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First published in Great Britain in 2020 by John Murray (Publishers)
First published in the United States of America in 2020 by Nicholas Brealey Publishing
Imprints of John Murray Press
An Hachette UK company

Cover image: Getty Images

A CIP catalogue record for this title is available from the British Library

UK eBook ISBN 978-1-529-31132-7
US eBook ISBN 978-1-529-36208-4

John Murray (Publishers)
Carmelite House
50 Victoria Embankment

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INTRODUCTION

I HAVE A confession to make: I'm an overweight, lazy slob. I love beer and fast food and the odd crafty fag outside a pub. I can't remember the last time I drank fewer than fourteen units of alcohol in a week. I have a gym membership but I rarely use it. After work I slump in front of the TV, with snacks. Sometimes I drive to a shop I could easily walk to, usually to buy beer. I take medication to keep my blood pressure under control and my body mass index (BMI) puts me in the 'overweight' category. Some lifestyle guru!

But here's another thing. Last year I cycled over 1,200 miles and ran the equivalent of fifteen marathons. I eat my five portions of fruit and vegetables a day, stay hydrated, watch my salt intake and don't eat meat. I fast regularly, go outdoor swimming, do Pilates and walk up eight flights of stairs to my desk instead of taking the lift. And despite my bad habits, my good ones seem to be winning. I'm fifty, but my 'biological age' was recently measured as forty-five. I'm living proof that you don't have to be a health and fitness fanatic to be reasonably healthy and fit.

What's my secret? I'm a science journalist with a background in biochemistry, and I have spent much of my working life keeping abreast of the latest thinking in biomedical science and writing about it for a non-specialist audience. As a result I'm able to evaluate health claims about nutrition, exercise, dietary supplements and more. I can spot a sensational headline a mile off, sniff out a fad and separate fact from fiction.

And I put what I know into practice. I'm not claiming to have invented the ideal health and fitness regime, but I lead a healthy but not too restrictive life, based on evidence. And that is the purpose of this book: to empower you to do the same. And I really mean 'empower'. Good health is something we should all strive for,

because the opposite – ill health – is not pleasant, and will eventually kill you.

Knowing what to do isn't easy. Biomedical science advances all the time, which is why we now routinely live to grand old ages and enjoy better health than our grandparents. But with this increase in knowledge come greater complexity and the risk of information overload. There's more advice out there than ever competing for your attention, but not always from the most authoritative sources.

My aim is to cut through the noise. I have rounded up the latest and most rigorous health research and converted it into useful, actionable advice on all the big health questions: we'll cover nutrition, diet, weight loss, hydration, exercise, preventative medicine, sleep and ageing. On the way I'll explain the basic science, debunk common myths, give you the tools you need to evaluate critically claims and counterclaims and help you to see beyond the headlines. Think of it as a manual for a healthy lifestyle.

If you have specific goals such as losing weight, improving your diet, getting fit, sleeping better or knowing which dietary supplements or superfoods are worth the money, you'll find advice in here. But the book is more than the sum of its parts. A mantra in health science is that we must consider the 'totality of the evidence'. That is what you'll get – a comprehensive and evidence-based guide to a heathier, fitter and, I hope, longer life.

Of course, lots of other people make similar claims. The world is not short of advice about what to eat and drink, how to stay fit, how to sleep better and live longer. But most of it is based on little more than myths, wishful thinking or unscientific mumbo-jumbo.

You can take this advice if you like. Or you can put your trust in the people whose job it is to work out what actually works: scientists. For every celebrity with a plan there are hundreds of nutritionists, exercise physiologists, sleep scientists and biomedical researchers doing the research and translating it into action. You won't read about it in glossy magazines or wellness blogs or see it on TV – it is mostly found in the pages of serious and technical scientific journals – but if you're really interested in

living a longer, healthier and happier life, this is the stuff you need to know.

First, though, a disclaimer. Contrary to what many self-appointed gurus will tell you, the road to health, fitness and wellness is neither simple nor easy. The science is often uncertain, contradictory or difficult to translate into concrete advice. Changing your lifestyle for the better inevitably requires some effort, willpower and sacrifice. If you want quick and easy fixes, this book is not for you. (But before you put it down and go in search of a quick and easy fix, let me tell you – for free – that anyone promising one is taking you for a fool and trying to part you from your money.) Do not let the certainty of sacrifice put you off. A modicum of effort can pay big dividends. And even just knowing that advice is based on good science can be a strong motivator to act.

That is also what sets this book apart. Resolving to change your life for the better is easy; actually changing it is hard. We've all made a resolution in January only to fall off the treadmill come February. But again, science can help. Understanding willpower, motivation and habit formation and how to harness them to your benefit is a big part of the battle.

And I promise it will pay off. You only get one life, and it's shrinking every day. If you enjoy being alive and well and want more of it, this is the book for you.

Sorting fact from fiction

Before we tuck into our main course of health advice, it is a good idea to have a starter of statistics: the science of drawing valid conclusions from raw data. Understanding a bit of mathematics can be key to debunking a sensationalist headline.

Consider the claim that taking multivitamin supplements prevents cancer. To see whether this holds up, you need to run an experiment where one group of people takes multivitamins and another doesn't. After a certain amount of time, you see how many cases of cancer occurred in the two groups. This is your raw data. To turn it into a scientifically valid conclusion, you need to put it through the statistical wringer.

You might think that if there were more cases of cancer in the non-vitamin group you've proved your point. But you haven't – the result could be due to chance. To rule this out you need to know the 'statistical significance' of the difference between the two groups. There is a standard equation for calculating this, which we don't need to concern ourselves with here. Suffice to say that the answer will be a number between 0 and 1.

For a result to be considered significant, that number needs to be at least 0.95. That means there is a 95 per cent probability that it did not happen by chance, and therefore reflects reality.

Some studies impose a higher threshold of 0.99 but 0.95 is the minimum standard of proof to look for. Bear in mind what that actually means: there is a one in twenty chance that it is a fluke. This is why multiple successful trials are needed to convince medical authorities that an effect is real.

Even if a finding is 99 per cent certain, that means there is a 1 per cent chance that it isn't. But that is not a licence to dismiss all scientific findings as 99 per cent certain means what it says: it is overwhelmingly likely to be true.

Statistical significance isn't everything. The second tip is to look at sample sizes – the number of people in your study. The bigger a sample size the more likely the result is to be valid. It's a bit like tossing a coin. Five tosses might give you four heads and a tail, but you'd be a fool to conclude that the probability of getting a head is therefore 0.8. Keep tossing the coin, say 100 times, and (assuming the coin is fair) that initial statistical noise will cancel out and a probability much closer to 0.5 will emerge.

Large sample sizes are also required to reveal small differences between intervening and doing nothing. If a nutritional study has a very low number of participants, say twenty or even fifty, take any conclusion with a very large pinch of salt.

The most important number to emerge from statistical analysis, however, is risk. In our example, that would be the risk of not taking multivitamins versus the risk of taking them.

But let's use a different example. Say you come across the finding that women who use talcum powder are 40 per cent more likely to develop ovarian cancer. Scary or what? It is easy to

interpret (or spin) this discovery as meaning that anyone using talc daily has a 40 per cent chance of ovarian cancer.

It doesn't, because 40 per cent is a 'relative risk' – the extra risk that you are taking by using talc. It means little unless you know the absolute risk, or how likely you are to get ovarian cancer if you don't use talc. That number is about 27 per 100,000, or 0.027 per cent. So a 40 per cent increase raises that to 38 per 100,000 – not negligible, but nothing to panic about.

Such rules of thumb can help anywhere you see a statistical claim. They have their limits – they are powerless to reveal when someone has cherry-picked their data or massaged their figures. But they are a good start when sorting out damned lies from statistics.

THE TRUTH ABOUT FOOD

THERE'S A FAMOUS scene in Woody Allen's 1973 film *Sleeper* in which two scientists in the year 2173 are discussing the dietary advice of the late twentieth century. 'You mean there was no deep fat, no steak, or cream pies, or hot fudge?' asks one, incredulous. 'Those were thought to be unhealthy,' replies the other. 'Precisely the opposite of what we now know to be true.'

'Incredible,' says the first.

We're not quite in incredible territory yet, but deep fat and cream pies are starting to look a lot less unhealthy than they once did. In the past few years, saturated fat – once the pariah of your plate – has been subject to a serious rethink.

And it is not just fat. The early twenty-first century has been a period of upheaval and soul searching for nutrition science. Almost everything we thought we knew has been challenged, and some of it overturned. Food groups once considered unhealthy are being rehabilitated, and vice versa.

This is probably familiar to anyone who keeps an eye on the latest news about diet and health with the goal of trying to eat well. It's confusing. For some reason the advice seems to keep on changing, so you end up not knowing if you are eating the right things.

One thing, however, remains absolutely beyond doubt. You are what you eat. Diet has a huge influence on your heath and is the bedrock of a healthy lifestyle. This chapter will bring you up to date on the latest thinking about some of the major food groups and nutrients, from fats and sugar to salt, meat, dairy and gluten, and conclude with some take-home messages.

But first, a health warning. Nutrition science is hard to do well, and rarely produces definitive answers. Focusing on a specific food group – fat, say, or fibre – does not capture the full complexity of what we eat over the course of our lifetimes, or how those different foods interact with each other and other lifestyle factors, which we'll look at in chapters to come.

Nonetheless, breaking down our complex diets into their component parts is a useful starting point for understanding the relationship between diet and health, and is the basis of official nutritional advice. To begin, let's start with perhaps the most interesting and misunderstood food group of all – fats.

THE TRUTH ABOUT FAT

For decades, dietary orthodoxy has been that fat is bad news. Not only is it the enemy of your waistline, it also clogs arteries and causes heart disease. The phrase 'a heart attack on a plate' was coined to describe the full English breakfast, swimming in grease. The idea that pigging out on such fare can lead to a heart attack is second nature to most of us; it is probably the single most influential piece of nutritional advice ever dished out.

There's no doubt that fatty food contributes to obesity – fat is the most calorie-dense of all the food groups – and being overweight is a risk factor for many diseases, including heart disease. But the idea that saturated fat is a direct cause of heart attacks appears to be melting away like a lump of lard in a hot pan.

What is a fat?

Fats are complex biomolecules that play various roles in the body, including energy storage and as components of cell membranes. A fat molecule is made up of three fatty acids bound to a molecule of glycerol. This unit is known as a triglyceride. There are dozens of different types of fatty acid, all with different properties.

The bulk of a fatty acid is a long string of carbon atoms with hydrogen atoms attached. In a saturated fatty acid, this chain does not have any carbon–carbon double bonds, meaning it has the maximum possible number of hydrogens: it is 'saturated'. Unsaturated fatty acids have at least one double bond. Fatty acids with more than one are called polyunsaturated, often used as a byword for health on food labels.

Triglycerides containing only saturated fatty acids are also called saturated; those with one or more double-bonded acids are unsaturated. As a rule, the more unsaturated a fat, the better it is for you – though this orthodoxy is being challenged. In terms of calories, however, there is no difference: saturated fats have just as much energy per gram as unsaturated fats.

Fats from animals tend to be saturated while those from vegetables are usually unsaturated. But this is only a rough guide. Meat, eggs and dairy contain unsaturated fats, while vegetables

also contain saturated fats. Some vegetable fats – notably palm oil, coconut oil and the cocoa butter used in chocolate – are higher in saturated fat than beef dripping or lard.

What about cholesterol? Strictly speaking, cholesterol is not a fat. But it is lumped together with fats in the category lipids, reflecting some commonalities. Neither fat nor cholesterol dissolves in water, for example. And cholesterol is a vital link between dietary fat and heart disease. Unlike saturated fat, it is almost exclusively found in animal products: meat, fish, seafood, milk and eggs. Cutting the cholesterol in your diet doesn't have much direct effect on blood cholesterol levels but can help indirectly because cutting down on cholesterol-rich foods will usually reduce your saturated fat intake.

Saturated fat: friend or foe?

Saturated fats are found in most foods, but are especially high in meat and dairy, as well as cakes, biscuits, pastries, chocolate, avocados, palm oil and coconut oil. The idea that eating them directly raises the risk of a heart attack has been a mainstay of nutrition advice since the 1970s. Instead, we are urged to favour the unsaturated fats found in vegetables and seafood.

This advice is driven by some pretty sobering figures on the toll of cardiovascular disease (a blanket term for diseases of the heart or blood vessels, including heart attacks, strokes, heart failure and angina). According to the World Health Organization (WHO), cardiovascular disease is the world's leading cause of death, killing more than seventeen million people annually, about a third of all deaths. It predicts that by 2030, that will have risen to twenty-three million a year.

In the US the official guidance for adults is that no more than 30 per cent of total calories should come from fat, and no more than 10 per cent from saturated fat. For a man eating the recommended 2,500 calories a day, that's about as much as is in 500 grams of beef mince (12 per cent fat), 130 grams of Cheddar cheese or 55 grams of butter. UK advice on saturated fats is the same: no more than 10 per cent of total calories. That is by no means an unattainable target: an average man could eat a whole twelve-inch pepperoni pizza and still have room for an ice cream before busting the limit.

Nonetheless, average adults in the UK and US manage to eat more saturated fat than recommended.

We used to eat even more. From the 1950s to the late 1970s, fat accounted for more than 40 per cent of dietary calories in the UK and US.[1] But as warnings began to circulate, people in Western nations trimmed back on foods such as butter and beef. The food industry responded, filling the shelves with low-fat cookies, cakes and spreads.

Gratifyingly, deaths from heart disease also went down. In the UK in 1961 more than half of all deaths were from coronary heart disease; now less than a third are (though cardiovascular disease is still the world's leading cause of death). But whether this is due to dietary changes is impossible to determine. Medical treatment and prevention improved dramatically, too. And even though fat consumption has gone down, obesity and its associated diseases have not.

To appreciate how saturated fat in food affects our health we need to understand how the body handles it, and how it differs from other types of fat.

When you eat fat (the triglyceride variety), it travels to the small intestine, where it is broken down into its constituent parts – fatty acids and glycerol – and absorbed into cells lining the gut. There they are bundled up with cholesterol and proteins and posted into the bloodstream. These small, spherical packages are called lipoproteins, and they allow water-insoluble fats and cholesterol (collectively known as lipids) to get to where they are needed in the body.

The more fat you eat, the higher the levels of lipoprotein in your blood. And that, according to conventional wisdom, is where the health problems start.

Lipoproteins come in two main types: high density and low density. Low-density lipoproteins (LDLs) are often simply known as 'bad cholesterol', despite the fact that they contain more than just cholesterol. LDLs are bad because they can stick to the insides of artery walls, resulting in deposits called atherosclerotic plaques that narrow and harden the vessels, raising the risk that a blood clot could cause a blockage. This state of affairs is called atherosclerosis – colloquially and not without reason known as

hardened arteries – and is the underlying cause of many cardiovascular diseases.

Of all types of fat in the diet, saturated fats have been shown to raise bad cholesterol levels the most. Paradoxically, the amount of cholesterol you eat matters much less. The reason it has a bad name is that it is found in animal foods that also tend to be high in saturated fat.

High-density lipoproteins (HDLs), or 'good cholesterol', on the other hand, help guard against arterial plaques. Conventional wisdom has it that HDL level is raised by eating foods rich in unsaturated fats or soluble fibre, such as whole grains, fruits and vegetables. This, in a nutshell, is the lipid hypothesis, possibly the most influential idea in the history of human nutrition and a major plank of the Mediterranean diet (see here).

Recently, however, the consensus around saturated fat has begun to weaken – though as yet the official dietary advice has not been changed. Doubts began to creep in about a decade ago when scientists pooled the results of twenty-one dietary studies that had followed a total of nearly 350,000 people for many years. Their analysis found 'no significant evidence' in support of the idea that saturated fat raises the risk of heart disease.[2]

A few years later an even bigger analysis revisited the results of seventy-two studies involving 640,000 people in eighteen countries.[3] Again, it failed to support the status quo, and the authors concluded that 'nutritional guidelines ... may require reappraisal'.

These doubts were reported widely, often with gusto. Many commentators interpreted them as a green light to resume pigging out on saturated fat. 'Eat Butter', declared the cover of *Time* magazine in 2014.

Can you safely ignore the old advice? For now the answer is an emphatic no. Other, less widely publicised analyses have supported the link between saturated fat and heart disease. There is also good evidence from animal research, where dietary control is possible to a degree that it is not in people. Such research repeatedly shows high saturated fat leads to elevated bad cholesterol and hardened arteries.

The results casting doubt on the orthodoxy could have arisen from other factors. It may be that in free-living humans going about their daily lives, the risk of developing heart disease depends on much more than simply the balance of saturated and unsaturated fat in the diet. Factors such as lack of exercise, alcohol intake and body weight may simply overwhelm the impact of fat.

Another key factor might be what people who cut down on saturated fat eat instead. All too often people consciously or unconsciously replace a large reduction in calories with something else. The problem is that the something else is often refined carbohydrates, especially sugars, added to foods to take the place of fat. This plays to the emerging idea that sugar is the real villain (for more on sugar, see here).

Then there are trans fats. Created by food chemists to replace animal fats such as lard, they are made by chemically modifying vegetable oils to make them solid at room temperature. Because they are unsaturated, and hence classed as 'healthy', the food industry piled them into products such as cakes and spreads. They also have chemical and physical properties appreciated by the food industry. They are highly resistant to rancidity and so extend the shelf life of foods. Restaurants love them because oils with trans fats can be heated and cooled repeatedly without breaking down.

However, it later turned out that trans fats cause heart disease. There is good evidence that they raise your LDL cholesterol (the bad form), and lower your HDL cholesterol (the good one), causing hardened arteries. In 2002 the US National Academy of Sciences concluded that the only safe amount of trans fat in the diet is zero.

All told, it is possible that the meta-analyses simply show that the benefits of switching away from saturated fat were cancelled out by replacing them with sugar and trans fats. But there is also emerging evidence that the impact of saturated fat and LDL is more complex than we thought.

At the moment all LDL is treated alike, but there are studies suggesting that casting it all as bad was a mistake. It is now widely accepted that LDL comes in two types – big, fluffy particles and smaller, compact ones. It is the latter that are strongly linked to heart-disease risk, while the fluffy ones appear a lot less risky. Crucially, eating saturated fat boosts fluffy LDL. What's more,

there is some research suggesting small (that is, *very* bad) LDL is elevated by a low-fat, high-carbohydrate diet, especially one rich in sugars.

Why might smaller LDL particles be riskier? In their journey around the bloodstream, LDL particles bind to cells and are pulled out of circulation. The hypothesis is that smaller LDLs don't bind as easily, so remain in the blood for longer – and the longer they are there, the greater their chance of causing damage. They are also more easily converted into an oxidised form that is considered more damaging. Finally, there are simply more of them for the same overall cholesterol level. And more LDLs may equate to greater risk of arterial damage.

Complex enough? Well, there's more. Not all saturated fats are the same. A study from 2012 found that while eating lots of saturated fat from meat increased the risk of heart disease, equivalent amounts from dairy reduced it.[4] The researchers calculated that cutting calories from meaty saturated fat by just 2 per cent and replacing it with saturated fat from dairy reduces the risk of a heart attack or stroke by 25 per cent. That sounds like actionable advice, but it is far too soon to swap meat for dairy. And in any case many dairy foods – cheese especially – are high in calories and salt.

This goes back to a common problem with nutrition science. Research on single nutrients can create a misleadingly simplified picture. People do not eat saturated fat but eat foods containing mixtures of saturated, unsaturated and polyunsaturated fats, plus many other nutrients. Teasing out the effect of one nutrient within that complex buffet is very difficult.

For this reason and others it is too soon to declare saturated fat innocent of all charges; much more research is needed before the nutrition rule book can be rewritten. So while dietary libertarians may be gleefully slapping a fat steak on the griddle and lining up a cream pie with hot fudge for dessert, the dietary advice of the 1970s still stands – for now. In other words, steak and butter can be part of a healthy diet. Just don't overdo them.

THE TRUTH ABOUT OMEGA-3S

There's at least one kind of fat that most of us should probably be striving to get more of: omega-3s. These are a family of fatty acids that are vital for our health. As a key ingredient of cell membranes they have wide-ranging benefits including protecting against cardiovascular disease and cancer.

Omega-3s are usually associated with oily fish, but that is a bit of a myth. The most important one is called alpha-linolenic acid (ALA), which cannot be synthesised in the body and so must be obtained from our diet. But it is not found in fish. The best sources are chia seeds, kiwi fruit, walnuts, flax seeds (linseed), rape (canola) and soybean oil, and seaweed. Leafy green vegetables are another good source.

There are two other really important omega-3s: eicosapentaenoic acid (EPA) and docosahexaenoic acid (DHA). Both can be made from ALA but only at low efficiencies that may not supply enough. Both can also be obtained directly by eating animal products, particularly oily fish. Algae make large amounts of EPA and DHA and these fatty acids accumulate up the marine food chain, with the highest levels found in predatory fish like mackerel and tuna.

For all three, average intake among adults in the US and UK falls far short of the recommended amount, largely due to the fact that many people eat little or no oily fish. Omega-3 is probably the only nutrient deficiency that is common in the West.

Worryingly, changes in farming methods are making some fish lower in omega-3s. Half of all fish consumed globally now come from aquaculture, and farmed fish have a different nutritional profile to wild varieties. Wild salmon, for example, is an excellent source of omega-3s because it feeds on smaller fish that have eaten omega-3-rich algae. But farmed fish are increasingly fed vegetable matter, suppressing their omega-3.

Many foods are fortified with omega-3 to address consumer concerns about not getting enough (and to shift more product, of course). But for some reason omega-3-fortified foods don't seem to deliver the same benefits as foods naturally high in omega-3.

Also be sceptical of omega-3 supplements or the fish oil capsules that boast a high omega-3 content. Recent studies indicate that – unlike eating actual fish – taking these does nothing to reduce

your risk of heart disease (for more on omega-3 supplements, see here).

Beside ALA the only other essential fatty acid is linolenic acid, which is chemically very similar. This is an omega-6 fatty acid, found in abundance in vegetable oils. Getting enough of this is not a problem. If anything, we eat too much. Excess omega-6 appears to interfere with metabolism of omega-3s, suppressing their health benefits.

Ironically, the omega-3-suppressing diet of farmed fish also increases levels of omega-6. In other words, eating too much vegetable oil and farmed oily fish may be bad for your health, which is not a message you will hear very often. But as with so many other things related to nutrition, the science is still not settled and focusing on single nutrients is likely to create problems elsewhere. The best response is to eat lots of vegetables, cut down on all fats – also a good idea for many other reasons – and try to eat wild oily fish rather than farmed.

THE TRUTH ABOUT CARBS AND SUGAR

The debate about saturated fat also touches on another food group whose reputation was seemingly sealed by twentieth-century research – albeit in the opposite direction.

The usual flip side of cutting down on saturated fat is to fill up on starchy foods. But some doctors now advocate the exact opposite: people who want to lose weight should stop worrying about fats and instead cut down on starch. If this is to be believed, it is not fat but carbohydrates we should be worried about. Potatoes, bread, pasta and rice – even the wholemeal varieties – make us fat and cause heart attacks and type 2 diabetes. Can that really be true?

What is a carbohydrate?

Starchy foods are part of a larger food group called carbohydrates, or carbs. They are a diverse bunch covering everything from simple sugars like glucose to tough, indigestible fibre. But what

is released depends on what you eat with it – fat or protein lowers the rate.

Releasing insulin to manage blood sugar is a perfectly normal metabolic process but it has its limits. When too much glucose hits the bloodstream at once, it overwhelms the body's ability to deal with it. Over time, this takes a toll. The pancreas works ever harder to pump out insulin but eventually becomes exhausted. Chronic release of insulin also causes cells to become increasingly insulin resistant. Eventually, this combination of a weakened pancreas and insulin resistance can progress to type 2 diabetes.

Insulin resistance also seems to be a bigger player in heart problems than we thought. One recent study found that for men, it is a bigger heart attack risk than high blood pressure, high cholesterol or being overweight.[7]

As a rule of thumb, the more complex the carb, the better it is for you because the slower it will release its sugars. Exactly how do you figure out which carb foods are best? One pointer is the glycaemic index (GI). The GI is a way of comparing how rapidly carbohydrates affect blood glucose levels compared with pure glucose, which is given a GI of 100. Foods with a high GI (above 70), such as peeled, boiled potatoes (89) or baguettes (95), hit the bloodstream fast and cause spikes in blood glucose. Foods with a low or moderate GI (55–70) like wholegrain breads release their glucose more slowly. Hence the health halo around wholemeal bread and pasta, brown rice, bran flakes and fibrous fruit and vegetables.

GI can be deceptive, though, because it doesn't tell you the absolute amount of carbohydrate in the food. On the one hand, even low-GI foods can cause blood sugar spikes. On the other, a boiled carrot has a high GI, but contains so little sugar that it has almost no impact on blood sugar – it has a low 'glycaemic load'. Fruits, vegetables, lean meat and grains all have a low glycaemic load. So does fat. Many nutritionists now consider the glycaemic load to be the measure that matters.

So is it time to overhaul official dietary advice? Probably not. The weight of evidence is still that starchy carbs are a healthier choice than fats, though the evidence is not as solid as it once looked.

Such nuanced evidence might well leave you scratching your head over what to eat. There isn't much left if you try to avoid both fat and carbs. A more moderate approach is to avoid saturated fat, added sugars and refined carbs, which leaves you more or less with an extra-oily Mediterranean-style diet, high in whole grains, fish, fruit, vegetables, nuts and vegetable oils.

Another option with some evidence on its side – albeit mostly anecdotal – is a light version of the Atkins diet. Cut down on starchy food and eat lots of non-starchy vegetables and less sugary fruits such as blueberries and raspberries. In place of carbs, fill up on meat, fish, full-fat dairy products, eggs and nuts. Anecdotally, people on this diet report less hunger while also losing weight. Their blood tests show improvements in glucose control, as well as blood pressure and cholesterol levels.

That may be down to a type of carb that we have hitherto neglected. Fibre, the largely indigestible structural material found in fruit, vegetables and whole grains, slows the absorption of sugars from the intestine and prevents the glucose spike. This is why healthier diets are not only low in refined carbs such as sugar, white flour and alcohol, they also contain plenty of fibre.

Poisonous potatoes?

Another reason to hold the carbs is that they may be carcinogenic, thanks to a compound called acrylamide. You may have heard the advice to avoid roast potatoes. Acrylamide is the reason for this.

As an industrial compound, acrylamide is classified as an extremely hazardous substance. The International Agency for Research on Cancer (IARC) lists it as a probable carcinogen.

Acrylamide is not added to food or found in uncooked foods. It is produced by cooking, specifically something called the Maillard reaction, which occurs between proteins and sugars when they are heated above 120 °C. The reaction produces a mixture of thousands of different chemicals that give many browned foods their appetising flavour. But acrylamide is anything but appetising. In the body, it is converted into another compound, glycidamide, which can bind to DNA and cause mutations. Animal studies clearly show that acrylamide causes all sorts of cancers.

Browning starchy foods such as potatoes produces particularly high levels of acrylamide, hence the warning about roasties. Bread is another source, especially when toasted. The chemical can also be present in breakfast cereals, biscuits and coffee.

It's hard to study the effects of acrylamide in people, but there's no reason to think that it does not damage our DNA. Quantifying the risk is difficult, however, but it probably pales in comparison to other well-known carcinogenic lifestyle factors such as smoking, obesity and alcohol. People who work in the food industry are often exposed to high levels of acrylamide, but do not have higher rates of cancer.

If you want to minimise acrylamide exposure, cut back on crisps, chips and biscuits. These are major sources of acrylamide and have the added downside of being high in sugar and/or fat. When frying, baking, toasting or roasting starchy foods, the UK Food Standard Agency's advice is to 'go for gold': aim for a golden yellow colour rather than brown. If you like your roast potatoes brown and crispy, you may have to eat them less often.

Another way of reducing exposure is to not keep raw potatoes in the fridge. At low temperatures, an enzyme called invertase breaks down the sugar sucrose into glucose and fructose, which can form acrylamide during cooking. Frozen food doesn't carry this risk, as sucrose doesn't get broken down at very low temperatures.

You can also blanch potatoes before roasting or frying them. This removes half the sugar, resulting in lower levels of acrylamide.

THE TRUTH ABOUT ADDED SUGARS

Imagine you are sitting at a table with a bag of sugar, a teaspoon and a glass of water. You open the bag and add a spoonful of sugar to the water. Then add another, and another, and another, until you have added twenty teaspoons. Would you drink the water?

Even the most sweet-toothed kid would find it unpalatably sickly. And yet that is the amount of sugar you are likely to eat today, and every day – usually without realising it.

What is added sugar?

Added sugar, or 'free sugar', refers to sugar added to food and drink (either by you or by food manufacturers) plus any sugars found in fruit juices, honey, maple syrup and so on.

The sugar added to food by manufacturers is usually either table sugar, which is sucrose, or high-fructose corn syrup. Sucrose is made up of a molecule of glucose and a molecule of fructose bonded together; they are split during digestion. High-fructose corn syrup, a mixture of glucose and fructose, is often portrayed as unhealthier than sucrose, but most researchers now agree that they are largely the same.

Calculating how much free sugar is in your diet is difficult. Food labels don't distinguish between natural and added sugar – a loophole the food industry is in no hurry to close.

Public health enemy number one?

Sugar was once a luxury ingredient reserved for special occasions. But in recent years it has become a large and growing part of our diets. If you eat processed food of any kind, it probably contains added sugar. Three-quarters of the packaged food sold in US supermarkets has had sugar added to it during manufacturing. You can find it in all sorts of unlikely places: sliced bread, salad dressings, soups, cooking sauces and many other staples. Low-fat products often contain a lot of added sugar.

It's hardly controversial to say that all this sugar is probably doing us no good. Now, though, sugar is being touted as the true villain of the piece: as bad as if not worse than fat, and the major driving force behind obesity, heart disease and type 2 diabetes. Some researchers even contend that sugar is toxic or addictive.

The WHO wants us to cut consumption radically; in 2017 it issued recommendations that adults and children should reduce their intake of 'free sugars' to less than 10 per cent of total energy intake, and preferably below 5 per cent. That would mean cutting current consumption by two-thirds, to about eight teaspoons a day for men and six for women. Many countries including the UK have introduced some form of sugar tax to incentivise people to cut down. But is sugar really that bad? Or is it all a storm in a teacup – with two sugars, please?

When nutrition scientists talk about sugar they are generally not fretting about sugars found naturally in food such as fruit and vegetables, or the lactose in milk. Instead they are worried about added sugar, usually in the form of sucrose (table sugar) or high-fructose corn syrup.

Our early ancestors would have been totally unfamiliar with these refined forms of sugar, and until relatively recently sugar was a rare and precious commodity. Only in the 1700s, after Europeans had introduced sugar cane to the New World and shackled its cultivation to slavery, did it become a regular feature of the Western diet. In 1700, the average English household consumed less than two kilograms of table sugar a year. By the end of the century that amount had quadrupled, and the upward trend has continued largely unbroken ever since. Between the early 1970s and the early 2000s, adults in the US increased their average daily calorie intake by 13 per cent, largely by eating more carbohydrates, including sugar. Today, yearly sugar consumption in the US is close to forty kilograms per person – more than twenty teaspoons a day.

The sugar rush has many causes, but one of the most important was the invention of high-fructose corn syrup (HFCS) in 1957. HFCS is a gloopy solution of glucose and fructose that is as sweet as table sugar but is typically about 30 per cent cheaper. Once this source of sweetness was available, food manufacturers added it liberally to their products. The motivation was to enhance palatability and hence increase sales.

Unfortunately, it is a guilty pleasure. Not all scientists see eye to eye on the health effects of sugar but there is one point on which most agree: we don't actually need it. You cannot live without essential fats, proteins and some carbohydrates. But sugar is an entirely dispensable food. All that unnecessary sugar adds calories to our diet, so it is no surprise that the rise in consumption coincided with the rise of obesity and related problems such as type 2 diabetes. In 1960, around one in eight US adults was obese; today more than a third are. Since 1980, obesity levels have quadrupled in the developing world to nearly one billion people. One study found that for every additional 150 calories' worth of


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stroke deaths were down by 80 per cent. Finland, another salt-guzzling nation, achieved similar gains in the 1970s.

A pinch of salt?

However, there are still some uncertainties. One oft-voiced concern is that sodium may not be the whole story and that other minerals in the diet – notably potassium – also play a role in blood pressure. That is true, but in a good way. Potassium blunts the blood pressure effects of sodium, and the effect is mediated by the ratio of potassium to sodium rather than the absolute amounts.

However, modern diets are not only laden with sodium but also depleted in potassium. So an effective way to boost the health benefits of salt reduction is to switch to high-potassium table salt or eat more high-potassium foods such as bananas, oranges and black beans. However, if you have kidney disease, heart disease or diabetes, seek medical advice as an increase in potassium may be harmful.

Another perhaps more worrying possibility is that salt reduction may have unintended consequences. Big reductions in salt intake may bring about hormonal changes that also increase the risk of cardiovascular disease. This is known as the 'J-shaped curve' hypothesis because, if plotted on a graph, both extremes of salt intake are associated with an increase in mortality.

The evidence for this is still quite thin on the ground, though a recent study done in the Netherlands found that a low sodium intake of less than three grams a day was associated with an increased risk of stroke.[12] However, the researchers say that more research is needed. And in any case even if low levels of sodium are risky, you'd be hard pressed to achieve them without serious efforts to cut back on salt. Your best bet is to make those efforts.

A spicy solution

One way to cut down on salt is to use spices as a substitute. Spices have been added to national dietary guidelines in the US and Australia because adding them to food makes it easier to cut down on salt.

Spices may even bring health benefits of their own. They are often rich in polyphenols, a group of plant compounds thought to

eating no more than 500 grams of red meat per week, roughly the equivalent of four burgers.

More recently, the WHO looked at the evidence and concurred that red meat – by which it means all meat from mammals, including pork – is a probable carcinogen, and processed meat a definite carcinogen.[14] Processed means salting, curing, smoking, drying or fermenting. As well as the obvious ones, this category also included jerky, biltong and canned meat. The WHO did not look at poultry (white) meat or fish, but other evidence suggests there is no link between these and cancer.[15]

The reason processed meats cause cancer appears to be added preservatives such as nitrites and nitrates, which are converted into carcinogens called N-nitroso compounds (NOCs) in the gut. However, this cancer risk needs to be put in perspective. Consider the recent finding that a bacon sandwich a day raises the likelihood of bowel cancer by 20 per cent. You could be forgiven for thinking that it means a daily bacon butty gives you a one in five chance of bowel cancer. But remember that this number is a relative risk: how much more likely you are to get bowel cancer if you regularly eat bacon sandwiches than if you do not. For an average person, the chance of getting bowel cancer at some point in their life is around 5 per cent. So a 20 per cent increase means raising the absolute risk from 5 to 6 per cent. That's big enough not to ignore, but not so big that it demands a total avoidance of bacon.

Cancer isn't the only worry. The science isn't fully settled yet, but diets high in saturated fats and cholesterol – which are found plentifully in meat – have been shown, as we have seen, to increase the risk of heart disease.

Indeed, two large studies published in 2012 found that the risk of dying from all causes – including heart disease – was 13 per cent higher for people eating 85 grams of red meat per day, and 20 per cent higher for those eating 85 grams of processed meat (which may be especially bad because it also contains high levels of salt and sugar).[16] That would translate to roughly a year off life expectancy for a forty-year-old man who eats a burger a day. That's a lot of lives potentially being shortened. In the UK, 40 per