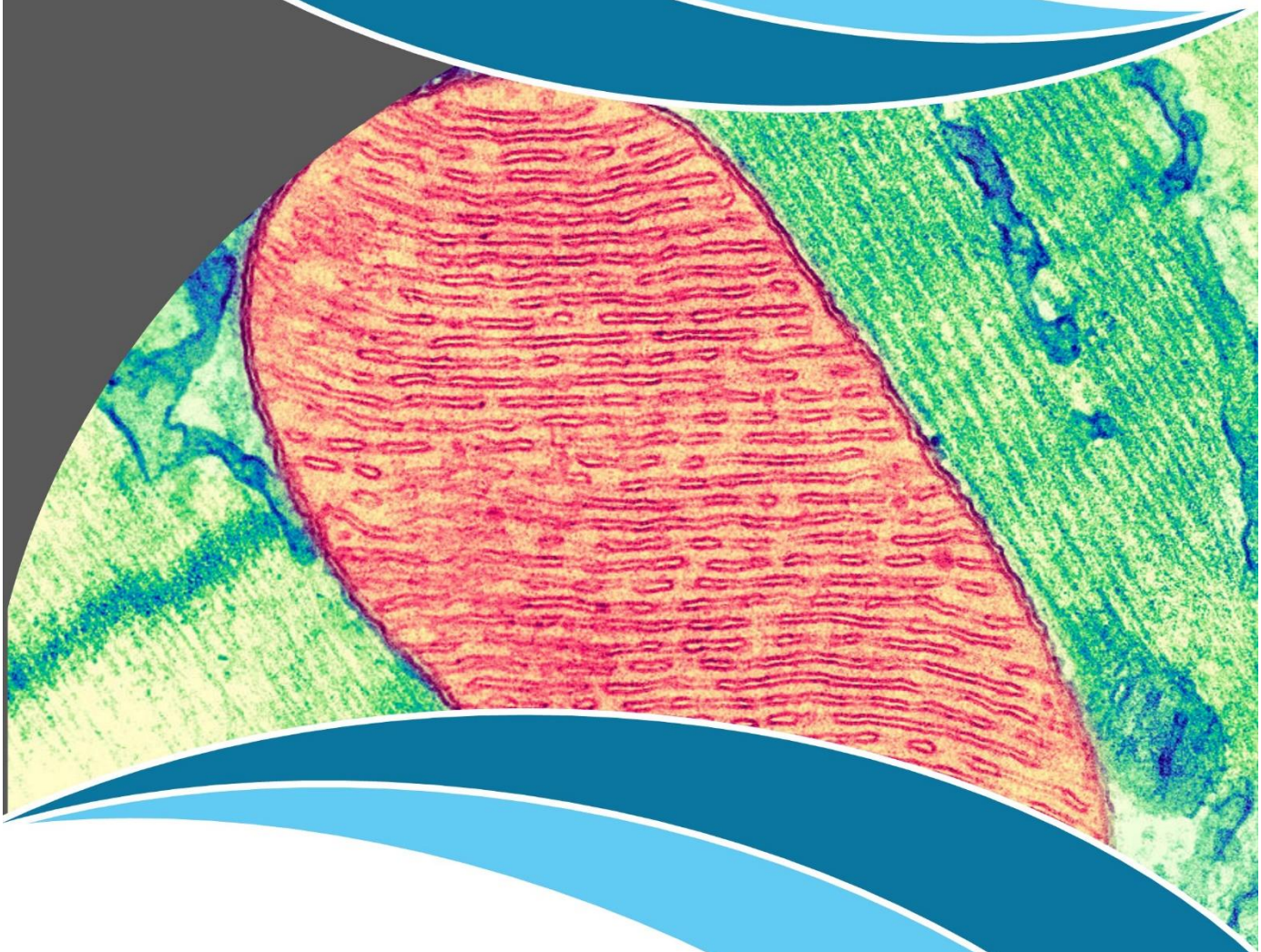
A SMORGASBORD OF EXCEEDINGLY UNUSUAL BEHVIOURS

A Talk on the Metabolic Approach to Cancer



Arden Forest Wellness

Chapter 1

(Slide 1: A Smorgasbord of Exceedingly Unusual Behaviours)

By way of setting the scene for this talk, I know that there are going to be a couple of moments in it that are likely to make you feel like you've suddenly hit a bump in the road. Moments that will make you stop; that will make you ask some pretty fundamental questions.

(Slide 2: Introducing the Metabolic Approach)

The aim of my talk today, introducing the Metabolic Approach to Cancer, is just to give you some food for thought.

(Slide 3: Nick Crawford)

I'm Nick Crawford, an independent quantum biochemist. My independence allows me to be totally candid which would not be the case if I was a doctor or worked in academia or industry.

And why today? Why not last year?

(Slide 4: The Glucose Ketone Index, scientific paper)

On the 14 July 2026, this scientific paper was released that gave us the final piece of the Metabolic Approach to Cancer jigsaw. We will obviously look at this final piece in some detail later.

The publishing of this paper has not been without some controversy, and it was apparently placed in embargo. The reason for the embargo is something that we may well get to talk about later as it extends some of the concepts that relate to cancer, to Type 2 diabetes, coronary heart disease and Alzheimer's.

But before we start, here's the obligatory disclaimers.

(Slide 5: Disclaimer 1)

This talk is intended for educational purposes only and should not be taken as medical advice.

(Slide 6: Disclaimer 2)

There are some cancers for which the metabolic approach is not appropriate.

(Slide 7: DNA Mutation 1)

Let's just have a look at the state-of-the-art.

The body's DNA blueprint comprises thousands of genes, each of which do something different. There are over two hundred that are responsible for various aspects of growth; they are called protooncogenes which can be likened to the accelerator in a car. Normally, when working properly, they just go about their business accelerating and decelerating growth as necessary.

But if one or two or three of these genes become harmfully mutated, it can be like the accelerator pedal getting stuck open. They become fully blown oncogenes. They can produce uncontrolled cell growth and uncontrolled cell division which together is termed uncontrolled cell proliferation. Uncontrolled cell proliferation is also called cancer.

(Slide 8: DNA Mutation 2)

On the other hand, there are over one thousand tumour suppressor genes whose role it is to regulate cell growth and cell division and hence prevent uncontrolled cell proliferation. They can be likened to the brakes on a car, slowing down cell growth. But, if they become harmfully mutated, it can be like the car's breaks suddenly not working.

(Slide 9: DNA Mutation 3)

In addition to oncogenes and tumour suppression genes. there are over 150 genes whose job it is to repair DNA damage. These are sometimes called caretaker genes and can perhaps be likened to the car's service engineer. If this engineer becomes ill and your car misses its services, problems can go undiagnosed and unfixed resulting in a breakdown.

It takes one or two or more harmful mutations from each of the above to start cancer.

(Slide 10: Cancer 1)

There are two hundred distinct types of cancer usually named after the part of the body like lung cancer, breast cancer, prostate cancer etc.

(Slide 11: Cancer 2)

With countless numbers of subtypes and variations.

(Slide 12: Cancer 3)

This means that just about everyone's cancer is different.

(Slide13: Cancer 4)

So, with what looks like an almost limitless number of diseases…

(Slide 14: Cancer 5)

… is it any wonder that progress has been frustratingly slow?

(Slide 15: Richard Nixon)

This is former president of the United States of America, Richard Nixon, who in 1971 declared war on cancer and pledging what today would be $13 billion to have conquered it in 5 years. After all, the USA had just put men on the moon, and they were walking on water.

(Slide 16: Sajid Javid)

51 years later, this was our health secretary, Sajid Javid, similarly declaring war on cancer.

(Slide 17: Total Cancer Deaths Worldwide)

It's a war that we are not winning - worldwide.

(Slide 18: All cancer deaths per year England)

Or in England.

(Slide 19: Otto Heinrich Warburg)

This is Otto Heinrich Warburg; he won the Nobel Prize for medicine in 1931 for discovering how oxygen is used in the conversion of food into energy.

During the late nineteen twenties, he was professor of Biology at the Kaiser Wilhelm Institute in Berlin.

(Slide 20: Cancer graphic)

At the institute, Otto was researching how cancer cells in particular obtain and use energy and what role oxygen plays in all this.

What he found was:

(Slide 21: Cancer cells consume huge amounts of glucose)

That cancer cells consume huge amounts of glucose, 50-200 times as much as normal cells. Even considering the fact that cancer cells are multiplying rapidly, this is an outrageously large amount. This is the big one. We will come back to this regularly.

Incidentally, this fact has never been disputed; everyone that has researched cancer in the meantime has identified this very same peculiarity.

(Slide 22: Cancer's acid microenvironment)

He also found that the cancer tumour microenvironment was acidic, which is strange, as by and large the human body is slightly alkaline.

(Slide 23: Lactic Acid)

He also established that one of the acids was lactic acid: the same acid which gives that burning sensation in your muscles if you are working too hard for your lungs to keep up.

(Slide 24: Succinic Acid)

There was an acid that he missed – succinic acid. We will meet this again later.

(Slide 25: Oxygen)

He also found that cancer cells, very unlike normal cells, don't seem to use or need the oxygen from the air that we breathe. The oxygen seemed to hang around causing and being part of all kinds of mischief.

(Slide 26: Oil droplets)

As if all this wasn't enough, he also found that the cancer cells contained rather more oil and fat droplets than you'd expect when comparing them to normal cells.

(Slide 27: A smorgasbord of exceedingly unusual behaviours)

What a smorgasbord of exceedingly unusual behaviours!

(Slide 28: The Biology of Cancer book cover)

This book, The Biology of Cancer, by Robert Weinberg, is the most widely read and definitive text on cancer biology. Robert Weinberg was a pioneer in cancer research and was instrumental in finding the first oncogenes and tumour suppressant genes.

(Slide 29: The Biology of Cancer book page 53)

On page 53, he calls all Warburg's observations, 'Exceedingly unusual behaviour'. Which he called The Warburg Effect.

Robert, however, does not mention these ever again in the 822 pages that follow. Which really is exceedingly unusual behaviour.

(Slide 30: Crick & Watson in the limelight and Warburg on a stamp)

As time went by, with Crick and Watson's discovery of the structure of DNA and the thinking that cancer was the result of mutations of this DNA, poor Warburg and his effect was lost in time and consigned to a postage stamp.

Chapter 2

(Slide 31: Building blocks and energy)

I just want to talk for a while about what we eat in order to survive.

At the most fundamental level, any organism needs two things to live:

The stuff with which to make its physical self – the building blocks, and

The energy to put these building blocks together, keep them in good repair and to make the living thing operate and do something useful.

We obtain both the building blocks and the energy from the food that we eat, the fluid that we drink and the air that we breathe.

(Slide 32: Carbs, protein and fat)

I want to concentrate on the macronutrients of food which are carbohydrates, proteins and fats. Although important, we will not be concerning ourselves with the micronutrients of vitamins and minerals.

(Slide 33: Carbs, protein and fat + oxygen and water)

Of course, we need oxygen and water. We'll come on to those in a moment.

(Slide 34: Hidden macronutrient - fibre)

Oh, and then there's the hidden macronutrient, the all-important dietary fibre.

So, from which macronutrients do we get the first element: the building blocks?

(Slide 35: Protein and fat)

That's easy, protein and fat. But unless we are growing or bodybuilding, we generally don't need much of these, just enough to cover the requirements for repair and maintenance. We won't be dealing much with this element, the building blocks, today.

So, moving on to the second element, from which macronutrients do we get the energy?

(Slide 36: Carbs and fat)

That's equally as easy, it's carbs and fat. And it's this element, the energy, that I want to concentrate on. In fact, I want to concentrate largely on the energy element for the remainder of the talk.

(Slide 37: Eatwell Guide)

This is the Government's Eatwell Guide. It's on display, or should be, in all surgeries and hospitals. We will be becoming very familiar with this during this presentation.

If you have a close look, you can see that the sector labelled Oil & spreads is really very small. Indeed, we are constantly being told by the government and the NHS to reduce our fat intake. So, I'm going to put oils and fats to one side for the moment and concentrate on the other element of energy: carbs.

(Slide 38: Energy - Carbs)

Please bear with me during the next bit; to quote Fergal Sharkey, ‘it will all be worth it in the end’.

(Slide 39: Glucose & Fructose)

Carbs, or carbohydrates, the source of much energy for the body, are themselves made of building blocks. The two fundamental building blocks are glucose and fructose. There is a third, galactose, but we won’t concern ourselves with that one, today.

I’ve shown glucose as a red hexagon and fructose as a green pentagon. We’ll stick with this throughout the talk.

In the main, we don’t come across glucose or fructose as single molecules; they to hang around in pairs or larger groups.

(Slide 40: Starch and Sucrose)

Here are the two most common arrangements in the food that we eat. Starch, which comprises long chains of solely glucose molecules; and sucrose, table sugar, which is one glucose molecule joined to one fructose molecule.

(Slide 41: Carbs 1, glucose and fructose)

So, carbs are mainly glucose with a smaller amount of fructose.

(Slide 42: Carbs 2, starch and sugar)

Another way to look at carbs is that they are mainly starch with a smaller amount of sucrose.

(Slide 43: Carbs 3, grains, potatoes and fruit)

Or another way, carbs are healthy bread and breakfast cereals, potatoes in jackets, vitamin-packed fruit, and of course naughty sweets and cake.

(Slide 44: Eatwell Guide)

Okay, here’s the Eatwell Guide again. As you can see, it’s segmented into fruit and veg, potatoes and breads, beans and peas, dairy, oils and spreads. It’s not segmented into the scientific bits that we are discussing; carbs, fat, protein and fibre.

(Slide 45: Eatwell Guide 2)

So, here’s the segments divided into the scientific elements of carbs, fat, protein and fibre.

(Slide 46: Eatwell Guide 3)

Let’s continue to concentrate on the carbs.

(Slide 47: Eatwell Guide 4)

When we pull all the carbs together, we find that the Government’s Eatwell Guide recommends that just over 50% of our food intake comes from carbs.

(Slide 48: Eatwell Guide 5)

That’s lots and lots of glucose and a smattering of fructose.

(Slide 49: That's a bit strange)

Now that's a bit strange considering Warburg's findings regarding cancer and glucose. Something's not right.

(Slide 50: Why are we being advised)

Why are we being advised to eat large amounts of glucose when cancer feeds off glucose?

Chapter 3

(Slide 51: Sanity check 1: What does history say about our diet?)

During this talk, I'll do a few sanity checks just in case you think that I've lost plot and gone off the rails.

What does history say about our diet?

(Slide 52: Evolution of Homo Sapiens 1)

I want to talk about the evolution of us; humans; Homo Sapiens. Now, if you don't subscribe to evolution, it's not a problem. If I mention evolution, you can just drop 'creator' in its place and all will be just fine.

It looks from this drawing as if Homo Sapiens evolved from apes; we didn't; it's just tradition to show an evolutionary illustration like this.

(Slide 53: Evolution of Homo Sapiens 2)

I'll just zoom in a bit closer to show the last 2.5 million years. If you saw an Australopithecus man nonchalantly walking down the road, you wouldn't think, "Oh, my goodness, another ape escaped from the zoo!" He would be more like us than an ape. A bit shorter, although that's in dispute, and a tad hairier, certainly. He'd be walking upright and not knuckle-walking or swinging through the trees like our ape-cousins. That said, there are evolutionary differences, brain size and head shape for instance. 2.5 million years, or 100,000 generations, is regarded as an acceptable period of time for this amount of evolution - meaningful evolution - to have taken place when considering organisms as large and complex as us.

(Slide 54: Hunter gatherers)

Our ancestors in these times were nomadic hunter gatherers.

(Slide 55: Hunter gatherer food)

They ate:

(Slides 56, 57,58, 59, 60: Hunter gatherer - food)

Lots of meat.

Lots of fish.

Lots of peas and beans, nuts and seeds.

Lots of leafy greens.

A bit of fruit – hence the rather smaller tick.

It's worth reflecting on fruit for a moment. Back then, fruit was not like it is now; adorning our supermarket shelves; large, beautiful, sweet and juicy; the result of a century of selective breeding. They were small, bitter and ugly and relatively rare as the tree or bush needed to be in season. And, to add insult to injury, our ancestors would have had third dibs after the birds and squirrels and only then if your friend would give you a leg up.

(Slide 61: Essential?)

So, based on this, and putting water, oxygen and fibre to one side, what would we say our essential macronutrients were?

(Slide 62: Fat)

Fat, yes.

(Slide 63: Protein)

Proteins, yes.

(Slide 64: Carbs – spaghetti)

Carbs?

(Slide 65: Carbs – spaghetti with cross)

No.

(Slide 66: But surely glucose is essential?)

Hold on, surely glucose is essential, I was taught it at school.

(Slide 67: Any glucose we do need 1)

Yes, we need a bit of glucose. But you don't need to eat it.

Any glucose that we do need…

(Slide 68: Any glucose we do need 2)

…the liver makes it from the protein and the fat that we eat. It's a process called de novo gluconeogenesis.

(Slide 69: Garden of Eden)

And so it was; life was fine and dandy in our beautiful Garden of Eden until…

(Slide 70: Agricultural Revolution)

Around about 10,000 years ago, something happened. It was the result of the combined stresses of a couple of million years of repetitive climate and landscape changes. It was the agricultural revolution. The domestication of livestock and the growing, milling and baking of grains and the end of nomadic lifestyles.

(Slide 71: Evolution of Homo Sapiens 3)

Let's have another look at the evolution chart. On this scale, the 10,000 years since the onset of the agricultural revolution appears as one pixel!

(Slide 72: 10,000 years is not enough)

The 10,000 years, which represents about 400 generations, is not enough for any meaningful evolution to have taken place.

(Slide 73: Food, carbs: Bread, Cereal, Potatoes, Pizza, Pasta, Rice)

And look what our poor caveman bodies, relentlessly, on a day-in-day-out basis, must deal with:

Bread, Cereal, Potatoes, Pizza, Pasta, Rice.

So, I think that it is fair to say that eating carbs, certainly to anywhere near the levels most of us do, is not essential.

Chapter 4

(Slide 74: Otto Heinrich Warburg + Glucose)

With this in mind, let's return to Otto Warburg and his finding that cancer cells often devour glucose as if it's going out of fashion – a finding, as I have already mentioned, that has never been disputed in any research, or study.

(Slide 75: Eatwell Guide 5)

Does that square with the Eatwell Guide with its advice to eat more than half your plate of glucose?

(Slides 76, 77, 78, 79, 80, 81, 82, 83, 84, 85, 86, 87, 88: The Eatwell Guide in various locations)

An Eatwell guide, that is almost ubiquitous in medical and educational settings throughout the UK.

(Slide 89: Exceedingly Unusual Behaviour)

Which I find Exceedingly Unusual Behaviour.

(Slide 90: Eatwell Guide)

As an aside, you might be interested to know from where this Eatwell Guide came.

(Slide 91: Dietary Reference Values Book)

It came from this 1991 book, 'Dietary Reference Values for Food Energy and Nutrients for the United Kingdom', published by the UK Government. It was the first time that a recommended breakdown of all three macronutrients was provided.

(Slide 92: Table 1.2)

And here they are, on page 20, almost exactly the same figures that we use today.

You'd think that these hugely important figures had been the result of much scientific research, theory and practice. But you'd be wrong.

(Slide 93: 1.3.6.b)

What they apparently did in the years before the book's publication, was to ask groups of people whether or not they felt well and whether or not they were suffering from any diseases. If they said that they felt well and were not obviously suffering from any diseases, the researchers asked them what they ate and they used this data as the model for a healthy diet. No science, just what the average, so-called healthy person ate in the 1970s and 1980s.

(Slide 94: The Balance of Good Health)

From that came the Balance of Good Health in 1994.

(Slide 95: The Eatwell Plate)

Followed by the Eatwell Plate in 2007.

(Slide 96: The Eatwell Guide)

And the current Eatwell Guide in 2016.

(Slide 97: US MyPyramid)

The United States are also in on the act. This was their food pyramid. I say was, because it's changed very recently; March this year, 2026 in fact. My understanding is that it was this 2015 report…

(Slide 98: Heart Disease and Stroke Statistics - 2015)

… that prompted a very reluctant change.

(Slide 99: Heart Disease and Stroke Statistics – 2015, page e81, 1)

Buried on page e81 are three game-changing statements:

Firstly, the admission that fat consumption has little effect on cardiovascular disease!

Secondly, that the consumption of the demon of fat, that being saturated fat, actually has no bearing on coronary heart disease, stroke or cardiovascular disease.

(Slide 100: Heart Disease and Stroke Statistics – 2015, page e81, 2)

And thirdly, that higher consumption of carbohydrates and lower consumption of fat, increases…

I'll say that again, higher consumption of carbohydrates and lower consumption of fat, increases risk of coronary heart disease.

So, in about turn of biblical proportions, they are now advising decreasing the consumption of carbs whilst increasing the fat intake. Which is great news.

(Slide 101: Inverted pyramid)

So based on this pivotal report, what did they do? I think this is great; it's truly inspired; I'd have done the same. They waited 11 years then cunningly flipped the pyramid upside down.

If you remember from their old pyramid, the foods like bread and all the grains, that they were advocating eating in abundance, were located at the pyramid's base – the biggest part. And foods that they viewed as the worst, the fatty foods, were located at the pyramid's apex – the smallest part.

By cunningly flipping the pyramid upside-down whilst keeping the location of most foods the same, meant that they could encourage us to do exactly the opposite of what they had been saying. They now want us to eat less grains and eat more fatty food!

I wonder if anyone has noticed? What happens when we in the UK catch up? Turn the Eatwell Plate upside down?

Chapter 5

I think at this point it's time for another sanity check.

(Slide 102: Sanity Check 2)

Would you believe that modern medicine makes use of cancer's love of glucose – the Warburg Effect?

(Slide 103: PET Scanner)

This is a PET scanner, one of the most useful diagnostic tools in the oncologist's armoury.

(Slide 104: Glucose hexagon)

We've encountered this red hexagon before; it's a molecule of glucose.

Now, please don't be unduly alarmed; I'm going to show you the chemical structure of glucose.

(Slide 105: Glucose structure)

I want you to concentrate on the O-H at the very bottom right.

(Slide 106: Glucose structure with F18)

I've replaced the O-H group with a single atom. It's an atom of a chemical called fluorine.

(Slide 107: Glucose structure with F18 & radiation signs)

It's not, however, ordinary fluorine, it's a radioactive flavour of fluorine. Because it's radioactive, we can use a detector to see where it's going. Now, here's the thing; the body can't immediately tell the difference between normal glucose and this radioactive one! That's the key. We can use this to see where in the body glucose is going and where it's being used.

(Slide 108: PET injection)

This radioactivity is why the PET scan syringe is made of thick metal and why they take so much care when they inject it into you.

And what do you get when you trace where the glucose goes?

(Slide 109: PET scan)

This is what you get.

The red hotspots are where glucose is being used the most, and blue where glucose is being used the least.

Let me first point out the brain, which uses a large amount of glucose as it never switches off even when lying very still in a PET scanner.

You can clearly see the bladder as this is where the spent radioactive glucose is waiting to be eliminated from the body.

You can also make out the kidneys and the liver. They show up a little as they are part of the elimination process that rids the body of the radioactive glucose.

Now. Let's look at the other bright red regions. These are areas in the body that are devouring glucose; these are active cancers; in the heart (it's a rare one - primary synovial sarcoma of the heart); in the right retroperitoneum probably in the ascending colon; in the pelvic wall; and in the left femoral neck. They flip the left and right on the transverse plane images as the standard is to view the body cross sections as if looking up from the floor and through the patient's feet. Presumably that's how a doctor first sees a hospital patient – feet first!

(Slide 110: 'The images produced by a PET scan are a dramatic visualisation of cancer's grotesquely voracious appetite for glucose.')

Whilst we are catching our breaths, here's a fabulous quote from Travis Christofferson's book Tripping Over the Truth: 'The images produced by a PET scan are a dramatic visualisation of cancer's grotesquely voracious appetite for glucose.'

(Slide 111: Intermission)

Let's just take a few seconds to assimilate everything.

Chapter 5

(Slide 112: Star of the Show)

And now, I want to introduce to you the star of the show:

(Slide 113: Ketones)

Ketones

(Slide 114: Egyptian famine)

This is Joseph and there's a famine in Egypt. But don't panic, the human body is well designed to deal with famines. Here's how it works...

(Slide 115: Glucose low)

When glucose in the body becomes low due to reduced food intake...

(Slide 116: Glucose low, fat high)

...The body starts to use stored fat. That's, I suppose, obvious and to be expected. But here's the thing.

(Slide 117: The blood brain barrier)

There's something called the blood brain barrier. It acts as a protective filter between your body's blood supply and the cells of your brain and spinal cord. It's very selective about what it allows through, and what it doesn't.

(Slide 118: Glucose and the blood brain barrier)

Glucose is allowed through alright. But we are in a famine, so there's not much glucose knocking around so I've greyed it out a bit.

(Slide 119: Fat and the blood brain barrier)

What about fats? No, they do not pass freely across the blood brain barrier.

(Slide 120: protein and the blood brain barrier)

What about protein? Well, not generally. So that's not going to help much in our time of famine.

So, what's to do? The brain is too important to have glucose as the only source of energy.

(Slide 121: Glucose down, ketones up)

Here's where we introduce ketones. Ketones are made by the liver when glucose (and glycogen) supply is low. They are made from the fat that we all store for just this moment in time; a famine.

(Slide 122: Ketones and the blood brain barrier)

These small, energy-storing molecules can freely pass through the blood brain barrier.

(Slide 123: Ketones and the body)

Ketones aren't just for the brain and spinal cord, though, they can be used everywhere in the body. Our bodies love to use ketones for energy. It's far more efficient to use ketones than it is to

use heavy old glucose. Indeed, during our caveman days we spent most of our time using ketones rather than glucose. We call this being in ketosis.

Chapter 6

I want to introduce you to another star in a moment. But just before then I want to consider the words energy and metabolism.

(Slide 124: Energy & Metabolism)

These two words can generally be used interchangeably. Metabolism, however, is probably a better term to use moving forward as we use it solely in connection with the conversion of food into energy in the body.

(Slide 125: What Warburg didn't know)

As a way of preparing for the new star's entrance, let's look at what we know now that Warburg did not.

(Slide 126: What Warburg didn't know – the scientists)

And it's thanks to these scientists and of course many others.

(Slide 127: What Warburg didn't know - Mitochondria)

What we now know is what exactly what our second star does. The star, or stars, are mitochondria.

So, what are mitochondria?

(Slide 128: Cell 1 – complex one)

We are all made up of microscopic subunits called cells. We are what is called multicellular beings. We each have approximately 30 trillion of them.

Here's a rather bewildering artist's impression of one. Radio 4 liken cells to huge cities with all sorts going on.

(Slide 129: Cell 2 – simple one)

Here's a simpler version. You can see the nucleus in the middle within which is stored most of our DNA; our chromosomes; our blueprint for life.

(Slide 130: Mitochondria)

And here are a couple of mitochondria. There's only two shown here, but most cells have hundreds or thousands within each of them.

(Slide 131: Mitochondria – The powerhouse of the cell)

Mitochondria are also known, by Radio 4 amongst many others, as the powerhouse of the cell.

(Slide 132: Mitochondria – Food input)

It is within these mitochondria that the majority of the work of converting the carbohydrates, proteins and fats that you've eaten into usable energy takes place. Afterall, your body can't use a potato or some cheese without it being converted!

(Slide 133: Mitochondria electron micrograph 1)

Here is a beautiful electron microscope micrograph of a mitochondrion – singular of mitochondria.

They have an outer membrane that, as you can see, is continuous.

They also have an inner membrane that is very close to the outer until it folds in on itself creating these wonderful structures called cristae. What they do is to increase the surface area of the inner membrane because it is on this inner membrane that a lot of the work in converting carbs, proteins and fats into usable energy takes place.

(Slide 134: Mitochondria electron micrograph 2)

Here's another fabulous electron micrograph.

(Slide 135: Mitochondria electron micrograph 3)

Mitochondria hang around in groups, and they fuse and then split depending upon the energy demands.

(Slide 136: Transmission Electron Microscope)

This is a transmission electron microscope. I show it to illustrate the sort of equipment that you need to obtain pictures like the ones we've just seen. Quite obviously, Otto Warburg had nothing of the kind; nothing that could reveal such small structure in exquisite detail.

But that's not all; you need be able to prepare the specimen that you wish to examine in a transmission electron microscope. This process is called ultramicrotomy.

(Slide 137: Ultramicrotomy)

Samples to be examined and photographed need to be sliced almost impossibly thin; often to a thickness of less than a tenth of the wavelength of light. So, you can't just put a sample on a glass slide and whack it in like you can with an ordinary light microscope. It's a right palaver of:

1) Fixing or stabilizing the organic material.
2) Removing all water.
3) Embedding it in epoxy resin.
4) Shaping the resin.
5) Sectioning the resin into the tiniest of slithers.
6) Floating the slithers so they can be lifted away and placed on a slide.

Just to get one specimen suitable for the electron microscope requires the dexterity of a watchmaker and the patience of a saint. It's a process that's continually evolving leading to the production of increasingly detailed images.

(Slide 138: Mitochondrion 3)

And when you get it right, you are rewarded; and the results can be breath-taking.

Chapter 7

With these beautiful mitochondria in mind, I would like to keep our focus on the food; energy that the cell can use. For normal cells, we've covered most of this already.

(Slide 139: Normal cell energy 1, glucose.)

They can of course use glucose for energy – it's the main source in our regular, western-world diets.

(Slide 140: Normal cell energy 2, fats.)

Of course, they can use fats; even though the governments of the World are asking us to cut down on them.

(Slide 141: Normal cell energy 3, proteins.)

I'm going to give proteins a small tick as we don't really use proteins for energy unless things have gone very wrong and the famine has outlasted our fat stores.

(Slide 142: Normal cell energy 4, ketones.)

Yes, very much so when it comes to ketones. As we now know, some the fat that we have stored is converted into ketones and sent coursing around our bodies, especially when glucose is low.

(Slide 143: Mitochondrion 4.)

And this all happens due to our wonderful mitochondria with all their fabulous, intricate internal structures of folded cristae.

(Slide 144: Mitochondria 5, pancreatic cancer.)

But what's happened here? What's happened to the beautiful cristae? These mitochondria in pancreatic cancer are damaged.

We have a saying; a motto; a dogma in biochemistry, 'that structure equals function'. If, therefore, you lack the structure, you lack the function. Just like a protein enzyme whose unique shape makes a certain chemical reaction happen in your body, if the enzyme's shape becomes disrupted, it's function will cease and that particular chemical reaction will stop. Same here; if the structure of the mitochondria is upset, then it cannot be expected to perform its duties.

(Slide 145: Mitochondria 6, kidney cancer.)

Same. Broken mitochondria in kidney cancer. Look at the internal structure, it's all over the place. Some mitochondria appear to have no internal structure at all.

(Slide 146: Mitochondria 6, myeloid leukaemia.)

And again, this time with acute myeloid leukaemia, an aggressive cancer of the blood. Look at the shapes of some of the mitochondria. Thet are supposed to be regular and somewhat oval; not all higgledy piggledy.

(Slide 147: Mitochondria 6, glioblastoma 1.)

Mitochondria in the brain tumour, glioblastoma, showing similar defects in structure.

(Slide 148: Mitochondria 6, glioblastoma 2.)

Another slide of glioblastoma mitochondria showing little internal structure, therefore little normal function.

(Slide 149: Mitochondria 6, glioblastoma 2.)

And a final one for glioblastoma displaying a very odd shape.

(Slide 150: Cell oil droplets)

Whilst we are looking at some electron micrographs, here's one displaying the abnormally large oil droplets that you find in cancer cells showing that cancer cells can't handle oil. They simply leave it, untouched, to wallow about in and around the cell.

(Slide 151: Cell energy)

Ok, with defective mitochondrial structure and therefore dysfunction in mind, let's do the same exercise with cancer cells as we did with normal cells.

(Slide 152: Cancer cell energy 1: glucose)

Let's look at glucose first. I have to tell you that over 90% of all glucose is processed, or as we say, metabolised, in the mitochondria. But that leaves 10% or so that is metabolised in the cancer cell, but outside of the broken mitochondria. I have, therefore, given glucose a small tick.

(Slide 153: Cancer cell energy 2: glucose)

But this does mean that cancer can metabolise glucose, albeit very inefficiently. So, what cancer cells do is to simply increase glucose metabolism – they really crank it up – one hundred times maybe!

(Slide 154: Cancer cell energy 3: glucose)

What does this cranking up of glucose metabolism mean? The usage of inordinate amounts of glucose by cancer cells. It means lots of waste product, lactic acid, that you don't tend to see in normal cells. This is exactly what Otto Warburg found. Hugely increased glucose consumption and lactic acid in abundance.

(Slide 155: Cancer cell energy 4: glucose)

And from where does all this glucose come? The weight that you lose and the food that you eat.

(Slide 156: Cancer cell energy 5: glucose)

Because of the upregulation of glucose in cancer cells, we will swap the small tick for a really large one.

What about fats? Can broken mitochondria metabolise fats?

(Slide 157: Cancer cell energy 6: fats)

No, they can't. There's a chemical, an enzyme called the mitochondrial trifunctional protein, that's needed to break down fats. This enzyme happens to be embedded in the mitochondrial inner structure, which is broken. So, cancer cells can't use oils or fats for energy.

Now, what about proteins?

(Slide 158: Cancer cell energy 7: proteins)

This is Thomas Seyfried, professor of biochemistry from Boston College in the United States. He is one of the pioneers of the metabolic approach, and it is him and his research team that have put the final pieces together in the Metabolic Approach to Cancer jigsaw.

When we considered normal cells, we noted that they don't normally use proteins for energy. But Thomas Seyfried and his team established that this is not always the case for cancer cells; they can use proteins; in a fashion.

(Slide 159: Cancer cell energy 8: proteins – 20 amino acids)

This complication is a diagram of the building blocks of all the proteins that make you. They are the twenty amino acids.

(Slide 160: Cancer cell energy 9: proteins - glutamine)

I'm going to highlight the one in the bottom left – glutamine.

Because part of the glutamine metabolism does not use the internal structure of the mitochondria, cancer cells can indeed use glutamine for energy.

(Slide 161: Cancer cell energy 10: proteins - glutamine)

So, it gets a tick. But again, like glucose in cells with broken mitochondria, it's not very efficient, therefore only gets a small tick.

(Slide 162: Cancer cell energy 11: proteins - glutamine)

So, what's cancer to do? Exactly the same as it did with glucose, it increases glutamine metabolism – it really cranks it up – one hundred times maybe!

(Slide 163: Cancer cell energy 12: proteins - glutamine)

What happens as a result? The usage of inordinate amounts of glutamine by cancer cells means lots of waste product. But this time it's succinic acid as has been found by scientists after Otto.

(Slide 164: Cancer cell energy 12: proteins - glutamine)

And from where does all this glutamine come? The weight that you lose and the food that you eat.

(Slide 165: Cancer cell energy 13: proteins - glutamine)

Because of the upregulation of glutamine, and just as we did for glucose, we will swap the small tick for a large(ish) one as it's still not as efficient as glucose.

Now, what about ketones?

(Slide 166: Cancer cell energy 14: ketones)

No, many cancer cells cannot utilize ketones. At least to nowhere near the degree that normal cells can. And the reason is that, amongst other things, cancer cells are missing an important enzyme that is part of the process of breaking down ketones for use. The enzyme rejoices in the name, succinyl-CoA:3-oxoacid CoA transferase or simply SCOT. In fact, people who have the rare disorder called SCOT deficiency can't do the Metabolic Approach to Cancer as they can't use ketones and rely on glucose to feed the brain.

(Slide 167: Cancer cell energy 15: oxygen)

If you notice, I've added one final row; oxygen. After all, it is a very important nutrient.

(Slide 168: Cancer cell energy 16: oxygen)

Of course, normal cells love it. They can't get enough of it. So, I'm going to give it a big tick.

Now, what about cancer cells? How are they with oxygen?

The answer is, not good. Cancer cells can't use oxygen. You see, the oxygen that we breathe is taken up and used by the mitochondrial inner membrane, the cristae. Which are, of course, broken. So, the oxygen just hangs around; kicking its heels.

(Slide 169: oxygen; highly reactive substance)

Now, when oxygen just hangs around, as it does in a cancer cell, it demonstrates its highly reactive…

(Slide 170: oxygen; a toxic substance)

Toxic nature.

(Slide 171: Reactive oxygen species 1)

Let's take a closer look.

(Slide 172: Reactive oxygen species 2, superoxide)

When left to their own devices, oxygen molecules will take electrons from wherever they can find them. They will also grab onto hydrogen atoms that are always floating about, especially in highly acidic cancer cells. This creates what are called Reactive Oxygen Species or ROS – reactive oxygen species are also called free-radicals, the things that antioxidants go after. These reactive oxygen species have wonderful names like superoxide…

(Slide 173: Reactive oxygen species 3, hydroxyl)

Hydroxyl…

(Slide 174: Reactive oxygen species 4, peroxide)

And hydrogen peroxide.

(Slide 175: Reactive oxygen species 5, 3 x ROS)

Let's assume that we only have a few oxygen molecules and thus only a few reactive oxygen species as would be the case in most cancers due to a limited blood supply.

(Slide 176: Reactive oxygen species 6, 3 x ROS + glutathione)

Cancer cells can deal with this because they, like normal cells, can produce their own antioxidant called glutathione.

(Slide 177: Reactive oxygen species 7, glutathione)

And as if by magic, the reactive oxygen species were no more.

(Slide 178: Reactive oxygen species 8, multitudes of oxygen)

But what if we could bypass the limited blood supply and flood the cancer cells with oxygen? Of course, the normal cells would think that Christmas had come early; they'd love it. But what of cancer cells?

(Slide 179: Reactive oxygen species 9, multitudes of ROS)

We'd get lots of reactive oxygen species.

(Slide 180: Reactive oxygen species 10, multitudes of ROS + glutathione)

Of course, the cancer cells would send in the glutathione. But the amount of this is limited.

(Slide 181: Reactive oxygen species 11, multitudes of ROS)

So, it does not make much difference. Even after the glutathione has done its thing, there's still a lot of reactive oxygen species knocking around.

(Slide 182: Reactive oxygen species 11, Cell with multitudes of ROS)

In their unbridled travels inside the cancer cells, these reactive oxygen species migrate to the cancer cells' outer wall; its membrane; the only thing that's holding it together.

(Slide 183: Reactive oxygen species 11, Cell destroyed)

And bingo, the reactive oxygen species set off chain reactions all along the cell membranes that cause them to fail. The cancer cells just fall apart.

(Slide 184: Cancer cell energy 17: cancer cell oxygen)

Let's now complete the energy chart that we've been working on?

Cancer cells hate oxygen when in excess. We will talk about how we make use of this in a few moments.

(Slide 185: Can we take advantage of all this?)

Can we use all this to our advantage?

Chapter 8

(Slide 186: The Metabolic Approach)

Yes, The Metabolic Approach

(Slide 187: The Metabolic Approach definition)

The metabolic approach is a non-toxic, lifelong cancer management framework that easily integrates with other treatments.

Let me just for a moment propose an analogy. If you were going to attempt to take control of something, you might have a couple of options:

(Slide 188: Castle under siege)

You might put it under siege and throw everything you have at it.

(Slide 189: Castle ruins)

But it's likely you'd end up with a ruin on your hands.

(Slide 190: Starve them out)

My contention is to be patient and starve them out.

Then after a couple of weeks without food and wallowing in their own waste...

(Slide 191: SAS)

We send in the troops to finish them off.

Chapter 9

(Slide 192: The Metabolic Approach Stage 1)

The Metabolic Approach to Cancer is split into three main stages.

(Slide 193: Press)

Stage 1 is called the Press stage.

(Slide 194: Calorie-restricted low-glucose diet)

The Press stage is a calorie-restricted low-glucose diet – a ketogenic diet if you like. But it's not to be mistaken for the Atkins diet that suggests increasing your protein intake. The Metabolic Approach to Cancer recommends keeping your protein level to about what it was before. So, you should…

(Slide 195: Enjoy all meats)

Enjoy all meats.

(Slide 196: Enjoy all seafood)

Enjoy all seafood.

(Slide 197: Enjoy all oils and fats)

Enjoy all oils and fats.

(Slide 198: Enjoy most dairy)

Enjoy most dairy.

(Slide 199: Enjoy all green veg.)

Enjoy all green veg.

But you must…

(Slide 200: Remove sugar 1.)

Remove sugar.

(Slide 201: Total Sugars)

This means looking at all food labels and identifying the Total Sugars figure. It should be very low; less than 1% or 1g in 100g.

(Slide 202: Remove grains.)

Remove grains. That's all bread, pasta, rice, breakfast cereals, etc.

(Slide 203: Remove HS tubers.)

Remove high-starch tubers. That's potatoes and all its derivatives: chips, crisps etc.

(Slide 204: Remove HS legumes 1.)

Remove high-starch legumes. That's beans, chickpeas, lentils, etc.

(Slide 205: Total Carbs.)

This means looking at all the food labels and identifying the Total Carbs figure which should be lower than 5% or 5g in 100g.

(Slide 206: Supplementation.)

Although you don't need to supplement, it's a good idea. You will need: a good multivitamin, omega 3, both EPA and DHA, vitamin D3, calcium and B complex.

(Slide 207: The Glucose Ketone Index.)

And now we come to the reason for now for The Metabolic approach to Cancer. And it's based upon what's called the Glucose Ketone Index (GKI) and the targets that those following should aim for.

(Slide 208: Keto Mojo.)

To measure your GKI, you need a piece of equipment that will measure both your blood glucose and your blood ketones and give the results to you in the same units; millimoles per litre, for example, as the Keto Mojo does. The Keto Mojo uses the smallest drop of blood, and you get the result in under a minute.

(Slide 209: GKI Chart.)

Once you have your GKI, you can chart it against targets.

If your GKI is largely above twenty, that's a problem; for anyone, not just those with cancer. Nobody should have a GKI of twenty or above for any period more than say twelve hours per week. The evidence is now so compelling that keeping your GKI lower than twenty for the vast majority of the time will substantially lower your risk of getting not only cancer but type 2 diabetes, coronary heart disease and even Alzheimer's.

If you are really needing to control and aggressive, out of control cancer, the current guidelines suggest keeping in deep ketosis with GKI of two or less until control has been established.

The guidelines for slightly less aggressive cancers suggest keeping your GKI to four or less until control has been achieved.

Once control has been achieved, then the GKI target can be relaxed to somewhere between four and twenty. We recommend around eight which for most people gives scope for about 40g of carbs to be allowed into your diet every day, which is nice.

(Slide 210: GKI Chart.)

Here's mine. It was my mother's 90th birthday in August. We spent a little while in Liverpool and relaxed the regimen a little as you can see. You can't tell from this chart, but a 24-hour water-only diet got me right back in.

(Slide 211: Body Composition and cachexia.)

One of the most important things to keep a careful eye on when applying the Metabolic Approach to Cancer is uncontrolled weight loss or cachexia.

(Slide 212: Body Composition – SECA.)

We keep a very close watch on this using our SECA body composition scanner. The important aspect of this is to closely watch the skeletal muscle mass percentage. It's not a case of simply maintaining body weight, it's a case of maintaining skeletal muscle mass. We also test for grip strength as this is a great way to detect loss of strength and therefore muscle mass.

(Slide 213: Body Composition – Smart scales, Keto Mojo and Grip tester.)

For those operating the Metabolic Approach to Cancer remotely from us, we advise the purchase of a smart scale that can measure skeletal muscle mass percentage, the Keto Mojo and a grip tester.

(Slide 214: Body Composition - Charts.)

We also have an online tracking system in which you upload your test results, and it will automatically alert you to issues that it picks up.

Chapter 10

(Slide 215: The Metabolic Approach stage 2.)

Moving on to The Metabolic Approach to Cancer stage 2.

(Slide 216: Pulse.)

This is called the pulse stage.

(Slide 217: Glutamine Inhibitor.)

It's the glutamine inhibitor stage, that we introduce when the patient has adjusted to the low-carb, ketogenic diet.

The reason that this stage is called the pulse stage is that for some people, the glutamine inhibition can't be used all the time. Whilst it's true that glutamine feeds cancer, it's also important for your normal cells. They don't need a lot, but they do need some.

(Slide 218: Berberine.)

We use a potent but natural substance called berberine as the glutamine inhibitor. There are some others, but they are still in research and not yet available.

Chapter 11

(Slide 219: The Metabolic Approach stage 3.)

And finally, to stage 3

(Slide 220: The Final Insult.)

This is the final insult during which we send in our equivalent to the SAS

(Slide 221: Hyperbaric Oxygen Therapy.)

Hyperbaric Oxygen Therapy or HBOT for short.

(Slides 222, 223, 224, 225, 226, and 227 Our HBOT Chamber)

Here's our huge, medical-grade, two-seater hyperbaric oxygen chamber with its large cinema screen for Netflix etc.

(Slide 228: Sanity Check 3.)

I just would like to conduct one last sanity check regarding hyperbaric oxygen therapy that might surprise you as some mainstream modern cancer therapies make use of reactive oxygen species, the same thing that HBOT makes in cancer cells that can't use oxygen.

(Slide 229: Radiotherapy LINAC machine.)

This is a radiotherapy machine.

(Slide 230: Radiation.)

There's no doubt that out of the spout comes high energy radio waves – X-rays and the like. But, when you hear many people talk about this, they say that it's this high energy radiation that damages the cancer's DNA and causes these cells to die. Well, that's not the only way, nor is it the main way.

(Slide 231: Microwave.)

This is a microwave oven, and it works by using less-energetic microwaves to set the water molecules in food into vibration that we perceive as heat.

(Slide 232: Cancer Cell with Water.)

This is a cartoon of a cancer cell and like all cells, it contains a lot of water.

(Slide 233: Cancer Cell with Radiation.)

Radiation therapy concentrates beams of high energy radio waves, usually high-energy X-rays, into your cancer cells.

(Slide 234: Cancer Cell with molecules vibrating.)

This, like the microwave oven, sets the water molecules into vibration. But, unlike the oven, because the radiation is so much more energetic, it doesn't just set the water molecules into vibration, it rips electrons off them. It ionises them.

(Slide 235: Cancer Cell with oxygen.)

Now, with electrons floating around, it's oxygen that will grab onto them creating reactive oxygen species.

(Slide 236: Cancer Cell with reactive oxygen species.)

Just like before, the reactive oxygen species make their way to the cells' outer membranes…

(Slide 237: Cancer Cell and lipid peroxidation.)

And sets the outer membrane chain reaction, lipid peroxidation, into process.

(Slide 238: Cancer cell destroyed.)

And the cancer cell is destroyed.

Chapter 12

(Slide 239: The Metabolic Approach.)

So, let's have a quick summary of the metabolic approach to cancer.

(Slide 240: A thorough assessment.)

Before we do anything, we conduct a thorough assessment of the patient and their condition.

(Slide 241: Water-only fast.)

We then, assuming all is OK, ask the patient to commence a 24 to 48-hour water-only fast to speed up the transition into ketosis aiming for a GKI typically between two and four.

(Slide 242: Calorie-restricted, low-carbohydrate diet.)

Then it's time for the patient to start the calorie-restricted, low-carbohydrate diet.

(Slide 243: Body composition monitoring.)

At this time, we commence the body composition monitoring to ensure the patient remains on track in reducing the risk of uncontrolled weight loss.

(Slide 244: Glutamine inhibitor.)

When the patient has got used to the low-carbohydrate diet and their GKI has stabilized, we then introduce the glutamine inhibitor on a week-on, week-off schedule to begin with. The amount of time spent on this glutamine inhibition can be increased or decreased as necessary to maintain as much skeletal muscle mass as possible.

(Slide 245: Hyperbaric Oxygen Therapy.)

It's then time to commence the hyperbaric oxygen therapy.

(Slide 246: Intensive element.)

This intensive element of low-carbohydrate diet, glutamine inhibitor and hyperbaric oxygen therapy lasts until the cancer is under control. Normally, one to two months.

(Slide 247: Maintenance.)

After which, it's the maintenance protocol of a slightly less calorie-restricted low-carbohydrate diet, still using the glutamine inhibitor and reducing the number of hyperbaric oxygen sessions. This protocol we recommend as a new lifestyle.

(Slide 248: Non-toxic.)

One of the beauties of this whole approach is that, unlike the other standard-of-care treatments, it's completely non-toxic!

(Slide 249: Standalone solution.)

And it's a standalone solution that works with a patient's current treatments.

(Slides 250 - 263.)

And, if you want to know what research has been done supporting the Metabolic Approach to Cancer, the list just goes on and on...

Chapter 13

(Slide 264: Loose ends.)

Now, there's some loose ends that we really should cover.

(Slide 265: How come we don't know about the metabolic approach?)

How come we don't know about the metabolic approach?

(Slide 266: Not in NHS and NICE guidelines.)

It's because none of this Metabolic Approach is in the NHS and NICE guidelines. The question then is why not?

(Slide 267: No intellectual property in nutrition or HBOT.)

It's because there's no intellectual property in nutrition or hyperbaric oxygen therapy, therefore, no money to fund what NICE and the NHS deem as the necessary studies.

(Slide 268: No money in it for big pharma.)

Because there's no intellectual property, there's no money in it for big pharma.

(Slide 269: No accolades for scientists.)

Because there's no intellectual property and nothing new, there's no accolades for scientists who themselves are often funded by big pharma.

(Slide 270: Our habits)

Then, of course, it's our habits. We've had it drummed into us that carbs are good; and fats, particularly saturated fats, are bad.

(Slide 271: Huge reliance on grains)

Our economy has such a huge reliance on grains. If we move away from grains, which we all should, the world economy would collapse.

(Slide 272: No capacity for more livestock)

We've got no more capacity for livestock.

(Slide 273: Medical dogma)

Medical dogma plays such a large part in this as well – you try getting doctors to budge on anything. I'm doing them a misjustice; even if they wanted to, they wouldn't be allowed to.

(Slide 274: Our natural scepticism)

And lastly, our natural scepticism doesn't help.

(Slide 275: Okay, where's the evidence?)

Okay, where's the hard evidence? You might say.

(Slide 276: There is plenty)

There is plenty out there but it's all small scale. This bit of one report is of a glioblastoma patient from Devon who with the very best standard-of-care available, would normally have been expected to live for less than a year. He lived for over a decade. We've also got patients with metastatic cancer who are now in remission to the surprise of their oncologist.

(Slide 277: Randomised studies)

The whole healthcare system is geared towards preservation of the status quo and limiting exposure to litigation. The authorities achieve this by insisting that every new therapy or new drug has to have gone through several, large scale, randomised, placebo-based, double-blind studies.

(Slide 278: 100's of millions)

Which cost hundreds of millions.

(Slide 279: Clearly aimed at approving drugs)

So, the system is clearly aimed at approving new, patentable, therefore financially rewarding drugs.

Chapter 14

(Slide 280: The Eatwell Guide)

Just going quickly back to The Eatwell Guide.

(Slide 281: Low-Carb Diet Guide)

We've created our own. Our Low-Carb Diet Guide.

(Slide 282: Exceedingly usual behaviour)

I believe that the Metabolic Approach to Cancer should be exceedingly usual behaviour!

(Slide 283: Google search)

If you would like to know more about The Metabolic Approach to Cancer, please type "Metabolic Approach to Cancer Solihull" into Google and you will find us.

(Slide 284: The End)

Thank you all so much for your attention.

(Slide 285: Disclaimers)

I'll now throw it open to the floor for any questions or comments.

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