Nigee's Guide to Losing Body-fat Healthily

(It's not another "diet" book, honest!)

by
"Nigeepoo"

Foreword

I said I was never going to do this - write a book, that is. I mean, aren't there enough "diet" books out there already? Aren't all "diet" book authors just out to make a fast buck? Mind you, this isn't a "diet" book, well, not as you know it.

However, after giving a friend a 4-hour explanation on Healthy Body-fat Loss, occasionally interrupted by her 3 kids and 2 dogs, she said that I really ***must*** write a book as my advice was unique, logical and easy to understand and deserved a wider audience.

So, you have Emily to thank/blame/whatever for what follows. So, who is Nigeepoo?

My name's Nigel and I was a Principal RF Design Engineer with Thales (formerly Racal) until 30th September 2006 when I took early retirement. So what's an ex-Electronic Engineer doing writing a book about Losing Body-fat Healthily? The story starts in 1997. I'd just come through an acrimonious divorce and I was tired, bloated, 17st 7lb and depressed.

Then a pamphlet dropped through my letterbox. It was from Agora Lifestyles, promoting a book by a Dr. Robert C. Atkins (***who?***). I didn't actually buy his book but I read the pamphlet from cover to cover, and it described sleepiness after meals containing carbohydrates. All my life I have felt very sleepy after starchy meals but never knew why. So, despite my disbelief that Atkins' diet could work, I cut out bread, pasta, potatoes, rice, cereals etc - all the things we were told were good for us as they were "low-fat".

Within days, I was like a new man! No more sleepiness, body-fat slowly disappearing and no more heartburn after eating or drinking carbs. I was a total convert. By nature I'm very curious (which is why I'm an Engineer), so I wondered how the Atkins diet could possibly work. In 1999, I got Internet access at work and was delighted to see that there were people out there (some of them doctors) other than Atkins who were saying much the same thing.

I still didn't understand how the diet worked so I studied some biochemistry web-sites to get an understanding of human metabolism. In November 2002, I joined the Muscletalk forum after e-mailing the site owner James criticising an article on ketogenic diets that he had written. Username "Nigeepoo" was born. This was the beginning of a new phase in my learning. From there I found a US & then Canadian bodybuilding forum which allowed me to learn even more about optimum nutrition. In January 2003, the BBC series "Diet Trials" studied the Atkins diet amongst others. At the end of the series, viewers were referred to a BBC Nutrition & Fitness message board where I've been posting ever since. As a result of various recommendations, I bought some books on running, diet & nutrition, metabolism and biochemistry. I also surfed various journals, looking for studies on ketogenic diets and the effects of different proteins, fats and carbohydrates on test subjects.

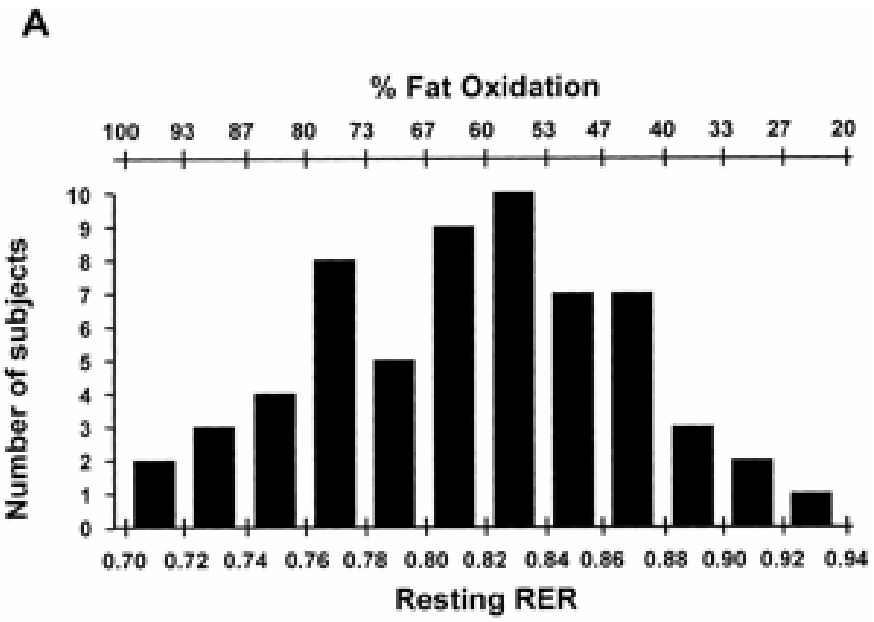
If there is one thing that I have learned, it's that **everyone is different.** I shall make all statements of significance bold for emphasis. So, on with the book.....

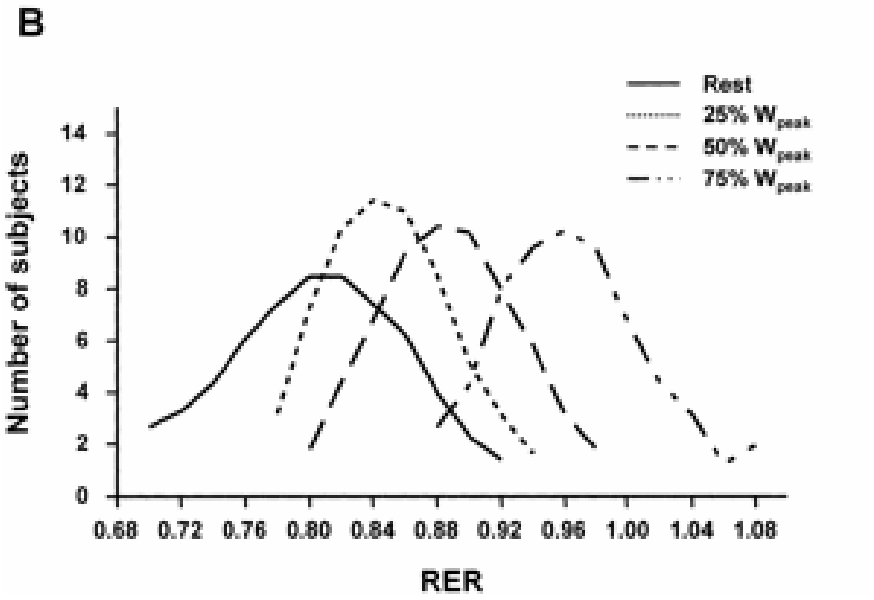
Everyone is Different

You, the reader, are unique. I don't know your metabolism or activity level so I couldn't possibly recommend a suitable diet for you. Before you throw this book away in disgust, let me explain. The point of this book is to give you the tools to work out for yourself what your body needs when it needs it and what it doesn't need when it doesn't need it.

Any person or book that says "eat X% of calories from proteins, Y% of calories from carbs and Z% of calories from fats" is assuming that everyone is the same. This applies to current "Healthy Eating" guidelines, which state that everyone should be eating 15% of calories from proteins, 55% of calories from carbs and 30% of calories from fats.

To illustrate just how different people are, click http://ajpendo.physiology.org/cgi/content/full/279/6/E1325 or copy & paste it into your Internet browser's address bar. Find Fig. 2. Here are the results, for those who don't have Internet access:- Used with permission.





What does RER mean? Respiratory Exchange Ratio (RER) is the ratio of carbon dioxide breathed out to oxygen breathed in. This ratio depends on the fuel that the body is burning for energy. If the body is burning 100% fat, RER = 0.7. If the body is burning 100% carbohydrate, RER=1.0. If the body is burning 100% carbohydrate anaerobically (flat-out sprinting), RER > 1.0.

The top diagram is a histogram of fasted RER & % fat oxidation vs. number of subjects. At the left-hand end of the histogram, there are two cyclists with a fat oxidation of 93 - 100%. At the right-hand end of the histogram, there is one cyclist with a fat oxidation of 20 - 27%.

There's a helluva big difference between burning 93 - 100% fat at rest and burning 20 - 27% fat at rest. Interestingly, average fat oxidation is 66%, which means that carb oxidation is 34%. So, on average, at rest, people burn twice as much energy from fat as from carbs. Hmm! So why do current "Healthy Eating" guidelines recommend almost twice as much energy from carbs as from fats for sedentary people? Because they're crap?

As exercise intensity increases, the peak in the histogram shifts to the right as shown in the lower diagram. At 25% full work-load, mean fat oxidation is ~53%. At 50% full work-load, mean fat oxidation is ~37% and at 75% full work-load, mean fat oxidation is ~13%. I suspect that at 100% full work-load, mean fat oxidation is 0% i.e. 100% of energy is being obtained from carbs when running flat-out. Someone on a high-fat, low-carb diet like Atkins induction would keel over with hypoglycaemia if they exercised for any length of time at this rate.

As there is so much variation from person to person, you, the reader must establish your own optimum proportions of proteins, fats and carbohydrates in your diet and these will vary depending on when and how much exercise you do. It all sounds horribly complicated but it isn't.

You apply the principle of "**Eat, monitor and adjust accordingly**" to quote Toxic Toffee (Muscletalk Moderator). The eating bit I will advise on in the next chapter. The monitoring bit does not actually involve the use of scales.

Hang on, isn't this book all about losing excess weight? Er, NO actually. Remember the old joke about the best way of losing 5lbs of ugly flab being to cut your head off? Well, if you cut your head off, you've lost 5lbs, but you're dead. Not exactly a good way to lose weight, then. As your body is made up of water, muscles, fat, bones, cartilage, tendons, organs, skin etc and your scales can't tell the difference between one thing and another, losing weight the wrong way can make you less healthy.

If you starve yourself (or skip breakfast for instance) or go for a run before breakfast, as your body is lacking in **glycogen** (see later) and **amino acids** (see later), a corticosteroid hormone called cortisol is secreted which converts muscle tissue into amino acids and then glucose. It also suppresses your immune system and eats away at your bones.

Unless you have a lot of bulky muscle on your frame, it's body-fat you should be losing, and to monitor this, either use a tape-measure around your waist, or check which hole your belt is on, or strip down to your underwear and jump up and down in front of a full-length mirror. As Big Les (another Muscletalk Moderator) says, "**If it jiggles, it's fat**".

So, what happens if you eat too many carbs but your body isn't burning them? Initially, carb intake tops-up liver and muscle glycogen stores. The liver can store about 70g of glycogen and muscles can store about 400g of glycogen. Once glycogen stores are full though, more carb intake passes through the lipogenesis pathway - this basically means that carbs are turned into fat - which you end up wearing as body-fat. But there's even worse news. On the way to being worn as body-fat, fat is in the blood as **triglycerides**. This is bad news for the cholesterol particles in your blood. See the

chapter on **cholesterol**. What happens if you eat too few carbs? As stated above, a "sugar-burner" taking in insufficient carbs will become hypoglycaemic. See later.

Just discussing weight again for a moment, it's often said that all diets are the same, as weight loss is all about calories. This is true - sort of. Click http://www.second%2dopinions.co.uk/do%2dcalories%2dreally%2dcount.html or copy & paste into your Internet browser's address bar. Weight loss is ***mostly*** determined by (calories eaten - calories excreted) - calories burned. Click http://www1.wfubmc.edu/news/newsprintfriendly.htm?ArticleID=1869 or copy & paste into your Internet browser's address bar. So, if all you're interested in is weight loss, put this book down and just count calories. If however, you wish to lose body-fat without losing muscle, you need to know how to determine what proportions of proteins, carbs and fats to eat. You need to know the difference between good carbs & bad carbs and good fats & bad fats. You need to know the optimum times to eat proteins, carbs and fats. You need to know the difference between good exercise and bad exercise.

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Good carbs, Bad carbs

As mentioned in Chapter 1, the body's carb requirements vary from person to person and increase with intensity of exercise done. So what are the best carbs to eat? Complex ones from "wholegrain" cereals? No.

Simple vs. Complex

TV ads for breakfast cereals bang on about the wholegrain goodness of complex carbohydrates. The terms "Simple" and "Complex" actually refer purely to the chemical structure of a carbohydrate and have nothing to do with how fast they turn into blood glucose in the body. The Glycaemic Index (GI) (or Glycemic Index if you're American) relates to how fast carbohydrates turn into blood glucose in the body. Click http://www.mendosa.com/gilists.htm or copy & paste into your Internet browser's address bar to learn about GI and see a list of 750 foods and their GI & GL (GL = Glycaemic Load = GI/100 x carb content per serving). If you don't have Internet access, I'm afraid I'm not putting the full list here! Here are a few extracts. Note: a GI of 55 is low; a GL of 10 is low.

Food	GI	Portion (g)	GL
Baguette, white, plain (France)	95±15	30	15
Wholemeal (whole wheat) flour bread (mean of 13 studies)	71±2	30	9
White wheat flour bread (mean of 6 studies)	70±0	30	10
Coarse rye kernel bread, 80% intact kernels and 20% white wheat flour	41	30	5
Bürgen ® Soy-Lin, kibbled soy (8%) & linseed (8%) loaf	36±4	30	3
Cornflakes (mean of 5 studies)	81±3	30	21
Shredded Wheat (mean of 2 studies)	75±8	30	15
Grapenuts (mean of 2 studies)	71±4	30	15
Weetabix (mean of seven studies)	70±2	30	13
Instant Porridge (mean of 2 studies)	66±1	127	9
Porridge made from rolled oats (mean of 8 studies)	58±4	127	7
Alpen Muesli	55±10	30	10
All-Bran (mean of 4 studies)	42±5	30	8
Baked Russet Burbank potato (mean of 4 studies)	85±12	150	26
Instant Mashed potato (mean of 6 studies)	85±3	150	17
Sweet potato (mean of 5 studies)	61±7	150	17
New potato (mean of 3 studies)	57±7	150	12
Boiled potato, boiled in salted water, refrigerated, reheated (India)	23	150	8
Chickpeas (Garbanzo beans), boiled (mean of 4 studies)	28±6	150	8
Chana dal (Gram dhal)	5±3	50 (dry)	1
Instant rice, white, cooked 6 min	87	150	36
Rice, boiled in salted water, refrigerated 16-20h, reheated (India)	53	150	20
Instant rice, white, boiled 1 min	46	150	19
Sucrose (table sugar) (mean of 10 studies)	68±5	12	8
Glucose (mean of 11 studies)	99±3	12	12
Fructose (fruit sugar) (mean of 6 studies)	19±2	12	2

The last three items in the list are all simple carbs. As you can see, many so-called complex carbs turn into blood sugar faster than table sugar. As fructose has such a low GI, does this mean that we can eat as much of it as we like? No.

When we eat fructose, it passes from the small intestine into the portal vein and goes straight to the liver. As liver cells contain an enzyme called fructokinase (which has a high affinity for fructose),

all dietary fructose is absorbed by the liver where it tops-up liver glycogen. Liver glycogen is also topped-up by glucose (obtained from starchy carbs). Once liver glycogen stores are full, any excess fructose is converted to.....**triglycerides**.

Why is GI important? When we eat natural carbohydrates and they slowly raise blood glucose levels, pancreatic beta cells secrete a hormone called insulin, which allows glucose to pass into cells (by moving Glu-T4 transporters inside the cells). As glucose enters cells, glucose levels in the blood slowly fall back to normal. It's a negative feedback loop. Humans evolved on a diet of natural carbohydrates and so the secretion of insulin never had to change blood glucose levels very rapidly.

When unnatural high-GI carbs are eaten, blood glucose levels rise faster than the body is used to. This results in over-secretion of insulin (hyperinsulinaemia). This then shuttles too much glucose into cells and results in.....low blood glucose. Rapidly-falling and low blood glucose levels cause feelings of severe hunger and cravings to eat more carbs. It's a vicious circle. Hyperinsulinaemia has other bad effects on the body. Click http://www.mercola.com/2001/jul/14/insulin.htm or copy & paste into your Internet browser's address bar to learn about Insulin and its Metabolic Effects.

Unfortunately, a weakness of GI is that adding fats and some proteins to high-GI foods lowers the GI but ***can*** increase hyperinsulinaemia. Saturated fats and omega-6 polyunsaturates raise the insulin secretion produced by carbs. Monounsaturates have little effect and omega-3 polyunsaturates lower the insulin secretion produced by carbs.

There ***is*** another index called the Insulin Index (II). Click http://www.mendosa.com/insulin_index.htm or copy & paste into your Internet browser's address bar to learn about II. If you don't have Internet access, here is an extract:-

Food	**Glycaemic Score**	**Insulin Score**
BREAKFAST CEREALS		
All-Bran	40	32
Porridge (Oatmeal)	60	40
Muesli	60	40
Special K	70	66
Honeysmacks	60	67
Sustain	66	71
Cornflakes	76	75
CARBOHYDRATE-RICH FOODS		
White pasta	46	40
Brown pasta	68	40
Grain [rye] bread	60	56
Brown rice	104	62
French fries (chips)	71	74
White rice	110	79
Wholemeal bread	97	96
White bread	100	100
Potatoes	141	121
PROTEIN-RICH FOODS		
Eggs	42	31
Cheese	55	45
Beef	21	51
Lentils	62	58

Fish	28	59
Baked beans	114	120
FRUIT		
Apples	50	59
Oranges	39	60
Bananas	79	81
Grapes	74	82
SNACKS AND CONFECTIONS		
Peanuts	12	20
Popcorn	62	54
Potato crisps	52	61
Ice cream	70	89
Yoghurt	62	115
Mars bar	79	112
Jellybeans	118	160
BAKERY PRODUCTS		
Doughnuts	63	74
Croissants	74	79
Cake	56	82
Crackers	118	87
Cookies	74	92

The above list has a few surprises. Some proteins produce a large insulin response. Whey (milk protein is 20% whey and 80% casein) produces a large insulin response. Another effect of excess insulin secretion is that fat-burning is reduced and fat-storage is increased, not exactly what someone who wants to lose body-fat really wants! Therefore, consumption of foods with an II greater than 60 should be minimised for optimum fat loss. So baked beans ("my carbohydrates are ***exceedingly*** complex") are out. Overcooking foods raises GI (& II) and baked beans are well-overcooked in a sugary sauce. On the other hand, refrigerating foods lowers GI (& II) by changing the structure of the starch, even if the food is subsequently re-heated.

As the terms "simple" and "complex" are meaningless in terms of carbohydrates' effects in the body, I prefer to use the terms "**slow**" and "**fast**". In a nutshell, **slow** carbs are good and **fast** carbs are bad. These terms can be applied to proteins, too. Egg is **slow** and Whey is **fast**. Sticking to mostly **slow** foods keeps blood glucose and insulin levels stable, which results in better appetite control and better health, too.

It was soaring blood insulin levels that were sending me to sleep all those years ago. Hyperinsulinaemia results in amino acids being shuttled into cells, too. However, L-tryptophan isn't shuttled into cells, so the level of this amino acid rises relative to others in the blood. As L-tryptophan competes with other amino acids to cross the blood-brain barrier, now that the competition has been removed, a lot of L-tryptophan enters the brain. Here, it's converted into 5-hydroxytryptophan (5-HTP) and then serotonin. High serotonin levels in the brain cause sleepiness. As serotonin is also a "feel-good" substance, it's no wonder that carbs are addictive - they really ***do*** make you feel good. For more information on hyperinsulinaemia, click http://www.allyourstrength.com/ArticleDetail.aspx?ID=78 or copy & paste into your Internet browser's address bar.

So remember, "**Right carbs, right amounts, right times**" to quote ToxicToffee.

3

Good fats, Bad fats

In a similar way to the previous chapter, there's a lot of bad information in the media about fats. Saturated fats are usually described as "bad" and polyunsaturates are usually described as "good". This is simplistic. ***Everything*** is bad in excess, even polyunsaturates. The thing about fats is that there are four basic types (saturates, monounsaturates, omega-6 polyunsaturates and omega-3 polyunsaturates), and these must be consumed in roughly the right proportions for optimum health. Suffice it to say, the majority of people in the West do not eat them in anywhere near the right proportions. So, what exactly ***are*** fats?

FATS 101

Fats are an ester of glycerol and fatty acids.

1 molecule of glycerol + 3 molecules of fatty acid = 1 molecule of triglyceride (fat) + 3 molecules of water.

It's the fatty acids that determine whether a fat is sat, mono etc. The four different types of fatty acid all have a CH_3 at one end and a COOH at the other. The difference is in the middle section.

Saturated fatty acids have a middle section consisting of all CH_2's. Here's a diagram for Stearic acid (the predominant fatty acid in beef fat):-

```
  H H H H H H H H H H H H H H H H H H O
H-C-C-C-C-C-C-C-C-C-C-C-C-C-C-C-C-C-O-H
  H H H H H H H H H H H H H H H H H H
```

Monounsaturated fatty acids have one C=C bond in the middle, which happens to be 9 from the left-hand end resulting in monounsaturates also being referred to as omega-9's, as omega is at the **end** of the Greek alphabet. Here's a diagram for Oleic acid (the predominant fatty acid in olive oil):-

```
  H H H H H H H H      H H H H H H H O
H-C-C-C-C-C-C-C-C=C-C-C-C-C-C-C-C-C-O-H
  H H H H H H H H H H H H H H H H H H
```

Omega-6 polyunsaturated fatty acids have two or more C=C bonds in the middle with the last one being 6 from the left-hand end. Here's a diagram for Linoleic acid (the predominant fatty acid in sunflower oil):-

```
  H H H H H      H      H H H H H H H O
H-C-C-C-C-C=C-C-C=C-C-C-C-C-C-C-C-C-O-H
  H H H H H H H H H H H H H H H H H H
```

Omega-3 polyunsaturated fatty acids have three or more C=C bonds in the middle with the last one being 3 from the left-hand end. Here's a diagram for Alpha-linolenic acid (the predominant fatty acid in flax-seed oil):-

```
  H H      H      H      H H H H H H H O
H-C-C-C=C-C-C=C-C-C=C-C-C-C-C-C-C-C-C-O-H
  H H H H H H H H H H H H H H H H H H
```

These diagrams are a bit misleading. Where there is a C=C bond, there are two H's on the "underside" only of the molecule. This asymmetry causes the H's to repel each other and bend the molecule into a V-shape at each C=C bond. C=C bonds with H's on the same side are known as "cis" bonds. The above molecule is really cis, cis, cis (c,c,c) Alpha-linolenic acid. The other type of C=C bond is known as "trans" and looks like the following diagram:-

```
  H H H   H H   H H   H H H H H H H O
H-C-C-C=C-C-C=C-C-C=C-C-C-C-C-C-C-C-O-H
  H H   H H   H H   H H H H H H H H
```

This is a diagram of trans, trans, trans (t,t,t) Alpha-linolenic acid. As the H's are on opposite sides of the molecule, they do not repel each other and the molecule is straight as shown above. Note that saturated fatty acid molecules are naturally straight. Therein lies the problem with trans-fatty acids. They're straight like saturated fatty acids but have unsaturated bonds which are prone to oxidation. Click http://www.cyberlipid.org/perox/oxid0002.htm or copy & paste into your Internet browser's address bar. **WARNING!** Heavy-duty organic chemistry!

Our bodies take trans fatty acids and incorporate them into cell membranes as if they were saturated fatty acids. This results in atherogenicity (artery-clogging), damage to the immune system and other health problems. Trans-fatty acids are found in partially-hydrogenated vegetable oils and so any processed foods or cooking/spreading fats which has the word "**hydrogenated**" high-up in the ingredients list should be avoided. These are **bad** fats. There are also naturally-occurring trans-fatty acids made by bacteria in the stomachs of ruminant animals, like Conjugated Linoleic Acid (CLA). This looks a bit like the diagram below:-

```
  H H H H H     H   H H H H H H H H O
H-C-C-C-C-C=C-C=C-C-C-C-C-C-C-C-C-C-O-H
  H H H H H H H   H H H H H H H H H
```

This has one of the C=C bonds shifted to the left and also has one cis bond and one trans bond, so the molecule is always bent. CLA has certain beneficial properties but medical studies show mixed results. It's certainly not artery-clogging, so don't let anyone put you off eating butter from grass-fed cows (e.g. New Zealand butter) by suggesting that it has nasty trans-fats in it. CLA is a **good** trans-fat.

Anyway, back to diet. I've read that saturated fat consumption should be about 10% of total calories. This is because, even though sat fats are not essential (our bodies can manufacture sat fats), this guarantees adequate levels of sex hormones in the body. I've also read that total polyunsaturate consumption should be about 5% of total calories, with a ratio of omega-6 (O6) to omega-3 (O3) of 2:1. As O3's are only found in greater quantities than O6's in flax-seeds (a.k.a. linseeds) and oily fish, and many people eat way too little or no oily fish (and who, other than body-builders and some vegetarians/vegans, eats flax-seeds?), the O6:O3 ratio in the West is about 20:1. This is due to the widespread consumption of meats, eggs & milk from grain-fed animals, grains, nuts and seeds. So, it's not surprising that there are high rates of heart disease and other inflammatory diseases in the West, as O6's end up in series 1 & 2 prostaglandins and series 2 prostaglandins are pro-inflammatory. O3 fats end up in series 3 prostaglandins, which are anti-inflammatory.

So eat up yer oily fish if you're not vegetarian or vegan. Otherwise, eat up yer ground-up flax-seeds!

Monounsaturates can make up 15% to 35% of total calories, depending on activity levels. Remember from the histogram in Chapter 1, **sedentary** people on average burn twice as much energy from fats as from carbs. So, if energy from protein is 25% say, 25% of energy can come

from carbs and 50% can come from fats i.e. a 2:1 ratio of fats:carbs. The cyclists at the left-hand end of the histogram in Chapter 1 would do best on 25% protein, 5% carbs, 70% fat when sedentary, whereas the cyclist at the right-hand end of the histogram would do best on 25% protein, 60% carbs, 15% fat when sedentary. When active, more carbs are needed by everyone.

Which fats contain which fatty acids? Click http://www.manitobaharvest.com/nutrition/index.asp?itemID=183 or copy & paste into your Internet browser's address bar to see a Comparison of Dietary Fats chart. Click http://www.apag.org/oleo/fatsoils.pdf or copy & paste into your Internet browser's address bar to see tables of Properties and Composition of Vegetable and Special Oils, and Properties and Composition of Animal and Marine Fats and Oils.

For high-temperature cooking, saturates are the least likely to oxidise (when they're on fire, they're oxidising!), followed by monos, then omega-6's and then omega-3's. An oil doesn't have to be smoking to be oxidising. Alpha-linolenic acid oxidises at room temperature, which is why linseed oil is used to varnish cricket bats and soften putty. The best non-animal fat for high-temperature cooking is therefore Coconut Oil, followed by Palm Oil and then Olive Oil. Extra-virgin Olive Oil has a lower smoking point than refined Olive Oil (due to higher levels of free fatty acids), but has higher levels of polyphenol antioxidants, which is good.

Good cholesterol, Bad cholesterol & Coronary Heart Disease

Cholesterol & coronary heart disease get mentioned in the media quite a lot. Unfortunately, most of what you see & hear is either wrong or oversimplified to the point that it misses out what's actually happening. Fat and/or cholesterol doesn't stick to the insides of artery walls like grease on the inside of a drainpipe. This article sets about explaining what cholesterol is and what really happens to artery walls, and also tells you how to minimise your risk of getting a heart attack or stroke.

What is cholesterol?

Cholesterol is a large, waxy molecule ($C_{27}H_{45}OH$) consisting of a hydrocarbon (fat-soluble) tail, a middle section consisting of four carbon rings (the steroid bit) and an alcohol (water-soluble) group on the end. Cholesterol is a powerful anti-oxidant and is what bile acids, mineralcorticoids, glucocorticoids, and sex hormones are made from.

Cholesterol is "chauffeured" around the body in lipoprotein "limousines". Lipoproteins are lipo (fat-soluble) at one end and protein (water-soluble) at the other end and form a spherical shell around their contents with the lipo end pointing inwards and the protein end pointing outwards. The shell is akin to the body of the limo. Inside the limo, there are apo(lipo)proteins which are akin to the driver, as they determine where the particles end up. The passengers are cholesterol, cholesteryl esters, phospholipids and triglycerides. These limos come in different variants, like chylomicrons, VLDL, LDL, IDL and HDL, the difference being the type and amount of apoprotein and the relative proportions of cholesterol and other ingredients, and there are sub-groups of each type also. Complicated, innit? To learn about chylomicrons etc, click http://www.umanitoba.ca/faculties/medicine/units/biochem/coursenotes/blanchaer_tutorials/LipTutWeb/pages/choices.htm or copy & paste into your Internet browser's address bar.

The different variants are affected by how much triglycerides there are circulating in the blood. High serum triglycerides (caused by a chronic over-consumption of sugary & starchy carbohydrates for the activity level) result in cholesterol-depleted, triglyceride-rich particles and low serum triglycerides result in cholesterol-rich, triglyceride-depleted particles. Therein lies the problem. As cholesterol is a powerful anti-oxidant, small, dense (Type B) cholesterol-depleted particles are more prone to oxidation than large, buoyant (Type A) cholesterol-rich ones.

LDL that becomes oxidised is now **bad** LDL and is swallowed-up by scavenger macrophages. These then swell-up into foam cells which embed themselves into the intima of artery walls. Other processes also occur which cause cholesterol and calcium to build-up as a plaque in the media of artery walls. To see a cross-section through a typical artery wall, click on http://www.health%2dheart.org/acceuil.htm or copy & paste into your Internet browser's address bar and scroll down to the bottom of the page.

This plaque build-up pushes the inner artery wall inwards making the artery narrower in cross-section, impeding the flow of blood through it. This can cause angina pectoris (pain in the chest) as the heart muscle is starved of oxygen. Eventually, especially if there are low levels of anti-inflammatories in the blood, the plaque can rupture, causing chunks of plaque to circulate and block-up narrow coronary arteries causing a heart attack, or narrow cerebral arteries causing a stroke. Unoxidised LDL is not bad and is not swallowed-up by scavenger macrophages.

It's possible to reduce serum triglycerides quite drastically by eating long-chain omega-3 fats from oily fish. These inhibit the conversion of glucose into triglycerides, but beware. Inhibiting the

conversion of glucose into triglycerides can result in increased blood glucose levels (which is not good – see below) if sugary/starchy carb intake is too high. Solution? Reduce your sugary/starchy carb intake to suit your activity levels.

Why do foam cells embed themselves into the intima of artery walls?

Arteries are muscular tubes. They open & constrict to control the flow of blood through them. When you get cold, they constrict to reduce the flow of blood to the skin to prevent excessive heat loss. When you get hot, they open to increase the flow of blood to the skin to increase heat loss.

Foam cells don't go just anywhere. They embed themselves into damaged areas of artery walls. This is a good thing otherwise damaged artery walls could rupture, causing a haemorrhage.

What damages artery walls?

1) Chronically-high blood pressure.
2) Chronically-high blood glucose.
3) Chronically-high blood free radicals.
4) Chronically-high blood homocysteine.
5) Chronically-low blood anti-oxidants.
6) Chronically-high blood pro-oxidants.
7) Chronically-low blood anti-inflammatories.

How can I reduce damage to artery walls?

1) Have blood pressure (BP) tested regularly. There's one problem with having your BP taken in a GP's surgery and that is "white-coat hypertension" where the stress of having your arm squeezed by the cuff sends your BP up! If you buy your own BP monitor (Lloyds pharmacy sell a fully automatic BP monitor with standard cuff for £9.99), you can become accustomed to using it and overcome white-coat hypertension.

2) Have blood glucose (BG) tested regularly. If you're lucky, you may be able to request a HbA1c test. This shows accumulated damage to red blood cells by blood glucose.

3) Don't smoke! Apart from lung cancer, chronic obstructive pulmonary disease & emphysema, smoking is **the** No.1 best way to damage your arteries and get them clogged-up.

4) Take a good B-complex tablet each day containing B6, B12 & Folic acid, which lowers homocysteine levels.

5) Ensure that your diet is high in natural anti-oxidants from coloured veggies (beta-carotene), fruits (Vitamin C + bioflavonoids), tomatoes (lycopene), sesame seeds (gamma-tocopherol), Brazil nuts (selenium), alcohol and/or red wine in moderation, green tea in moderation, extra-bitter chocolate in moderation, onions/garlic etc.

6) In men and non-menstruating women, excess iron in the blood is pro-oxidant, so don't supplement with iron. Menstruating women have the opposite problem, so they should supplement with iron.

7) Take about 2g/day of long-chain omega-3 fats, or about 20g/day of flaxseed oil if male, or about 10g/day of flaxseed oil if female.

What about Benecol & Flora Pro-Activ?

These yoghurts & spreads contain plant sterols or stanols, which reduce total serum cholesterol by up to 15%. However, as explained above, it's LDL **quality** that counts, not **quantity**, and there is no evidence to show that these foods actually save any lives.

What about statins?

Statins (HydroxyMethylGlutarate Coenzyme-A Reductase inhibitors) reduce total serum cholesterol. They also have anti-inflammatory and anti-clotting effects by reducing levels of the non-sterol derivative mevalonate and its subsequent products. Click http://www.cholesterol%2dand%2dhealth.com/Synthesis%2dOf%2dCholesterol.html or copy & paste into your Internet browser's address bar to see the cholesterol synthesis pathway. Statins **do** save lives in people who have already had a heart attack and also in men between the ages of 30 and 60. However, younger or older men and all women do not get reduced number of deaths, though heart-attack deaths may be slightly lower. I would strongly recommend anyone who is taking statins to supplement with 100mg/day of Coenzyme-Q10, as the synthesis of this vital substance is suppressed by them. Note that omega-3 fats also have anti-inflammatory and anti-clotting effects as well as anti-arrhythmia effects and don't suppress the production of Co-Q10. Click http://www.allyourstrength.com/ArticleDetail.aspx?ID=173 or copy & paste into your Internet browser's address bar.

What about eating cholesterol?

As cholesterol is eaten, the liver secretes less to compensate. As an average egg contains about 250mg of cholesterol, you can eat two eggs per day (500mg of cholesterol per day) without significantly affecting cholesterol levels in the blood. Click http://www.ajcn.org/cgi/reprint/32/5/1051.pdf or copy & paste into your Internet browser's address bar. So why do current "Healthy Eating" guidelines recommend no more than 3 eggs per week? Because they're…….?

Losing Body-fat healthily

Having established what's good and what's bad, here are some guidelines for losing body-fat healthily. Please note that these guidelines have not been evaluated by any health agency and don't exactly conform with current "Healthy Eating" guidelines either, but then you know what I think of current "Healthy Eating" guidelines!

1) Eat 6 meals, or 3 meals and 3 "snacks" per day to keep your metabolic rate high. Skipping breakfast is **not** an option.

2) All meals and "snacks" should contain proteins, fats and fibrous carbs i.e. all meals and snacks should be "balanced".

3) **Slow** sugary/starchy carbs can be eaten at breakfast and in meals/snacks before and after workouts/exercise/intense activity. If/when you are sedentary, keep slow carbs to a minimum. Keep **fast** sugary/starchy carbs to a minimum all the time (except for the odd treat) as they cause wild swings in blood glucose and insulin levels. **The more intense exercise you do, the more slow sugary/starchy carbs you can eat.**

4) Drink >1.5 litres of no added sugar watery drinks per day. If your urine is too dark, you need to drink more.

5) Balance your omega-6 and omega-3 essential fatty acids (EFAs) by eating omega-3 rich oily fish (sild, sardines, pilchards, mackerel, herring, salmon, trout, fresh tuna etc) or powdered linseeds (a.k.a. flaxseeds) or supplement with enough fish oil capsules to get ~2g of long-chain omega-3s per day. As conversion of alpha-linolenic acid into long-chain omega-3s is only about 36% in women and about 16% in men, ~10g/day for women or ~20g/day for men of flaxseed oil, or ~20g/day for women or ~40g/day for men of powdered linseeds is required.

6) Eat 2 to 3 portions of low-sugar fruits per day - preferably before meals/snacks. Minimise consumption of high-sugar fruits.

Proteins are:- Meats, poultry, fish, eggs, cheese, Quorn, seeds, nuts, legumes (peas, beans & lentils).

Fibrous carbs are:- All veg that grows above ground level, bulbs (onions, leeks, garlic), non-sweet fruits (tomatoes, peppers, olives, aubergines, avocados, cucumbers/courgettes etc).

Slow sugary/starchy carbs are:- All-bran, no added sugar muesli, non-instant oats, sweet potatoes, lightly-boiled new potatoes cooled and refrigerated, brown Basmati rice, non-overcooked legumes & root veggies, dark, wholegrain pumpernickel-style rye breads and Bürgen/Vogel's soya & linseed bread.

Fast sugary/starchy carbs are:- White wheat breads, wholemeal wheat breads, most breakfast cereals including Shredded wheat, Weetabix, Cheerios, Grapenuts etc, cereal bars, sugar, sweets, chocolate, cakes, biscuits, jacket potatoes, overcooked root veggies, sweetcorn and overcooked legumes like baked beans.

Low sugar fruits are:- Berries, stone fruits, citrus, tart apples, pears, dried fruit salad (apple rings, apricots, pear halves, peach halves and prunes without added sugar).

High sugar fruits are:- Ripe bananas, grapes, pineapples, sweet apples, sultanas, raisins, currants and dried fruits with added sugar.

Fats are sats, monos and the EFAs mentioned above. Butter is high in sats and monos. Olive oil & spreads are high in monos. Sunflower oil & spreads are very high in omega-6 EFAs - avoid. Don't fry or roast with high-EFA oils as they oxidise. Meats contain sats, monos and some EFAs. Minimise consumption of foods with the word "**hydrogenated**" in the ingredients list.

Note: Some foods fall in-between categories like unripe bananas which are medium speed sugary/starchy carbs.

Depending on your insulin sensitivity, speed of metabolism and exercise levels, you may get away with eating some fast sugary/starchy carbs - you'll just have to suck it and see.

6

Too much carbs, Not enough carbs

As stated in previous chapters, eating too much carbs raises triglycerides and makes you fat. What happens if you eat too few carbs? Interestingly, the proportions of fat & carbs that the body uses for fuel can be shifted. Referring to the histogram in Chapter 1, as glycogen stores are filled, fat-burning decreases and carb-burning increases. The opposite happens as glycogen stores are depleted.

It seems obvious then that to maximise fat-burning, glycogen stores should be depleted as much as possible. This is how low-carb, high-protein & fat diets work. Is there a danger if glycogen stores become fully-depleted? It all depends.....

As blood glucose levels fall, glucagon (which is secreted by pancreatic alpha cells when blood glucose levels drop below normal) converts liver glycogen into blood glucose. When liver glycogen stores are fully-depleted, ketones (by-products of the increased fat-burning) in the blood rise. As glucose contains 4kcals/g and D-3-hydroxybutyrate contains 5kcals/g, tissues like the brain, nerves, kidneys and muscles (but not the liver) can switch over to ketones as a fuel which can replace 60% of the energy that glucose was providing. This is an adaptation to starvation which reduces muscle wastage. The presence of ketones in the blood is known as ketosis. See the next chapter for an explanation of why ketosis won't hurt you - unless you starve yourself.

Lack of liver glycogen means that blood glucose has to be produced from amino acids like glutamine etc. This process is called gluconeogenesis and is also controlled by the hormone glucagon. Gluconeogenesis is a slow process. If intense exercise is done (which shifts the balance of fuel usage towards carbs), glucose can be used-up faster than it can be produced. This results in **hypoglycaemia**, which makes the sufferer feel light-headed, dizzy, headachy, hungry etc. At least this results in symptoms that make the sufferer eat some carbs. Eating too much carbs produces no physical symptoms at all.

Hypoglycaemia is not good, as the body produces the stress hormone cortisol in a panic attempt to raise blood glucose levels. Cortisol catabolises (breaks down) muscle into amino acids and also slowly eats away at bones and skin.

This means that the more intense exercise you do, the more sugary/starchy carbs you need, which is what I said in the last chapter on body-fat loss guidelines.

Ketogenic diets and Ketoacidosis

Everybody knows that ketogenic diets like Atkins' destroys your kidneys and rots your bones, right? WRONG! Everybody knows that diets like Atkins' cause ketoacidosis which is a very dangerous condition requiring urgent hospital treatment, right? WRONG!

There's a lot of nonsense spoken about ketogenic diets by people who really should know better. I suspect that they have been taught wrongly at uni or med school. As Diana Schwarzbein MD said in the intro' to her first book (click http://www.schwarzbeinprinciple.com/pgs/dr_schw/sp_I_intro.html or copy & paste into your Internet browser's address bar).

"In medical training, I was taught that a low-fat diet high in complex carbohydrates prevented weight gain and disease. I believed what my professors said. Early on, I advocated low-fat diets. But this soon changed."

Ketosis is not ketoacidosis.

Consider the following four cases:- Note: Figures are from "Introduction to Nutrition and Metabolism" By David A Bender (Senior Lecturer in Biochemistry, UCL)

1) Healthy human, fed state: Glycogen stores are **replete**. Serum glucose = ~5.5mmol/L. Serum fatty acids = ~0.3mmol/L. Serum ketones = 0mmol/L. No gluconeogenesis is taking place. Amino acid pool is **replete**. Cortisol level = **normal**. **No loss of muscle mass**.

2) Healthy human, starvation for 7 days: Glycogen stores are **depleted**. Serum glucose falls to ~3.5mmol/L. Serum fatty acids rise to ~1.2mmol/L. Serum ketones (mainly D-3-hydroxybutyrate) rise to ~4.5mmol/L (not high enough to cause acidosis). Gluconeogenesis is occurring. Amino acid pool is **depleted**. Cortisol level = **high**. M**uscle mass slowly decreasing**. THIS IS BAD KETOSIS.

3) Healthy human, low-carb diets: Glycogen stores are **depleted**. Serum glucose falls to ~5mmol/L. Serum fatty acids rise to ~1.2mmol/L. Serum ketones (mainly D-3-hydroxybutyrate) rise to ~4.5mmol/L (not high enough to cause acidosis). Gluconeogenesis is occurring. Amino acid pool is **replete** (due to high protein intake). Cortisol level = **normal**. **No loss of muscle mass**. THIS IS GOOD KETOSIS.

4) Human with untreated type 1 diabetes: Glycogen stores are **depleted**. Due to lack of insulin, the Glu-T4 transporters in cells cannot move to the surface so glucose cannot enter cells. Serum glucose = >20mmol/L. This causes major damage to kidneys, arteries, eyes, nerves etc by cross-linking with proteins (glycosylation) resulting in major disability and eventual death. As the body is FORCED to run off fatty acids & ketones, metabolic processes are out of control and ketones rise to MUCH higher levels than in 2) or 3) (I don't know how high exactly) resulting in acidosis and eventual death. THIS IS KETOACIDOSIS. I don't know about cortisol levels but they are pretty irrelevant as death by other means occurs.

Conclusions: Low-carb diets are ***similar*** to starvation in that serum glucose levels are lower than normal and glycogen stores are depleted. The body is ENCOURAGED to burn less glucose and more fatty acids but the metabolic processes are all **under control**. The big difference between low-carb dieting and starvation is in the amino acid pool and cortisol levels.

Useful advice for vegetarians & vegans

Firstly, by vegetarian, I mean someone who does not eat the flesh of animals. This includes fish and chicken.

A vegan MD, Dr. Michael Greger (click http://www.drgreger.org/talks/#nutrition or copy & paste into your Internet browser's address bar) has a very informative and witty lecture on "Optimum Vegetarian Nutrition: Surprising New Research on Omega 3's and B12".

In a nutshell, vegetarians & vegans have the ***same*** mortality rates as omnivores – as shown in a study of 28,000 subjects. Vegetarians & vegans have the ***same*** rate of heart disease as omnivores but ***double*** the rate of degenerative brain diseases like Alzheimer's. WHY?

There are two reasons.

1) Vegetarians and vegans don't eat oily fish and most don't eat cracked or ground-up linseeds either. This means that the ratio of O6 to O3 fats in their diet is way too high. This ***increases*** the risk of diabetes, certain cancers and heart disease.

2) Only animal produce naturally contains Vitamin B12. Lack of B12 in the diet raises the level of homocysteine in the blood, which attacks artery walls. This ***raises*** the risk of heart disease and Alzheimer's.

What to do?

1) Eat 3 (for women) or 6 (for men) heaped tablespoonfuls of powdered brown or golden linseeds per day, or add 11ml (for women) or 22ml (for men) of flax-seed oil to food per day, or take 10 (for women) or 20 (for men) 1000mg flax-seed oil capsules per day.

2) Supplement with vegan B12.

'Nuff said.

Gluten - a pain in the .....?

Remember the advert "I'm feeling a bit bloated". "Here, have some Bifidus Digestivum."? I wonder what percentage of the population suffers from bloating, tummy pains, constipation, IBS, or a failure to properly absorb the nutrients from their food?

People with Coeliac Disease (CD) or Dermatitis Herpetiformis (DH) (intensely itchy spots on pressure points) have to avoid gluten as much as possible, as it produces an allergic reaction, with antibodies that attack the body (auto-immune disease).

However, gluten is implicated in other conditions due to molecular mimicry. Sjogren's syndrome (dry eyes & other bits) and cerebellar ataxia (brain damage) are mentioned in the huge article "Cereal Grains: Humanity's Double-Edged Sword" by Loren Cordain (click http://www.thepaleodiet.com/articles/Cereal%20article.pdf or copy & paste into your Internet browser's address bar).

Anecdotal evidence suggests that there ***are*** conditions other than CD or DH which can benefit from switching from gluten-containing grains (wheat, rye, oats, barley, spelt) to non-gluten-containing ones (rice, corn, quinoa, buckwheat, millet). Luckily, supermarkets like Waitrose and Sainsbury's have a large "Free from" section nowadays, which makes finding gluten-free substitutes for breads, cakes, biscuits, breakfast cereals etc a lot easier.

10

Good exercise, Bad exercise

As stated at the beginning of this epic work, it's all about body-fat loss, not weight loss. Exercise improves fitness, I'm not arguing about that. However, as a way of losing body-fat, it's not very good. You have to burn 3,500kcals to lose just one measly pound of body-fat. That's a helluva lot of exercise, and as stated before, too much exercise or exercise done at the wrong time can result in muscle loss.

In fact, just plodding away on a treadmill, apart from being incredibly boring, is not actually the most efficient way to burn body-fat. Cardio "bunnies" read on.

The smart way to burn body-fat is to combine Endurance Training with Weight (Resistance) Training. Click http://www.exrx.net/FatLoss/WT&End.html or copy & paste into your Internet browser's address bar.

"At the conclusion of the study, the "endurance only" group lost a total of 3.5 lbs of which 3 lbs was fat ***and a half pound was muscle loss***. On the other hand, the "endurance and weight resistive" group lost 8 lbs with an actual fat loss of 10 lbs ***and an increase of 2 lbs. of lean body weight***." That's the way to do it!

The other smart way to burn body-fat is to do High-Intensity Interval Training (HIIT). Click http://www.exrx.net/FatLoss/HIITvsET.html or copy & paste into your Internet browser's address bar.

"The HIIT group lost over 3 times as much subcutaneous fat as the ET group despite of only expending less than half as many calories."

Also, click http://www.musclemedia.com/training/hiit.asp or copy & paste into your Internet browser's address bar. You need to be pretty fit to do HIIT - it's not for couch potatoes.

Finally, about "toning". Toning means to build muscle and burn body-fat to reveal the muscle. It's not actually possible to control where your body burns fat from, so it's not possible to "tone" a specific body-part, though it ***is*** possible to build muscle in specific places by intense exercise with weights. Anyone who promises you a toned tum & bum is after your money. 1000 sit-ups will just give you a sore tum, not a toned one, so don't do it!

Good combinations, Bad combinations

Some people believe in food combining i.e. don't eat protein with carbohydrate as protein needs acid conditions to digest and carbohydrate needs alkaline conditions to digest. This theory assumes that the human digestion system is like a barrel where all foods are digested at the same time. This isn't the case. The following is cribbed (with minor edits) from a message board so I hope I'm not breaking copyright by printing this. Click http://forum.bodybuilding.com/showpost.php?p=1447655 or copy & paste into your Internet browser's address bar.

DIGESTION 101

The order you eat foods in does not make a difference to digestion. Once foods hit your stomach, the peristaltic motion (that is - the muscles in your stomach wall contracting) mix it all together regardless. Also, the different enzymes that are released are released regardless of the order that you eat your food.

In your stomach:-

The presence of food in your stomach stimulates:-
1. Gastrin - this is what is responsible for the eventual release of HCl - or stomach acid.
2. Pepsinogen - this is converted to pepsin by the acid in your stomach. Pepsin is important in the digestion of proteins.

In the small intestine:-

Once food hits the small intestine the pancreas and gall bladder are stimulated:-
1. Pancreas - It secretes many enzymes which help digest proteins, starches and triglycerides (fats).
2. Liver/gall bladder - This is responsible for making and secreting bile. This is important in fat digestion. It is stimulated more when you eat fatty foods.

The small intestine itself is also important, but it actually does not secrete anything. It acts to further digest the carbohydrates, proteins and fats, due to enzymes that are bound to the wall of the intestines, and then acts to absorb these things.

So - digestion occurs in two parts - the LUMINAL phase - which involves all of the enzymes that are secreted by the stomach, pancreas and liver. And the MEMBRANOUS phase which is that which occurs because of the enzymes attached to the intestinal wall. It does not matter when you eat carbohydrates or proteins or fats during a meal, because the simple stimulus of food in your digestive tract will cause the secretion of the luminal enzymes (although as you increase your fat, you will stimulate more fat enzymes to be released).

Carbohydrates:-

Starches are the only type of carbohydrates to undergo luminal phase of digestion. This results from enzymes (called amylases) that are released from the pancreas. These act to break down the long starches into shorter polysaccharides (intermediate chains called dextrins). These are then cleaved again to form Disaccharides or trisaccharides (such as maltose or maltotriose).

Sugars and the trisaccharides and disaccharides from the starches are then further digested in the Membranous phase. This involves enzymes (such as lactase - for the breakdown of lactose, sucrase for the digestion of sucrose and maltase for the breakdown of maltose) that are bound to the

intestinal wall. So - these enzymes act on lactose, sucrose and the di and trisaccharides from the breakdown of starch to form glucose, galactose and fructose. These are them absorbed across the intestinal wall and enter the blood to go to the liver.

The liver then takes up most of the glucose/galactose and all of the fructose and converts it into glycogen or fats while the rest stays in the blood for the rest of the body.

Proteins:-

These are broken down in a similar fashion as carbs. But - the enzymes involved in protein breakdown are secreted by the stomach (pepsin and chymosin) and the pancreas. There are lots of different enzymes involved in protein breakdown (because of the large variety of amino acids). So - digestion of proteins begins in the stomach with the secretion of HCl and pepsin which begin to cleave the long protein molecules. This then continues in the small intestines with the secretion of pancreatic enzymes.

These smaller chains of amino acids (called peptides) are then either broken down by MEMBRANOUS phase enzymes on the intestine cells to form amino acids or are absorbed as dipeptides or tripeptides and then convert to simple amino acids by the cells. The amino acids are then released into the blood and are taken to the liver.

In the liver, some of the amino acids go straight into circulation for the muscles, some are used directly for protein synthesis, but the rest are processed to enter the pathway of energy metabolism, carbohydrate formation or fatty acid formation.

Fats:-

This is a little different. Fat is harder to digest because it does not absorb in the fluids in your gut. The digestion of fat is divided into four stages:-
1. Emulsification - This begins in the stomach and involves the warming and mixing of the fats. This breaks the fats into globules. The bile acids from the liver are then secreted into the intestines and make the fat droplets even smaller.
2. Hydrolysis - Enzymes from the pancreas (lipases) then act on the fats to form smaller molecules.
3. Micelle formation - These smaller molecules (free fatty acids, cholesterol, single chain fats etc) combine with bile to form tiny, droplets called micelles.
4. Absorption - The micelles then attach to the intestinal wall and all the components (except the bile) are then absorbed. These are then packaged (into things called chylomicrons) and secreted by the intestinal cells into tiny tubes in your intestinal wall called lacteals which take the fats straight to your heart, which then enters your back to your heart, which then pumps it to the body. These are then taken up by the liver or the fat cells.

These processes in the intestine take a while to complete (depending on what you eat) and so eating one thing 5 minutes after the other will have no effect.

Having said all that, there are certain combinations of food that are less desirable than others, but not for reasons of malabsorption.

Don't eat high-GI carbs together with whey, saturated fat or omega-6 fats. High-GI carbs produce a large glucose and insulin response. Whey, saturated fats and omega-6 fats magnify the insulin response. This reduces fat-burning and increases fat-storage. Guess what? Virtually all junk foods are a combination of high-GI carbs and sat & omega-6 fats. The only time that an insulin spike is desirable is if you have just finished a workout and are bulking (building muscle). That particular

insulin spike would be obtained by swallowing whey with glucose and/or maltodextrin but not accompanied by fat.

The only other dodgy food combination is fruit and protein. Fruit passes through the GI tract rapidly (possibly due to the fibre and simple sugar content stimulating peristalsis) and if eaten with or just after slow-digesting foods like meat or eggs, results in the protein passing through the small intestine too rapidly resulting in incomplete digestion and subsequent fermentation in the colon, producing smelly flatulence!

Eggs

Eggs are a very nutritious food and should be eaten freely as part of a healthy diet.

How much protein is there in an egg/white/yolk?

Click http://www.nutritiondata.com/facts%2dC00001%2d01c201n.html and set serving size to 1 large (50g) to see what nutrients there are in a raw whole egg.
Click http://www.nutritiondata.com/facts%2dC00001%2d01c201o.html and set serving size to 1 large (33g) to see what nutrients there are in a raw egg white.
Click http://www.nutritiondata.com/facts%2dC00001%2d01c201p.html and set serving size to 1 large (17g) to see what nutrients there are in a raw egg yolk.

The amino acid scores for whole egg , white and yolk are 136, 145 and 146 respectively. Eggs are a good source of complete proteins. This is because the yolk and white need to contain everything necessary for a growing chick embryo.

Can I drink eggs raw to save time?

There are three potential problems with this:

1) Salmonella poisoning. Unless you're pretty sure of the hens that the eggs came from, there is a risk of poisoning from raw eggs. This doesn't apply to pasteurised eggs from suppliers like http://www.eggnation.co.uk/ for example.
2) Poor absorption of egg white protein. According to http://jn.nutrition.org/cgi/content/full/128/10/1716 , only 51% of raw egg white protein is absorbed during digestion compared to 91% for cooked egg white protein. According to http://ajpgi.physiology.org/cgi/content/full/277/5/G935 , the figures are 65% and 94% respectively. The second study used 200g of white and one yolk. I don't believe that there is a problem with the absorption of raw egg yolk, though problem 1 still remains. Pasteurised egg white protein is well-absorbed.
3) Poor biotin absorption. Raw egg white contains a glycoprotein called avidin which binds to biotin (Vit. B7) in the yolk and prevents its absorption. Cooking or pasteurisation denatures (changes the 3-D structure of) the avidin and renders it harmless.

What about all the cholesterol in egg yolks?

Current "Healthy Eating" guidelines state that we should eat no more than 3 egg yolks/whole eggs per week. This is based on the erroneous assumption that dietary cholesterol always increases serum cholesterol and that this is always a bad thing. According to http://www.ajcn.org/cgi/reprint/32/5/1051.pdf , adding or not adding 500mg of dietary cholesterol from two large eggs per day made no significant difference to serum cholesterol or triglycerides in 116 healthy male subjects. Some went up and some went down.

Eddie Vos at http://www.health%2dheart.org/cholesterol.htm reckons that you'd have to eat 20 whole eggs per day to get as much dietary cholesterol as the liver produces each day (5g).

Egg yolks do contain some fat and this should be factored into your total diet.

There **is** a problem with modern eggs though, and it's caused by the food that's fed to the hens. Grains contain about 50 times more Linoleic acid (omega-6) than Alpha-Linolenic acid (omega-3) and this raises the omega-6:omega-3 ratio of the eggs that the hens lay. Hens eating a natural diet of bugs, grubs and vegetation lay eggs with a 1:1 ratio of omega-6:omega-3, but grain-fed hens lay eggs with an omega-6:omega-3 ratio of >10:1. A high omega-6:omega-3 ratio in the diet is associated with increased risk factors for heart disease, cancer and insulin resistance (pre-type 2 diabetes). Therefore, if large numbers of cheap eggs are eaten, it's advisable to supplement with omega-3 fats.

Any good egg recipes?

Try
http://www.bbc.co.uk/apps/ifl/food/recipes/queryengine?templatestyle=refine_by_1&config=db&scope=recipes&page=1&pagesize=15&SuppressCaching=0&attrib_26=keywords&oper_26=eq&val_26_1=Eggs

Coconut Oil

As mentioned previously, Coconut Oil is ***the*** most stable oil for use at high temperatures.

What's in coconut oil?

According to http://www.manitobaharvest.com/nutrition/index.asp?itemID=183 , coconut oil is ~91% saturated fatty acids, ~7% monounsaturated fatty acids, ~2% omega-6 fatty acids and zero omega-3 fatty acids. According to McCance and Widdowson's "The Composition of Foods", the fatty acid composition of coconut oil is as follows:-

Name (:0 = sat, :1 = mono, :2 = poly, n6 = omega-6)	Quantity (%)
Caprylic Acid (C8:0)	7.5
Capric Acid (C10:0)	7.1
Lauric Acid (C12:0)	47.7
Myristic Acid (C14:0)	15.8
Palmitic Acid (C16:0)	9.0
Stearic Acid (C18:0)	2.4
Arachidic Acid (C20:0)	1.0
Palmitoleic Acid (C16:1)	0.4
Oleic Acid (C18:1)	6.6
Linoleic Acid (C18:2 n6)	1.8

Won't all that saturated fat give me coronary heart disease?

Whether or not you get coronary heart disease depends on your **whole diet.** According to http://www.ajcn.org/cgi/reprint/34/8/1552.pdf , Pukapukans were getting 26% (male) to 30% (female) of total calories from saturated fats. Tokelauans were getting 47% (male) to 49% (female) of total calories from saturated fats. Tokelauans had total serum cholesterol 35-40mg/dL (0.9-1.03mmol/L) higher than Pukapukans.

As Tokelauans were getting about seven times more energy from saturated fats than the 7% that current healthy eating guidelines recommend, they must have been dropping like flies from coronary heart disease or strokes, right? Wrong. To quote "Vascular disease is uncommon is both populations and there is no evidence of the high saturated fat intake having a harmful effect in these populations." How come? Well, if you look at the rest of the Tokelauans' diet, you'll see virtually no refined sugar or cereal products. They weren't eating junk.

When Tokelauans migrated to New Zealand, their sat fat intake **fell** to ~41% of total calories but as they were eating more refined carbs & sugar, their lipid profile got **worse.**

What are the benefits of coconut oil?

Medium-chain fatty acids are metabolised rapidly without passing through the liver and provide a quick source of energy for muscles. There is evidence that medium-chain fatty acids stimulate the thyroid gland to secrete more T4 & T3 which can be an aid when cutting. There is also evidence that Lauric Acid has anti-bacterial & anti-viral properties. Coconut oil is also good for the skin when rubbed in.

Where can I buy coconut oil?

Don't buy cheap coconut oil. It's almost certainly Refined, Bleached & Deodorised which detracts from its health benefits. The best coconut oils are Organic Virgin Oils. Two good on-line sources are http://www.fresh%2dcoconut.com/ and http://www.revital.co.uk/product_search.cfm?searchString="Nutiva+Organic+Extra+Virgin+Coconut+Oil"

Linseeds/Flax-seeds & Flax-seed Oil

"Where flax is eaten...health abounds!" Mahatma Ghandi. These little seeds pack a quadruple-whammy of protein, omega-3 EFA, soluble fibre and minerals & vitamins.

What's in flaxseeds & flaxseed oil?

Click http://www.nutritiondata.com/facts%2dC00001%2d01c20p1.html and set serving size to 100g to see what nutrients there are in flaxseeds.
Click http://www.nutritiondata.com/facts%2dC00001%2d01c21xq.html and set serving size to 100g to see what nutrients there are in flaxseed oil.

How do I eat flaxseeds & flaxseed oil?

Flaxseeds have a fibrous seed coat which swells-up when wet and passes through our guts undigested. To get the benefit of the protein, omega-3 essential fatty acid and minerals in flaxseeds, the seeds need to be crushed, cracked, chopped-up, sliced-up or ground-up using a coffee grinder, adjustable pepper grinder or most simply, a blender. The resulting powder can be mixed with liquids or sprinkled on foods, though extra fluid must be drunk as the soluble fibre absorbs lots of water. Although whole flaxseeds will keep at room temperature, once ground, it's advisable to keep them refrigerated to minimise oxidation of the exposed fat. Flaxseed oil must be kept refrigerated at all times after opening and must ***never*** be used for cooking. It's O.K. sprinkled over hot food as long as the food is eaten shortly afterwards. Oxidised flaxseed oil tastes bitter and has lost any health benefits it had when fresh, so it should be discarded or used to varnish something or thin down putty. Unoxidised flaxseed oil has a nutty kind of taste or it may taste a bit like tea. Ground-up flaxseeds hardly taste of anything.

How much flaxseeds & flaxseed oil do I need to eat each day?

As men are much poorer converters of alpha-linolenic acid (the omega-3 fatty acid in linseeds) into the longer-chain omega-3 fatty acids than women (see http://www.nutritionsociety.org.uk/bjn/088/bjn0880355.htm and http://www.nutritionsociety.org.uk/bjn/088/bjn0880411.htm), men need to eat approximately 50g of ground flaxseeds per day and women need to eat approximately 25g per day. The daily amount of flaxseed oil for men is approximately 20g and the daily amount for women is approximately 10g.

Where can I buy flaxseeds & flaxseed oil?

Flaxseeds and linseeds are the same thing and they come in different colours. The cheapest linseeds are brown ones which are often found sold as bird seed in pet shops, but can also be found in Holland & Barrett. There are also golden linseeds, which is the type most often found in supermarkets. Linusit & Granovita are two well-known brands. Granovita organic flaxseed oil is a good brand and it comes in dark glass bottles to keep the light out as light causes oxidation of alpha-linolenic acid.

Supplements

We're often told that a "healthy balanced diet" (whatever ***that*** is) contains all the vitamins & minerals that we need. As the comedienne Gina Yashere might say "I don't fink so!"

Food ain't what it used to be. See http://observer.guardian.co.uk/foodmonthly/story/0,,1481443,00.html for an exposé of modern food.

In fact, according to http://www.ajcn.org/cgi/content/abstract/85/3/860 , 87% of the UK white population at age 45 are deficient in 25-hydroxyvitamin D in the Winter & Spring and 61% are ***still*** deficient in the Summer & Autumn despite the sun being strong enough to produce cholecalciferol (Vitamin D3) in the skin. This is because it's impossible to get enough D3 from our Standard English Diet unless we were to eat an Eskimo diet of >90% seal, whale & oily fish. Due to concerns about getting skin cancer, people cover themselves up or use high factor sun-block and get very little D3 from sunlight.

What about RDAs?

RDAs are basically the amounts of a vitamin, mineral or EFA that are required to prevent deficiency diseases. They are not the same as the amounts required for optimum health. Talking about Vitamin D3, the RDA is 200iu/day. I was taking 400iu/day for several years, so I should not have been deficient in D3. However, as my skin burns easily and I also have Broadband Internet access, I rarely ventured out during daylight hours so I got no D3 from sunlight.

Over a period of years, my mental faculties deteriorated, resulting in me having to take early retirement at the end of September 2006. As a result of a recommendation on the BBC Healthy living Message Board in January 2007, I increased my D3 intake from 400iu/day to 1,600iu/day. In the middle of February 2007, I noticed an awakening in my mental processes. This has continued and I am now well on the way to normal mental function. Nothing else changed in my supplement regime. I now take 2,000iu/day of D3 (20iu/kg body-weight/day), as well as getting 2 minutes/day full body exposure in front of a UV lamp.

I believe that many people are deficient in the mineral magnesium, resulting in muscle cramps, stress & irritability. Thanks to the modern "fad" of low-fat diets, modern animal feeding methods and the increasing use of omega-6 rich oils, I believe that many people are very deficient in omega-3 EFAs, resulting in a whole plethora of inflammatory conditions, from asthma, eczema & painful joints to coronary heart disease, strokes & cancer.

Blood glucose, insulin & diabetes

Diabetes is afflicting an increasing percentage of the population as time goes by. Even athletes like Sir Steven Redgrave can get it. This article tries to explain the workings of the body's blood glucose (BG) regulation system and what can go wrong with it.

How does the body regulate blood glucose?

At any given moment, there is about 4.5g of glucose circulating in your blood (5mmol/L x 180g x 5L). As the brain alone uses about 6g of glucose per hour in the absence of ketones, BG could fall to zero within an hour if we ate no sugary/starchy carbs. If we ate a mere 5g of glucose, BG level could double. As low BGs are fatal and high BGs damage proteins by a process called glycosylation (a bit like caramelisation), the body keeps BG levels within fairly tight limits by the use of a negative feedback (NFB) control system.

How does a negative feedback control system work?

NFB systems consist of a non-inverting (more in => more out) part, which in this case are one type of pancreatic cells called the islets of Langerhans (the type that secrete insulin are called beta cells), as increasing BG level results in increasing insulin level. It's actually more sophisticated than that. Beta cells can store insulin and dump it into the blood if there is a sudden increase in BG level. This is analogous to the accelerator pump in a carburettor, which dumps petrol into the engine if you slam your foot on the accelerator pedal, i.e. it produces a rapid response. The dumping of insulin from beta cell storage is known as the Phase 1 insulin response. If this (or the accelerator pump) fails, there is a lag in the response; which will become significant later.

Increasing BG level results in increasing insulin secretion from beta cells and is known as the Phase 2 insulin response.

The other part of a NFB system is the inverting (more in => less out) feedback part, which in this case is split into three parts, all working in parallel. They are:

1. Liver - increasing insulin level results in decreasing hepatic glucose production
2. Muscles - increasing insulin level shifts GLUT-4 transporters which shuttle glucose from blood into cells, decreasing BG level
3. Fat cells - increasing insulin level shifts GLUT-4 transporters which shuttles glucose from blood into cells, decreasing BG level

What can go wrong?

There are three main types of diabetes:

1) Type 2 diabetes. This by far the most common (about 95% of all cases) and has been linked with abdominal obesity. Type 2 diabetes has two main mechanisms going on. The first is a progressive insulin resistance of target tissues (firstly liver, then muscles and then fat cells in that order) possibly caused by increased levels of saturated fatty acids being fed to the liver from abdominal fat stores, and chronically-high BG and insulin levels caused by chronically over-consuming high glycaemic load carbohydrates, possibly accompanied by large amounts of saturated fat and/or large amounts of omega-6 fat. Omega-3 fats increase insulin sensitivity and monounsaturates seem to be neutral. A sedentary lifestyle lowers the sensitivity of muscle cells to insulin. Insulin resistance also

has a hereditary link. It may also be linked to a Vitamin D deficiency, see http://care.diabetesjournals.org/cgi/content/abstract/29/10/2244

Insulin resistance weakens the feedback in the NFB system, resulting in further increased BG and insulin levels. Increased BG level causes increased damage to beta cells by glycosylation. Increased insulin level causes further insulin resistance as target tissues become increasingly insensitive (a bit like louder and louder music making you progressively deafer and deafer). Eventually, beta cells become too damaged to secrete sufficient insulin and insulin levels begin to fall. This results in a massive rise in BG level and this is now full-blown Type 2 diabetes.

There are five main treatments for Type 2 diabetes:

* Lifestyle interventions - reduced intake of high glycaemic load carbohydrates and/or increased intake of omega-3 fats and/or increased intake of Vitamin D3 and/or increased intense exercise and/or loss of abdominal fat.
* Sulphonylureas - drugs which stimulate beta cells to secrete even more insulin. Unfortunately, that's a bit like flogging a dying horse as it doesn't address the problems caused by weakened feedback and eventual beta cell failure is inevitable, resulting in the need for insulin injections.
* Biguanide drugs such as Metformin - increase insulin sensitivity in target tissues. This strengthens the feedback in the NFB system, which results in reduced BG and insulin levels. This combined with lifestyle interventions can return the NFB system to normal operation.
* Thiazolidinediones - also increase insulin sensitivity in target tissues, e.g. muscle and fat, as well as possibly improving the secretory function of beta cells.
* Insulin injections take the strain off beta cells, but may worsen insulin resistance of muscle tissues resulting in increasing obesity.

2) Type 1 diabetes. This is much less common (about 5% of all cases) and is caused by an autoimmune disease. One possible mechanism is as follows: Due to an increase in Zonulin (see http://en.wikipedia.org/wiki/Zonulin), the gut becomes more permeable than it should (Leaky Gut Syndrome) and allows protein fragments to pass into the blood. These are destroyed by antibodies. However, if a protein fragment happens to have the same sequence of amino acids as a protein in our own body, the antibodies then set about destroying parts of our own body. Examples of this are gluten (proteins found in wheat, rye, barley and oats) producing antibodies in the blood that can destroy the gut causing Coeliac Disease or skin cells causing Dermatitis Herpetiformis or mucous membranes causing Sjogren's Syndrome or brain cells causing Cerebellar Ataxia. As there is an association between the consumption of cows' milk and the incidence of type 1 diabetes (see http://diabetes.diabetesjournals.org/cgi/content/abstract/49/6/912), it is quite possible that, in susceptible individuals, fragments of casein protein enter the blood in this way resulting in antibodies that destroy pancreatic beta cells.

Another possible mechanism is autoimmune attack after a viral infection.

Once all of the beta cells are destroyed, no insulin is secreted and insulin injections are required. If some beta cells survive, there is a possibility that normal BG level can be maintained if sugary/starchy carbohydrate intake is much reduced.

3) Latent Autoimmune Diabetes of Adulthood (LADA). The percentage of cases with this is unknown as it is often misdiagnosed as type 2 diabetes. This is a slow developing diabetes that is more like type 1 in origin (autoimmune with antibodies) but is often misdiagnosed as type 2 because of the age at diagnosis and the relatively slow progression of the disease (slow compared to type 1 but fast compared to type 2). See http://www.postgradmed.com/issues/2005/03_05/nabhan.htm . It is believed that Sir Steven

Redrave has this type of diabetes. Whether his autoimmune disease was triggered by a huge intake of milk (to build those Olympic-winning muscles) we will never know.

What else can go wrong?

As stated earlier, loss of the Phase 1 insulin response can occur. This usually happens when beta cells are chronically over-secreting insulin due to a chronically-high intake of sugary/starchy carbs and are unable to store any. This results in a lag in insulin response. This isn't a problem if low glycaemic load carbs are eaten and BG levels change only a little or very slowly. However, if high glycaemic load carbs are eaten, this produces a large and rapid rise in BG level. If a NFB loop with a lag in it is presented with a sudden change in input level, its output overshoots. This results in too much insulin being secreted, which eventually results in low BG levels! This is known as rebound hypoglycaemia. The solution? Stick to low glycaemic load carbs.

Where does blood glucose come from if I haven't eaten?

When no sugary/starchy carbs are being digested, BG starts to fall. Adrenaline and noradrenaline (catecholamine hormones) are secreted by the adrenal medulla into the blood and also by sympathetic neurons. Like glucagon (see below), they stimulate the mobilisation of glycogen and triacylglycerols (stored fats) by triggering the production of cyclic AMP (adenosine monophosphate). Adrenaline and noradrenaline differ from glucagon in that their glucose-producing effect is greater in muscle glycogen than in liver. They also inhibit the uptake of glucose by muscle. Instead, fatty acids released from adipose tissue are used as fuel. Adrenaline also stimulates the secretion of glucagon and inhibits the secretion of insulin. Thus, catecholamines such as adrenaline and noradrenaline increase the amount of glucose released into the blood by the liver and decrease the utilization of glucose by muscle.

Pancreatic alpha cells secrete glucagon. This hormone mobilises the conversion of liver glycogen into glucose. The liver only stores about 70g of glycogen, but when combined with water, a larger mass of glucose can be generated. Eventually, liver glycogen stores become depleted and BG level falls again. Glucagon also stimulates gluconeogenesis in the liver, which is the production of glucose from non-carbohydrate precursors, like the conversion of glucogenic amino acids, such as glutamine, into glucose. This causes slow muscle wastage unless there is sufficient protein intake to provide the necessary amino acids. When BG falls to about 3.3mmol/l, the pituitary gland kicks-in and secretes ACTH (adrenocorticotropic hormone) which stimulates the release of cortisol from the adrenal cortex. Cortisol further stimulates gluconeogenesis in the liver. When BG level falls to about 2mmol/l, the pituitary secretes GH (Growth Hormone) which has an anti-insulin effect.

What else does insulin do?

Insulin has many metabolic effects in the body apart from lowering BG level. It's a very anabolic hormone and an insulin spike is usually desired post workout to maximise the uptake of glucose and amino acids by muscle cells. There's nothing wrong with the occasional short-term insulin spike. It's chronically high insulin levels that cause long-term health problems like high blood pressure, high total cholesterol with low HDL-c & clogging of arteries.

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Even ***I*** could understand this – and I'm blonde! – Emily P. Kent.

Un-put-downable – he'd covered it in sticky tape! – Eileen C. Surrey.