adjacent cells.

Insulin has a huge number of functions, including:

- Insulin causes GLUT4 within muscle cells to move to the cell wall, allowing it to act in those cells to transport glucose across the membrane and into the cell itself. While some amount of glucose absorption can happen during everyday activity due to GLUT1, amongst others, GLUT4 stimulation caused by the presence of insulin in the blood rapidly increases this process
- Insulin stimulates an increase in glycogen synthesis in the cells of the liver, which in turn increases the rate at which the liver absorbs glucose
- Insulin increases protein synthesis, helping to lower blood amino acid levels and increasing the rate at which proteins are made
- Insulin can also stimulate an increase in a process called de novo lipogenesis in the liver. This is the process by which glucose is converted into pyruvate which then ultimately becomes a triglyceride, which can be packaged into VLDL and ultimately stored in adipose tissue. This process is incredibly unlikely to lead to meaningful fat gain, as the amount of triglycerides produced from glucose over the course of a day, even if carbohydrate intake is exceedingly high, is almost certainly to be no more than 5-6g
- Additionally, insulin is able to help triglycerides from the blood enter muscle cells for use as energy, or adipose tissue for storage. It would however, be incorrect to think that insulin is **required** for fat to be stored. If insulin is not increased due to carbohydrate consumption but fat is consumed and enters the blood, Acylation Stimulating Protein (ASP) in the adipocytes themselves allows fat to be stored

In type 2 diabetes, insulin is not able to increase GLUT4 translocation in muscle cells, or increased glycogen synthesis in the liver, and therefore blood levels of glucose gradually rise and stay elevated. This can cause inflammation and damage to arterial walls, nerve damage and organ damage to boot. When insulin doesn't do its job, you have problems.

Insulin in effect helps to reduce blood sugar, and it does this through the steps above, as well as by adopting one other means, and that is to counteract the action of its opposite hormone – glucagon.

2.7.10. Glucagon: An overview

Glucagon is the polar opposite to insulin. Its role is to facilitate the breakdown and release of stored energy and it does this via three means. Firstly, it releases triglycerides from adipocytes to allow them to travel to the liver for conversion to glucose, or to muscle cells for direct use in the production of energy. Secondly, it will break down liver glycogen into glucose, and finally it will break down proteins around the body into amino acids, which can be converted into glucose and used for energy.

Importantly, glucagon has very little effect on skeletal muscle or the glycogen stored there because muscle cells do not have a high concentration of glucagon receptor sites.

As you can see, if insulin does not 'stop' this process from happening – blood glucose will continue to stay elevated, not only due to glucose not being removed, but from additional glucose entering the blood from the liver.

2.7.11. The fasted and fed states

In between meals, glucagon will gradually release fatty acids from your adipocytes and glycogen from the liver to make glucose, which is then deposited in the blood to keep blood glucose levels stable. When you eat, inulin's **first** role is to stop this happening, so that more glucose is not deposited in the blood from your body's stores. Then it gets to work depositing what you have already eaten.

Another issue in diabetes is that this process doesn't happen. Glucose which you have eaten is not as efficiently absorbed, but moreover, glucose is still being released from the liver, increasing blood glucose to potentially harmful levels over time.

To bring this all together, consider that the body has two key states – fasted and fed.

Fed state: For the first couple of hours after a meal, nutrients will be steadily reaching your bloodstream from the intestines; meaning that as the nutrients are absorbed, blood glucose, blood amino acid levels and triglycerides are elevated. These nutrients will be transported around your body to various cells and tissues which might immediately need them, and what is not needed is either stored (fat and glucose) or converted to something else and stored (protein) to return blood levels to baseline or homeostasis.

Insulin is the primary driver of this. In the fed state, it shuts off the breakdown and release of glycogen, it promotes the formation of new glycogen, it activates protein synthesis in various tissues, and it promotes the storage of fatty acids in either muscle cells or adipocytes. Glucagon is supressed here.

Fasted state: A few hours after a meal (we cannot really give a specific number as it depends entirely on the composition of the meal, but 3-4 hours for a standard sized mixed meal is about right for descriptive purposes), blood glucose starts to drop. The glucose from the meal is no longer present, and your pancreas 'senses' that levels are going to reduce to below what they should ideally be.

At this time, insulin secretion is suppressed which then in turn amplifies glucagon release. Glucagon gets to work promoting the release of glycogen from the liver (**not the muscle cells**

as it cannot) into the bloodstream for use around the body during day-to-day activity. A lot of this glucose is used by the brain, and by red blood cells which are another obligate glucose user. At the same time, glucagon encourages the release of amino acids from various tissues and from other proteins like enzymes or hormones – these are then either used in the liver to make more glucose, or they remain in the blood and are used in protein synthesis later. Finally, glucagon encourages the breakdown and release of triglycerides into the blood. The fatty acids can be used directly by various cells around the body (especially your heart) to produce energy, and the glycerol backbone from the triglyceride makes its way to the liver and is used to produce more glucose.

Note: Of course, though glycogen cannot be broken down and released to the blood during the fasted state, you are still able to break it down and use it within the muscle cells which have it stored to perform exercise. This is because the cellular hydrolysis of glycogen and subsequent usage of glucose to produce energy is not dependent on glucagon. Whether your muscle uses stored glycogen or the newly freed fatty acids depends on the intensity of the activity you are performing – whether you are strolling to the kitchen or sprinting for the finish line, your muscle needs to get energy from somewhere, and the reason why it 'chooses' one or the other is the subject of a different section.

This is an incredibly brief overview, but we hope it's provided a pretty simple insight into how insulin and glucagon work together in the fasted and fed states. Insulin isn't likely to store glucose (or sugar) as fat to any meaningful level, and if it didn't get released and do its job then glucagon would start to raise your blood sugar levels and kill you – insulin is **not** a 'bad hormone' and increases in it are not something to be avoided.

As a final note here, we hope that this section has shown you just how capable your body is of maintaining homeostasis. If you eat a **huge** amount of carbohydrate, your body is able to store a lot in the muscle, then what is not stored is cleared up by the liver. So long as, over the course of a day, your energy intake is even, some of that stored carbohydrate will stay stored and some will be released for use and nothing bad will happen. On the flipside, if you do not eat for a long period of time you do not induce hypoglycaemia because your liver glycogen stores will stabilise blood glucose levels, and if this is not sufficient your liver is able to absorb freshly released glycerol from triglycerides in your bodyfat, convert these to glucose and keep everything as it should be.

In fact, it is only after multiple days of fasting that glucose levels would meaningfully decrease, and at this point your body would enter a state of ketosis. This, again, is far beyond the scope of this course, but ketosis is a state whereby there is insufficient glucose to keep blood sugar levels stable and fuel your brain, and as such, the fatty acids released from triglycerides can be used to make ketone bodies in the liver, which can provide an emergency backup. If you wish to learn more on ketosis, we will cover it on our Practical Academy course.

2.7.12. Fructose metabolism

Before we can go further we need to finish discussing the 'unusual sugar', fructose. Fructose is unusual because it has a different metabolic fate than glucose and because this different fate can potentially have an impact on our health.

Fructose is found in its free form in fruits, honey and in small amounts in some vegetables. Generally speaking, it's not likely that you would ever historically have consumed a lot of it. In the modern world, however, fructose intake is significantly higher than it ever has been before due to one factor – sucrose.

As you already know, carbohydrates are absorbed in the intestine and then deposited at the cells in the liver. Some amount of glucose is absorbed immediately for glycogen synthesis but the rest makes its way to the bloodstream. Once there, insulin is released which increases the rate at which glycogen in synthesised in the liver, meaning that glucose is rapidly sucked up into the cells there.

Fructose does not require insulin for rapid absorption, and therefore almost no fructose ever makes it to the bloodstream. This means, incidentally, that fructose has little to no effect on blood sugar and it also does not impact insulin levels. This is why agave nectar, which is almost 100% fructose, is marketed as being ideal for those with poor insulin control.

What happens is that fructose is converted to glucose under normal conditions, but in overfeeding it's also used to produce triglycerides. In fact, intakes of over around 75g of fructose per day seem to cause worsening of non-HDL cholesterol and total triglyceride levels. With that said, amounts of around 50g do not seem to, which would make at least some logical sense as the liver is able to store at least this much as glycogen anyway (not to say that all of that fructose would be stored, some would be converted to glucose, too).

This is, however, only truly relevant in situations of calorie surpluses. If you are consuming a diet which is in negative calorie balance, though an increased amount of triglycerides may indeed be produced (because your body requires that energy to perform its daily tasks) you will ultimately oxidise more fat than you synthesise, and therefore while losing fat, consuming in excess of 75g fructose shouldn't greatly impact your health.

Of course, this would entail eating around 150g of table sugar or equivalent (or an insane amount of fruit – a typical 140g apple contains only around 10g fructose) and this is probably a bad idea for dental health and satiety if nothing else.

2.7.13. Summing up digestion, absorption and utilisation

Very briefly, here's everything we have talked about, summed up simply:

- When you eat something, it is broken down into its constituent amino acids, monosaccharides and triglycerides then absorbed from the intestine
- Monosaccharides and amino acids enter the portal vein and reach the liver
- Fructose and galactose are converted to glucose in the liver, and this glucose (as well as some glucose which was eaten) are either used to make liver glycogen or used to

fuel the things the liver needs to do. The rest of the glucose reaches the blood and is sent around the body to be stored in muscle cells or used immediately by various tissues like the brain or red blood cells

- Amino acids are either taken by the intestine or liver for use in protein synthesis, used to make glucose in the liver, or transported around the body for use in protein synthesis
- Triglycerides use the lymphatic system to sidestep the liver, and are transported around the body. They are either broken down into fatty acids and glycerol to use for energy, or stored as bodyfat
- In between meals, these things are then released to use for energy, or used to fuel the cells which have stored them (for example, muscle glycogen isn't released, it's used in the cells which have stored it)
- If you overeat, you store more than you release over time and gain fat
- If you under-eat, you release more than you store over time and lose fat
- By increasing dietary protein, you are able to utilise more protein to build muscle, and will not break muscle down so much to provide energy or necessary amino acids
- Certain fatty acids can be harmful if overeaten, and a balanced approach is necessary
- Sugar is not the enemy but it contains fructose and shouldn't be eaten in enormous amounts – especially if you are eating more than you need to maintain
- Everything is checks and balances, and you can't really beat the system

We will finish this module by discussing how these substrates are actually used as energy, because they differ in the way that the body can utilise them in a way which explains why we recommend a higher carbohydrate intake in a given calorie intake for those engaged in intense exercise.

2.7.14. Cellular energy production

We will not go into great amounts of detail on this as it's not 100% relevant for what you will actually **do** with your nutrition, but it's important to give you a background understanding as this allows us to understand why things are the way that they are.

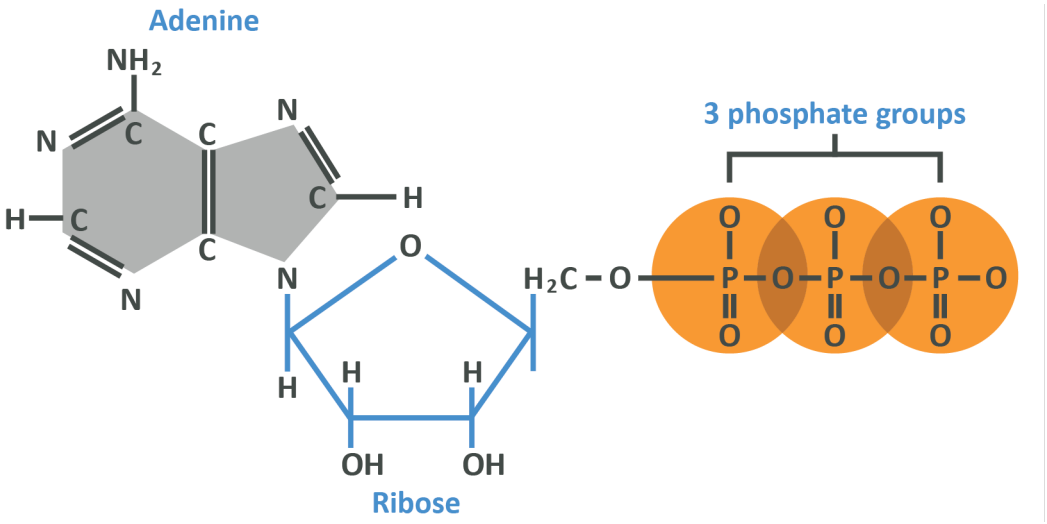
When you consume any of the three macronutrients, they can be used within your cells for energy. Your muscle cells use this energy to move, your nerves use it to send signals and your cells use it to make proteins. You basically need it for **everything**.

- **Protein:** Can be converted into glucose and used to produce energy
- **Fats:** Stored as triglycerides, the fatty acids can be used for energy and the glycerol can be used to make glucose, or synthesise more triglycerides

- **Carbohydrates:** Broken down to glucose which is either used as energy, or in very small amounts converted to fatty acids which can then be used in the same manner as fatty acids that have been consumed

The process by which this occurs is hugely more complex than anything we have discussed so far, but ultimately either glucose or fatty acids make their way to the cells requiring energy – either from the blood or from that cells' storage sites, and those cells then use internal machinery called mitochondria to convert the substrate into a tiny molecule called Adenosine TriPhosphate (ATP). ATP is the thing which actually 'gives' energy to something – and it does it in the following way:

Fig. 33



Above you see a molecule of ATP. On the left is an adenine group which is made of hydrogen, nitrogen and carbon. That is linked to a ribose (remember we mentioned this when talking about the different sugars we consume?) which follows a pretty similar configuration, indicating it is a sugar. These two aren't really so important for this discussion – place your attention to the right-hand side where you see 3 phosphate groups.

Each phosphate group is bound to the next, and it is these bonds which are important.

Within your cell are numerous molecules of Adenosine Di-Phosphate (ADP). You should now know that di- denotes 2, and should therefore be able to deduce that ADP is the same as ATP but with one fewer phosphate group attached.

When cells put glucose or fatty acids through a certain set of reactions, some energy is released, which allows ADP to pick up another phosphate and effectively 'store' the energy which has just been produced. Think of ADP like a flat battery – your cells can use glucose or fatty acids to charge that battery by attaching another phosphate.

When your cell then needs the energy, the bond between the 2nd and 3rd phosphate is broken, releasing it, and re-synthesising a flat battery ADP and a spare free phosphate.

The problem is that ATP is difficult to store a lot of. Because of this, your body needs to be able to continually recycle the ADP-ATP-ADP-ATP process, and that means it needs to convert those substrates into energy. It can do this using either glucose or fatty acids, but because glucose is what is used most of the time, we will cover this first.

Glucose is used for cellular respiration, which is a 3-stage, incredibly complicated process starting with glycolysis, moving on to the Krebs cycle and finishing with the electron transport chain which converts one molecule of glucose into up to a maximum of 38 molecules of ATP (though realistically it's going to be closer to 30 or so). Don't worry, we are not going to go through all of this in detail, but we will be giving you a brief overview of it so you can understand where things are.

Note: You can also produce ATP using one other thing – the PhosphoCreatine Energy System. This is the 'fastest' means by which your body replaces ATP, and is therefore used for intense, often maximal effort such as exertions lasting less than 5 seconds or so. This would be hugely useful for powerlifters, throwers, jumpers, sprinters and athletes involved in martial arts or other combat sports. In order to replace the third phosphate, your body is able to 'steal it' from a molecule of phosphocreatine which is stored within muscle cells and therefore does not need glucose.

Muscle cells will always have some amount of phosphocreatine (creatine bound to a phosphate) but those who supplement creatine will have a larger amount. This is the primary means by which creatine can improve performance, and will be discussed in more depth during the module on supplements. After this, we need to consider respiration.

2.7.15. Respiration (aerobic)

The first stage of respiration is glycolysis, the breaking down of glucose which happens within the cytoplasm (cytosol – the liquid interior) of the cell. The first stage involves simply breaking down the 6-carbon chain of glucose into 2 3-carbon chains called pyruvates. This is an anaerobic process, meaning that oxygen is not required to help it – this will become important later on.

At this stage, 1 glucose and 2 ATP's get used up to produce 4 ATP's and 2 pyruvates. As a side product, free hydrogens are released which could raise the acidity in the cell and must therefore be 'cleaned up'. This cleaning up is done by molecules called NAD+ which are free in the cell, which collect the hydrogens to become NADH which then enters the mitochondria. In short, the first stage can be summed up as so:

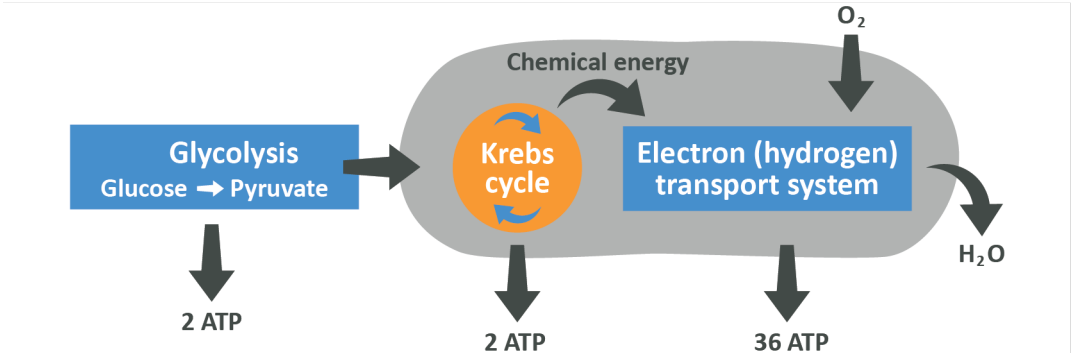
- **1 Glucose + 2 ATP + NAD + = 2 Pyruvate + 4 ATP + NADH**

The pyruvates are then oxidised (meaning that this reaction needs oxygen) into acetyl CoA. This acetyl CoA then enters the mitochondria and is used to produce 2 more ATP's in the set of reactions known as the Krebs cycle. The Krebs cycle also produces more NADH and some FADH (a similar molecule, for our purposes here, consider them both 'electron carriers') which along with the original NADH are sent into the next set of mitochondrial reactions called the electron transport chain.

So far we have produced 4 ATP's and around 10 units combined of NADH and FADH which have been sent to the electron transport chain. If the above made no sense, just remember that one glucose leads to these things.

The 10 NADHs or FADHs which have been sent to the electron transport chain are finally used to produce up to another 34 ATP's – the bulk of what the mitochondria can produce for a single glucose. Like the Krebs cycle, this stage also requires oxygen.

Fig. 34



On the left you see the initial glycolysis which gives pyruvate, NADH and ATP (remember that to get NADH, the NAD+ which is always present in the cell gets used up). The pyruvate then enters the Krebs cycle in the form of acetyl CoA and releases NADH and FADH as well as ATP, then finally all of the NADH just produced enters the electron transport chain where a ton more ATP is made, also releasing the now unburdened NAD+ to use again.

The net result is up to 38 ATP's generated.

This is the way in which carbohydrates are used for energy during day-to-day living and low intensity exercise. It's a high-yield but relatively slow process which demands oxygen to work (which is why you breathe heavily when you run for a long time).

Sometimes, though, you need energy quickly, often faster than you could supply oxygen. This means that energy must sometimes be produced in the absence of oxygen, through what is known as anaerobic respiration.

2.7.16. Respiration (anaerobic)

Anaerobic respiration occurs almost exclusively in the muscle tissue, because it is only likely to be these whichever work in the absence of oxygen. Other tissues will use the above method to produce energy from glucose.

Anaerobic respiration starts off with the same glycolysis process as aerobic respiration, with glucose being broken into 2 pyruvates in the cytosol. This yields 2 pyruvates and 2 ATP, but because there is no oxygen available to oxydise the pyruvate into acetyl CoA, and there is no electron transport chain for NADH to go into, they build-up.

This build-up of NADH means that there is a depletion of NAD+ going on (as NADH is NAD+ with an added H+ or hydrogen ion) which is a problem. If NAD+ cannot accept the spare electrons, glycolysis cannot occur. What happens now is that pyruvate becomes fermented

into lactic acid (sometimes known as lactate) by taking on some of the spare electrons from NADH, resulting in there being more NAD+ and re-allowing glycolysis to occur.

In short, glycolysis adds hydrogen electrons to an NAD+ and produces pyruvate. The resultant NADH can then give the electrons back and become NAD+ again, while turning the pyruvate into lactate. This means that glycolysis can repeat over and over. Crucially, this is **far** less efficient than aerobic respiration per unit of glucose, but it is vital if you are ever going to perform exercise without oxygen. Without this happening, you could not perform well in a sprint or set of bench press.

The lactate/lactic acid which is produced is then able to be re-absorbed into the blood or surrounding tissues, converted back into glucose or other molecules like amino acids, and stored or used as per normal.

Interestingly, despite what you may have believed, lactic acid is **not** the thing that gives you 'the burn'. What gives you the burn is the build-up of free H+ ions.

2.7.17. Fatty acid oxidation

The final means our cells can use to produce energy is through fatty acid oxidation which, as you can probably guess, is dependent on oxygen. This is one of the primary means by which your body fuels your day-to-day life, and it is also a useful 'energy system' for extremely long, low intensity exercise.

Of the triglycerides you consume, around 95% of the energy is contained in the fatty acids. The glycerol backbone can also either be used to make energy in the liver, or to make glucose to use in the above manner.

As for the fatty acids, recall that the key stage of respiration which produced ATP was the electron transport chain – here we will basically be doing the same thing with fatty acids. The first stage is to oxidise the long chains of fatty acids into chunks which are 2 carbons in length – Acyl CoA. This then enters a cascade of reactions to produce Acetyl CoA.

Now that the fatty acid has been oxidised into acetyl CoA, you may have predicted that the process released hydrogens to be picked up by NAD+ and carried into the mitochondria for use in the Electron Transport Chain (ETC). We can also then use the acetyl CoA in the Krebs cycle to make more ATP **and** more NADH and FADH to send to the ETC. You should already be aware that this is creating a ton of ATP. In fact, a 16-link fatty acid like palmitic acid can be broken into 8 acetyl CoA's, and so can be used to produce 107 ATP molecules. This is an incredibly efficient process!

This is precisely why fats have more calories per gram than carbohydrates, but it's a very, very slow process and so when carbohydrates are readily available, your body will in general prefer to use these.

2.7.18. In summary

When you are at a state of very low activity, or performing extremely long duration exercise, the majority of the energy used by your body is going to come from aerobic respiration or fatty acid oxidation. While some tissues like your heart which can only use fatty acids or your

brain and red blood cells can only use glucose, your muscle cells, liver and many other cells can use both. During day-to-day life or very low intensity exercise, your body could use either fatty acid oxidation or aerobic respiration to fuel what it is doing, and the one it chooses primarily depends on your diet.

If you consume more glucose, you will use more glucose. If you consume more fats, you will use more fatty acids.

During exercise, the primary fuel for muscle cells will always be glucose, though at very low levels fatty acids can be used too.

2.7.19. So… how do we gain and lose fat?

As we have repeated over and over, overeating calories causes fat gain. You may or may not have noticed, however, the specific nutrients you consume which provide those calories are not equally likely to be stored as fat.

Protein which is consumed in excess of need is converted to glucose to use as energy (or store as glycogen to be used for energy later) and some amount of it can be processed and used for energy directly. Glucose can be used or stored as glycogen to use as energy later, while a very, very small amount can be converted into fatty acids either in the liver or in the fat cells, and triglycerides can be stored directly in fat tissue.

This **could** lead you to think that a low-fat diet is the way forward, and that eating as much carbohydrate and protein as you like won't lead to fat gain, but you would be wrong. The simple way to put this is that your body and the systems it uses to process nutrients for energy production, storage and survival are **way** more complicated than that, and there is almost always a feedback loop or other mechanism which, no matter what you eat, brings you at least 95% of the way back to the 'calories in vs. calories out' approach, when fat loss or gain is the concern. If you overeat on a diet which is almost entirely coconut oil, butter and lard, you'll gain fat. If you overeat on a diet which is almost entirely rice, potatoes and pasta, you'll gain fat, and of course you'll gain fat in the middle ground, too.

The mechanism for storing that fat, as in where the stored fat itself actually comes from may differ, but the end result is always the same, and that's all you really need to remember. If you ever hear that X food (for example, pasta or cheese) is more fattening than another food, that claim is wrong. If you ever hear that X nutrient (for example, sugar, fructose, or fat) is more fattening than another nutrient, then again that claim is pretty much wrong too.

Though we will concede that an **extremely** minor difference to fat gain would be seen if one diet was very high-fat and the other very high carbohydrate with both having the same calorie load, in that the fat would be easier to store. This would have to be an extreme diet which was artificially low-fat or carbohydrate, created with refined foods, and it's not really that practically useful. For fat loss, there would be no difference at all.

The point remains, that although you can't store carbohydrates or protein meaningfully as fat, you can certainly gain fat if you overeat them and calories are always king. The long

answer as to why you can gain fat while eating macronutrients which can't really be converted into fat is as follows:

- As you know, weight loss or gain comes down to energy balance. If energy in > energy out, you'll gain weight. This can then be broken down further into nutrient-specific balance. If fat in > fat out, you're gaining bodyfat, and therefore fat gain can be considered as a positive fat balance
- Positive fat balance is achieved when fat intake exceeds fat storage, and ultimately that comes down to energy balance. When you consume fat in excess of need, what is not oxidised is then stored immediately in either adipose tissue, in small amounts in muscle tissue and potentially around the organs. When you consume carbohydrate and/or protein, the amount of fat which you oxidise will be reduced accordingly
- Importantly, though, if you are eating a certain amount of calories, the amount of fat you eat will be reduced as the amount of carbohydrate/protein you eat increases. If you overeat fat, you gain fat because you store that fat – but if you overeat carbohydrates you gain fat **still** because you store the fat which you consumed
- After a high-fat meal, you oxidise the fat in the meal and store any remaining fat
- After a high carbohydrate meal, you oxidise the carbohydrates in the meal and store any remaining fat

Regardless of which way you play it, for a given calorie intake, the fat you gain will be the fat you have consumed, at an amount roughly equal to the calorie surplus you have created. This does assume a given protein intake, though. Because protein is used for protein synthesis, and because it requires some amount of conversion before it can be used as energy (and because of a few other hormonal effects), protein intake can indeed affect your body composition changes for a given calorie level.

The same thing goes for fat loss, too, assuming protein intake is set at the right level, the amount of carbohydrate and fat doesn't really matter outside of exercise performance and overall preference. This is important, though – go too low on fat and you can harm health, go too low on carbohydrates and you can impair exercise performance and overall perceived energy. The balance that is right for you is individual, this is what is key and now we will work out how to find it.

2.7.20. Summing up the theory; estimating ideal daily intake

What we have done here is given you a huge amount of background information to justify the following recommendations. It's important to know, however, that this is not the end. Once you have these numbers you need to apply them to your own life, and the way to do **that** will be laid out in the following modules. It could be implied, if we didn't include this pre-amble, that the way to eat which is the most health-promoting, would be to weigh and measure all of your food while paying close attention to the labels, and then track this to make sure you are reaching the 'correct' intakes for each nutrient, but that may not always be the case.

Of course, many people **do** indeed track their macronutrient intake and we will discuss this in later modules because doing so is effective as a tool and invaluable as an educational process, but that doesn't make it the only way. There are methods of ensuring you get as close as is practically relevant without weighing anything, so please don't feel overwhelmed at this point.

In module 1 we showed you how to estimate your calorie needs. Please use the below to now convert that into your macronutrient needs. The steps to take are in order, but the following flowchart should make this a little easier to work out.

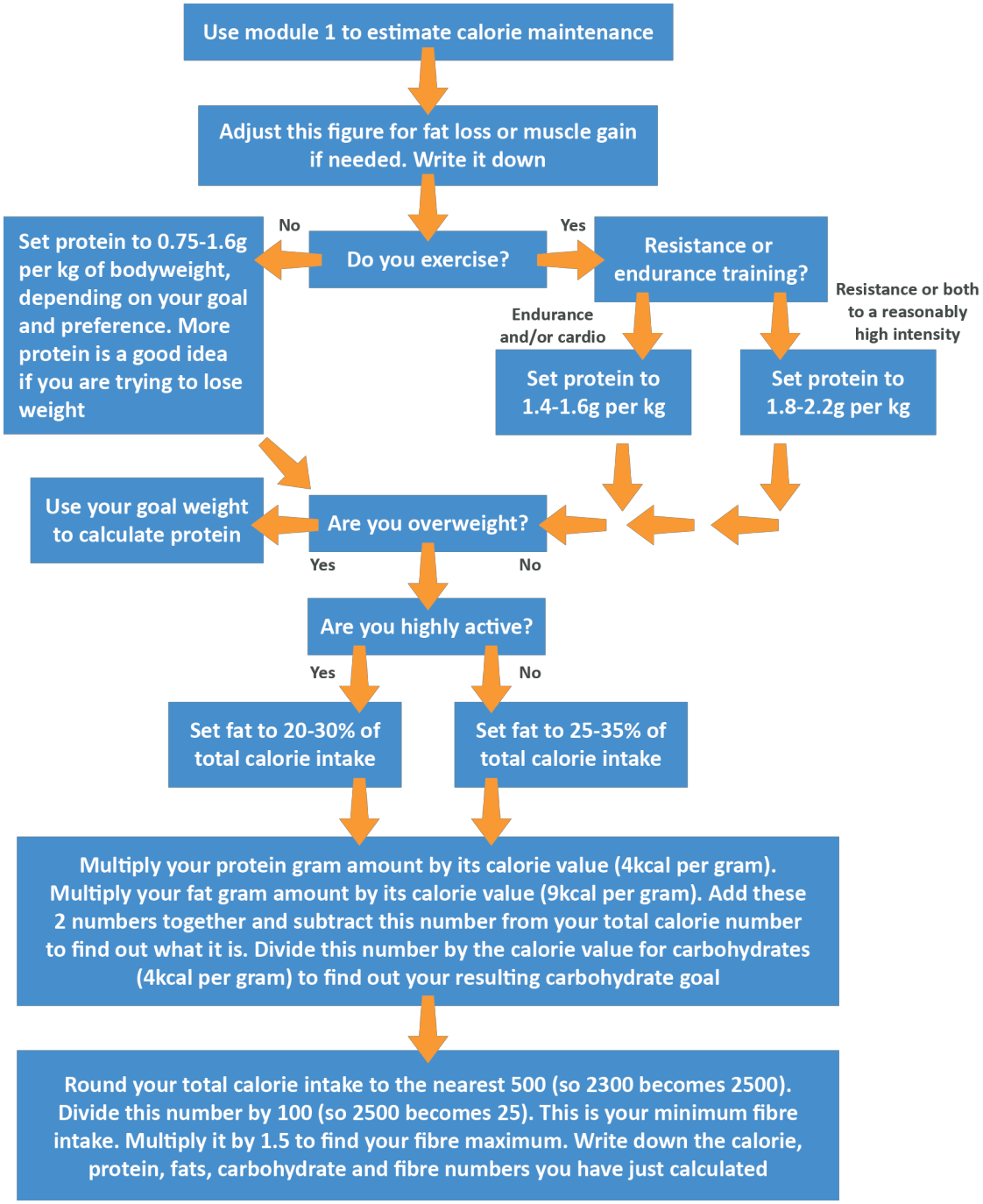
- **Calories:** As per module 1
- **Protein:** 0.75-2.2g per kg of bodyweight or 'goal weight', from high quality sources wherever possible, broken up over 2-5 roughly even meals per day, with at least one of these somewhere within 2 hours pre and post-workout
- **Fats:** 20-35% of total calorie intake, split evenly between saturated, mono-unsaturated and polyunsaturated fats, ensuring at least 6.5% of your total calories are polyunsaturated fats and ensuring you get sufficient Omega-3 fatty acids (we recommend at least 1, but ideally 2-3g)
- **Carbohydrates:** The remainder of your calories, with ideally no more than 50g coming from fructose
- **Fibre:** 10-15g per 1000 calories consumed. This is the first time fibre has been mentioned in this module and this recommendation as well as the role of fibre will be explained later in more detail, so please take our word for it for now

Confused? Use these steps…

As a baseline, the UK Eatwell Guide recommends the following for a typical man and woman, both relatively sedentary:

	Men	Women
Energy	2500kcal	2000kcal
Protein	55.5g	45g
Carbohydrate (at least)	333g	267g
Fat (less than)	97g	78g
Fibre	30g	30g

Fig. 35



An example of how this looks can be found in the Foundation Academy video content associated with this module. In the next module, we will turn our attention to micronutrients, the nutrients which we consume in very small (micro) amounts.

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