Clubwell®

ebook

Fat, Fibre & Flora

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Introduction:
The Three Things Nobody Talks About Together

Everyone is talking about protein.

Protein shakes. Protein bars. High protein diets. Protein targets. If you spend any time in health circles, you could be forgiven for thinking that protein is the only macronutrient worth caring about. And yes, protein matters. Enormously. I am not about to tell you otherwise.

But here is what I have come to believe after years of reading the science, speaking with some of the world's leading researchers, and spending the better part of a decade building a health community dedicated to getting this right. The conversation about protein has crowded out something more important. Three things, to be precise.

Fat. Fibre. Flora.

These three elements sit at the centre of almost everything that goes wrong in the modern body. And almost nobody talks about them together, in the same breath, as the interconnected system they actually are.

I spent twenty-five years being obese while following official dietary advice. I ate low-fat food. I avoided butter and replaced it with margarine. I chose skimmed milk, low-fat yoghurt, reduced-fat everything. And I got fatter, sicker, and more confused with every passing year. When my parents were diagnosed with type 2 diabetes and Alzheimer's, I stopped trusting the official

narrative and started reading the science myself. What I found made me furious.

The fat advice was wrong. Not a little bit wrong. Catastrophically wrong. Based on cherry-picked data, funded by the sugar industry, and built into dietary guidelines that fifty years later are still making people sick.

The fibre advice was almost non-existent. We were told to eat some. We were not told what it actually does in the body, why it is the most important food for our gut bacteria, how it triggers the same satiety hormones that Ozempic mimics, or why stripping it from our food supply may be one of the most damaging things the food industry has ever done.

And the flora, the community of bacteria, fungi, and other organisms living in your gut, barely rated a mention. Yet we now know that this inner ecosystem influences your weight, your mood, your immune system, your risk of cancer, your risk of Alzheimer's, and your likelihood of developing virtually every chronic disease that defines modern ill health.

This short book is my attempt to change that. It is not a diet book. It is not a plan. It is an education. I want you to understand what fat actually does when you eat it and why saturated fat was framed for a crime it did not commit. I want you to understand what fibre does in the body at a cellular and hormonal level, not just that it keeps you regular. And I want you to understand the extraordinary community of organisms living inside you and what happens when you stop feeding them properly.

Once you understand these three things and how they connect, the way you eat will change. Not because someone told you to. Because the knowledge itself makes the old way of eating seem, quite frankly, absurd.

Let us begin...

Part One:
Fat

Why the most essential substance
in your body became the most
feared food on the planet.

Chapter One:
The Great Fat Lie

Everyone is talking about protein.

Protein shakes. Protein bars. High protein diets. Protein targets. If you spend any time in health circles, you could be forgiven for thinking that protein is the only macronutrient worth caring about. And yes, protein matters. Enormously. I am not about to tell you otherwise.

In 1955, President Dwight D. Eisenhower had a heart attack. He was the most powerful man in the world, and the nation watched in horror. Heart disease was rising fast across America, and someone needed to find an explanation.

That someone was a scientist called Ancel Keys.

Keys had a theory. Dietary fat, he believed, raised cholesterol, and raised cholesterol caused heart disease. He had been pushing this idea for years, but few were listening. Eisenhower's heart attack changed everything. Suddenly, the question of what caused heart disease was a political and public health emergency, and Ancel Keys was ready with his answer.

In 1958, he published what became known as the **Seven Countries Study**. It appeared to show a clear correlation between fat consumption and heart disease deaths across seven countries. The graph was compelling. The

data looked convincing. Keys became famous. His hypothesis became doctrine.

There was just one problem. He had data from twenty-two countries, not seven. He selected the seven that supported his theory and discarded the fifteen that did not. When researchers later analysed the full twenty-two country dataset, the correlation largely disappeared.

John Yudkin, a British scientist and professor of nutrition at Queen Elizabeth College in London, saw this clearly. Yudkin argued that the real culprit was not fat but sugar. He published his findings. He was methodical, evidence-based, and right. And he was destroyed for it.

The sugar industry, which had been watching the fat debate with great interest, hired scientists to attack Yudkin's work and fund research that shifted the blame firmly onto fat. Internal documents revealed decades later showed that the Sugar Research Foundation paid three Harvard scientists to publish a review in 1967 that downplayed sugar's role in heart disease and emphasised fat as the problem. One of those scientists later became the head of nutrition at the United States Department of Agriculture and helped shape the dietary guidelines that would define what millions of people ate for the next half century.

The guidelines duly followed. Low fat became the message. Governments adopted it. Food companies loved it because removing fat and replacing it with sugar and refined starch made food cheaper, more addictive, and longer lasting on shelves. A multi-billion-pound industry built itself on the back of the low-fat label.

And people got fatter. And sicker. And the rates of heart disease, type 2 diabetes, obesity, and metabolic syndrome continued to climb.

We now have substantial evidence that the original hypothesis was flawed. The Women's Health Initiative, the largest randomised

controlled trial of a low-fat diet ever conducted, followed nearly 50,000 women for over eight years. The low-fat group showed no significant reduction in cardiovascular disease, no reduction in breast cancer, and no meaningful weight loss compared to the control group. Eight years. Fifty thousand women. And the diet they had been told would save their lives did nothing of the sort.

The PURE study, published in The Lancet in 2017, followed 135,335 people across eighteen countries. It found that higher fat intake was associated with a 23 per cent lower risk of total mortality. Higher carbohydrate intake was associated with a 28 per cent higher risk of mortality. The findings were so contrary to official guidelines that the study caused an uproar. The evidence was solid. The uproar continued anyway.

The Minnesota Coronary Experiment is perhaps the most revealing of all. Conducted in the 1960s, it replaced saturated fat with vegetable oil in the diets of hospital and nursing home patients. Cholesterol levels dropped. Exactly as the diet-heart hypothesis predicted. But mortality increased. The people whose cholesterol dropped most had the highest risk of death. These findings were suppressed for decades, unpublished until 2016 when researchers discovered the original data.

Let that sink in. An experiment that showed saturated fat replacement with vegetable oil increased mortality was hidden for fifty years.

This is the context in which I want you to understand everything that follows. **The dietary advice on fat was not just wrong. It was built on biased science, funded by industries with commercial interests, and then defended long after the evidence turned against it.** Understanding this history matters because it shapes how you feel about fat today, and that feeling is almost certainly making you less healthy.

Chapter Two:
The Journey of Fat

Most people have no idea what actually happens to fat when they eat it. They know, vaguely, that it goes in the mouth and either gets used or stored. The details are a mystery. And in that mystery lives the fear.

So let me take you through it. Because once you understand the journey fat takes from your fork to your cells, the idea that dietary fat is clogging your arteries becomes not just wrong but biologically implausible.

It starts in your mouth. Saliva contains an enzyme called **lingual lipase** that begins the process of breaking fat apart even before you swallow. Not much happens here, but the process has begun. As the food moves to your stomach, gastric lipase continues the work, breaking apart fat molecules in the acidic environment.

Then comes the critical stage: the small intestine.

When fat arrives in the duodenum, the liver and pancreas respond. The gallbladder releases bile, a substance produced by the liver and stored ready for exactly this moment. Bile is extraordinary. It is essentially a natural detergent. It takes the fat, which does not dissolve in water, and emulsifies it into tiny droplets called micelles, making it accessible to the digestive enzymes. Pancreatic lipase then breaks the fat down into its component fatty acids and monoglycerides, small enough to be absorbed into the cells lining the small intestine.

Inside those intestinal cells, something remarkable happens. The fatty acids are reassembled into triglycerides and packaged into structures called

chylomicrons. Chylomicrons are large, spherical particles. Think of them as the delivery trucks of the dietary fat world. And every single one of them carries a protein on its surface called ApoB-48.

Here is the crucial point, and I want you to read this carefully because it changes everything.

These chylomicrons do not go into your bloodstream directly. They enter the lymphatic system. Specifically, they enter tiny lymphatic vessels inside each intestinal villus called lacteals. From there, they travel through the lymphatic network, up through the thoracic duct, and enter the bloodstream near the collarbone. They have bypassed the liver entirely.

Once in the bloodstream, chylomicrons deliver their triglycerides to muscle cells and fat tissue, where the fatty acids are either used for energy or stored. As they shed their contents, the chylomicrons shrink and are eventually cleared by the liver.

The reason this journey matters so much is what it tells us about arterial plaques.

Arterial plaques are built from LDL particles that penetrate the arterial wall, become oxidised, trigger an inflammatory response, and accumulate as foam cells. This process is well established. But the LDL particles that cause this damage carry a protein called ApoB-100. Not ApoB-48. Not the protein that chylomicrons carry.

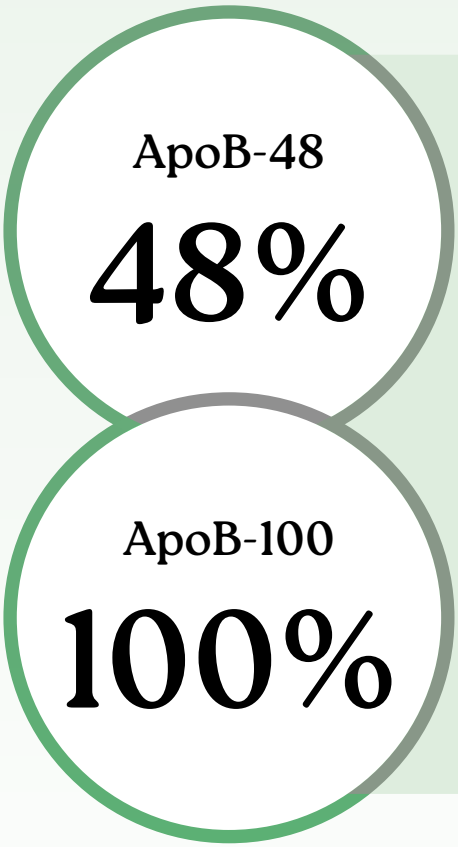
Chylomicrons are physically too large to penetrate the arterial endothelium. They cannot get through. The dietary fat delivery system is structurally incapable of directly depositing fat into your artery walls. The vehicle simply does not fit through the door.

This is not a fringe argument. This is basic physiological fact. Dietary fat uses the ApoB-48 route. Arterial plaques are built from ApoB-100 particles. These are different proteins on different particles produced by different processes in different organs.

Where do ApoB-100 particles come from? The liver. And what drives the liver to produce them in excess? That is the question the next chapter answers. But I will give you a hint: it is not the butter you had on your toast.

Chapter Three:
ApoB-48 vs ApoB-100 - The Case That Was Never Made

ApoB-48 and ApoB-100 are not just two versions of the same thing. They are fundamentally different proteins with different origins, different sizes, different destinations, and crucially, very different relationships with cardiovascular disease.



The names are not arbitrary.

ApoB-48 is 48% of the full ApoB protein sequence.

ApoB-100 is the complete version, 100% of the protein.

This difference in size matters enormously because the full-length ApoB-100 has a receptor-binding region that ApoB-48 lacks. That receptor-binding region is what allows LDL particles to be taken up by cells throughout the body, and when those cells are saturated, it is what allows the particles to persist in the bloodstream long enough to cause damage.

ApoB-48 is made in the intestine and goes on chylomicrons. It carries dietary fat through the lymphatic system and delivers it to cells. Once the fat is delivered, the remnant particles are cleared by the liver quickly and efficiently.

ApoB-100 is made in the liver. It goes on VLDL particles, which the liver secretes into the bloodstream. As VLDL particles shed their triglycerides, they shrink and become IDL, and then LDL. It is this LDL, carrying ApoB-100, that has the potential to penetrate the arterial endothelium and contribute to plaque formation, particularly when the LDL particles are small and dense.

So the question we should have been asking all along is not how much dietary fat are you eating. **The question is: what is driving your liver to produce excess ApoB-100 particles?**

The answer is insulin. Specifically, chronically elevated insulin, driven by a diet high in refined carbohydrates and sugar.

When you eat refined carbohydrates or fructose, your liver works overtime. Fructose in particular is almost entirely metabolised by the liver, and when consumed in excess, it triggers a process called de novo lipogenesis, where the liver converts excess carbohydrate into fat. The liver then packages this fat into VLDL particles and releases them into the bloodstream. More VLDL means more LDL downstream. And a liver under metabolic stress tends to produce smaller, denser LDL particles, the type most strongly associated with arterial damage.

Here is the irony. The dietary advice that blamed fat for heart disease, and told people to replace fat with carbohydrates and sugar, was actually driving the very mechanism it claimed to be preventing.

There is another dimension to this that rarely gets discussed. Not all LDL is the same. There are large,

buoyant LDL particles, sometimes called pattern A, and small, dense LDL particles, sometimes called pattern B. These two types of LDL have very different risk profiles. Large buoyant LDL is largely benign. Small dense LDL is the type that penetrates arterial walls, oxidises, triggers inflammation, and contributes to plaque.

What drives the production of small dense LDL? A diet high in refined carbohydrates, fructose, and excess calories. What drives the production of large buoyant LDL? Saturated fat. Yes, saturated fat raises LDL, but it preferentially raises the large buoyant type that is not associated with cardiovascular risk in the same way.

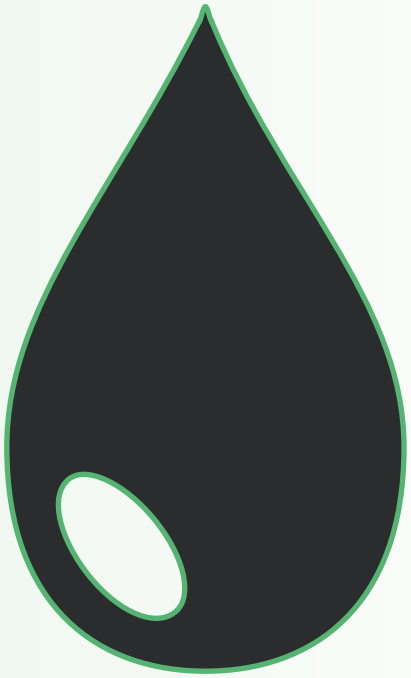
This is why the total LDL number, the single figure that has terrified patients and driven statin prescriptions for decades, is such a poor predictor of cardiovascular risk. It lumps together two very different particles with very different risk profiles. Measuring total LDL without knowing the particle size and number is a bit like judging the danger of traffic by counting total vehicles without distinguishing between lorries and bicycles.

I am not anti-statin. They clearly help certain high-risk populations. But the dietary policy that has shaped what 67 million British people eat every day was built on a misunderstanding of the very mechanism it claimed to be addressing. And that misunderstanding has cost lives.

The science on ApoB-48 and ApoB-100 is not new. Biochemists have known this for decades. What has failed is the translation of that science into the dietary guidelines, food labelling systems, and public health messaging that shapes what people actually believe and eat.

Chapter Four:
The Fats We Should Fear

I want to talk about the fats that actually deserve your concern. Not butter. Not olive oil. Not the fat in your steak or your egg yolk.



The fats that should be keeping you up at night are the ones sitting in your kitchen cupboard right now, probably next to your frying pan, probably labelled as vegetable oil or sunflower oil, and almost certainly getting a green or amber light on the traffic light labelling system.

Industrial seed oils. This is the category of fats the food industry pushed aggressively as we moved away from natural animal fats in the twentieth century. **Soybean oil. Sunflower oil. Corn oil. Rapeseed oil. Cottonseed oil.** These oils did not exist in the human food supply in any meaningful quantity until the twentieth century. They were made possible by industrial processing, specifically solvent extraction using hexane, a component of petrol, followed by degumming, bleaching, deodorising, and chemical treatment to make a product that is stable enough to sit on a shelf.

These oils are extraordinarily high in linoleic acid, an omega-6 polyunsaturated fatty acid. This matters because polyunsaturated fatty acids are chemically unstable. They have multiple double bonds

in their molecular structure, and those double bonds are highly vulnerable to oxidation.

When you heat these oils, particularly to cooking temperatures, the oxidation accelerates dramatically. The oils break down and produce toxic compounds including 4-hydroxynonenal (4-HNE), malondialdehyde, and acrolein. These aldehydes are cytotoxic. They damage DNA. They contribute to oxidised LDL formation. They are not substances the human body was designed to encounter, certainly not in the quantities that result from daily cooking with seed oils.

Here is a statistic that I find genuinely alarming. Analysis of human fat tissue has shown that the linoleic acid content of body fat increased by 136 per cent between 1959 and 2008. The composition of human body fat has changed dramatically within a single lifetime. That increase tracks almost perfectly with the rise in seed oil consumption over the same period. **We are literally incorporating these oils into our own tissues.**

And yet the traffic light system gives sunflower oil amber. Extra virgin olive oil, a fat consumed by Mediterranean populations for thousands of years with exceptionally well-documented health benefits, gets red. Cold-pressed rapeseed oil, which at least contains some omega-3 alongside its omega-6, gets amber. Meanwhile, the oils that are producing toxic aldehydes in your kitchen every time you fry something get a gentler rating than an avocado.

The omega-6 to omega-3 ratio matters too. In the human diet our ancestors evolved eating, this ratio was somewhere between 1:1 and 4:1. In the modern Western diet, driven by ubiquitous seed oil consumption, the ratio is estimated to be between 15:1 and 20:1. This chronic excess of omega-6 relative to omega-3 promotes inflammation at a systemic level. Both types of fatty acids compete for the same enzymes in your body. When omega-6 dominates, it wins. And omega-6 metabolites tend to be pro-inflammatory, while omega-3 metabolites tend to be anti-inflammatory.

Then there are trans fats. Artificial trans fats, created by partial hydrogenation of vegetable oils to make margarine and shortening,

are now widely banned after overwhelming evidence of their harm. They raise LDL, lower HDL, increase inflammation, and are clearly linked to cardiovascular disease. The fact that these were the very products pushed as healthy alternatives to butter for decades is one of the most damning episodes in the history of nutritional science.

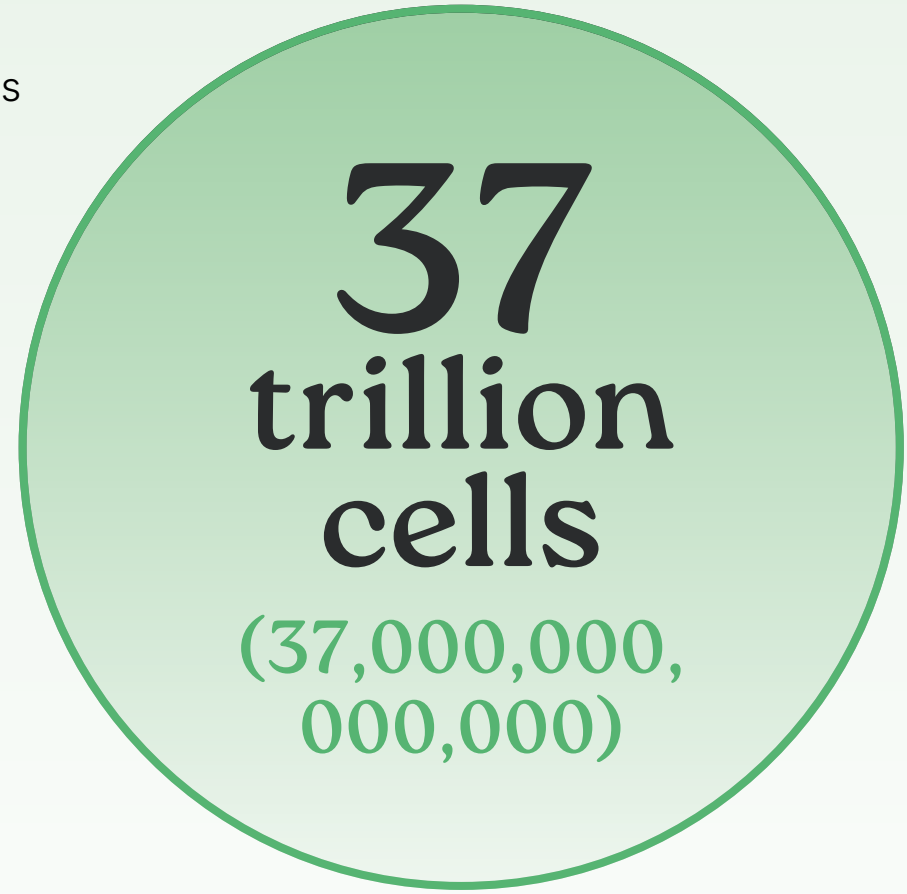
What should you cook with instead? Butter, ghee, coconut oil, beef tallow, and extra virgin olive oil for lower heat cooking. These are stable fats that do not produce toxic aldehydes at cooking temperatures. These are the fats humans have been eating for most of our evolutionary history. These are the fats getting punished by the traffic light system.

The system is not just wrong. It is upside down.

Chapter Five:
The Fats We Should Love

Fat is not your enemy. Fat is not even neutral. Fat is essential. Let me tell you what I mean by that, because essential has a specific meaning in nutrition: it means your body cannot make it. You have to eat it. And if you do not get enough of it, things go wrong at a fundamental level.

Every single cell membrane in your body is made from fat. You have approximately 37 trillion cells. Each one has a membrane built primarily from phospholipids, which are fat molecules. The quality of that membrane, its fluidity, its permeability, its ability to allow nutrients in and waste out, depends on the quality of the fat you eat. A membrane built from stable, natural fats functions very differently from one built from oxidised, processed fats.



Your brain is approximately 60 per cent fat by dry weight. The myelin sheath that insulates your nerve fibres and

allows signals to travel at speed is made from fat. DHA, a long-chain omega-3 fatty acid, is the most abundant fatty acid in the brain and is critical for neurological function, memory, and mood regulation. You cannot build a functional brain without dietary fat.

Fat is also the only way you can absorb vitamins A, D, E, and K. These are called fat-soluble vitamins for a reason: they require fat to be dissolved, transported, and absorbed. A low-fat diet does not just starve you of fat. It deprives you of four essential vitamins. Vitamin D deficiency alone, which is epidemic in the UK given our climate and our low-fat dietary advice, has been linked to increased risk of multiple sclerosis, type 1 diabetes, cardiovascular disease, depression, and immune dysfunction.

Cholesterol deserves a special mention because it has been so thoroughly demonised. Cholesterol is not a poison. It is a biological essential. Every cell in your body makes cholesterol and uses it to maintain membrane integrity. Your liver makes cholesterol. Your adrenal glands use cholesterol to make cortisol, adrenaline, and aldosterone. Your sex organs use cholesterol to make oestrogen, testosterone, and progesterone. Your skin uses cholesterol to make vitamin D when exposed to sunlight. Your liver uses cholesterol to make bile, without which you cannot digest fat at all.

The idea that eating cholesterol raises your blood cholesterol was also largely wrong. The liver tightly regulates cholesterol production based on dietary intake: if you eat more, it produces less. If you eat less, it produces more. For most people, dietary cholesterol has a relatively modest effect on blood cholesterol, and the evidence that blood cholesterol itself is a reliable predictor of cardiovascular risk is considerably weaker than we were led to believe.

Now let me walk you through the fats worth prioritising.

Saturated Fat

Saturated fat has been the most unfairly maligned macronutrient in dietary history. Butter, cheese, red meat, full-fat dairy, coconut oil, eggs: all demonised on the basis of evidence that has not held up under scrutiny. The meta-analyses that looked at this most rigorously have consistently failed to find a strong relationship

between saturated fat intake and cardiovascular mortality in healthy populations. When you replace saturated fat with refined carbohydrates, as the dietary guidelines effectively recommended, outcomes get worse, not better.

Saturated fat raises LDL cholesterol, but as we discussed in the previous chapter, it preferentially raises the large buoyant type that is not meaningfully associated with arterial damage. It also raises HDL, the lipoprotein associated with cardiovascular protection. The net effect on cardiovascular risk is, at best, neutral, and may well be beneficial depending on what it is replacing.

Monounsaturated Fat

This is where the evidence is most consistently positive. Extra virgin olive oil is the star of this category. The Mediterranean diet, which is high in olive oil, has some of the most robust evidence in nutritional science behind it, including the PREDIMED trial, which showed a significant reduction in cardiovascular events compared to a low-fat diet. Avocados and most nuts are also high in monounsaturated fat. These are stable fats, less vulnerable to oxidation than polyunsaturated fats, and associated with reduced inflammation and improved cardiovascular markers across multiple studies.

Omega-3 Fatty Acids

Omega-3 fatty acids, particularly EPA and DHA found in oily fish, and ALA found in walnuts, flaxseed, and chia seeds, are genuinely anti-inflammatory. They counterbalance the pro-inflammatory effects of excess omega-6. DHA is critical for brain health and cognitive function. EPA has particularly strong evidence for reducing triglycerides and supporting cardiovascular health. The conversion of plant-based ALA to the active forms EPA and DHA is inefficient in the human body, which is why oily fish is such a valuable food.

Salmon, mackerel, sardines, herring, anchovies: these are some of the most nutrient-dense foods available. They contain omega-3, vitamin D, vitamin B12, selenium, and high-quality protein in a single package. In the UK, where oily fish consumption is far below what it should be and vitamin D deficiency is widespread, these foods should be dietary staples.

The bottom line on fat is simple. The human body requires fat, specifically the right fats, to function. Fear of fat has made us sicker. It drove us toward refined carbohydrates and seed oils, both of which have caused far more metabolic damage than any natural fat ever could. Eat butter. Eat olive oil. Eat avocados and eggs and full-fat yoghurt and oily fish and nuts and cheese. Avoid the industrially processed seed oils that are genuinely doing you harm.

Your body has been waiting for permission to eat this way. Consider it given.

Part Two:
Fibre

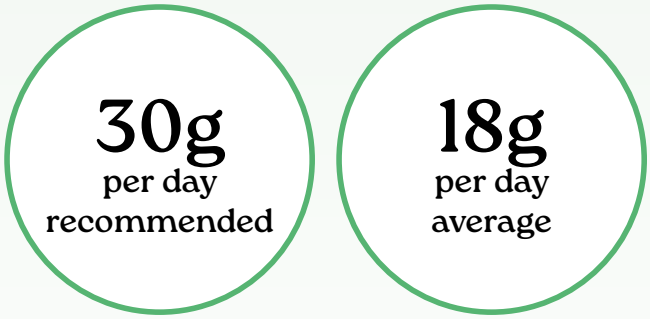
The substance we stripped from our food supply without knowing what we were throwing away.

Chapter Six:
The Missing Ingredient

If I had to identify the single most consequential thing that happened to the human food supply in the twentieth century, it would not be the rise of sugar, though that has been catastrophic. It would not be the shift to seed oils, though that has been deeply damaging. It would be the systematic removal of dietary fibre from the foods we eat every day.

This was not a conspiracy, exactly. It was an economic and technological process that happened incrementally, driven by the demands of industrial food production, extended shelf life, and the preferences of a market that had been taught to associate white, refined, smooth food with quality and status. Wholegrain bread gave way to white bread. Brown rice gave way to white rice. Whole oats became instant oats. Real vegetables became vegetable-flavoured products with the fibre extracted. The fibre went in the bin, and what remained was sold as progress.

The numbers tell the story. Our Palaeolithic ancestors are estimated to have consumed somewhere between 50 and 100 grams of dietary fibre per day. The Hadza people of Tanzania, one of the last remaining hunter-gatherer communities, eat over 100 grams of fibre daily.



The current UK recommendation is 30 grams per day. The UK average actual intake is around 18 grams.

We are getting less than two thirds of what public health authorities recommend, and roughly one fifth of what the human gut evolved consuming.

This matters because the gut microbiome, the community of bacteria that lives in your large intestine, runs on fibre. It is the primary fuel source. When fibre intake falls, the bacteria that depend on it starve. The community collapses. Diversity drops. And as diversity drops, so does the production of every beneficial substance those bacteria were making: butyrate, propionate, acetate, serotonin precursors, immune-modulating signals, and compounds that maintain the integrity of the gut lining itself.

As Professor Robert Lustig of the University of California puts it: **if you do not feed your bacteria, they feed on you. Starved of dietary fibre, gut bacteria turn to the next available food source: the mucus layer that coats and protects your gut wall. As that mucus layer degrades, the barrier between your gut contents and your bloodstream weakens. Gut permeability increases. Things get through that should not. The immune system fires. Inflammation becomes the baseline state of your body.**

I want to talk about what we actually lost when we lost fibre, beyond the vague idea that it keeps you regular. Because the fibre story is one of the most extraordinary stories in nutritional science, and most people know almost none of it.

Soluble Fibre

Soluble fibre dissolves in water and forms a gel. Glucomannan, pectin, beta-glucan from oats, and psyllium husk are all examples. When you eat soluble fibre, it forms a viscous matrix in your stomach and small intestine that slows everything down. Gastric emptying slows. Nutrients are released more gradually. Blood sugar rises more gently. Satiety signals go up. The gel physically coats the gut wall and acts as a barrier, slowing the absorption of glucose and fructose into the bloodstream.

Soluble fibre also binds bile acids in the small intestine. Bile acids are made by the liver from cholesterol and released to aid fat digestion. When fibre binds them and carries

them out of the body in stool, the liver has to make more. To do so, it draws on circulating cholesterol. This is the primary mechanism by which soluble fibre lowers LDL cholesterol, a mechanism so well established that EFSA has approved a specific health claim for glucomannan.

Insoluble Fibre

Insoluble fibre does not dissolve. It adds bulk to stool, draws water into the colon, and physically stimulates the gut wall, speeding transit time through the large intestine. This is what most people think of when they think of fibre, the roughage, the stuff that stops you getting constipated. And yes, it does that. But calling it roughage is like calling a smartphone a calculator. Technically accurate, wildly incomplete.

Insoluble fibre also feeds different bacterial species from soluble fibre, contributing to a broader and more diverse microbiome. Cellulose, lignin, and hemicellulose are the main forms. Vegetables, wholegrains, nuts, and seeds are the primary sources.

The human gut works best when it gets both types. Soluble fibre for the upper gut: the gel, the blood sugar braking, the bile acid binding, the satiety. Insoluble fibre for the lower gut: the bulk, the transit, the mechanical stimulation, the feeding of a wider bacterial population. Real whole plants deliver both. Processed food delivers neither.

Chapter Seven:
What Fibre Actually Does

I want to tell you about a set of receptors in your gut wall that most people have never heard of, and why understanding them changes the way you think about food entirely.

Deep in the lining of your small intestine and throughout your colon are specialised cells called enteroendocrine cells. These cells make up only about one per cent of the cells lining your gut, but they are responsible for producing more than twenty different hormones that regulate almost everything: appetite, digestion, glucose metabolism, and the signals your gut sends to your brain.

Among the most important of these enteroendocrine cells are L cells. L cells produce GLP-1 (glucagon-like peptide-1) and PYY (peptide YY), two of the most powerful satiety hormones in the human body. When L cells release GLP-1, it slows gastric emptying, reduces appetite, and creates a powerful sense of fullness. When PYY is released, it travels to the hypothalamus in the brain and suppresses hunger.

These are the hormones that Ozempic and its siblings mimic. The pharmaceutical industry has made billions of pounds from drugs that activate the GLP-1 pathway. And fibre activates the same pathway, through the same receptors, naturally.

Here is how.

L cells have receptor proteins on their surface called FFAR2 and FFAR3, free fatty acid receptors 2 and 3. These receptors are specifically designed to respond to short chain fatty acids: butyrate, propionate, and acetate. When your gut bacteria ferment dietary fibre, these short chain fatty acids

are the end product. They bind to FFAR2 and FFAR3, triggering L cells to release GLP-1 and PYY.

This is the chain: you eat fibre, gut bacteria ferment it, short chain fatty acids are produced, FFAR2 and FFAR3 are activated, L cells release GLP-1 and PYY, you feel full, you eat less, your blood sugar is regulated.

Nature designed this system. It has been operating in humans for hundreds of thousands of years. We did not need a pharmaceutical company to create it. We needed to keep eating fibre.

But the action of fibre on appetite and blood sugar does not only happen at the level of gut hormones. It also happens mechanically, much faster, through the gel it forms.

Soluble fibre, particularly glucomannan, forms a thick gel in the stomach that physically distends the stomach wall. This activates mechanoreceptors, specifically piezo channels, pressure-sensitive receptors that fire when the gut wall is stretched. These signals travel rapidly to the brain and register as fullness. This mechanical satiety signal arrives within minutes of eating, long before any hormonal signal has had time to take effect.

Robert Lustig has a useful model for thinking about this. Soluble fibre forms a lattice structure across the intestinal wall. This lattice slows the absorption of glucose, fructose, and fat into the bloodstream. Insoluble fibre plugs the gaps in that lattice, making it a more complete and effective barrier. Together, they create what Lustig calls a wall of protection between what you eat and what your bloodstream receives.

A whole apple contains both **pectin (soluble)** and **cellulose (insoluble)**. The fibre matrix it creates in your gut dramatically slows the absorption of the natural sugars it contains. Apple juice, which has had all the fibre removed, delivers those same sugars with no matrix, no barrier, no protection. The blood sugar response is entirely different, even though the sugar content is similar. The fibre is not a footnote to the apple. The fibre is what makes the apple a food rather than a sugar delivery mechanism.

The evidence for fibre and disease prevention is not marginal. The Lancet published a comprehensive meta-analysis in 2019, the

Reynolds et al. analysis, covering 185 prospective studies and 58 clinical trials involving over 135 million person-years of data. The findings were unambiguous. People eating the most dietary fibre had significantly lower rates of heart disease, stroke, type 2 diabetes, colorectal cancer, breast cancer and all-cause mortality. The dose-response was clear: more fibre, better outcomes. There was no upper limit above which benefits plateaued.

This is among the most comprehensive bodies of evidence in nutritional science. The effect sizes are substantial. And yet the average British adult is eating 18 grams of fibre per day instead of the recommended 30, let alone the 50 to 100 grams our gut was designed to receive.

Chapter Eight:
The Resistant Starch Revolution

If I asked you to describe resistant starch, you might struggle. It is not quite fibre and it is not quite starch. It sits between the two, and that in-between status is precisely what makes it so remarkable.

Here is the simplest way to understand it.

This is different from traditional fibre, which was never digestible in the first place. Your body never had the enzymes for cellulose or inulin. Resistant starch starts as ordinary starch that your body could theoretically digest. The structure of the molecule is what puts it out of reach.

When resistant starch arrives in your colon, your gut bacteria get to work. And here is where it becomes genuinely exciting. While most fermentable fibres produce a mix of short chain fatty acids, resistant starch is particularly good at producing butyrate. This matters enormously because butyrate is the primary fuel source for colonocytes, the cells that line your colon. Without adequate butyrate, colonocytes are essentially running on empty. The gut lining weakens. Permeability increases. Inflammation follows.

Resistant starch also specifically feeds a bacterial species called Akkermansia muciniphila. Akkermansia has become one of the most studied bacteria in metabolic health research over the past decade, and for good reason. It maintains the mucus layer of the gut lining, supports insulin sensitivity, and is found in significantly lower quantities in people with obesity, type 2 diabetes, and metabolic syndrome.

Low Akkermansia is emerging as a biomarker for poor metabolic health. Resistant starch is one of the best dietary tools for feeding it.

Where do you find resistant starch in food?

Green bananas are one of the richest natural sources. Unripe bananas contain predominantly resistant starch. As the banana ripens, an enzymatic process converts that resistant starch into regular digestible sugar, which is why a ripe banana tastes sweet and a green one does not.

Raw potato starch is another excellent source. Critically, it must be raw. The moment you cook it, the starch gelatinises and becomes digestible. But raw potato starch stirred into cold water or a smoothie delivers resistant starch with almost no digestible carbohydrate. This is why raw potato starch behaves very differently in the body from a cooked potato.

Cooked and cooled starches are perhaps the most surprising and democratic source. When you cook starchy foods and then cool them, a process called retrogradation occurs. The starch molecules rearrange themselves into a tighter, more crystalline structure that resists digestion. Cold potato salad contains significantly more resistant starch than a hot baked potato. Cooked and cooled rice is a better butyrate source than freshly cooked rice. Even reheating the cooled food retains some of the resistant starch content, though eating it cold maximises the effect.

I want to be clear about the timeline. Resistant starch takes considerably longer than soluble fibre to produce its effects. It takes six to eight hours for food to travel from your mouth to your colon. Fermentation then takes additional hours. The FFAR2 and FFAR3 signal from resistant starch fermentation may take twelve to twenty-four hours to fully register. This is not a fast appetite control tool. It is a chronic background signal. People who consistently eat resistant starch and soluble fibre have persistently higher butyrate levels, a stronger gut lining, better metabolic health over time.

Think of it like compound interest. Each day you eat resistant starch and fibre; you are making a deposit. The benefits are not always felt meal to meal but accumulate meaningfully

over weeks and months. This is precisely why resistant starch and soluble fibre work so beautifully together: soluble fibre does the fast work, the immediate gel, the blood sugar blunting, the quick satiety signal. Resistant starch does the slow work, the butyrate production, the Akkermansia feeding, the long-term gut lining maintenance. They are operating on completely different timescales and doing completely different jobs.

Chapter Nine:
Fibre and the Brain

Here is something that upends almost everything we think we know about mental health. Ninety percent of your serotonin is not in your brain. It is in your gut

Serotonin is the neurotransmitter most associated with mood, wellbeing, and emotional stability. It is the target of the most commonly prescribed class of antidepressants, SSRIs, which work by keeping serotonin in circulation longer. And the vast majority of it is produced not in the brain but by enteroendocrine cells in the gut wall, specifically cells called enterochromaffin cells that line the intestinal epithelium.

Why does this matter for fibre?
Because the production of gut serotonin is influenced by your gut bacteria, and your gut bacteria are fed by dietary fibre.

The chain works like this. When gut bacteria ferment dietary fibre, they produce short chain fatty acids. Those short chain fatty acids interact with enterochromaffin cells and stimulate the production and release of serotonin. They also produce tryptophan metabolites: tryptophan is the amino acid precursor to serotonin, and gut bacteria play a key role in how tryptophan is metabolised and how much of it is available for serotonin synthesis. A depleted microbiome means disrupted serotonin metabolism. That disruption has measurable effects on mood.

The research connecting dietary fibre intake and mood in women is striking. Women who eat the most dietary fibre consistently report significantly lower rates of anxiety and depression than those eating the least. Women are twice as likely as men to experience

anxiety and depression, and part of the reason may be hormonal: oestrogen levels directly influence the composition of the gut microbiome. Hormonal fluctuations during the menstrual cycle, perimenopause, and menopause all affect the bacteria responsible for mood regulation. Feeding those bacteria with fibre is not optional for women's mental health. It is foundational.

The vagus nerve is the communication superhighway of the gut-brain axis. It runs from the brainstem all the way down to the colon, carrying signals in both directions. The majority of signals, roughly 80 per cent, actually travel upward from the gut to the brain, not the other way around. The gut is sending more information to the brain than the brain is sending to the gut. When the microbiome is healthy and well-fed with fibre, the messages it sends up the vagus nerve are calming and regulatory. When it is depleted, the signals change.

There is a brain side to this story as well. The brain has its own immune cells, called microglia. In a healthy brain, microglia exist in what researchers describe as M2 mode: calm, maintained, conducting routine surveillance and repair. In an inflamed brain, microglia shift to M1 mode: activated, aggressive, releasing inflammatory cytokines that damage neuronal connections and have been linked to depression, cognitive decline, and neurodegenerative disease.

Butyrate, produced by gut bacteria fermenting resistant starch and other fermentable fibres, crosses the blood-brain barrier. In the brain, butyrate has anti-inflammatory properties and has been shown in animal studies to shift microglia from M1 (inflammatory) toward M2 (maintenance) mode. Human studies on this are still developing, but the mechanistic pathway is established. The butyrate your gut bacteria make from fibre is doing work in your brain.

This is a profound shift in how we need to think about mental health. I am not suggesting that fibre alone prevents or treats depression. The causes of mental health conditions are complex and multifactorial. But the idea that what we eat has no bearing on how we feel is demonstrably wrong. The gut produces the precursors to the neurotransmitters that regulate

mood. Gut bacteria regulate the production of those precursors. Fibre feeds the gut bacteria. The chain is real. The science is solid. And it is being almost entirely ignored by the mainstream conversation about mental health.

Eating more fibre will not cure depression. But a brain running on a gut that is chronically depleted of fibre and has a dysbiotic, inflamed microbiome is a brain working against itself. Addressing the gut environment is one of the most underutilised tools in mental health.

Part Three:
Flora

The inner ecosystem that runs almost everything, and what we did to destroy it.

Chapter Ten:
Your Inner Ecosystem

You are not as human as you think you are.

1, 2 skip a few...
38 trillion

In terms of cell count, your body contains approximately **30 to 37 trillion human cells**. It also contains approximately **38 trillion microbial cells**, most of them bacteria living in your large intestine. The numbers are roughly equal, with microbial cells winning by a narrow margin depending on the measurement. And your microbial community carries approximately **3.3 million unique genes**, compared to the 23,000 genes in the human genome.

The gut microbiome, that community of bacteria, fungi, viruses, and other microorganisms, is now understood to be one of the most important regulators of human health. It influences metabolism, immune function, inflammation, mental health, hormonal balance, the risk of cancer, cardiovascular disease, neurodegenerative disease, and the response to infections. In the last two decades, the science of the microbiome has produced one paradigm-shifting finding after another.

The most important single metric of microbiome health is diversity. Not any particular bacterium, not a specific ratio, but the sheer breadth and variety of species present. A diverse microbiome is a resilient microbiome, one that can perform a wider range of functions, maintain balance when challenged, and resist colonisation by harmful pathogens. A depleted microbiome, dominated by a small number of species, is vulnerable, functionally limited, and associated with almost every chronic disease that defines modern ill health.

The Hadza people of Tanzania offer one of the most illuminating comparisons. The Hadza are one of the last remaining hunter-gatherer communities, and they have been studied extensively by microbiome researchers because they represent something close to the pre-industrial human gut. The Hadza microbiome contains bacterial species that are essentially extinct in Western populations. Their diversity levels are dramatically higher than those of people eating modern Western diets. They eat over 100 grams of fibre per day, sourced from tubers, berries, honey, and wild plants. They experience almost none of the chronic diseases that are epidemic in the industrialised world.

This is not romanticising primitive life. It is evidence. The human microbiome evolved over hundreds of thousands of years in the context of high-fibre, highly diverse plant-food diets. We have changed that diet radically in the space of a few generations, and the microbiome has not had time to adapt.

What depletes the microbiome?

Antibiotics are the most dramatic cause. A single course of broad-spectrum antibiotics can eliminate up to 30 per cent of gut bacterial species, with some of them never returning. This is sometimes necessary, but the long-term consequences for microbiome diversity are significant and have been systematically underappreciated by medicine. Children who receive multiple courses of antibiotics in their first years of life show measurable differences in microbiome composition that persist into adulthood and are associated with higher rates of obesity, asthma, and autoimmune conditions.

Ultra-processed food is a more insidious threat because its effects accumulate slowly over years. These foods contain virtually no fibre, starving the bacteria that depend on it. They also contain emulsifiers, additives used to improve texture and shelf life, that have been shown to directly damage the gut lining and alter the microbiome composition. Professor Robert Lustig puts it bluntly: emulsifiers are detergents. They are chemically similar to washing-up liquid, and what detergent does to grease on a pan, emulsifiers can do to the protective mucus layer of your gut wall. Polysorbate 80 and carboxymethylcellulose, two of the most commonly used emulsifiers, have been shown in animal studies to increase gut permeability and trigger low-grade inflammation even at doses approved for food use. You are quite literally eating a substance designed to dissolve protective barriers, and your gut lining is paying the price.

Artificial sweeteners have been shown in multiple studies to alter the gut microbiome, potentially in ways that paradoxically impair glucose metabolism, the very thing they were meant to help. The evidence is still developing, but the assumption that they are inert has not held up.

Chronic stress alters the microbiome through its effects on gut motility, mucus production, and immune function. Poor sleep disrupts the circadian rhythm of gut bacteria, many of which have their own daily cycles. Even the method of birth matters: infants born by caesarean section, who are not exposed to the mother's vaginal microbiome during delivery, and those who are formula-fed rather than breastfed, start life with different microbiome profiles that have measurable effects on immune development and disease risk.

The modern world has been systematically hostile to the human microbiome in ways we did not fully understand until very recently. The good news is that the microbiome is also remarkably responsive to dietary change. Studies show meaningful shifts in microbiome composition within days of changing diet. The system is damaged but it is not static. Feed it properly and it responds.

Chapter Eleven:
Alessio Fasano and the Leaky Gut

Alessio Fasano is one of the most important scientists working on human health today. A gastroenterologist and researcher based at Harvard Medical School, Fasano spent decades studying coeliac disease before making a discovery that has changed how we understand autoimmune conditions, inflammatory disease, and the gut's role in systemic health.

He discovered zonulin.

Zonulin is a protein produced in the gut that regulates the tight junctions between enterocytes, the cells lining the small intestine. These tight junctions are exactly what their name suggests: extremely tight connections between adjacent cells that control what passes through the gut wall and what does not. In a healthy gut, these junctions are selective gatekeepers. They allow nutrients, vitamins, and water through and keep everything else out.

When zonulin is elevated, tight junctions open. The gut becomes more permeable. Things that should stay in the gut can pass into the bloodstream: bacterial fragments, lipopolysaccharides (the outer membrane components of certain bacteria), incompletely digested food particles, toxins. When these substances enter the bloodstream, the immune system does exactly what it is supposed to do: it mounts a response. But when that response is chronic, driven by a continuously permeable gut delivering a constant trickle of

foreign material into the circulation, it becomes systemic inflammation. And systemic inflammation is the common thread running through virtually every chronic disease we know.

Fasano proposed what he calls the three-legged stool model of autoimmune disease. For autoimmune disease to develop, he argues, you need three things: genetic predisposition, an environmental trigger, and intestinal permeability. Remove any one of the three legs and the stool falls. Without gut permeability, even a person with the genetic predisposition and the environmental trigger may not develop the disease. Gut permeability is not just a consequence of poor health; it may be a prerequisite for the development of many autoimmune and inflammatory conditions.

The environmental triggers that elevate zonulin and open the tight junctions include gluten (particularly in genetically susceptible individuals), certain gut bacteria, infections, non-steroidal anti-inflammatory drugs, and prolonged stress. But there is another trigger that is chronically underappreciated: a depleted microbiome starved of dietary fibre.

This is where Lustig's observation becomes critical. **If you do not feed your bacteria, they feed on you.** The mucus layer that coats the gut wall and sits between the gut contents and the epithelial cells is produced by goblet cells in the gut lining. It is the first physical barrier between your gut bacteria and your bloodstream. This mucus layer is built from large proteins called mucins, which are heavily laden with complex sugar chains called glycans. And here is the key point: dietary fibre is also built from networks of complex sugars, polysaccharides from plant cell walls. The gut bacteria that ferment fibre possess exactly the enzymatic tools needed to break apart polysaccharide chains. When fibre disappears from the diet, those same bacteria do not starve quietly. They switch targets. The sugar chains coating your mucus layer look close enough to dinner, and some species make the switch, degrading the mucus and exposing the epithelium beneath.

One bacterial species is particularly worth knowing: **Akkermansia muciniphila.** The name tells the whole story.

Akkermansia after Dr Akkermans, the Dutch microbial ecologist who identified it. Muciniphila from the Latin mucin, the primary protein in gut mucus, and the Greek philus, meaning loving. Mucin-loving. When Akkermansia is present at healthy levels, fed by resistant starch and dietary fibre, it maintains the mucus layer. When the microbiome is depleted and fibre intake is low, Akkermansia levels fall, the mucus layer thins, and what Fasano's model predicts follows: the tight junctions weaken, permeability increases, and the immune system begins firing at things it was never meant to see.

A landmark study by Desai and colleagues published in Cell in 2016 demonstrated this in compelling fashion. Mice fed a fibre-free diet showed dramatic degradation of the gut mucus layer, with gut bacteria consuming the mucus as an alternative energy source. The degraded mucus layer left the epithelium exposed and vulnerable, increasing susceptibility to infection and inflammation. Restore the fibre and the mucus layer recovers. Remove it again and the degradation returns.

The clinical implications of Fasano's work extend across a remarkable range of conditions. Increased gut permeability has been documented in type 1 diabetes, multiple sclerosis, rheumatoid arthritis, lupus, Crohn's disease, ulcerative colitis, coeliac disease, Parkinson's disease, and Alzheimer's disease. This does not mean leaky gut causes all of these conditions. The relationships are complex and still being mapped. But the pattern is consistent enough, and the mechanism plausible enough, that gut barrier integrity has emerged as one of the most important therapeutic targets in modern medicine.

Fasano's work is not fringe science. He is a Harvard professor whose findings have been published in the world's leading medical journals and replicated across multiple research groups. The idea that the gut is a sealed barrier is wrong. The idea that gut health is central to systemic health is right. And the primary tool for maintaining gut barrier integrity is the thing we have been systematically removing from our food: dietary fibre.

Chapter Twelve:
The Microbiome and Chronic Disease

The science of the gut microbiome and chronic disease is moving faster than almost any other area of medicine. What I am about to tell you represents our current best understanding, but I want to be clear: we are at the beginning of this story, not the end. The findings are sometimes preliminary, the mechanisms often complex, and the translation from laboratory findings to clinical practice is still unfolding. What I can tell you is that the pattern emerging from this research is consistent, compelling, and points unmistakably in one direction.

In my previous book I traced a single thread running through most of the chronic diseases that define modern ill health: insulin. Chronically elevated insulin, driven by a diet high in refined carbohydrates and sugar, is the root cause of metabolic syndrome, type 2 diabetes, fatty liver disease, cardiovascular disease, and much of the obesity epidemic. That argument stands. But insulin is not the only thread. The microbiome is a second pathway, distinct from insulin but frequently running alongside it, and in many cases amplifying it. A dysbiotic gut increases systemic inflammation, which worsens insulin resistance. Insulin resistance starves the beneficial bacteria that need a stable metabolic environment to thrive. The two pathways are not separate stories. They are two

sides of the same breakdown. Understanding both gives you a far more complete picture of why the modern body fails, and what to do about it.

The microbiome is not just involved in gut health. It is involved in almost everything.

Obesity

One of the most striking demonstrations of the microbiome's influence on body weight came from faecal microbiota transplant (FMT) experiments. Researchers transplanted the gut bacteria from obese humans into germ-free mice, mice raised in sterile conditions with no microbiome of their own. The mice developed significantly more body fat than mice that received transplants from lean donors, despite eating the same amount of food. The microbiome itself was driving fat storage. When the experiment was reversed, transplanting bacteria from lean individuals into obese ones, metabolic improvements followed. This is not correlation. It is causation, demonstrated in controlled experiments.

Type 2 Diabetes

People with type 2 diabetes consistently show a depleted microbiome with reduced diversity and a shift in bacterial composition away from butyrate-producing species toward pro-inflammatory ones. Lower levels of Faecalibacterium prausnitzii, one of the most abundant and important butyrate producers in a healthy gut, are found repeatedly in metabolic disease. Studies have shown that microbiome composition can predict the development of type 2 diabetes before it develops, suggesting that dysbiosis is not merely a consequence of metabolic disease but potentially a contributor to its development.

Cardiovascular Disease

The microbiome influences cardiovascular health through multiple pathways. One of the most studied involves TMAO (trimethylamine N-oxide). Certain gut bacteria metabolise choline and carnitine, found in red meat, eggs, and fish, into trimethylamine, which the liver converts to TMAO. Elevated blood TMAO levels have been associated with increased cardiovascular risk in some studies, though the picture is complicated and the research is ongoing. More clearly established

is the role of microbiome-derived short chain fatty acids in regulating blood pressure, inflammation, and cholesterol metabolism, all through pathways that improve cardiovascular outcomes when the microbiome is healthy.

Mental Health

The gut-brain axis, which we explored in the previous section from the fibre angle, is increasingly recognised as a genuine bidirectional pathway with major implications for mental health. Studies have found that people with depression have measurably different microbiome profiles from those without. Specific bacterial species have been identified that are consistently lower in depressed populations. FMT experiments in animals have shown that transplanting bacteria from depressed humans induces depressive behaviours in recipient animals. And small human trials of specific probiotic strains have shown modest but real improvements in mood and anxiety measures.

Cancer

The connection between the microbiome and colorectal cancer is now well established. Certain bacterial species, particularly Fusobacterium nucleatum, are found in much higher concentrations in colorectal tumours than in surrounding healthy tissue, and high levels of this bacterium are associated with worse outcomes. The production of butyrate by gut bacteria has anti-cancer properties in the colon, which is one reason high fibre intake is consistently associated with reduced colorectal cancer risk. The microbiome also appears to influence the response to cancer immunotherapy, with patients having certain microbiome profiles responding significantly better to treatment than others.

Alzheimer's Disease

The gut-brain axis is increasingly implicated in neurodegenerative disease. Research has found that people with Alzheimer's disease have different microbiome profiles from cognitively healthy controls. The potential mechanisms include microbiome-driven neuroinflammation via the vagus nerve, the production of amyloid by gut bacteria (amyloid plaques being the hallmark of Alzheimer's pathology), and the role of butyrate and other short chain fatty acids in maintaining the

blood-brain barrier and regulating microglial activity. The research is early but consistent enough to be taken seriously.

My mom was diagnosed with Alzheimer's, which is one of the reasons I have spent so many years researching this field. I cannot tell you that dietary intervention would have changed their trajectory. But I can tell you that the science increasingly points to gut health, inflammation, and the microbiome as factors in neurodegeneration, and that the tools for supporting gut health are available to all of us right now.

Chapter Thirteen:
How to Feed Your Flora

Here is the practical question. If the microbiome is this important, and if its decline is driven primarily by what we eat, then what do we actually do about it? The answer is less complicated than you might expect. The fundamentals are the same things humans have been eating for most of our history, before the food industry decided it knew better.

Dietary Diversity

The single most important dietary variable for microbiome health is not any particular food or supplement. It is diversity. Different bacteria eat different things. A diet that contains a wide variety of plant foods feeds a wide variety of bacterial species and produces a diverse, resilient microbiome. A diet dominated by a narrow range of ultra-processed foods feeds almost nothing and produces a depleted one.

Tim Spector, Professor of Genetic Epidemiology at King's College London and one of the world's leading microbiome researchers, has popularised the goal of eating 30 different plant foods per week. This sounds daunting but becomes surprisingly manageable when you realise that herbs and spices count, that different coloured varieties of the same vegetable count separately, and that a small amount of each plant contributes meaningfully. A handful of mixed seeds, a sprinkle of herbs, a varied salad: diversity accumulates quickly once you start thinking this way.

Polyphenols, the compounds that give plants their colours and flavours, are themselves a major food source for gut

bacteria. Different bacterial species metabolise different polyphenols into different beneficial compounds. Dark berries, red cabbage, red onion, dark chocolate, green tea, extra virgin olive oil, coffee: all are exceptionally rich in polyphenols and all directly feed and diversify the microbiome. The colour of your diet is a rough but useful proxy for its polyphenol content. Eat the rainbow, not because it sounds nice, but because different colours represent different polyphenol classes feeding different bacterial communities.

Fermented Foods

Fermented foods are one of the oldest forms of food preservation and one of the best sources of living beneficial bacteria. Yoghurt, kefir, sauerkraut, kimchi, miso, tempeh, and kombucha all contain live bacterial cultures that contribute to microbiome diversity. A landmark randomised controlled trial by Wastyk and colleagues at Stanford, published in Cell in 2021, compared a high-fibre diet with a high-fermented-food diet over ten weeks. Fermented foods produced faster and more significant increases in microbiome diversity and reductions in inflammatory markers than fibre alone, at least in the short term.

The interpretation matters here. Fibre is the long-term investment. It feeds the bacteria already present and helps them establish and diversify over months and years. Fermented foods are the seeds. They introduce new species and produce rapid short-term improvements in diversity and inflammation. Both are needed. Think of fermented foods as seeding a garden and fibre as the fertiliser that determines whether those seeds take root and flourish.

Prebiotics

Prebiotics are substances that specifically feed beneficial bacteria. Inulin and FOS (fructooligosaccharides) feed Bifidobacteria and Lactobacilli. Resistant starch feeds Faecalibacterium prausnitzii, Roseburia, and Akkermansia. GOS (galactooligosaccharides) in legumes and human breast milk feed early colonisers. The key is to think about variety: different prebiotics feed different bacteria, so consuming a range is more beneficial than high doses of any single one.

What to Avoid

Antibiotics are sometimes medically necessary and should be taken when prescribed for genuine bacterial infections. But they should not be taken for viral infections, where they are ineffective, and their microbiome-disrupting effects should be taken seriously. If you do take a course of antibiotics, a deliberate effort to rebuild microbiome diversity in the weeks and months following is sensible.

Emulsifiers found in ultra-processed foods, particularly polysorbate 80 and carboxymethylcellulose, have been shown to damage the gut lining and alter microbiome composition. These are in products ranging from ice cream to bread to salad dressings. Reading labels and choosing products without long ingredient lists of additives is one of the most impactful things you can do for your microbiome.

Artificial sweeteners are another area of legitimate concern. The evidence is mixed, but multiple studies have shown changes in microbiome composition following consumption of saccharin, sucralose, and aspartame. Whether these changes are meaningful at typical consumption levels is still being debated, but the assumption of complete safety for the microbiome does not appear to be justified.

Chronic stress, poor sleep, and sedentary behaviour all negatively affect microbiome diversity. The gut is not isolated from the rest of your lifestyle. It responds to cortisol, to disrupted circadian rhythms, and to physical inactivity. A comprehensive approach to microbiome health includes sleep, stress management, and movement alongside diet.

Part Four:
Bringing it all together

Why fat, fibre, and flora
are one system, not three
separate topics.

Chapter Fourteen:
Fat, Fibre and Flora as a System

I want to bring this all together, because the real insight of this book is not about fat alone, or fibre alone, or the microbiome alone. It is about how these three things function as a single integrated system, and what happens when any part of that system is disrupted.

Let me start with the most elegant connection of all.

Flora eat fibre and produce fat.

Your gut bacteria ferment dietary fibre and produce short chain fatty acids: butyrate, propionate, and acetate. These are, chemically speaking, fats. They are the fats your inner ecosystem manufactures from the fibre you eat. Butyrate fuels the cells lining your colon. Propionate travels to the liver and influences cholesterol and glucose metabolism. Acetate circulates in the bloodstream and signals to the brain that you are full.

So when you eat fibre, you are not just getting fibre. You are feeding your flora so that they can produce the fats that run almost every aspect of your gut health and contribute to your metabolic health and your brain chemistry. The fibre is the raw material. The flora are the factory. The short chain fatty acids are the product. Cut the fibre and the factory stops producing.

The dietary fat connection runs in the other direction. The type of fat you eat directly influences

which bacteria thrive in your gut. Diets high in saturated fat from whole food sources, meat, dairy, eggs, appear to support different bacterial communities from diets high in industrial seed oils. The polyunsaturated fats in omega-3 rich foods (oily fish, walnuts, flaxseed) have anti-inflammatory effects that extend to the gut environment, supporting the conditions in which beneficial bacteria flourish. Seed oils, high in pro-inflammatory omega-6 linoleic acid, appear to do the opposite.

There is a third connection that runs through bile acids. Bile acids are produced by the liver from cholesterol and released into the small intestine to emulsify dietary fat. Without them, fat digestion is impaired. But the story does not end there. Gut bacteria transform primary bile acids into secondary bile acids, a process that produces a remarkable range of biologically active compounds that influence gene expression in the gut and liver, regulate glucose metabolism, and affect appetite. The type of gut bacteria present determines which secondary bile acids are produced and in what quantities.

Fibre plays a role here too. Soluble fibre binds primary bile acids in the small intestine and carries them out of the body. The liver then synthesises new bile acids from cholesterol to replace them, which is the mechanism by which fibre lowers LDL cholesterol. But the amount and type of fibre consumed also affects how many bile acids reach the colon for bacterial transformation, which in turn affects the composition of secondary bile acids and their downstream effects.

Fat digestion, bile acid production, fibre-mediated bile acid binding, microbial transformation of bile acids, downstream metabolic effects: these are not separate systems. They are one system, with fat, fibre, and flora as the three essential players.

Now consider what the modern Western diet does to this system.

It replaces natural fats with industrial seed oils, which are pro-inflammatory and structurally compromise cell membranes. It removes fibre from the food supply through processing, starving the microbiome of its primary fuel. And in starving the microbiome, it causes the flora to degrade the mucus layer that protects the gut wall, increasing permeability

and triggering chronic immune activation.

Each disruption amplifies the others. Low fibre means fewer short chain fatty acids, which means a weaker gut lining, which means more permeability, which means more inflammation, which means a more disrupted microbiome, which means even fewer short chain fatty acids. Poor quality fats produce an inflammatory environment that suppresses beneficial bacteria, which further reduces short chain fatty acid production, which further weakens the gut lining.

The system has positive feedback loops. When it is running well, each element supports the others. When it is disrupted, each dysfunction makes the others worse. This is why chronic disease so often comes in clusters: metabolic syndrome with cardiovascular disease and type 2 diabetes and fatty liver and depression and autoimmune conditions arriving together, as expressions of a single underlying system breakdown.

Protein matters. I want to say that clearly. Adequate protein intake is essential for muscle maintenance, immune function, enzyme production, and tissue repair. I am not arguing against protein. But protein does not fix a broken gut. Protein does not feed your microbiome. Protein does not produce butyrate or regulate your GLP-1 response or maintain your gut lining or reduce neuroinflammation or support the production of the serotonin precursors your brain depends on.

The health conversation that focuses overwhelmingly on protein is missing three quarters of the picture. Fat, fibre, and flora, understood as the interconnected system they are, may be the most important nutritional framework for preventing and reversing the chronic diseases that are overwhelming modern healthcare.

Chapter Fifteen:
Your Action Plan

I do not believe in rigid programmes. I have tried enough of them myself. What I believe in is understanding, because understanding changes behaviour in ways that willpower never can. If you have read this far, you understand what fat, fibre, and flora actually do. Now let me make it practical.

On Fat

Stop fearing natural fats. Butter, extra virgin olive oil, full-fat dairy, eggs, avocados, oily fish, nuts and seeds, coconut oil: these are foods the human body knows what to do with. They have been part of human diets for hundreds of thousands of years. Eat them without guilt.

Remove industrial seed oils from your kitchen. Check labels. Sunflower oil, vegetable oil, soybean oil, corn oil: these should not be your primary cooking fats. Use butter or ghee for high-heat cooking. Use extra virgin olive oil for lower heat and dressings. These are stable fats that do not oxidise and produce toxic compounds at cooking temperatures.

Eat oily fish at least twice a week. Salmon, mackerel, sardines, herring, anchovies. These are among the most nutritionally complete foods available. If you genuinely cannot stand oily fish, a high-quality omega-3 supplement (triglyceride form, not ethyl ester) is a reasonable alternative, but whole food is always preferable.

Do not worry about your total cholesterol number in isolation. What matters is the particle type, the LDL to HDL ratio, your triglyceride levels, and your insulin sensitivity. If you are concerned about cardiovascular risk, ask your doctor for a full lipid panel

including particle size or ApoB levels, not just a total cholesterol figure.

On Fibre

The target is 30 grams of dietary fibre per day as a minimum. Most people are getting 18 grams. The gap is not insurmountable but it requires intentional effort. Here is how to close it.

Start every meal with a fibre-first approach. Eat your vegetables and salad before the rest of your plate. This simple change means the fibre goes in first, the gel forms first, and the blood sugar braking begins before the carbohydrates arrive. The research on eating order and blood sugar response is consistent and clinically significant.

Eat legumes every day if you can. Lentils, chickpeas, black beans, kidney beans, edamame: these are some of the most fibre-dense, nutrient-rich foods available and they are among the most thoroughly studied foods for metabolic health and longevity. A single serving of lentils provides around 8 grams of fibre. Half a tin of chickpeas provides around 7 grams. They are inexpensive, versatile, and the single biggest dietary change most people can make for their gut health.

Add resistant starch deliberately. Cook rice or potatoes the night before and eat them cold or reheated the following day. Keep green bananas in the house. These changes are small but cumulative.

Eat 30 different plant foods per week. Count herbs and spices. Count different colours of the same vegetable. Count seeds and nuts. Make it a game. The diversity matters as much as the quantity.

On Flora

Eat fermented food often. A portion of natural yoghurt, a tablespoon of sauerkraut, a glass of kefir, a portion of kimchi. These do not need to be large amounts. Consistency matters more than quantity. You are seeding your garden regularly rather than doing one dramatic intervention.

Be thoughtful about antibiotic use. Take them when genuinely necessary and complete the course. But do not push for them when they will not help, and if you do take a course, make a deliberate effort to rebuild microbiome diversity in the weeks following with diverse plant foods and fermented foods.

Read ingredient lists. Ultra-processed food with long lists of additives, emulsifiers, and preservatives is directly hostile to your gut lining and microbiome. You do not need to be perfect, and I am not asking you to be. But choosing minimally processed whole food as your default, rather than as the special occasion, makes an enormous cumulative difference.

Sleep seven to eight hours. Manage stress as best you can. Move your body regularly. These are not the main topic of this book but they are not optional for microbiome health. The gut responds to your whole life, not just your diet.

The Bigger Picture

The health of the nation is not a mystery. We are eating food that was designed for profit rather than for the human body. We removed the fat that cells are built from and replaced it with refined carbohydrates that drive insulin and seed oils that drive inflammation. We stripped the fibre that feeds the ecosystem living in our guts. We filled the spaces left by real food with ultra-processed products that the microbiome cannot ferment and the gut lining cannot tolerate.

The consequences are the chronic disease epidemic we are living through. Not inevitable. Not genetic destiny. Largely preventable, and in many cases reversible, through the seemingly radical act of eating the way human beings actually evolved to eat.

Fat, fibre, and flora are not three separate topics. They are one conversation about what it takes to be genuinely healthy in a food environment that is working against you. Understanding that conversation is the most important thing I can give you.

Now, go and eat something real

A Note on the Science

Throughout this book I have tried to be clear about the quality of the evidence I am citing. The science of nutrition is not perfect. Studies are often observational, confounded by lifestyle factors, and difficult to conduct as randomised controlled trials in the way that drug trials can be. I have tried to distinguish between well-established mechanisms, consistent epidemiological findings, and more preliminary or contested areas of research.

The key studies I have drawn on include the PURE study (Dehghan et al., The Lancet, 2017), the Reynolds et al. meta-analysis on dietary fibre (The Lancet, 2019), the Wastyk et al. fermented food RCT (Cell, 2021), the Desai et al. fiber-deprivation study (Cell, 2016), and the work of Alessio Fasano on intestinal permeability, published across multiple journals including Gut, Annals of the New York Academy of Sciences, and Clinical Reviews in Allergy and Immunology. The Women's Health Initiative dietary modification trial (Howard et al., JAMA, 2006) and the Minnesota Coronary Experiment analysis (Ramsden et al., BMJ, 2016) are key references for the fat chapters.

The science of the gut microbiome is moving rapidly. Some findings that are compelling today may be revised as the field matures. I have tried to present the current best understanding while acknowledging where certainty is limited. The mechanistic science, the physiology of fat digestion, the action of FFAR2 and FFAR3, the role of butyrate in colonocyte health, the tight junction biology: this is well-established physiology. The epidemiological and interventional evidence is strong in most areas, though as always in nutritional science, interpretation requires care.

I am not a doctor or a scientist. I am a businessman who became furious about what has happened to people's health and spent years trying to understand why. I have done my best to represent the science accurately, but please consult healthcare professionals for any personal health decisions, and please do your own reading. The curiosity that led you to this book is your most powerful health tool.

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