

Inflammation update:

Salk discovery puts mitochondria at the heart of things

Myopathy and Myalgia:

lessons from athletes that medical research is missing

A thiamine case report:

post-COVID neurological symptoms finally fixed

Niacin-heart disease

warning: plus CFS, inulin and “leaky brain” in our news and research

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**Sleep and the microbiome:
the unsuspected links
between the gut, disrupted
circadian rhythms - and
metabolic syndrome**



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Just grit your teeth and carry on

Britain's dental care system is in a mess. The public eats WAY too much sugar, and the links between poor oral health and chronic disease are now well-established. But millions of Brits can't get to see a dentist. But never fear, the Government is on it: they're adding fluoride to our drinking water in a move dubbed "the biggest fluoridation expansion since the 1980s".

We've got fluoride available in toothpaste and bags of fluoride in our tea, so I've never understood why we need to mass-medicate our drinking water. Much of Europe has rejected fluoridation, with good reason. A study from Denmark, which has the healthiest teeth on the continent, last year confirmed an association between fluoride transferred from the mother during her pregnancy to the foetus and damage to foetal brain development.

The University of Southern Denmark reported: "This association has previously been supported by a multitude of studies, but a new study led by Prof Philippe Grandjean, MD, now documents the linkage in a joint study of more than 1500 mother-child pairs from cohorts in North America, now also including births in Denmark".

Grandjean says that fluoride is beneficial when it is in contact with the enamel surface of the teeth, but that ingestion of the fluoride does not add any benefit. "Pregnant women who ingest fluoride will pass it on to the foetus without obtaining any benefit at all. In contrast, the fluoride can reach the highly vulnerable foetal brain and should therefore be minimised or fully avoided".

Public "health"

But wait...this is such a great public health idea! (Sarcasm).

So many people in the UK can't get in to see a GP (wait times are up to four weeks in some places), that pharmacists have had to be empowered to diagnose and treat some common ailments. So why not take the fluoride example to its logical conclusion? Let's have statins added to our water supply. Yes, do shudder: they're thinking about it. The idea's been floated in British medical journals since at least 2004.

And an anti-hypertensive, maybe? Oh and how about something for diabetes?

Meanwhile, medics elsewhere have revealed that rheumatoid arthritis can be prevented! I got excited when I saw the news from King's College London. Maybe they'd discovered fish oil supplements and

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eating real food. But no. It's a once-a-week INJECTION of the drug abatacept. Which is an immune suppressant. It turns down T-cell activity with the aim of blocking auto-immune activity.

Once a week injections. And they're proud of it. Prof Andrew Cope said it was "the largest rheumatoid arthritis prevention trial to date" and the first to show a treatment "effective in preventing the onset of disease in people at risk". Yes, this is pharma-driven orthodox medicine's idea of preventive medicine.

He added: "There are currently no drugs available that prevent this potentially crippling disease". Implication: only drugs can prevent disease. (By the way, Bristol-Myers Squibb, who manufacture the drug under the brand name Orencia, funded the study.)

Drugs as prevention: Big Pharma's marketing success

The idea that health is delivered at the point of a needle continues to be a sales and marketing triumph for the pharmaceutical industry. The scam they're running is the idea that humans are frail, vulnerable creatures inevitably prone to all sorts of diseases and mechanical breakdowns without life-long interventionist medical care. And once you get past 60, well you're bound to need parts replaced, too: hips, knees, you name it. Doctors seem to be brainwashed into accepting these lies from day one of medical school. Never mind the robustness and vigour inherited from millions of years of evolution that we KNOW can be conserved by a modicum of effort to personalise diet and exercise.

In Britain right now, the NHS has children scheduled to be given 31 vaccinations by the age of 15. This is NOT healthcare. This is out of control.

SIMON MARTIN, EDITOR

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“QUOTE
of the
MONTH”

"These disease processes are real, but within the current medical model the 'realness' of a disease is determined only by its connection to a pharmaceutical and the ability of the pharmaceutical to temper some surrogate marker of that disease process. When there are no pharmaceuticals that return the patient to health (because, given the chemistry, how could there be?), it is an easy jump to assume the patient is feigning their symptoms".



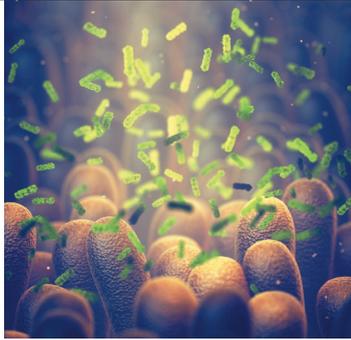
Dr Chandler Marrs, PhD, founder and editor of www.hormonesmatter.com, in her article on muscle pain and weakness in this issue.

in this issue

MARCH 2024

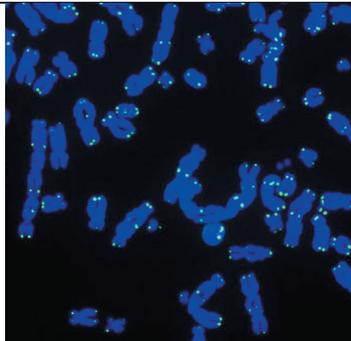
20 Sleep and the microbiome: the unsuspected links between diet, metabolic syndrome and “the delicate orchestration of sleeping patterns”.

He is that very rare individual, a clinical nutritionist who has had a review paper published in a peer-reviewed journal. Even better, his review is the first of its kind to examine the unsuspected links between gut bacteria, serious sleep disorders and metabolic syndrome. **ADRIANO DOS SANTOS** talks to *IHCAN* editor **SIMON MARTIN** about the path to publication and the implications of his ground-breaking paper, the importance of butyrate, the genesis of GABA and other neurotransmitters...and much more.



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56 Treating Myopathy and Myalgia: lessons from athletes

Can we learn some things about neuromuscular pain and injury from sports physiology that we cannot from medical research? Athlete and research scientist **Dr CHANDLER MARRS** slams the standard medical approach for presupposing “a certain futility”: the disease process is inevitable and cannot be halted or healed, just “managed”. This is not what the exercise physiology research says.



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ALEX ZODIAC describes his recovery from a post-COVID syndrome that started with exercise intolerance and progressed into some kind of neurological disturbance for which doctors had no answers.

Link between high levels of niacin and heart disease

Niacin, used even in commercial medicine to lower triglycerides and the so-called “bad” LDL cholesterol, is now said to potentially fuel inflammation and cardiovascular disease through a new pathway just discovered by Cleveland Clinic researchers.

Cleveland is saying it is “high” or “excess” levels of the B vitamin that could potentially cause problems. The Clinic has itself been using pharmaceutical versions of niacin in extended-release capsules and tablets, such as ENDUR-AMIDE, Niaspan, NiaVasc and Slo-Niacin, a chemically-synthesised OTC product rated the “number one-selling” niacin product in the US, which contains nicotinic acid in either 250mg, 500mg or 750mg strength.

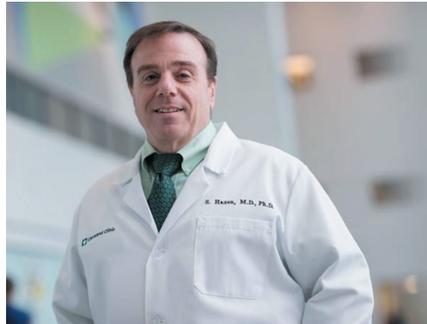
In trials, a “pharmaceutical” dose of niacin is often given in association with a statin, which has made it difficult to tease out its effects. In one study following 25,000 patients, the intervention group were not only given a statin AND niacin, but also laropiprant, a drug that was previously used to decrease the flushing caused by niacin! However, Leslie Cho, MD, Director of the Women’s Cardiovascular Centre at the Cleveland Clinic, is on record saying that niacin has only a very limited role when used judiciously in specific types of patients.

“We have known for a long time that niacin increases the risk of diabetes - it doesn’t cause diabetes, it just brings it forward”, Dr Cho says. “Niacin can also cause more gout”.

The Cleveland Clinic researchers have not published a recommendation for a “safe” dose of niacin, saying it is impossible to quantify without a blood test for the metabolite formed in response to excess doses, but team leader Stanley Hazen, MD, PhD, is nevertheless warning people not to take any supplements that have niacin in them on the grounds that as so many processed foods have niacin added, people are at risk of getting too much.

In the UK-led 25,000-patient international study co-ordinated by the University of Oxford, the dose of niacin used was a massive 2g of extended release. UK recommended daily allowances are 16.5mg for men and 13.2mg for women.

Hazen’s team has discovered a link between 4PY, a breakdown product from excess niacin, and heart disease. Higher circulating levels of 4PY were strongly associated with development of heart attack, stroke and other adverse cardiac events in large-scale clinical studies. The researchers also showed in



Be careful with niacin: Cleveland Clinic’s Dr Stanley Hazen.

preclinical studies that 4PY directly triggers vascular inflammation which damages blood vessels and can lead to atherosclerosis over time.

The study used blood samples and medical records from 503,325 people in the UK and was published in *Nature Medicine*. It also details genetic links between 4PY and vascular inflammation.

“What’s exciting about these results is that this pathway appears to be a previously unrecognised yet significant contributor to the development of cardiovascular disease”, said Dr Hazen.

“What’s more, we can measure it, meaning there is potential for diagnostic testing. These insights set the stage for developing new approaches to counteract the effects of this pathway”.

Niacin (vitamin B-3) is very common in a Western diet. “For decades, the United States and more than 50 nations have mandated niacin fortification in staple foods such as flour, cereals and oats to prevent pellagra and other niacin deficiency syndromes”, he said. The UK “fortifies” white flour with niacin, and it is relatively high in processed breakfast cereals.

Rise in CV disease

While this has successfully reduced pellagra deaths to a dramatic degree, he notes, there is now wide availability of foods naturally rich in niacin, and average dietary intake of niacin has increased to levels far exceeding recommended ranges. He wonders whether flour and cereal fortification, despite its remarkable success in preventing nutritional deficiencies since World War II, might have had the unintended consequence of contributing to the overall rise in cardiovascular disease over the past 75 years.

“In our studies, the highest quartile of 4PY levels was associated with an approximately

twofold increased incidence of major adverse cardiovascular events compared with levels in the lowest quartile”, Dr Hazen says. “One in four people in our cohorts had high levels of 4PY and are at significantly higher risk for adverse cardiovascular events”.

In light of this, he and his co-authors contend, it might be reasonable to ease niacin fortification mandates to allow for nonfortified flour and cereal options to be available.

“Those with high levels of 4PY should cut back on the carbs and eat more protein and whole foods - more of a Mediterranean style diet”.

Dr Hazen compares our intake of niacin as multiple taps pouring water into a bucket. One of those sources is tryptophan, which the body uses to manufacture niacin. Once that bucket is filled, it begins to spill over. The human body then needs to process that spill-over and produce other metabolites, including 4PY.

“The main takeaway is not that we should cut out our entire intake of niacin - that’s not a realistic approach”, he says. “Given these findings, a discussion over whether a continued mandate of flour and cereal fortification with niacin in the US could be warranted”.

Broader use of over-the-counter supplements made with different forms of niacin have also become popular because of research showing an NAD-led increase in mitochondrial biogenesis.

The new findings also might help explain why niacin is no longer a go-to treatment for lowering cholesterol. Niacin was one of the first treatments prescribed to lower LDL the so-called “bad” cholesterol. However, eventually niacin showed to be less effective than other cholesterol-lowering drugs and was associated with other negative effects and higher mortality rates in previous research.

“Niacin’s effects have always been somewhat of a paradox”, Dr Hazen said. “Despite niacin lowering of cholesterol, the clinical benefits have always been less than anticipated based on the degree of LDL reduction. This led to the idea that excess niacin caused unclear adverse effects that partially counteracted the benefits of LDL lowering. We believe our findings help explain this paradox. This illustrates why investigating residual cardiovascular risk is so critical; we learn so much more than what we set out to find”.

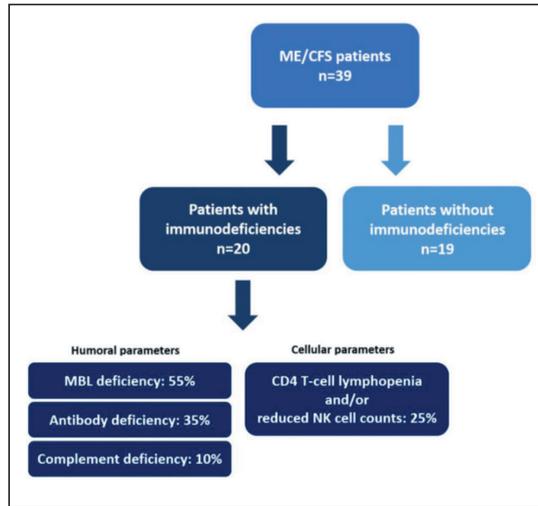
Chronic fatigue syndrome patients predicted to double due to long COVID – and leaky gut is one of the reasons

The number of ME/CFS patients is expected to rise drastically due to long-term effects of the COVID-19 pandemic, say scientists at the Medical University of Vienna, who have identified possible biomarkers that could improve the diagnosis and treatment of long-lasting and debilitating fatigue.

Up to 80,000 people in Austria are estimated to suffer from ME/CFS, or myalgic encephalomyelitis/chronic fatigue syndrome. The number of ME/CFS patients is expected to rise drastically due to long-term effects of the COVID-19 pandemic. However, research in the field has neither identified mechanisms of disease onset nor causal treatment approaches.

A new study by Eva Untersmayr-Elsenhuber and her team from MedUni Vienna’s Centre for Pathophysiology, Infectiology and Immunology builds on earlier research on immune disorders and the intestinal barrier function in patients with ME/CFS.

The team shows that ME/CFS patients can be divided into subgroups based on the function of their immune systems. The study was able to identify biomarkers in the patients that indicate immune system disorders or reduced intestinal barrier function. As a result, differences relevant to clinical care were identified in ME/CFS patients that would have remained undetected without the previous immunological stratification of the ME/CFS patient group.



“In our study, we see that the immunological evaluation of ME/CFS patients is of crucial importance. Patients suffering from immunodeficiencies are characterised by an altered innate immune function. In ME/CFS patients with an intact immune system, the intestinal barrier function was reduced”, explains Untersmayr.

“The study stratified ME/CFS patients into two groups based on their immunological status...”

* reduced C4a levels in ME/CFS patients with immunodeficiencies suggested a subgroup-specific disease pattern impairing innate immunity. It remains elusive if this deficiency contributes to disease onset due to a failure of innate immunity in situations of (viral) host

defence or is a result of the ongoing disease.

* ME/CFS patients without immunodeficiency exhibited mucosal barrier leakage, indicated by elevated levels of the marker LBP, potentially contributing to low-grade inflammation”.

According to the researchers, this not only provides a more detailed insight in different disease mechanisms, but also indicates that depending on the patient’s immune competence, some treatment approaches might be more suitable than others.

In order to advance research, the first ME/CFS Biobank in Austria is currently being set up at MedUni Vienna with the support of the WE&ME Foundation. ME/CFS Biobank Austria will collect tissue samples, which will be made available for future research

projects. The team has been coordinating with research groups in the UK, the Netherlands and Germany.

25% of those affected are bedridden

The MedUni team describes ME/CFS as a “severe multisystemic disease”. The Austrian experience is that it often leads to a high degree of disability: 60% per cent of patients are unable to work full-time and 25% are bedridden. According to current studies, between 26,000 and 80,000 people in Austria suffer from chronic fatigue. Due to COVID-19, this number could double in the next few years, they say. The links between infection with SARS-CoV-2 and ME/CFS are also the subject of intensive research.

“Use faecal transplant for recurrent C. Diff”: say new US guidelines

The American Gastroenterological Association has published the first comprehensive evidence-based guideline on the use of faecal microbiota-based therapies for gastrointestinal disease. It is recommending FMT for patients with recurrent C. Diff infection, but has stopped short of promoting its use in IBS and IBD.

Key takeaways:

- Prevention with faecal microbiota-based therapies can be considered in patients after the second recurrence (third episode) of *C. difficile* infection (CDI) or in select patients at high risk for either recurrent CDI or a morbid CDI recurrence.
- In adults hospitalised with severe or fulminant CDI not responding to antimicrobial therapy, consider select use of



conventional FMT as adjuvant treatment.

- Conventional faecal microbiota transplant as treatment for IBD or IBS should only be considered in the context of clinical trials.

“As a general gastroenterologist, this guideline has changed my practice. I now have a much better understanding of FMT and how to position faecal microbiota-based therapies in practice. This guideline has made me a better doctor for my *C. diff* patients”.

The guideline covers the use of conventional faecal microbiota transplant, performed most commonly via colonoscopy, as well as recently FDA approved therapies such as live faecal microbiota delivered via enema and live faecal microbiota spores delivered in an oral capsule.

Inulin positively alters microbiome in four weeks

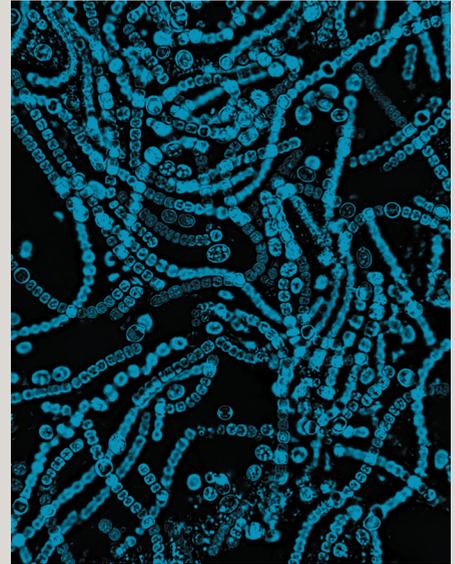
While fructans is supposedly bad for IBD, being one of the fermentable carbohydrates avoided in the therapeutic low-FODMAP diet, a new study has found that supplementing with inulin-type fructans may be good for children whose IBD is as yet sub-clinical.

The inulin prebiotics increased faecal and mucosal bifidobacteria as well as increasing levels of butyrate-producing bacteria. The researchers tracked short-term alterations in the microbiome of children with IBD - finding an enrichment of Bifidobacteria and the key butyrate producer Anaerostipes.

The US team ran a double-blind, placebo-controlled trial at the Children's Hospital of Philadelphia. Participants included children, 6-21 years old, with subclinical active colonic IBD as defined by a faecal

calprotectin levels and clinical remission. Participants were randomly assigned 1:1 to consume prebiotic oligofructose-enriched inulin (OI) or maltodextrin placebo for eight weeks.

"The global faecal and mucosal microbiome composition was significantly altered by prebiotic intake and specific inulin-induced increase in relative abundance of Bifidobacterium and Anaerostipes in the faecal samples were identified at week four", the team reported. It was led by senior author Lindsey Albenberg, DO, from the Centre for Paediatric Inflammatory Bowel Disease at Children's Hospital of Philadelphia. Two months after discontinuation of OI, microbial composition returned to baseline levels.



Why inulin is a premier prebiotic: Vienna study on personalised dietary supplements

A groundbreaking study led by David Berry and Alessandra Riva from the Centre for Microbiology and Environmental Systems Science (CeMESS) at the University of Vienna claims to have "significantly advanced" our understanding of prebiotics in nutrition and gut health. The study, published in *Nature Communications*, reveals the extensive and diverse effects of inulin on the human gut microbiome.

The researchers used fluorescence-labelled nanoparticles to track the interaction of inulin with gut bacteria. These inulin-grafted nanoparticles, when incubated with human stool samples, yielded a surprising result: a wide range of gut bacteria, far more than previously assumed, can bind to inulin.

"Most prebiotic compounds are selectively utilised by only a few types of microbes", explains David Berry, the lead researcher. "But actually, we found that the ability to bind to inulin is really widespread in our gut microbiota". Using a state-of-the-art technique to identify cells actively synthesising proteins, the team discovered that a diverse group of bacteria actively responds to inulin, including some species not previously associated with this capability, such as members of the Coriobacteriia class.

"Inulin supplements have been on the market for years, but precise scientific evidence of their health-promoting effects has been lacking", says Berry. "We used to think that inulin mainly stimulates Bifidobacteria, the so-called 'good bacteria,' but now we know that the effect of inulin



"We used to think that inulin mainly stimulates Bifidobacteria, the so-called 'good bacteria,' but now we know that the effect of inulin is much more complex. Our study is a trailblazer for the future of microbiome-based medicine: with our method, dietary supplements can be personalised, precisely designed, and scientifically substantiated in the future". - Prof David Berry, Department of Microbiology and Ecosystem Science, Division of Microbial Ecology, University of Vienna.

is much more complex. Our study is a trailblazer for the future of microbiome-based medicine: with our method, dietary supplements can be personalised, precisely designed, and scientifically substantiated in the future".

Every person's microbiota reacts differently

"Interestingly, when comparing stool samples from different individuals, we noticed significant differences in the microbial communities that respond to inulin", says Alessandra Riva, also a leader of the study. "These findings highlight the importance of considering individual differences in the development of dietary recommendations and microbiome-based interventions", she explains. The CeMESS research not only contributes to a better understanding of prebiotic metabolism

in the human digestive tract but also to a better framework for its investigation. "Our approach to marking and sorting cells based on their metabolic activity is relatively new", says Riva.

Prebiotics are defined as "non-digestible food components that promote the growth of beneficial microorganisms in the gut". Inulin, one of the most popular commercial prebiotics, is naturally abundant in foods such as bananas, wheat, onions and garlic. When we consume these foods, inulin reaches our large intestine, where it is broken down and fermented by gut bacteria.

Studies have shown that inulin may have positive effects on human health, such as anti-inflammatory and anti-cancer properties. However, the complex nature of the human gut, home to about 100 trillion microbes, poses a challenge in deciphering the exact effects of supplements like inulin.

Fasting-mimicking diet reduces human's biological age 2.5 years: a new Longo study

A new University of Southern California study led by Dr Valter Longo shows how cycles of a fasting-mimicking diet reduce insulin resistance, liver fat, immune system ageing, and biological age in clinical trial patients.

The study, published in *Nature Communications*, adds to the body of evidence supporting the beneficial effects of the fasting-mimicking diet (FMD).

The FMD is a five-day diet high in unsaturated fats and low in overall calories, protein and carbohydrates, and is designed to mimic the effects of a water-only fast while still providing necessary nutrients and making it much easier for people to complete. The diet was developed by the laboratory of USC Leonard Davis School Prof Valter Longo, the senior author of the new study.

“This is the first study to show that a food-based intervention that does not require chronic dietary or other lifestyle changes can make people biologically younger, based on both changes in risk factors for ageing and disease and on a validated method developed by the Levine group to assess biological age”, Longo said.

Previous research led by Longo has indicated that brief, periodic FMD cycles are associated with a range of beneficial effects. They can:

- Promote stem cell regeneration: 2015 study in *Cell Metabolism* involving yeast and mice, then a pilot clinical trial in “generally healthy adults” concluded, “FMD cycles induce long-lasting beneficial and/or rejuvenating effects on many tissues including those of



A new University of Southern California study led by Dr Valter Longo shows how cycles of a fasting-mimicking diet reduce insulin resistance, liver fat, immune system ageing, and biological age in clinical trial patients.

the endocrine, immune, and nervous systems in mice and in markers for diseases and regeneration in humans”.

- Lessen chemotherapy side-effects: 2020 paper in *Nature Communications* showed that “Short-term fasting protects tumour-bearing mice against the toxic effects of chemotherapy while enhancing therapeutic efficacy”.

- Reduce the signs of dementia in mice: “FMD cycles delay cognitive decline in AD models in part by reducing neuroinflammation and/or superoxide production in the brain” reported a *Cell Reports* paper published in 2022.

In addition, in 2017 research, Longo and colleagues “compared subjects who followed three months of an unrestricted diet to subjects who consumed the FMD for five consecutive days per month for three months. Three FMD cycles can lower the risk factors for cancer, diabetes, heart disease and other age-related diseases in humans”.

The Longo lab also had previously shown

that one or two cycles of the FMD for five days a month increased the healthspan and lifespan of mice on either a normal or Western diet, but the effects of the FMD on aging and biological age, liver fat, and immune system aging in humans were unknown until now.

Lower disease risks and more youthful cells

The new study analysed the diet's effects in two clinical trial populations, each with men and women between the ages of 18 and 70. Patients who were randomised to the

fasting-mimicking diet underwent three-four monthly cycles, adhering to the FMD for five days, then eating a normal diet for 25 days.

The FMD comprises proprietary formulations belonging to USC and its associated company L-Nutra (www.prolonfmd.com) of vegetable-based soups, energy bars, energy drinks, chip snacks, tea, and a supplement providing high levels of minerals, vitamins, and essential fatty acids., portioned out for five days. Daily foods are individually boxed to allow the subjects to choose when to eat while avoiding accidentally consuming components of the following day. Patients in the control groups ate either a normal or Mediterranean-style diet.

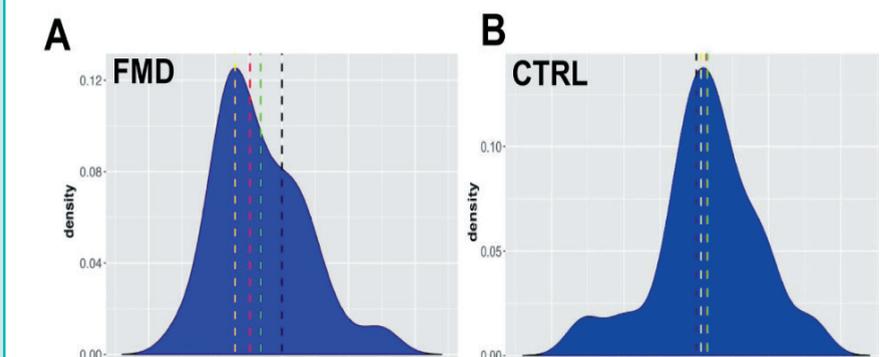
An analysis of blood samples from trial participants showed that patients in the FMD group had lower diabetes risk factors, including less insulin resistance and lower HbA1c results. Magnetic resonance imaging also revealed a decrease in abdominal fat as well as fat within the liver, improvements associated with a reduced risk of metabolic syndrome. In addition, the FMD cycles appeared to increase the lymphoid-to-myeloid ratio - an indicator of a more youthful immune system.

Further statistical analysis of the results from both clinical studies showed that FMD participants had reduced their biological age - a measure of how well one's cells and tissues are functioning, as opposed to chronological age - by 2.5 years on average.

“This study shows for the first time evidence for biological age reduction from two different clinical trials, accompanied by evidence of rejuvenation of metabolic and

Fig. 3: FMD reduces biological age between enrollment and completion of trial independent of weight loss.

From: [Fasting-mimicking diet causes hepatic and blood markers changes indicating reduced biological age and disease risk](#)



immune function”, Longo said.

“Although many doctors are already recommending the FMD in the United States and Europe, these findings should encourage many more healthcare professionals to recommend FMD cycles to patients with higher than desired levels of disease risk factors as well as to the general population that may be interested in increased function and younger age”, he said.

“I always say that fasting doesn’t really mean anything. Fasting is like saying eating. As we all know now, if you say, ‘Oh, is eating good for you?’ Well, yes and no. So it can be very good or very bad. It depends on what you eat, and fasting is the same way. It can be very good or very bad, depending on what you do, how long you do it for, who you are, etc. So, we’d been trying to move away from words that don’t mean anything, like intermittent fasting, and really started saying, ‘Pay attention, be careful’, because fasting-based intervention can be very powerful or very damaging”. - Prof Valter Longo, PhD, Director of the Longevity Institute at UCLA.

Fasting and calorie restriction keys to offset ageing

Prof Valter Longo’s studies focus on the fundamental mechanisms of ageing in simple organisms and mice and on how these mechanisms translate to humans.

The Longo laboratory has identified some of the key genetic pathways that regulate ageing in simple organisms and has demonstrated that the inactivation of such pathways can reduce the incidence or progression of multiple diseases in mice and humans. His laboratory has also developed both dietary and genetic interventions that protect normal cells while sensitising cancer cells to chemotherapy - interventions now being tested in many US and European hospitals.

Dr Longo is Professor of Gerontology and Biological Sciences and Director of the Longevity Institute at the University of Southern California Los Angeles, one of the leading centres for research on ageing and age-related disease. Dr Longo is also the Director of the Longevity and Cancer Program at the IFOM Institute of Molecular Oncology in Milan.

He has said: “We can argue, based on over 100 years of research, that nutrition is by far the most powerful way to alter how fast we age, and also alter whether we will develop age-related disease like diabetes, cancer, cardiological and neurodegenerative



diseases”.

The Longo laboratory has previously published key findings on a five-day periodic dietary intervention called the Fasting Mimicking Diet (FMD), and showed in randomised clinical trials that FMD reduces the risk factors and markers

associated with ageing and diseases. Dr Longo’s most recent studies focus on the use of FMD interventions to activate stem cell- based regeneration to promote longevity.

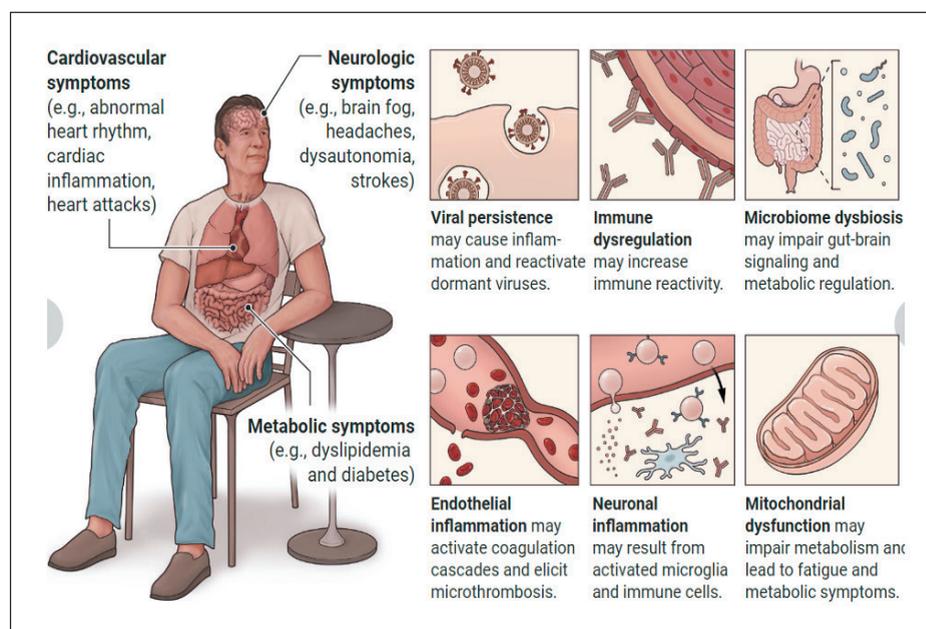
Prof Longo’s core objectives are: to offer treatment and other health services to patients with serious diseases and to those who seek to halt the onset of such diseases; to educate the public - both adults and youth - about how to live a long and healthy life; to sponsor research to develop innovative and creative treatment strategies that are affordable and accessible to all; and to identify ways to prevent specific diseases.

To achieve these goals, he donates all profits from his books to research and programs, made possible by his foundations, Fondazione Valter Longo in Milano and Create Cures Foundation in Los Angeles. These books include the best seller *The Longevity Diet* and the two Italian books *Alla tavola della longevità* (*At the Table of Longevity*), and *La longevità inizia da bambini* (*Longevity Begins in Childhood*).

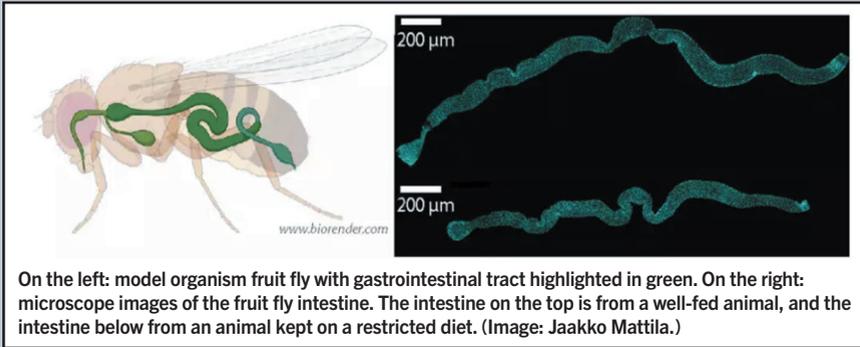
Solving the puzzle of Long COVID

“The pandemic has laid bare a blind spot in epidemiology and surveillance...This approach does not account for the burden of long-term health loss due to infectious illnesses, which obscures their true toll”.

“Long COVID is a multisystemic disease with sequelae that affect almost all organ systems. Various putative mechanisms that underlie these sequelae are not mutually exclusive and may explain the myriad health effects seen in long COVID. Therapeutics that target these pathways, such as antivirals, anti-inflammatory agents, microbiome restoration and anticoagulant drugs, may ameliorate symptoms”. - A. Mastin/*Science*.



Intermittent fasting fights ageing through effects on stem cells



On the left: model organism fruit fly with gastrointestinal tract highlighted in green. On the right: microscope images of the fruit fly intestine. The intestine on the top is from a well-fed animal, and the intestine below from an animal kept on a restricted diet. (Image: Jaakko Mattila.)

The capacity of intestinal stem cells to maintain cellular balance in the gut decreases upon ageing. Researchers at the University of Helsinki have discovered a new mechanism of action between the nutrient adaptation of intestinal stem cells and ageing. The finding may make a difference when seeking ways to maintain the functional capacity of the ageing gut.

Intermittent fasting prolongs animal lifespan and the researchers now believe this is because stem cell function is preserved.

The Helsinki team says the cellular balance of the intestine is carefully regulated, and it is influenced, among other things, by nutrition: ample nutrition increases the total

Intestinal cells are in direct contact with ingested food and the gut microbiota. They are therefore susceptible to damage, requiring continuous renewal. Intestinal stem cells maintain tissue renewal by proliferating and differentiating, thus replacing dead or damaged cells in the gut. With ageing, the ability of stem cells to regenerate the cells of the gut is reduced, resulting in the loss of tissue function or uncontrolled cell growth, which can predispose, among other things, to tumour formation.

number of cells in the gut, whereas fasting decreases their number.

The relative number of different types of cells also changes according to nutrient status.

The questions of how the nutrition status of the gut controls stem cell division and differentiation, and how the nutrient adaptation of stem cells changes as during ageing have not been comprehensively answered. Nutrient adaptation refers to the way in which nutrients guide cell function.

The Helsinki group identified a new regulatory mechanism that directs the differentiation of intestinal stem cells under a changing nutrient conditions. Cell signalling activated by nutrients increases the size of stem cells in the fruit fly intestine. The size of the stem cells, in turn, controls the cell type into which the stem cells differentiate.

For efficient stem cell function, flexible regulation of their size is essential. The size of the cells dynamically increases or decreases, depending on the dietary conditions. Such flexibility enables stem cells to differentiate in accordance with the prevailing nutrient status.

The researchers found that the nutrient adaptation of stem cell size and the resulting

differentiation vary in different regions of the gut.

"Our observations demonstrate that the regulation of intestinal stem cells is much more region-specific than previously understood. This may be relevant to, for example, how we think about the pathogenetic mechanisms of intestinal diseases", says Jaakko Mattila, from the Faculty of Biological and Environmental Sciences.

Intermittent fasting may benefit intestinal stem cells

The researchers also observed that the ability of intestinal stem cells to react to a changing nutrient status is greatly reduced in older animals. They also found that, in older animals, stem cells are in a state where they are constantly large in size, which restricts their ability to differentiate.

With ageing, flexible regulation of stem cell size was markedly better preserved in animals kept on intermittent fasting.

In the past, intermittent fasting has been shown to prolong the lifespan of animals, and the results now obtained indicate that the improved preservation of stem cell function may underlie this prolongation.

According to the researchers, the mechanisms associated with the functioning, nutrient adaptation and ageing of human and fruit fly stem cells are fairly similar. "We believe that these findings have a broader significance towards understanding how to slow down the loss of tissue function caused by ageing by controlling the nutrient adaptation of stem cells. However, more information is needed on the effect of the mechanism on human intestinal stem cells. Our work on the nutrient adaptation of stem cells continues", says Prof Ville Hietakangas.

Medics finally wake up to dangers of junk food

They're still so afraid of offending the food industry that they can't say "junk food", but pharmaceutical-led medics are beginning to wake up to its dangers.

Physicians from Florida Atlantic University's Schmidt College of Medicine have taken a giant leap (for them) in asking, "Could ultra-processed foods be the new 'silent' killer?"

Hundreds of novel ingredients never encountered by human physiology are now found in nearly 60% of the average adult's diet and nearly 70% of children's diets in the US. The Florida doctors are late to the party but say in a commentary in the *American*

Journal of Medicine, "An emerging health hazard is the unprecedented consumption of these ultra-processed foods in the standard American diet. This may be the new 'silent' killer, as was unrecognised high blood pressure in previous decades"..

"Those of us practising medicine in the US today find ourselves in an ignominious and unique position - we are the first cohort of health care professionals to have presided over a decline in life expectancy in 100 years", said Dawn Sherling, MD, an associate professor of medicine.

Ultra-processed foods contain emulsifiers and other additives that the GI tract mostly

does not digest. They may be creating a dysbiotic microbiome that can promote disease.

"Additives such as maltodextrin may promote a mucous layer that is friendly to certain bacteria that are found in greater abundance in patients with inflammatory bowel disease", said Sherling. "When the mucous layer is not properly maintained, the epithelial cell layer may become vulnerable to injury, as has been shown in feeding studies using carrageenan in humans and other studies in mice models, using polysorbate-80 and cellulose gum, triggering immunologic responses in the host".



Liver health check



Understand your clients' liver performance by identifying a precise liver score.

THE LIVER CLINIC™



Liver health is fast becoming the new pressing health trend, so Equilibrium Labs has recently launched its Liver Clinic, helping give everyone fast access to rare diagnostic technologies to help address the growing problem of fatty liver, which is today costing the NHS more than £17 billion annually (according to the Royal Free), almost 10 per cent of their budget.

The good news is that non-alcoholic fatty liver disease (NAFLD) is preventable and reversible if you catch it in time. Through a combination of more accurate testing centring on FibroScan (specialised ultrasounds), as well as blood tests and lifestyle changes, you can help your clients

heal this master organ and reverse NAFLD.

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The power of testing

This unique offering gives people the power to diagnose, strengthen, heal and restore their liver by making informed choices to suit their lifestyle. They can then live a life where all of their interconnected systems work harmoniously together.

Dr Federica Amati, PhD in Clinical Medicine, Masters in Public Health, AfN Registered Nutritionist and Chief Science Officer at Equilibrium Labs, commented: “Early detection of a struggling liver with a simple, non-invasive scan like FibroScan is the perfect example of an effective preventative tool to help people reverse the damage done and work towards keeping this crucial organ healthy. Early pre-clinical and observational results show that de-

liver-ance could offer a real solution to help kick-start the reversal of fatty liver disease with measurable improvement, which is not only rare but also incredibly exciting and promising.”

Equilibrium Labs has a commitment to creating the best products and services to address fatty liver and to help them deliver on their proposition, the company has joined forces with The Private GP Group and selected Randox clinics to help extend their offering of making liver screening accessible to all.

By combining their powerful, natural, liquid elixir de-liver-ance with accurate testing, technology and nutritional/lifestyle advice, Equilibrium Labs is changing the game in helping to reverse liver damage and optimise this master organ and overall health.



If you would like to explore the possibility of becoming a Liver Clinic partner, please contact info@theliverclinic.com or visit <https://theliverclinic.com/>

“Leaky brain”: Trinity team discovers underlying cause of brain fog linked with long COVID - new scans provide objective proof

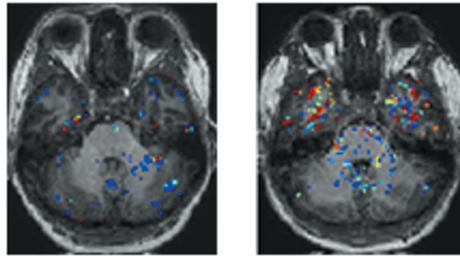
A team of scientists from Trinity College Dublin and investigators from FutureNeuro have shown that there is a disruption to the integrity of the blood vessels in the brains of patients suffering from long COVID and experiencing brain fog.

This blood vessel “leakiness” was able to objectively distinguish those patients with brain fog and cognitive decline compared to patients suffering from long COVID but without brain fog.

The team led by scientists at the Smurfit Institute of Genetics in Trinity’s School of Genetics and Microbiology and neurologists in the School of Medicine, reporting in *Nature Neuroscience*, have also uncovered a novel form of MRI scan that shows how long COVID can affect the human brain’s delicate network of blood vessels.

“For the first time, we have been able to show that leaky blood vessels in the human brain, in tandem with a hyperactive immune system, may be the key drivers of brain fog associated with long COVID. This is critically important, as understanding the underlying cause of these conditions will allow us to develop targeted therapies for patients in the future”, said Prof Matthew Campbell, Professor in Genetics and Head of Genetics at Trinity, and Principal Investigator at

Brain Fog (-) Brain Fog (+)



MRI scans in patients with long COVID with or without “brain fog”. Increased “leakiness” of the blood vessels is seen (here in the temporal lobe region of the brain) in those patients with brain fog (indicated by red signal).

FutureNeuro.

This project was initiated by a rapid response grant funded by Science Foundation Ireland (SFI) at the height of the pandemic in 2020 and involved recruiting patients suffering from the effects of long COVID as well as patients who were hospitalised in St James’ Hospital.

“The findings will now likely change the landscape of how we understand and treat post-viral neurological conditions. It also confirms that the neurological symptoms of long Covid are measurable with real and demonstrable metabolic and vascular changes

in the brain”, said Prof Colin Doherty, Professor of Neurology and Head of the School of Medicine at Trinity.

Beyond COVID-19

Prof. Campbell added: “Here, the team at Trinity was able to prove that every patient that developed long COVID had been diagnosed with SARS-CoV-2 infection, because Ireland required every documented case to be diagnosed using the more accurate PCR-based methods. The concept that many other viral infections that lead to post-viral syndromes might drive blood vessel leakage in the brain is potentially game-

changing and is under active investigation by the team”. That research may include multiple sclerosis, where a viral infection is also thought to play a part.

Dr Chris Greene, Postdoctoral research fellow and first author of the study, added: “Our findings have now set the stage for further studies examining the molecular events that lead to post-viral fatigue and brain fog. Without doubt, similar mechanisms are at play across many disparate types of viral infection, and we are now tantalisingly close to understanding how and why they cause neurological dysfunction in patients”.

Sandalwood oil by-product prevents prostate cancer development in mice

Researchers from Florida Atlantic University’s Schmidt College of Medicine and collaborators are the first to demonstrate *in vivo* the chemo-preventive properties of alpha-santalol against prostate cancer development using a transgenic mouse model.

Sandalwood oil has been used worldwide for centuries. Extracted from the core of sandalwood trees (*Santalum album*), in addition to containing esters, free acids, aldehydes, ketones and santenone, sandalwood oil is primarily (90% or more) made up of santalol – in equal amounts of two compounds, alpha and beta-santalol.

The study is the first to demonstrate that administering alpha-santalol reduced visible prostate tumours, protected the normal tissue, and delayed progression from a precancerous condition to a high-grade form of cancer.

The study, published in *Phytomedicine Plus*, showed that administration of alpha-santalol decreased the incidence of prostate

tumours by decreasing cell proliferation and inducing apoptosis, without causing weight loss or any noticeable side-effects.

Findings revealed that the area occupied by normal tissue in alpha-santalol-treated mice was 53% compared to 12% in control mice. This suggests that alpha-santalol protected the normal tissue and delayed progression from prostatic intraepithelial neoplasia, a precancerous condition, to poorly differentiated carcinoma, a high-grade form of cancer where cancer cells and tissue look very abnormal.

Mortality in prostate cancer patients is mainly attributable to advanced stages of the disease.

In previous studies, the researchers demonstrated the efficacy of alpha-santalol in suppressing growth and inducing apoptotic cell death in cultured human prostate cancer cells. Based on these observations, they selected a genetically engineered mouse model that resembles many features similar to human prostate

cancer, eliciting different lesion grades and cancer progression.

“Although our cellular studies provided important mechanistic insights, relevant *in vivo* models are vital for developing novel chemo-preventive agents for clinical use and to determine if alpha-santalol offers protection against prostate cancer development”, said Ajay Bommareddy, PhD, associate professor of pharmacology.

Additional findings showed alpha-santalol reduced the incidence of visible prostate tumours compared to control-treated mice.

In the UK, prostate cancer is the most common cancer in men. Prostate Cancer UK says about 1 in 8 men will get prostate cancer in their lifetime. More than 52,000 men are diagnosed with prostate cancer every year, and 12,000 men die from it – that’s one death every 45 minutes.

“Identifying agents that have the ability to selectively target cancerous cells and delay onset and progression of prostate cancer is greatly needed”, said Bommareddy.

World first: restoring the gut-brain axis — through the virome

Last year APC Microbiome Ireland scientists produced an atomic-level structure of a novel virus that attacks and kills bacteria in the human gut (see picture, right). These highly abundant viruses can account for up to 95% of gut viruses in some individuals. Researchers at APC collaborated with scientists at the University of York to complete the first ever structural atlas of a crassvirus (also referred to as crassvirales, bacteriophage, crass-like phage) – the most abundant group of viruses in the human gut, which can play an important role in shaping the gut microbiome, impacting health and disease.

A study by APC Microbiome Ireland SFI Research Centre reveals the virome is a viable treatment option to restore the microbiome-gut-brain axis during stress. The findings pave the way for new treatments for stress-related disorders.

The researchers based in University College Cork (UCC) have made what is claimed to be a world-first discovery in the role the human virome plays in stress management. The findings support the development of potential treatments to reduce stress and stress-related disorders through targeting the virome, the vast community of viruses hosted by every human body.

With one in eight people living with a mental disorder across the globe according to the WHO, stress-related psychiatric disorders such as depression and anxiety are extremely prevalent and are significant societal burdens.

Although most people think of viruses in their negative context, there is a rich ecosystem of other viruses known as bacteriophages that

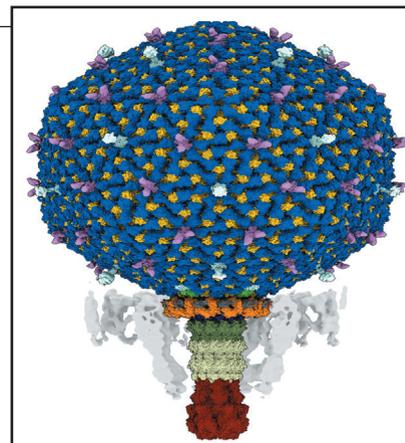
infect bacteria including pathogenic bacteria and can play a vital role in maintaining our health and well-being.

Over the past decade a new burgeoning field of research studying the human microbiome has taught us that the microbes living in our guts play a pivotal role in many important aspects of our physical and mental health. Indeed it has been shown that the composition of bacteria in the gut changes with stress and that targeting these bacteria may dampen down the effects of stress in animals and humans. However, up to now all the focus has been on bacteria and viruses have been neglected.

Dr Nathaniel Ritz, first author on the study, said: “Although bacteria in the gut have been the subject of growing research, the way the virome interacts with bacteria and how they affect stress-related health and disease status is largely unexplored. Our research focuses on this and highlights a link between

stress-related behaviours and the gut bacteriome and virome which opens up the potential to target the virome to treat and reduce the effects of stress”.

Prof John Cryan and colleagues teamed up with a leading bacteriophage lab of Prof Colin Hill at APC to decode the relative contribution of these gut viruses to stress. The study published in *Nature Microbiology* found for the first time that chronic stress led to changes in virome composition that was associated with behavioural, immune and bacteriome alterations. It supports increasing evidence that brain-gut interactions may play a key role in how stress is regulated. Understanding the biological consequences of chronic stress offers



Molecular surface of virion reconstruction
Source: Bayfield et al/*Nature* 2023.

potential to develop alternative, new therapies for stress-related disorders.

Next, they showed that through a faecal virome transplant (FVT), whereby they harvested viruses from healthy animals prior to stress and returned then to the stressed gut, was able to exert remarkable outcomes in preventing stress-associated behavioural and physiological outcomes. The research provides strong causal evidence that the viral component of the microbiome can be used to restore the microbiota-gut-brain axis during stress and a potential treatment strategy for stress-related psychopathologies.

He also cautions that “these are mice studies and validation in humans is now required to assess the potential of the FVT approach. However, given that the virome composition varies greatly among individuals, it may open the door for personalised medicine approaches for stress-related disorders in the future. We must acknowledge that not all viruses are bad, and they can play a key role in keeping the keep bad bacteria in our gut at bay, especially in times of stress”.

New microbiome tome from APC Ireland

Scientists at APC Microbiome Ireland are celebrating a new textbook.

It's the second edition (first edition was 2016) of *The Gut-Brain Axis Dietary, Probiotic, and Prebiotic Interventions on the Microbiota*.

It is edited by researchers Dr Niall Hyland and Prof Catherine Stanton from APC Microbiome Ireland, University College Cork and Teagasc. The authors presented a copy of the book (above) to Coral Black, University Librarian at University College Cork.

“The new volume brings together outstanding authors not only from around the world, but also from a breadth of scientific disciplines including neuroscience, microbiology, gastroenterology, nutrition, mental health



and exercise biochemistry. The 2nd edition is a timely update to the first reflecting the pace at which the microbiome field has accelerated since the 1st edition was published over five years ago. New models have been developed allowing further exploration of the microbiome-gut-brain

axis, providing more mechanistic insight than ever before, as we begin to see the evolution of investigative and pre-clinical findings translate into human trials. Reflecting this progress, the 2nd edition includes a case study on such translational success.

“Although the publication is targeted toward a scientific and medical readership, introductory chapters such as Correlating the Gut Microbiome to Health and Disease provide an accessible resource for those looking for an introduction to the microbiota-gut-brain axis. The second edition re-examines the microbiota-gut-brain axis across the lifespan, from early life to aging and brings up-to-date viewpoints and perspectives on how the microbiome interacts with the gut and the brain.”



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Saturday 22 June 2024 – LIVE at 155 Bishopsgate, London Headlined by Dr Leo Pruimboom and Dr Malcolm Kendrick

Dr Leo Pruimboom, PhD, a pioneer in clinical psychoneuroimmunology who earned a standing ovation for his talk at last November's IHCAN Summit, returns alongside GP and author of *The Clot Thickens*, Dr Malcolm Kendrick. He'll present on the theme: "How blood clots cause heart disease - the 170-year-old 'brand new' hypothesis". *Two more speakers to be confirmed.*

Saturday 21 September 2024 – VIRTUAL Headlined by Dr Dale Bredesen, Dr Allison Siebecker, Dr Cate Shanahan

We know you can't get enough of Dr Dale Bredesen, MD, the first to publish case reports documenting patients recovering from dementia and Alzheimer's - the disease for which there is allegedly "no cure". Dr Bredesen has proven otherwise with a personalised program inspired by functional medicine and heavily dependent on intervention with diet and supplements.

True to form, Dale has a LOT to discuss and update us on. This includes:

- New blood tests such as p-tau 217. These will allow us to "see it coming" and also to follow success.
- New trial - "we are a third through now" - at

six sites. What they are learning.

- New book - out next year.
- A unifying theory of neurodegenerative diseases, allowing us to treat more than just AD.
- New program - Precision Brain Health for Neurodegenerative Disease - at Pacific Neuroscience Institute. First of its kind.
- Atypical presentations of Alzheimer's, such as PCA and PPA.
- Therapeutic additions - what is working and what is not?

Dale told us: "Part of what we'll discuss will be that we have entered a 'Golden Age', with numerous advances, making Alzheimer's optional (believe it or not), and allowing us all to reduce the global burden of dementia.

As if that wasn't inspiring enough, we've teamed Dale with two giants of the nutritional therapy world - "SIBO Queen", naturopathic physician, Dr Allison Siebecker, ND; and Dr Cate Shanahan, MD, author of the amazing *Deep Nutrition*, an expert on dietary fats and seed oils. *One speaker to be confirmed.*

Saturday 16 November 2024, LIVE at 155 Bishopsgate, London, headlined by Dr David Unwin

More to update - this time in the world of blood sugar control and diabetes. We'll be hearing from "Low-carb GP" Dr David Unwin, whose "conventional" GP practice has a staggering success rate with putting people with Type 2 diabetes into remission. His clinical audits show diabetics on a low-carb lifestyle losing on average 10kg in weight, reducing HbA1c and seeing significant reductions in LDL cholesterol, triglycerides and blood pressure.

Dr Unwin is joined by Lucinda Miller, a specialist in children's health, autism and ADHD, and appearing for the first time, the compelling Adriano dos Santos, a clinical nutritionist and MSc sleep medicine researcher, who has just published the first systematic review on the microbiome, sleep and metabolic syndrome. Read his interview in this issue!

And remember, our LIVE event tickets don't just include world-class presenters, but also a large expo with some of the industry's biggest brands showcasing their new products and research, an "energizer" gluten-free breakfast, a three-course gluten-free buffet lunch, all your refreshments throughout the day, a goody box from our friends at The Natural Dispensary, and all the video recordings and audio downloads sent to you after the event. And you get the chance to network with practitioner friends, colleagues and new acquaintances.

- Book your early-bird ticket today at ihcansummit.co.uk or telephone 01279 810080 (normal BT landline rates apply). Tickets are limited and sold on a first-come-first-served basis.

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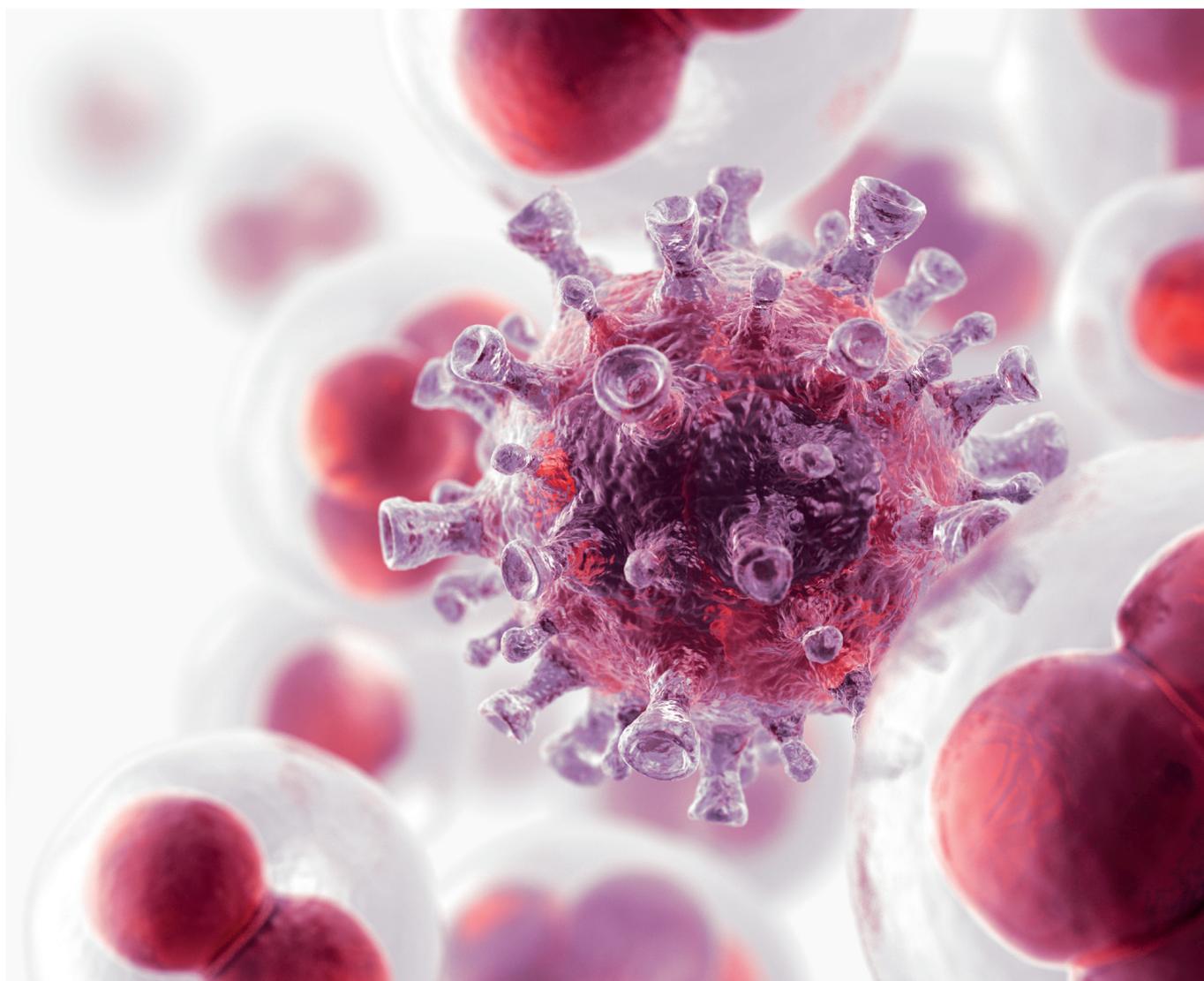
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Thursday, 25th	Bury St Edmunds
Thursday, 25th	York

June

Thursday, 6th	Bristol
Thursday, 6th	Norfolk
Thursday, 13th	Birmingham
Thursday, 13th	Newcastle
Thursday, 20th	London

May

Thursday, 2nd	Edinburgh
Thursday, 2nd	St Albans
Thursday, 16th	Belfast
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Thursday, 23rd	Brighton
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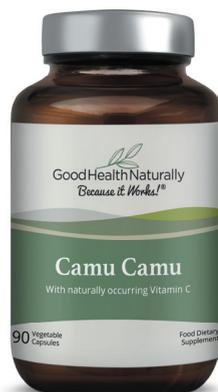


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Sleep and the microbiome: the unsuspected links between diet, metabolic syndrome and “the delicate orchestration of sleeping patterns”

He is that very rare individual, a clinical nutritionist who has had a review paper published in a peer-reviewed journal. Even better, his review is the first of its kind to examine the unsuspected links between gut bacteria, serious sleep disorders and metabolic syndrome. **ADRIANO DOS SANTOS** talks to *IHCAN* editor **SIMON MARTIN** about the path to publication and the implications of his groundbreaking paper, the importance of butyrate, the genesis of GABA and other neurotransmitters...and much more.



Nothing about Adriano dos Santos is particularly “ordinary” - a Brazilian-born nutritionist with a practice in the Hague, currently doing online consultations from the south of France while pursuing an MS in sleep medicine at the University of Bern, Switzerland, lined up for a three-month Fellowship at the University of Oxford with the rock star of sleep and circadian rhythm research Prof Russell Foster - but how exactly does an “ordinary” nutritional therapist manage to get a review paper published in a leading journal? Before we get to the paper itself - which is fascinating - I want to know how he made that happen and what his background is.

He completed a degree in sports science at the University of Sao Paulo, aged 21, then moved to London to follow a fascination with nutrition, training at Kingston University. “That’s where I actually immersed myself in clinical nutrition, physiology, biochemistry... I’d always been really fascinated about our biochemistry, and how our bodies are so cleverly designed, in a way to correct themselves”, he says. But after graduating in 2011, he realised there was still so much more to learn - “And there’s so many things that are unexplained in the orthodox view”. So he went straight in to “extra-curricular” disciplines such as functional medicine.

For the last five years he’s been completely focused on nutritional therapy, and especially on research into metabolic syndrome, nutrigenomics and gut health. During COVID time he met up with the “inspiring” Iris Kee, a CNM and IFM trained nutritional therapist also based in the Netherlands, who steered him towards the IFM education offerings. And it was post-COVID that he noticed a pattern in his patients that took him down another rabbit hole.

“It was just after COVID, and I started to wonder why I’ve been seeing all these patients suffering with sleep disruption. Not formal sleep disorders, which some of them may have had. Even though we were measuring methylation, endocrine, detoxification, digestion, the immune system, and really getting them well-balanced, they were still facing sleep disruption”. A naturally curious person, Adriano went after a deeper understanding of the brain-gut connection. He applied to universities offering a masters degree in neuroscience, or what they call “sleep medicine”. He was accepted by Oxford and Bern, but made a “hard choice” of Bern due to the geographical advantages. Your mileage may vary, but it shows where a nutrition degree might take you.

“Both of them are orthodox”, he says, “so you’re going to be in the same field, exploring →

→ neurophysiology and neuro-anatomy. And what struck me was that I was the only nutritionist in the cohort of neurologists, doctors and even nurses. So that's been kind of very exciting, however, very challenging as you can imagine".

So sports science, to nutrition degree to functional medicine, to very conservative, orthodox neurophysiology at Bern. "My motto is to never squander opportunities", he says. His thinking was "just immerse yourself in the knowledge you get there, and just bring to your own clinical practice approach, which is functional medicine, and integrate that, not just listen to those - I keep saying 'dinosaurs' of doctors - preaching what we know already, waiting for some double-blind clinical trials..." But he was prepared to put up with that, because he'd registered the value of studying the mechanisms underlying the gut, brain, sleep axis. "It's so important, and I think even more important nowadays, with so much sleep disruption going on in our society and all the health and economic consequences. And that's what led me to write this systematic review, not only for myself to get into the knowledge, but also to share with our colleagues in functional medicine that there is some knowledge on the orthodox side that we should consider. But there are fascinating facts that I brought into this review, which I think my colleagues will be very happy to read".

Happy, yes. I certainly was, because Adriano has put the science - our kind of science - back into sleep, uncovering the research into gut bacteria's impact on our physiological rest. "Sleep hygiene" is trendy. We're inundated with "experts" telling us to use blackout blinds and blue-blocker glasses and stop looking at screens late at night. Obvious things. At the other extreme we're saddled with University of California, Berkeley's Matthew Walker, a Professor of Neuroscience and Psychology and Director of the Centre for Human Sleep Science, fêted on talk shows and podcasts worldwide thanks to his book *Why we Sleep*. A book which, I have to say, doesn't answer its titular question and is "Riddled with Scientific and Factual Errors", as you can read in Alexey Guzey's critique at <https://guzey.com/books/why-we-sleep>.

So sleep, yes, I understand Adriano's intellectual curiosity, but why the focus on the microbiome? Its connection with sleep has been completely missed by the "experts". Back to the clinic...

"I noticed that a lot of my patients who came with sleep disruption also had some sort of microbial dysfunction, or they had an infection, or they had a leaky gut...it was always linked, to a certain extent, to their gut. That really puzzled me. OK, we know in clinical practice that there are some links and



"I noticed that a lot of my patients who came with sleep disruption also had some sort of microbial dysfunction, or they had an infection, or they had a leaky gut. . .it was always linked, to a certain extent, to their gut. That really puzzled me".

there is some evidence that sleep and the gut might be linked, but we still don't understand what the neurophysiology mechanism is. Our understanding of the microbiome continues to evolve, as we delve deeper into identifying the various bacteria it harbours and learning the unique properties these microorganisms possess.

"But that was the link, clinical practice with my patients. And that really got me triggered as to what was the mechanism behind this sleep disruption. And was that to do with the microbiome?"

"If we're talking about sleep disorders, say narcolepsy, or apnea, is there a link with our gut? Neurologists traditionally view these disorders through the lens of neurological conditions; however, considering the intricate connection between the brain and the gut, it's conceivable that the causality could, in fact, be reversed".

The other factor is that there are probiotic supplements being marketed for better sleep. Is this a real thing? "I went and tracked all the literature behind this claims, because I wanted

to understand whether this is just marketing. I traced all these clinical trials and was amazed to see how much robust evidence we have".

It also soon became apparent that the conventional world of "sleep medicine" (the clue is in the name) does not want to acknowledge this evidence.

Adriano decides to study sleep in more detail, goes to one of the top universities in the world to do a Master's in "sleep medicine", focused on the (evidence-based) idea that there's a link here between the microbiome, the brain and sleep. But he's now in a place where everybody is a neurologist or a doctor, and they don't want to know about the microbiome. So, I asked him, how did that go, when you presented this idea for your dissertation?

"Well just to start this, the systematic review isn't a part of my thesis! It's not even part of an assignment".

The story is baffling, if not unexpected. As he started his Master's Adriano decided to read his way into the subject, review the literature and while doing so, collect all the papers he could find on his special interest: the microbiome, gut health and sleep. With all that data collected, why not write a review? So he contacted his professor at the University of Bern, explained that he was doing this on his own initiative, and asked for support - maybe a supervisor, or an affiliation with one of their research groups?

"First of all, they were not supportive. 'Why are you studying the microbiome? We don't think it does so much'. 'It's not a field that is still developing'. All these kind of comments that just made me think, 'Well, we can't blame them; they are too focused only in the brain'. I talked with three professors and all of them were not encouraging, not really supportive".

So began a private search for a supervisor.

“Because for a systematic review you have to have at least two to three people engaged in the work to avoid bias. I got in contact with another researcher, presented the project and she jumped into it because she has been researching the microbiome and cancer treatment”. That turned out to be Dr Serena Galiè, PhD, from the European Institute of Oncology in Milan.

As Adriano says, that gives you an idea of how much bottle it takes to get published. “To get our job done in functional medicine”, as he puts it. If Adriano was a more compliant type, this review would never have been published. “It’s OK, you’re right, I’m just going to change the way I’m going to write this paper, because you guys don’t believe in the microbiome”. Wow!

So to the next hurdle: get it published. That was not easy either, despite having a well-established supervisor (who gets to be listed as co-author). First attempt was the official European sleep research journal. He thought he’d give it a go, despite knowing that many of the Bern professors were involved with it. The paper was rejected in about a week. Enough papers for the month, no room, they couldn’t accept another. “Exactly what I was expecting”, he says.

The MDPI journal *Nutrients* was no soft touch, but they did show interest. What followed was an “excruciating process of peer review, which was amazing, because the paper just got better”. Then acceptance, in January. “Let’s say it took me a little bit of energy to get that published!”

A familiar story: they say they’re not interested in nutrition or functional medicine, not interested in the microbiome, because “there’s no evidence”. Well here’s the evidence and I’m trying to publish it. No, you can’t publish it. No wonder we have such an uphill struggle, when we can’t get into the journals.

“Another thing we have to take into consideration”, says Adriano, “is that some of these professors behind this chokehold on the European sleep research journal are connected somehow to pharmacological companies and most of these professors run research labs which operate on grants after grants, and often from pharmaceutical companies. So then along comes this guy with his gut microbiome and sleep disorder and nutrition...”

Whereas they’re looking at the field as sleep MEDICINE: let’s find a medication for it.

Biological rhythm

So, into the review. One thing that struck me was the research on biological rhythm. This is obviously a key component of Adriano’s research, given that he has been accepted

The abstract

“The Microbiota–Gut–Brain Axis in Metabolic Syndrome and Sleep Disorders: A Systematic Review”, published in *Nutrients* as part of a special issue on Nutritional Intervention in Mental Health: online at <https://www.mdpi.com/2072-6643/16/3/390>.

Background: Over recent decades, a growing body of evidence has emerged linking the composition of the gut microbiota to sleep regulation. Interestingly, the prevalence of sleep disorders is commonly related to cardiometabolic comorbidities such as diabetes, impaired lipid metabolism, and metabolic syndrome (MetS). In this complex scenario, the role of the gut–brain axis as the main communicating pathway between gut microbiota and sleep regulation pathways in the brain reveals some common host–

microbial biomarkers in both sleep disturbances and MetS. As the biological mechanisms behind this complex interacting network of neuroendocrine, immune, and metabolic pathways are not fully understood yet, the present systematic review aims to describe common microbial features between these two unrelated chronic conditions.

Results: This systematic review highlights a total of 36 articles associating the gut microbial signature with MetS or sleep disorders. Specific emphasis is given to studies evaluating the effect of dietary patterns, dietary supplementation, and probiotics on MetS or sleep disturbances.

Conclusions: Dietary choices promote microbial composition and metabolites, causing both the amelioration and impairment of MetS and sleep homeostasis.



for an Oxford Fellowship to go deeper into this. The suggestion is that while diet is key, feeding time and rhythm is really important.

“Yes, it’s great to mention that. In this next research project, I will evaluate the effects of circadian misalignment as a consequence of Dim-Light in the Evening (DLE) on the liver transcriptome (using RNAseq) and whether DLE results in changes in the gut microbiome. The over-arching goal of this project is to identify the mechanisms by which circadian misalignment affects metabolic function, with the hypothesis that changes in the gut

microbiome may play an important role in mediating these effects. We will also look into the genes responsible for glucose and fatty acid metabolism and their interaction with our microbiome. Genes such as: Glucose-6-phosphatase (G6PC), Phosphoenolpyruvate carboxykinase (PEPCK), Glucokinase (GCK), Fatty acid synthase (FASN), Acetyl-CoA carboxylase (ACC), Carnitine palmitoyltransferase 1 (CPT1), Peroxisome proliferator-activated receptor alpha (PPARA).

“In my review, I found quite interesting a particular trial where they found butyrate

ameliorates the damage in intestinal mucosa caused by lack of sleep”. It seems that butyrate corrects sleep patterns and induces circadian alignment via immune system pathways.

“They started the trial with a sleep restriction, which is very useful in the sleep medicine world - they use the sleep restriction for a couple of days to reset the circadian rhythm, in the central clock, The suprachiasmatic nucleus (SCN). In the group where they gave probiotic supplementation they saw this significant increase in butyrate, which played an important role in the immune system and inflammatory processes etc.

“I also discuss the clinical trial done by Wang and his team from the Department of Cardiology, in Wuhan, China, where they illustrate beautifully well how the central clock genes, including CLOCK, BMAL1, Period circadian (PER1, PER2, PER3), and cryptochromes (CRY1 and CRY2), form a complex network that regulates the circadian rhythm and that these genes are also present in peripheral parts of our body too such as in the liver, gut etc. They showed that our central and peripheral clocks are triggered by light exposure and food intake and that the circadian clock genes are directly linked to genes involved in glucose and fatty acid metabolism via the liver transcriptome genes.

“What I found it so intriguing in their research is that the circadian phosphorylation of dynamin-related protein 1 (DRP1) is controlled by the central clock genes and this process influences mitochondrial dynamics, oxidative phosphorylation, and adenosine 5-triphosphate (ATP) production, essential for cellular energy metabolism. They also demonstrated that the ability of gut microbes to directly modulate the expression of important genes involved in circadian rhythm regulation, like Retinoic acid receptor-related orphan receptor alpha (Rev-ERBA) and Nuclear factor interleukin-3-regulated protein (Nfil3), through the DC-ILC3-STAT3 immune pathway. Similarly, the circadian expression of Toll-like receptor 9 (TLR9) by immune cells is also guaranteed through gut microbiota stimulation. That’s another example of robust evidence”.

What’s very interesting is that the review ties in metabolic syndrome. This disruption of the internal clock doesn’t just affect sleep, it affects the entire metabolism. And it looks like this is being driven by changes in the microbiome.

“In my review, you can see that is exactly what’s happening, with the microbiome being a metabolic and sleep homeostasis mediator. That was fascinating. We already have enough evidence about short-chain fatty acids (SCFAs), and we know that some of them, specifically the butyrates, are so important

for your metabolic homeostasis. But what was interesting to find some many evidences that a reduction in butyrate producers, including Faecalibacterium, Alistipes, Oscillobacter, Roseburia, and Pseudofavonifractor, in Metabolic syndromes (Mets) are key contributors to the pathogenic mechanisms. The SCFAs, including butyrate, propionate, and acetate, have a particular affinity for G-protein coupled receptors, namely FFAR2 and FFAR3, which regulate satiety and intestinal motility by stimulating the secretion of glucagon-like peptide (GLP) and peptide YY (PYY) from intestinal L-cells.

“Another fascinating example is the research of Zielinski et al, where they demonstrated that at the same time, microbial metabolites and bacterial cell wall components, like LPS, interact with the microglia cells from the innate immune compartment in the ENS, inducing an inflammatory cascade in the gut. In this regard, they showed that LPS from Gram-negative bacteria can significantly reduce the power of the EEG theta, increasing the duration of NREM and decreasing the duration of REMS, thus inducing host fatigue”.

“In my review, I found quite interesting a particular trial where they found butyrate ameliorates the damage in intestinal mucosa caused by lack of sleep”. It seems that butyrate corrects sleep patterns and induces circadian alignment via immune system pathways.

Microbiome production of butyrate: if you have enough, metabolic health follows. So now we see this interlocking connection between circadian rhythm – or the sleep clock - microbiome, butyrate and metabolic syndrome. Now Nature steps in, too, or rather the issue of a population exposed to artificial “daylight” round the clock.

“Going back to chronobiology”, says Adriano, “the clock genes are expressed in response to light exposure. That helps to synchronise the metabolic process with the day/night life cycle. So imagine our society with people exposed to light pollution. What is that doing to their metabolism? And I’m talking about glucose metabolism, protein

and fatty acids metabolism, and also about methylation or oxidative phosphorylation? We’ve seen results from clinical trials on ATP production - that’s a mitochondria issue already. That’s all to do with circadian phosphorylation, or the phosphorylation of clock proteins, which is done by one enzyme called dynamin-related protein 1. And this protein and the other clock genes mentioned earlier are light sensitive”.

Bear in mind that clock genes are not only present in the brain, but also the liver, and as Adriano explains in the paper, it’s not a giant leap to realise that they could be “controlling” metabolism - glucose, fatty acids, oxidative processes, methylation and so on. It’s actually quite scary, when you consider the way we unwittingly disrupt our clocks with bright light and blue light.

Part of my own sleep and anti-night cramp (from training) routine is to take supplements of GABA and magnesium. After reading the paper, I’m a little ashamed to realise that I shouldn’t have been looking at a “pharmaceutical” solution, but paying attention to feeding my microbiome with the right type of fibre. GABA is just one neurotransmitter “produced”, or activated, by gut bacteria.

“I hope my review illuminates instead of darkens our reader’s mood, literally! GABA and magnesium are great elements in our toolkit, but I would start with examining how much light exposure throughout the day and evening, light intensity vs exposure in the evening, immune system and gut microbiome responsible for GABA and serotonin production. Certain gut microbes can produce and metabolise these neuromodulators, directly impacting the interconnection of afferent neurons in the gut with the central nervous system. For example, Lactobacillus and Bifidobacterium species can convert glutamate to GABA, influencing sleep disorders and memory. *Clostridium sporogenes* contributes to the conversion of tryptophan to 5-hydroxy-tryptophan, which is then converted into the neurotransmitter serotonin. So eating specific food and increasing fibre content, and diversity is the key.”

So, how to address all this from a clinical point of view. We’ve got diet, we’ve got probiotics, we’ve got prebiotics. Fibre came up a lot in the review. Do we have enough research now to say, “If you put in this particular probiotic bacterium, that’s going to do something really wonderful?”

Adriano has four components in his answer.

“First, to establish a good gut-brain connection, we need to look at the neurotransmitters, the neuromodulators that are produced in the gut and brain, such as Acetylcholine, Serotonin, Dopamine, Norepinephrine, Hypocretin and Histamine. They are circadian regulated and any nutritional deficiency could cause an imbalance in their production”.

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“Secondly, we need to look at the health of our vagus nerve communication. Issues in this pathway could be neurologically driven or perhaps microbial modulated. That’s something I’ve seen in some of the papers that I analysed.

“Thirdly, we’ve got the endocrine system, the hypothalamic-pituitary-adrenal axis (HPA) to look at. We know that sleep homeostatic processes (the adenosine pathway) creates a sleep pressure which drives cortisol down and increases melatonin production. As they have antagonistic function, it’s important to reduce the cortisol production towards the evening hours.

“And then the last component I’d say is the immune system crosstalk. I covered quite a lot about the immune system, because it modulates our sleep”. Sleep restriction studies always show an increase in inflammatory markers.

Fibre is really important – for a healthy microbiome AND for sleep. He has also identified some helpful nutraceuticals including the TCM-based Tianwang Buxin Granules (TBG) supplement.

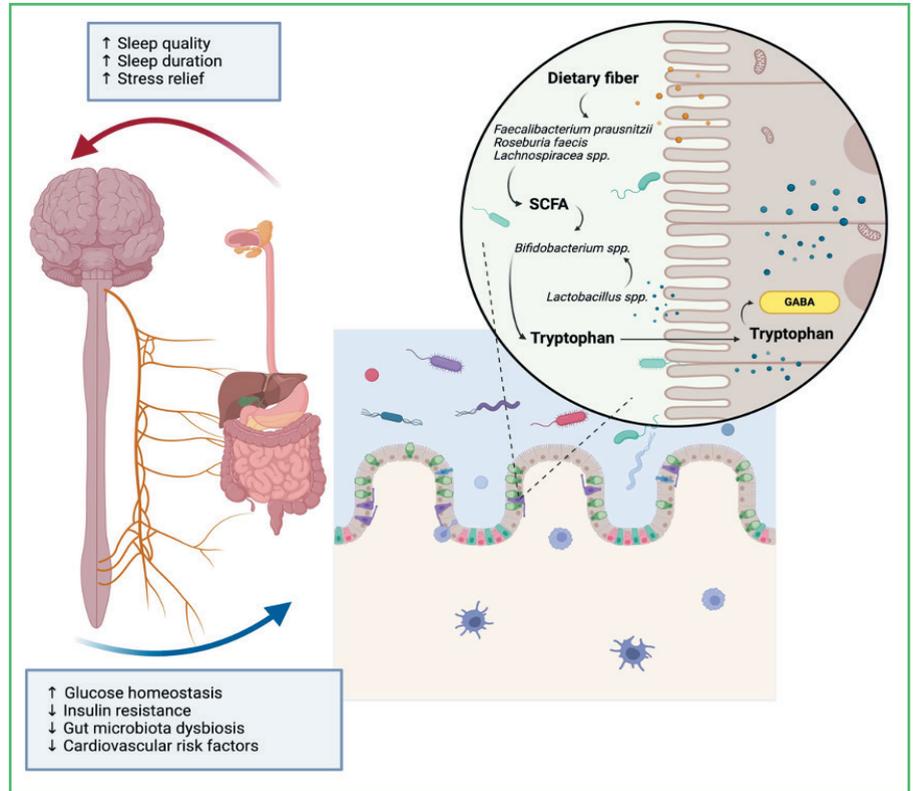
“That supplement was quite amazing in terms of how much it influences the intestinal flora in perimenopausal Insomnia. In the study of Yang et al., they show that this supplement promoted an increase in *Roseburia faecis*, *Ruminococcus*, *Prevotella copri*, *Fusicatenibacter saccharivorans* and *Blautia obeum* which directly correlates with a better sleep quality score. In another randomised double-blind, placebo-controlled study by Nishida et al, they found an improved sleep quality with *Lactobacillus gasseri* CP2305 tablets in young adults exposed to chronic stress.

“But going through solutions, it is butyrate that really struck me. And the research actually just confirmed what we’ve been talking about in functional medicine”.

So as a nutritionist seeing patients, would you change the diet, get fibre in, make sure they’re eating at the right time and so on, or would you now start by giving them butyrate. A kind of pharmaceutical approach?

“I would definitely start with the fibres in a very specific way”, he says. “Depending on the patient, because people react differently to different types of fibers. But definitely starting from increasing the nutrient consumption in terms of fibre-rich and nutrient dense food, and then perhaps the butyrate-producing probiotics”. The catch with butyrate supplements (postbiotics) is the poor absorption, according to some clinical trials.

“There are few studies on rectal administration of butyrate, but I think this sounds promising for certain health conditions. There was this randomised



controlled clinical trial in The Netherlands, where they investigated butyrate enema versus placebo enema. Before and after rectal administration, plasma samples were taken from several veins and arteries to analyse SCFA concentrations and fluxes from portal drained viscera, liver and the splanchnic area. They found that colonic administration is safe and beneficial in a variety of intestinal diseases, such as ulcerative colitis, Crohn’s disease, pouchitis, diversion colitis, radiation proctitis, and irritable bowel syndrome”.

Probiotics are also very much part of the protocol in his practice. Especially Bifidobacteria, Lactobacillus and the other SCFAs-producing bacterias.

Adriano also draws attention to a 2021 study using *Lactobacillus plantarum*, which is one almost all supplement companies are providing. This randomised, double-blind, placebo-controlled trial study showed positive effects of lactobacillus plantarum ps128 supplementation on depressive symptoms and sleep quality in insomniacs patients. As Adriano says: “We know depression leads to insomnia. Depressed patients all face sleep problems”.

It’s always incredible to me that we have a microbiome full of trillions of microbes - and yet dropping one specific bacterium into the mix can have such a profound effect. Sometimes, a simple supplement can be life-changing. Nobody can explain it, but we know it works.

Adriano concludes: “I firmly support the inclusion of nutrient-dense foods and

phytonutrients in our diet. The consumption of antioxidants present in foods, combined with sufficient fibre, plays a crucial role in supporting our microbiome. This symbiotic association is thoroughly recognised in scientific studies. Such a dietary regimen promotes the growth of a varied microbial community, enhancing overall microbial well-being rather than benefiting only particular strains. Nevertheless, it’s important to acknowledge that, in specific situations, the strategic application of supplements can lead to significant and beneficial changes in health”.

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About



ADRIANO DOS SANTOS, BSC, RNUTR, AFMCP, MBOG, RSM, ESIM, is a Functional Nutrition Practitioner who focuses on nutritional therapy for patients with metabolic syndrome, particularly those suffering from digestive issues and sleep disturbances.

He describes himself as half Brazilian/half British and has been living and working in the Netherlands for the past ten years. He shares his time between The Hague and Cannes, and sees clients both face-to-face and online. He gives lectures and workshops for companies on preventive medicine programs and does consulting work for nutraceutical companies. He has more than 20 years of experience in sports science and nutrition therapy altogether.

He is a Registered Nutritionist (rNutr) with the Association for Nutrition, a graduate of the Institute for Functional Medicine’s AFMCP (Applying Functional Medicine in Clinical Practice) program, and trained in Nutrigenomics and Pharmacogenomics at the DNAlife Institute.

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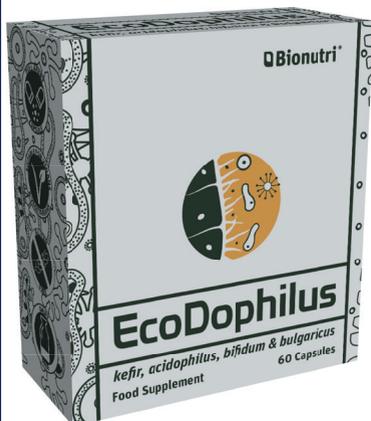
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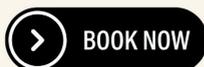


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HOW LONG HAVE YOU BEEN IN PRACTICE AND WHERE? WHAT IS YOUR MAIN MODALITY?

I have been practising for almost a decade. I had a successful fashion career until I received a cancer diagnosis which turned my life upside down. I used a combination of conventional, nutrition and lifestyle changes to transform my health and I've now been in remission for 12 years. The results I achieved from an integrative health approach were so inspiring, I quit my job and retrained in Nutrition, Naturopathy and Functional Medicine and I then completed Cancer Nutrition Training so that I could help others regain their health from complex illness. I have never looked back.

I opened my London clinic SJ Health in Chelsea and Chiswick in 2015, where I specialise in women's health issues, immune support and gut function. I have a special interest in assisting clients with cervical dysplasia,

the development of cervical cancer and its precursors. SJ Health is now a virtual clinic operating from West London. As well as one to one health packages and consultations, I offer other professional services such as health writing, speaking, workplace wellness and corporate nutrition education where I work with recognisable companies such as Adidas, Elemis Skincare, LVMH, Women for Women International, Squad Model Management and IMG models.

I split my clinical time with The Optimum Health Clinic (OHC), one of the world's leading integrative clinics specialising in fatigue-related illness, trauma, anxiety and sleep issues. I hold the positions of Senior Nutrition Lead and Registered Nutritional Therapist. I have worked with the brilliant OHC team for over six years and our approach combines nutrition, functional medicine and psychology, which we have been successfully using to support clients in over 50 countries for nearly 20 years. OHC is also an education centre with award-winning Therapeutic Coaching and Therapeutic Nutrition training programs for health professionals wanting to take a deeper dive into complex fatigue-related health concerns.

I use a powerful evidence-based combination of cutting-edge lab testing, nutrition, natural therapies and lifestyle medicine to understand root cause perspectives in order to build successful and achievable clinical roadmaps with my clients while keeping up with the latest scientific research.

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I have been using the range for over a decade both in clinic and personally. I have benefited not only from the clever formulas, but also from the very generous practitioner education and resources they offer such as the clinical protocols and the PureGenomics tool.

I was also initially attracted to the brand because of their transparency and our ethical values align especially regarding their sustainability goals by next year - I am in the business of supporting people's health as well as that of the planet.

It is so difficult to pick just one hero product because I use and recommend so many, but I'll start with the Daily Support Formula, which is a game-changer for so many.

HOW DOES IT FIT INTO YOUR WORK/WHAT DIFFERENCE DOES IT MAKE TO YOUR PRACTICE?

It's such a well-considered formula packed with active nutritional ingredients to support adrenal, psychological and nervous system function like magnesium, vitamin C, B1, B3, B6 and B12 and free-form l-tyrosine. It delivers powerful botanicals like lemon balm and the adaptogens ashwagandha, rhodiola and bacopa, which I witness making an enormous difference to stress management for my clients and it's even improved cognition, anxiety and mood - all of which affect the majority of my clients. Having this combination of factors in one supplement helps me and my clients because it reduces the number of daily tablets they need to take, and ultimately helps cut down on cost too if they can achieve a five-in-one effect from the one nutraceutical.

CAN YOU GIVE AN EXAMPLE OF HOW IT HAS MADE A DIFFERENCE TO ONE OF YOUR CLIENTS?

I was working with a client recently around cervical dysplasia and we identified that high levels of perceived stress were associated with her positive HPV-status and her general immunity. Her depleted immune response meant she had been positive to HPV for a number of years. Together, these put her at risk of cervical disease progression. Within about two weeks of using the Daily Support Formula she felt more resilient, better able to manage her stress response and had a noticeable difference to her mood and sleep. Supporting these mechanisms within a comprehensive HPV protocol over a number of months meant she was able to clear the HPV infection and reverse her CIN 2 status without a surgical intervention.



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Winner - OptiBac Immune Support +

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★ Highly commended - Oxford Healthspan Primeadine®

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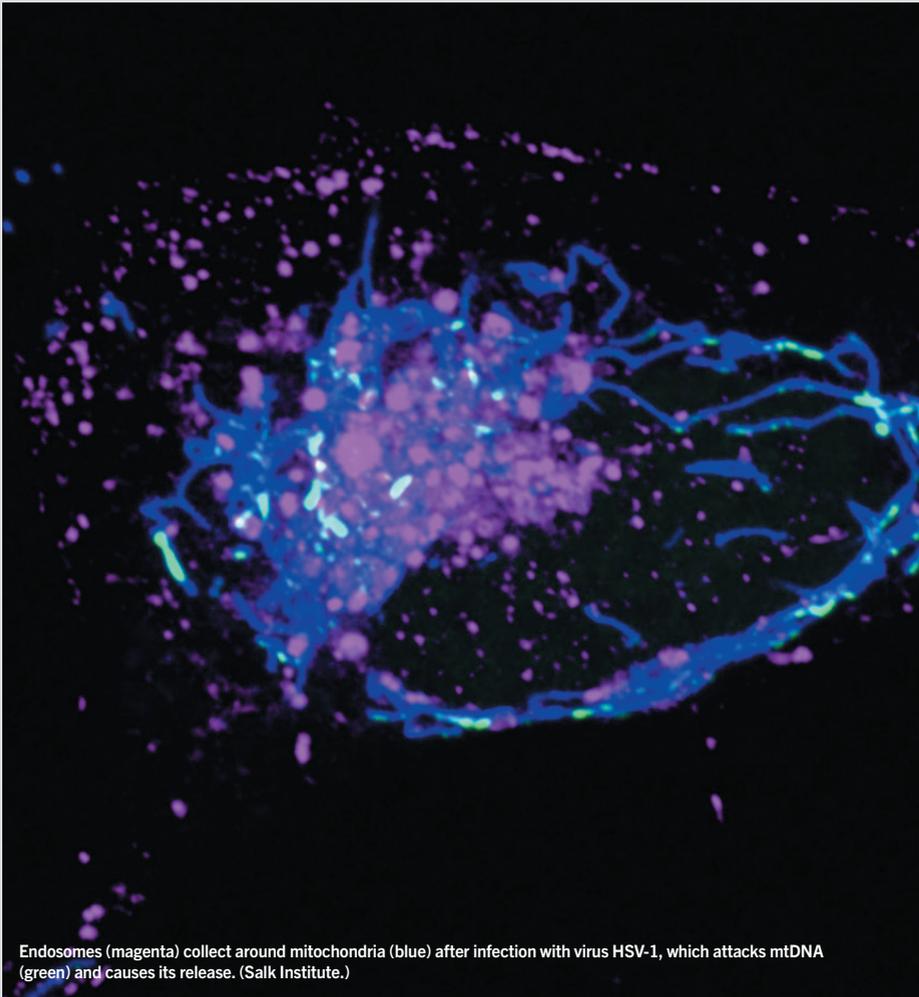


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Salk discovery puts mitochondria at the heart of inflammation



Endosomes (magenta) collect around mitochondria (blue) after infection with virus HSV-1, which attacks mtDNA (green) and causes its release. (Salk Institute.)

Salk Institute scientists have discovered a pathway leading from mitochondrial DNA replication stress to immune system activation and inflammation, finding that a fault in the autophagy system that disposes of dysfunctional mitochondrial DNA (mtDNA) can leak mtDNA into cells and prompt an inflammatory immune response.

Cells in the human body contain power-generating mitochondria, each with their own mtDNA - a unique set of genetic instructions entirely separate from the cell's nuclear DNA that mitochondria use to create life-giving energy. When mtDNA remains where it belongs (inside of mitochondria), it sustains both mitochondrial and cellular health - but when it goes where it doesn't belong, it can initiate an immune response that promotes inflammation.

Salk scientists and collaborators at UC San Diego have now discovered a novel mechanism used to remove improperly functioning mtDNA from inside to outside the mitochondria. When this happens, the mtDNA gets flagged as foreign DNA and activates a cellular pathway normally used to promote inflammation to rid the cell of

pathogens.

The findings, published in *Nature Cell Biology* last month, offer many new targets for therapeutics to disrupt the inflammatory pathway and therefore mitigate inflammation during ageing and diseases like lupus or rheumatoid arthritis.

"We knew that mtDNA was escaping mitochondria, but how was still unclear", says senior author Prof Gerald Shadel, director of the San Diego-Nathan Shock Centre of Excellence in the Basic Biology of Ageing. "Using imaging and cell biology approaches, we're able to trace the steps of the pathway for moving mtDNA out of the mitochondria, which we can now try to target with therapeutic interventions to hopefully prevent the resulting inflammation".

One of the ways our cells respond to damage and infection is through the innate immune system. While the innate immune response is the first line of defence against viruses, it can also respond to molecules the body makes that simply resemble pathogens - including misplaced mtDNA. This response can lead to chronic inflammation and contribute to human diseases and ageing.

Scientists have been working to uncover how mtDNA leaves mitochondria and triggers the innate immune response, but the previously characterised pathways did not apply to the unique mtDNA stress conditions the Salk team was investigating.

"We had a huge breakthrough when we saw that mtDNA was inside of a mysterious membrane structure once it left mitochondria - after assembling all of the puzzle pieces, we realised that structure was an endosome", says first author Laura Newman, assistant professor at the University of Virginia.

"That discovery eventually led us to the realisation that the mtDNA was being disposed of and, in the process, some of it was leaking out".

The team discovered a process beginning with a malfunction in mtDNA replication that caused mtDNA-containing protein masses called nucleoids to pile up inside of mitochondria. Noticing this malfunction, the cell then begins to remove the replication-halting nucleoids by transporting them to endosomes, a collection of organelles that sort and send cellular material for permanent removal. The endosome gets overloaded with these nucleoids, springs a leak, and mtDNA is suddenly loose in the cell.

The cell then flags that mtDNA as foreign DNA - the same way it flags a virus's DNA - and initiates the DNA-sensing cGAS-STING pathway to cause inflammation.

"Using our cutting-edge imaging tools for probing mitochondrial dynamics and mtDNA release, we have discovered an entirely novel release mechanism for mtDNA", says Uri Manor, former director of the Waitt Advanced Biophotonics Core at Salk.

"There are so many follow-up questions we cannot wait to ask, like how other interactions between organelles control innate immune pathways, how different cell types release mtDNA, and how we can target this new pathway to reduce inflammation during disease and ageing".

The researchers hope to map out more of this complicated mtDNA-disposal and immune-activation pathway, including what biological circumstances - like mtDNA replication dysfunction and viral infection - are required to initiate the pathway and what downstream effects there may be on human health.

How inflammation kills cancer: Salk research shows how telomeres talk to mitochondria and promote disease-cancelling inflammation

Salk Institute's research linking mitochondria to inflammation is just the latest in a series of discoveries from Prof Gerald Shadel's group. It follows a surprising finding, published last year, in which the researchers found that ageing triggers a natural, inflammatory anti-cancer mechanism.

Their continuing research suggests we're on the right track in having a cautious approach to inflammation - it is fundamentally a healing mechanism, until it gets out of hand.

As we age, the end caps of our chromosomes, called telomeres, gradually shorten. The structure and function of telomeres was only recently elucidated, winning a 2009 Nobel Prize for Elizabeth Blackburn and colleagues. Telomere testing is now widely used to establish biological age. Salk scientists discovered that when telomeres become very short, they communicate with mitochondria, triggering a complex set of signalling pathways that initiates an inflammatory response that destroys cells that could otherwise become cancerous.

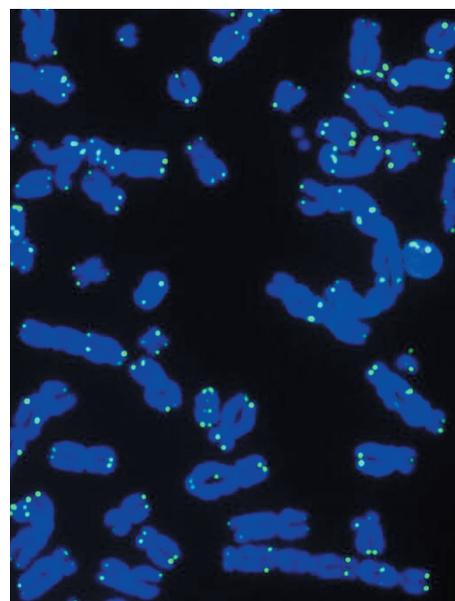
The discovery was the result of a collaboration between co-senior authors and Salk professors Jan Karlseder and Gerald Shadel, who teamed up to explore similarities they had each found in inflammatory signalling pathways. Karlseder's lab studies telomere biology and how telomeres prevent cancer formation. Shadel's lab studies the role mitochondria play in human disease, ageing, and the immune system.

"We were excited to discover that telomeres talk to mitochondria", says Karlseder. "They clearly synergise in well-controlled biological processes to initiate cellular pathways that kill cells that could cause cancer".

When telomeres shorten to a point where

they can no longer protect chromosomes from damage, a process called "crisis" occurs and cells die. This beneficial natural process removes cells with very short telomeres and unstable genomes and is known to be a powerful barrier against cancer formation. Karlseder and the study's first author Joe Nassour, previously discovered that cells in crisis are removed by autophagy, in which the body rids itself of damaged cells.

In this study, the team wanted to know how autophagy-dependent cell-death programs are activated during crisis, when telomeres are extremely short. By conducting a genetic screen using human skin cells called fibroblasts, the scientists discovered interdependent immune sensing and inflammatory signalling pathways - similar to the ones by which the immune system combats viruses - that are crucial for cell death during crisis. Specifically, they found that RNA molecules emanating from



Telomeres protect the ends of chromosomes from damage. This image shows telomeres (green) and DNA (blue) during DNA repair activities. (Salk Institute.)

short telomeres activate immune sensors called ZBP1 and MAVS in a unique way on the outer surface of mitochondria.

The findings demonstrate important links between telomeres, mitochondria, and inflammation and underscore how cells can bypass crisis (thereby evading destruction) and become cancerous when the pathways are not functioning properly.

"Telomeres, mitochondria and inflammation are three hallmarks of ageing that are most often studied in isolation", says Shadel. "Our findings showing that stressed telomeres send an RNA message to mitochondria to cause inflammation highlights the need to study interactions between these hallmarks to fully understand ageing and perhaps intervene to increase health span in humans".

"Cancer formation is not a simple process", says Nassour. "It is a multistep process that requires many alterations and changes throughout the cell. A better understanding of the complex pathways linking telomeres and mitochondria may lead to the development of novel cancer therapeutics in the future".



"Telomeres, mitochondria and inflammation are three hallmarks of ageing that are most often studied in isolation. Our findings showing that stressed telomeres send an RNA message to mitochondria to cause inflammation highlights the need to study interactions between these hallmarks to fully understand ageing and perhaps intervene to increase health span in humans".

- Prof Gerald Shadel, director of the Salk Institute's San Diego-Nathan Shock Centre of Excellence in the Basic Biology of Ageing.

Reservoirs of “zombie” SARS fragments continue to cause inflammation after the virus is destroyed

In a study published in Proceedings of the National Academy of Sciences, a UCLA-led multidisciplinary research team explores one way that COVID-19 turns the immune system against the body itself, with potentially deadly results.

Using an artificial intelligence system, the authors scanned the entire collection of proteins produced by SARS-CoV-2 and then performed an exhaustive series of validation experiments. They found that certain viral protein fragments, generated after the SARS-CoV-2 virus is broken down into pieces, can mimic a key component of the body’s machinery for amplifying immune signals. Their discoveries suggest that some of the most serious COVID-19 outcomes can result from these fragments overstimulating the immune system, thereby causing rampant inflammation in widely different contexts such as cytokine storms and lethal blood coagulation.

“What we found deviates from the standard picture of viral infection” said Prof Gerard Wong, at the UCLA Samueli School of Engineering. “The textbooks tell us that after the virus is destroyed, the sick host ‘wins’, and different pieces of virus can be used to train the immune system for future recognition. COVID-19 reminds us that it’s not this simple.

“For comparison, if one were to assume that after food gets digested into its molecular components, then its effects on the body are over, it would be very liberating; I wouldn’t have to worry about the half-dozen jelly donuts I just ate. However, this simple picture is not correct”.

The team found SARS-CoV-2 fragments can imitate innate immune peptides, a class of immune molecules that amplify signals to activate the body’s natural defences. Peptides are chains of amino acids like proteins, only shorter. These immune peptides can spontaneously assemble into new structures with double-stranded RNA, a special form of a molecule essential for building proteins from DNA, typically found in viral infections or released by dying cells.

The resultant hybrid complex of the immune peptides and double-stranded RNA kicks off a chain reaction that triggers an immune response.

In addition to their AI analysis, the researchers used state-of-the-art methods for elucidating nanoscale biological structures and conducted cell- and animal-based experiments. Compared to relatively harmless coronaviruses that cause the common cold, the team found that SARS-CoV-2 harbours

many more combinations of fragments that can better mimic human immune peptides. Consistent with that, additional experiments with multiple cell types all consistently show that fragments of the SARS-CoV-2 coronavirus prompt an amplified inflammatory response compared to those from a common cold coronavirus. Likewise, experiments with mice show that fragments from SARS-CoV-2 lead to huge immune response, especially in the lungs.

In future, “We may be able to look at the protein composition of this year’s coronavirus strains and figure out whether they’re potentially pandemic-capable or just going to cause the common cold”, Wong said.

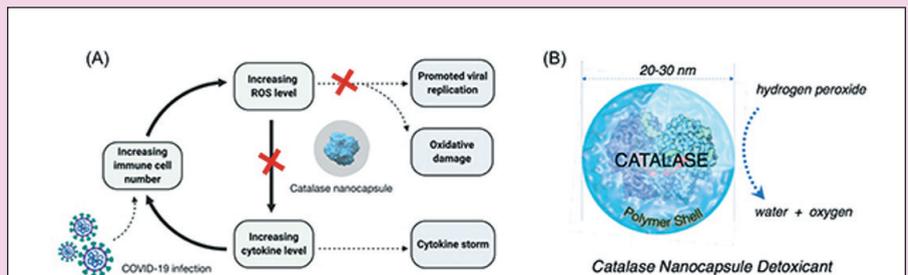
Wong and colleagues concentrated on three SARS-CoV-2 fragments. They found that, like the innate immune peptide, the SARS-CoV-2 fragments can organise double-stranded RNA into structures that stimulate the immune

system.

“We saw that the various forms of debris from the destroyed virus can reassemble into these biologically active ‘zombie’ complexes”, Wong said. “It is interesting that the human peptide being imitated by the viral fragments has been implicated in rheumatoid arthritis, psoriasis and lupus, and that different aspects of COVID-19 are reminiscent of these autoimmune conditions”.

The scientists also measured the entire set of genes expressed at the cellular level. By performing a comparison with internationally curated databases, the team found that the gene expression profile from cells exposed to SARS-CoV-2 “zombie” complexes closely resembled that from COVID-19 itself.

“What’s astonishing about the gene expression result is there was no active infection used in our experiments”, Wong said. “We did not even use the whole virus -



Catalase: a forgotten treatment for hyper-inflammation

In previous research at UCLA’s UCLA Samueli School of Engineering, researchers identified catalase – one of the most common antioxidant enzymes and widely available as a supplement – as a potential treatment for COVID-19.

Catalase is produced naturally and used by humans, animals and plants. Inside cells, the antioxidant enzyme kick-starts the breakdown of hydrogen peroxide, which can be toxic, into water and oxygen. The enzyme is also commonly used worldwide in food production as well as being a popular dietary supplement.

“There is a lot of focus on vaccines and antiviral drugs, and rightly so”, said Yunfeng Lu, a UCLA Samueli School of Engineering professor of chemical and biomolecular engineering. “In the meantime, our research suggests this enzyme could offer a very effective therapeutic solution for treatment of hyperinflammation that occurs due to SARS-CoV-2 virus, as well as hyperinflammation generally”.

Lu’s group conducted three types of tests, each addressing a different symptom of COVID-19.

First, they demonstrated the enzyme’s anti-inflammatory effects and its ability to regulate the production of cytokines, inflammatory proteins produced in white blood cells. Cytokines can also signal the immune system to attack the body’s own cells if too many are made - a so-called “cytokine storm” that is reported in some patients diagnosed with COVID-19.

Second, the team showed that catalase can protect alveolar cells, which line the human lungs, from damage due to oxidation.

Finally, the experiments showed that catalase can repress the replication of SARS-CoV-2 virus in rhesus macaques, a type of monkey, without noticeable toxicity.

“This work has far-reaching implications beyond the treatment of COVID-19. Cytokine storm is a lethal condition that can complicate other infections, such as influenza, as well as non-infectious conditions, like autoimmune disease”, said Dr Gregory Fishbein, a pathologist at the UCLA David Geffen School of Medicine.

rather only about 0.2% or 0.3% of it - but we found this incredible level of agreement that is highly suggestive”.

The findings may account for some peculiarities of COVID-19 infection. For instance, that fragments from SARS-CoV-2 lead to excessive inflammation could help explain why some seemingly healthy people experience severe COVID-19. Normally, the activity of enzymes varies a great deal between healthy individuals - with levels differing by as much as a factor of 10. It is ultimately enzymes that are responsible for cutting virus particles into smaller and smaller pieces.

Evidence that persistence of SARS-CoV-2 fragments may drive illness also reinforces emerging clues about which treatments may show promise.

“Our results suggest we may be able to manage COVID-19 by inhibiting certain enzymes or enhancing others”, Wong said. “One could even imagine a strategy also based on mimicry, by using biologically inactive decoys that look enough like these viral fragments to compete for double-stranded RNA, but form complexes that don’t activate the immune system”.

Remnant viral fragments are known to exist in other viral infections, but their biological activities have not been systematically studied.

“Results presented here indicate that there exist intrinsically proinflammatory sequences found in the SARS-CoV-2 proteome that are not found in common cold coronavirus homologs, sequences that strongly activate immune responses in a broad range of cell and tissue types connected to disease states in multiple systems”, the study concludes.



“We saw that the various forms of debris from the destroyed virus

can reassemble into these biologically active ‘zombie’ complexes. It is interesting that the human peptide being imitated by the viral fragments has been implicated in rheumatoid arthritis, psoriasis and lupus, and that different aspects of COVID-19 are reminiscent of these autoimmune conditions”.

— Prof Gerald Wong, professor of bioengineering at the University of California at Los Angeles.

Immune suppression during pregnancy may protect against inflammation: gut microbiome provides the signals

During pregnancy, a woman's immune system changes dramatically but researchers don't yet understand all the underlying mechanisms. A new study shows how the gut microbiota may play a role.

Researchers in China report that during pregnancy, changes in levels of cytokines - immune system proteins important in inflammation - may be linked to specific alterations in the mother's gut microbiome and in plasma and faecal metabolites.

“To the best of our knowledge, these associations were first explored in our study”, said first author Ting Huang, MD, from the First Affiliated Hospital of Jinan University in Guangzhou.

Pregnancy brings a raft of changes, including fluctuations in hormones, changes to a woman's body structure, and variations in the immune system.

Previous studies have identified changes to the gut

microbiome that can occur during pregnancy; they have also suggested that those changes may influence physiological processes in the mother through metabolites.

Disturbances in the microbiota, for example, have been connected to the promotion of preeclampsia, a dangerous pregnancy complication characterised by high blood pressure. However, it remains unclear how alterations in the gut microbiota during pregnancy affect maternal immunity.

To investigate, the researchers at Jinan compared the gut microbiota, metabolite profiles and immune system status of 30 healthy pregnant women to 15 healthy women who weren't pregnant.

All the women in the study were between 18 and 34. Faecal and blood samples were collected during or after the 37th week of pregnancy, and samples from non-pregnant women were collected

on the 14th day of the menstrual cycle.

Analyses showed that *Firmicutes* was the most dominant phylum of bacteria in both groups of women. However, *Bacteroidota* bacteria accounted for a smaller relative share of the microbial population in pregnant women than in non-pregnant women. Pregnant women also showed a higher relative abundance of both *Actinobacteriota* and *Proteobacteria*, compared to non-pregnant women.

The researchers similarly found a distinct difference in the cytokine levels in the two groups. Pregnant women had lower levels of cytokines that promote inflammation, and they showed higher levels of cytokines that act against

inflammation.

These results suggest that the immune system may be suppressed during pregnancy.

The researchers then identified hundreds of metabolites found in the plasma and faecal samples.

They found that each group - pregnant and non-pregnant women - had its own distinct

collection of metabolites, or metabolome. Notably, many of those metabolites were connected to bile acid secretion and metabolism, and bile acids have previously been tied to inflammation.

In further analyses, they found that some enriched metabolites in pregnant women were associated with lower levels of pro-inflammatory cytokines. Similarly, some of the depleted metabolites are associated with increases in pro-inflammatory cytokines.

Finally, the group identified a total of 46 connections among microbes, metabolites and cytokines. They found that some microbes enriched in pregnant women, for example, may inhibit an immune response by inhibiting pro-inflammatory metabolites.

Overall, Huang said, the results support the idea that gut microbes interact with host metabolites to change cytokine levels in the blood.



→ Cambridge study on fasting reveals arachidonic acid is NOT pro-inflammatory

Scientists at the University of Cambridge claim they may have discovered a new way in which fasting helps reduce inflammation.

In research published in *Cell Reports*, the team describes how fasting raises blood levels of arachidonic acid, which inhibits inflammation. The researchers say it may also help explain some of the beneficial effects of drugs such as aspirin.

Inflammation is our body's natural response to injury or infection, but this process can be triggered by other mechanisms, including by the so-called "inflammasome", which acts like an alarm within cells, triggering inflammation to help protect the body when it senses damage. But the inflammasome can trigger inflammation in unintentional ways - one of its functions is to destroy unwanted cells, which can result in the release of the cell's contents into the body, where they trigger more inflammation.

Prof Clare Bryant from the Department of Medicine said: "We're very interested in trying to understand the causes of chronic inflammation in the context of many human diseases, and in particular the role of the inflammasome.

"What's become apparent over recent years is that one inflammasome in particular - the NLRP3 inflammasome - is very important in a number of major diseases such as obesity and atherosclerosis, but also in diseases like Alzheimer's and Parkinson's, many of the diseases of older age people, particularly in the Western world".

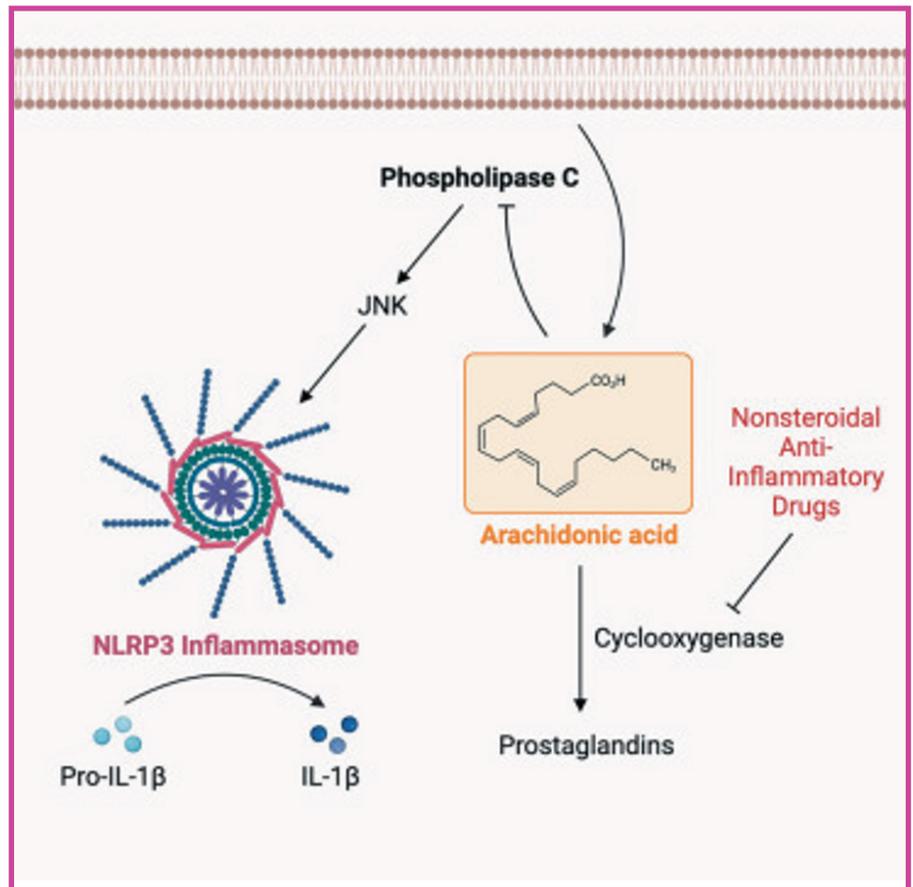
Fasting can help reduce inflammation, and to find out how that works a team led by Bryant and colleagues at Cambridge and the National Institutes for Health in the USA studied blood samples from a group of 21 volunteers, who ate a 500kcal meal then fasted for 24 hours before consuming a second 500kcal meal.

The team found that restricting calorie intake increased levels of the lipid arachidonic acid.



"It's too early to say whether fasting protects against diseases like Alzheimer's and Parkinson's, as the effects of arachidonic acid are only short-lived, but our work adds to a growing amount of scientific literature that points to the health benefits of calorie restriction. It suggests that regular fasting over a long period could help reduce the chronic inflammation we associate with these conditions. It's certainly an attractive idea".

– Prof Clare Bryant, University of Cambridge, Department of Medicine.



As soon as individuals ate a meal again, levels of arachidonic acid dropped.

When the researchers studied arachidonic acid's effect in immune cells cultured in the lab, they found that it turns down the activity of the NLRP3 inflammasome.

This surprised the team, as arachidonic acid was previously thought to be pro-inflammatory.

"This provides a potential explanation for how changing our diet - in particular by fasting - protects us from inflammation, especially the damaging form that underpins many diseases related to a Western high calorie diet", said Prof Bryant.

"It's too early to say whether fasting protects against diseases like Alzheimer's and Parkinson's disease, as the effects of arachidonic acid are only short-lived, but our work adds to a growing amount of scientific literature that points to the health benefits of calorie restriction. It suggests that regular fasting over a long period could help reduce the chronic inflammation we associate with these conditions. It's certainly an attractive idea".

The findings also hint at one mechanism whereby a high-calorie diet might increase the risk of these diseases. Studies have shown that some patients that have a high fat diet have increased levels of inflammasome activity.

"There could be a yin and yang effect going on here, whereby too much of the wrong thing is increasing your inflammasome activity and too little is decreasing it", said Prof Bryant. "Arachidonic acid could be one way in which this is happening".

The researchers say the discovery may also offer clues to an unexpected way in which so-called non-steroidal anti-inflammatory drugs such as aspirin work. Normally, arachidonic acid is rapidly broken down in the body, but aspirin stops this process, which can lead to an increase in levels of arachidonic acid, which in turn reduce inflammasome activity and hence inflammation.



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Refs: 1. C. González de Vega, C. Speed, B. Wolfarth, J. González. Traumeel vs. diclofenac for reducing pain and improving ankle mobility after acute ankle sprain: A multicentre, randomised, blinded, controlled and non-inferiority trial. *Int J Clin Pract.* 2013. doi: 10.1111/ijcp.12219.

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Inflammatory cascade in the foetus: researchers warn pregnant to avoid junk food

Research from the University of Washington School of Medicine shows that phthalates, a class of chemicals associated with plastics, can shed from the wrapping, packaging and even from plastic gloves worn by food handlers into food. And once consumed during pregnancy, the chemicals can get into the bloodstream, through the placenta and then into the foetal bloodstream.

The chemicals can cause oxidative stress and an inflammatory cascade in the foetus.

Previous research has indicated that exposure to phthalates during pregnancy can increase the risk of low birth weight, preterm birth and child mental health disorders such as autism and ADHD. This is the first study in pregnant women to show that diets higher in ultra-processed foods are linked to greater phthalate exposures, the authors say.

"When mums are exposed to this chemical, it can cross the placenta and go into foetal circulation", said senior author Dr Sheela Sathyanarayana, a UW Medicine paediatrician and researcher at the Seattle Children's Research Institute. This analysis involved data in the Conditions Affecting Neurocognitive Development and Learning in Early Childhood (CANDLE) research cohort: 1,031 pregnant individuals in Memphis, who were enrolled between 2006 and 2011.

Phthalate levels were measured in urine samples collected from during the second trimester of pregnancy.

The researchers found that ultra-processed food composed 10% to 60% of participants' diets, or 38.6%, on average.

Each 10% higher dietary proportion of ultra-processed food was associated with 13% higher concentration of di(2-ethylhexyl) phthalate, one of the most common and



harmful phthalates.

The researchers define ultra-processed foods as "foods" made mostly from substances extracted from real foods - such as oils, sugar and starch - but have been so changed from processing and the addition of chemicals and preservatives to enhance their appearance or shelf life that they are hard to recognise from their original form. These include packaged cake mixes, for example, or packaged french fries, hamburger buns and soft drinks.

When it comes to fast food, gloves worn by the employees and the storage, preparation, serving equipment or tools may be the main sources of exposure.

Both frozen and fresh ingredients would be subject to these sources, said lead author Brennan Baker, a postdoctoral researcher in

Sathyanarayana's lab.

This is the first study, researchers say, to identify ultra-processed foods as a link between exposure to phthalates and the socio-economics issues facing the mothers.

The mothers' vulnerability might stem from experiencing financial hardships and from living in "food deserts", where healthier, fresh foods are harder to obtain and transportation to distant markets is unrealistic. "We don't blame the pregnant here", said Baker. "We need to call out manufacturers and legislators to offer replacements, and ones that may not be even more harmful".

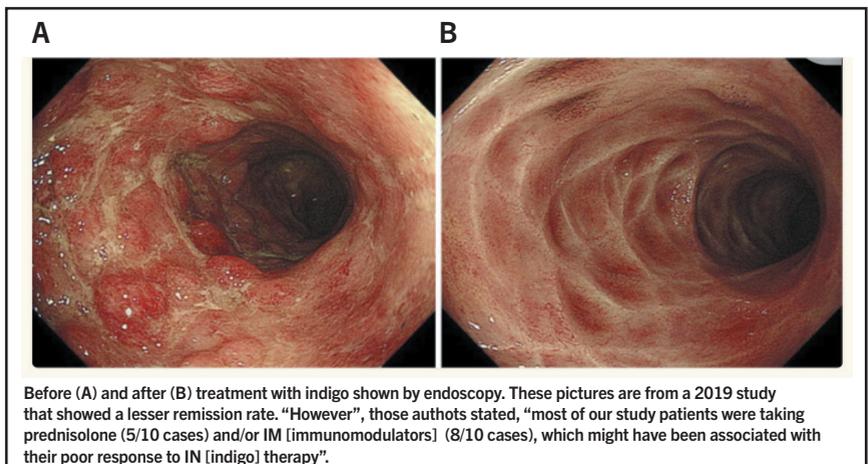
Sathyanarayana said that pregnant women should try to avoid ultra-processed food as much as they can, and seek out fruits, vegetables and lean meats.

Chinese herbal medicine shows 90% remission in ulcerative colitis

Indigo naturalis, the herbal remedy used in Traditional Chinese Medicine that has previously been shown to be very effective in inducing remission in patients with ulcerative colitis, has scored again in a single-centre open-label, randomised controlled study in Japan led by Dr Yuichi Matsuno, Kyushu University in Fukuoka.

Patients taking indigo for at least one year were enrolled and then randomised to either continue or discontinue the herb. At 52 weeks, the remission rate was 90% for the patients still taking indigo. For the patients who discontinued indigo, the remission rate was only 20%.

• Abstract presented at the annual Crohn's & Colitis Congress, January 25-27, Las Vegas.



Before (A) and after (B) treatment with indigo shown by endoscopy. These pictures are from a 2019 study that showed a lesser remission rate. "However", those authors stated, "most of our study patients were taking prednisolone (5/10 cases) and/or IM [immunomodulators] (8/10 cases), which might have been associated with their poor response to IN [indigo] therapy".

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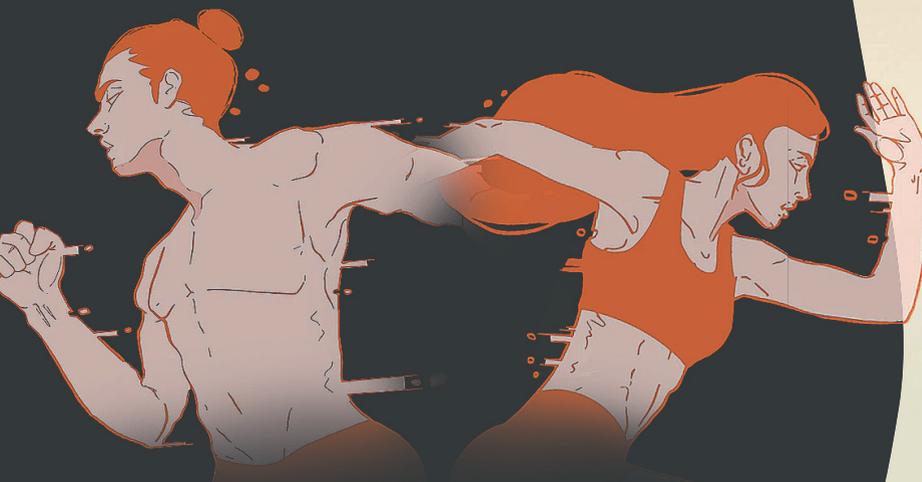
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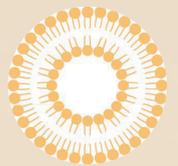
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Dr. Reckeweg (U.K.) Ltd was formed in 1990 for the distribution of the Dr. Reckeweg & Co. GmbH homeopathic range of products. The products have been manufactured to a very high standard at the family owned company in Bensheim, Germany for over 70 years and the motto of the company is 'quality admits no compromise'. A comprehensive range of homeopathic combinations are available, also tissue salts, ointments, oils, eye drops, nasal sprays, food supplements and veterinary products. The Dr. Reckeweg range of products is available to Practitioners only. Please ring 01204 650100 to open an account. The company are also sole importers for the Tegor/Most Beauty range of products manufactured in Spain to GMP standards and with European Licences. They include food supplements and beauty products. The supply of Dr. Reckeweg products is limited to Practitioners and Health Professionals only, whereas the Tegor/Most Beauty range are for general sale.

Please contact us for a catalogue.



NAME: Ify Akpuaka.

WEBSITE: The Modern Glow.co.uk.

INSTAGRAM: Instagram the_modern_glow.

QUALIFICATIONS: Reg. Nutritional Therapist and Yoga Teacher.

TRAINING: Studied at the College of Naturopathic Nutrition.

HOW LONG HAVE YOU BEEN IN PRACTICE AND WHERE?

I have been in practice since 2017 and work in Hurley and Reading in Berkshire and also online.

WHAT IS YOUR MAIN MODALITY?

I take an integrative approach, combining principles from both yoga and nutritional therapy to support overall wellbeing.

YOU'RE BEING NOMINATED BY CYTOPLAN BECAUSE YOU'RE ONE OF THEIR BEST CUSTOMERS. HOW LONG HAVE YOU BEEN USING THEIR PRODUCTS AND WHAT FIRST ATTRACTED YOU TO THEM?

I'm honoured to be nominated by Cytoplan as one of their top advocates. My success with clients wouldn't be possible without Cytoplan products; they simply deliver results. I'm drawn to their extensive product range and the company's ethos.

Since 2012, Cytoplan has been integral to my wellness journey. Initially introduced to me by a nutritional therapist,

their supplements became a game-changer. Despite dietary changes, it was the supplements that truly revitalised me. Within three months, my energy surged, my skin cleared, and I felt rejuvenated from the inside out.

Empowered by this transformation, I decided to train as a Nutritional Therapist and later established my practice, integrating yoga for a more holistic approach. I empathise with the demands of modern life and the toll it takes on well-being. I firmly believe in addressing underlying issues rather than superficial symptoms, supporting clients in their quest for optimal health.

CAN YOU GIVE AN EXAMPLE OF HOW THE PRODUCTS HAVE MADE A DIFFERENCE TO ONE OF YOUR CLIENTS?

One memorable client journey involved a woman struggling with weight loss amid a demanding job. Instead of fixating on weight and another restricted eating plan, we focused on restoring her

body's balance and reducing inflammation.

With minor tweaks to her diet and strategic supplementation, including Cytoplan's Omega 3 (which is great for heart health and reducing inflammation), Women's Whole Food Multivitamin to address nutritional shortfalls, CytoProtect GI Tract - which addressed the inflammation in her gut, and targeted liver supplements such as CytoProtect Liver Health, her transformation was remarkable. Not only did she shed nearly two stone, but her overall well-being flourished.

Witnessing her newfound confidence and vitality reaffirmed my commitment to guiding others on their wellness journey. It is so rewarding to know that I have been a part of her journey and helping her restore her glow!

In essence, Cytoplan's products have been instrumental in my personal and professional journey, empowering me to make a meaningful impact on others' lives.

DIET

Defining “low carb”: what’s your guess?

A review of 500-plus clinical trials finds growing scientific consensus on “how low you have to go”, but still there is no standardised definition.

Low-carb eating has doubled in popularity among US consumers over the last decade, while a 2023 UK survey saw 13% of respondents saying they were “Low-carb/ no carb”. Yet confusion persists around what exactly qualifies as a low-carb eating pattern.

A scoping review published in *Critical Reviews in Food Science and Nutrition* sheds new light on the topic – and highlights the fact that results from low-carb trials focused on weight loss are ignored on principle by health authorities coming up with dietary recommendations.

Of the more than 500 clinical trials reviewed, the majority defined a low-carb diet as either limiting carbohydrate intake to 30% or fewer of total calories, or eating fewer than 100 grams of carbohydrates daily. The review included results from 508 studies published between 2002 and 2022, over half of which were randomised controlled trials and nearly one-third of which were government-funded.

“The sheer volume of clinical trials on low-carb diets published over the last two decades was striking”, notes principal

investigator Dr Taylor Wallace. “Any perception that there is a lack of scientific evidence on low-carbohydrate eating patterns, or even a lack of government-funded evidence on the matter, clearly is not supported by the data”.

The review also found that 152 of the studies included in the analysis were designed to assess a low-carbohydrate diet’s effect on weight or body composition. Notably, these studies are often excluded from consideration in federal nutrition evidence review processes, such as updates to the Dietary Reference Intake (DRI) guidelines for carbohydrates. These guidelines are the basis of numerous public health and nutrition activities, including food and nutrition labelling, federal nutrition programs, patient counselling and public health education.

“While it may not be surprising to learn that so many studies assessing the impact of low-carb nutrition interventions are focused on weight-related outcomes, it is important to understand that translates into a wealth of clinical data that has no bearing on some of the most foundational tools in US dietary guidance”, adds Wallace. “It leaves a lot of the scientific evidence on the table”.

Additionally, the investigators noted

key gaps in the published literature. Of the studies using per cent of total calories to define low-carb, percentages ranged from zero to 50% of total calories from carbohydrates. And of the studies using the number of grams of carbohydrates consumed daily, many used thresholds well below 100g.

“With both consumers and public health officials interested in understanding the potential benefits of low-carbohydrate eating patterns, arriving at a standardised consensus definition is non-negotiable and urgently needed”, says Wallace.

• **IHCAN comment:** 100g of carbs a day is more “lower carb” - compared to the typical Western diet – than really low carb, which we would put at around 50g a day. Anyone wanting to go full-on keto might have to go even lower. That said, low-carb is not really a diet, even when the aim is losing bodyfat; it is more of a “lifestyle choice” to be sustained, so carbs can go higher. The UK’s most successful low-carb proponent, Dr David Unwin, presents at our November Summit. He has Type 2 diabetes patients who go low-carb and no longer have the disease. Tellingly, he doesn’t set a carb “allowance”, but focuses on helping patients reform their diets and take out high glycaemic load foods.

Arachidonic acid: physiological roles and potential health benefits

“It is time to shift the arachidonic acid (ARA) paradigm from a harm-generating molecule to its status of polyunsaturated fatty acid essential for normal health”. So begins a 2018 review.

Arachidonic acid is an omega-6 PUFA we get from food such as poultry, animal organs and meat, fish, seafood, and eggs, while another important source, particularly for vegans and vegetarians is from linoleic acid, also an omega-6.

As both the 2018 review and the new Cambridge research (reported on page 38) show, arachidonic acid has outgrown its reputation as an pro-inflammatory molecule.

The study’s abstract continues:

“ARA is an integral constituent of biological cell membrane, conferring it with fluidity and flexibility, so necessary for the function of all cells, especially in nervous system, skeletal muscle and immune system. Arachidonic acid is obtained from food or by desaturation and chain



elongation of the plant-rich essential fatty acid, linoleic acid.

“Free ARA modulates the function of ion channels, several receptors and enzymes, via activation as well as inhibition. That explains its fundamental role in the proper function of the brain and muscles and its protective potential against *Schistosoma mansoni* and *S. haematobium* infection and tumour initiation, development and metastasis.

“Arachidonic acid in cell membranes

undergoes reacylation/deacylation cycles, which keep the concentration of free ARA in cells at a very low level and limit ARA availability to oxidation. Metabolites derived from ARA oxidation do not initiate but contribute to inflammation and most importantly lead to the generation of mediators responsible for resolving inflammation and wound healing.

“Endocannabinoids are oxidation-independent ARA derivatives, critically important for brain reward signalling, motivational processes, emotion, stress responses, pain and energy balance.

“Free ARA and metabolites promote and modulate type 2 immune responses, which are critically important in resistance to parasites and allergens insult, directly via action on eosinophils, basophils, and mast cells and indirectly by binding to specific receptors on innate lymphoid cells.

“In conclusion, the present review advocates the innumerable ARA roles and considerable importance for normal health”.

DIET

Protein-rich breakfast boosts satiety and concentration

A new Danish study has found that a protein-rich breakfast can increase satiety and improve concentration.

An Aarhus University team explored how different types of breakfast affect satiety and concentration, following 30 obese women aged 18-30 for three days, during which the women consumed a protein-rich breakfast, a carbohydrate-rich breakfast or no breakfast at all.

The women's sense of satiety, hormone levels and energy intake were measured at lunchtime. Their total daily energy intake was measured as well.

"We found that a protein-rich breakfast with skyr (a sour-milk product) and oats increased satiety and concentration in the participants, but it did not reduce the overall energy intake compared to skipping breakfast or eating a carbohydrate-rich breakfast", says Mette Hansen, associate professor.

Combat obesity

Previous studies have shown that people who eat breakfast have a lower BMI than people who do not eat breakfast, and protein-rich foods have generally been shown to have an increased satiety effect compared to carbohydrate-rich and high-fat foods with the same calorie content. The idea was therefore to test whether a protein-rich breakfast could be a good strategy to achieving greater satiety during the day and thus reducing daily calorie intake.

However, the solution is not that simple, says Hansen: "The results confirm that protein-rich meals increase a sense of satiety, which is positive with regard to preventing weight gain. However, the results also suggest that for this nutritional strategy to be effective, it's not enough to just eat a protein-rich breakfast".

The potential of replacing a carbohydrate-rich diet with a protein-rich diet can clearly be seen in the satiating effects measured in the study. Several of the subjects had difficulty consuming the entire protein-rich breakfast consisting of skyr and oats.

"It's intriguing that there can be such a big difference in the satiety effect of two different meals with the same calorie content. Had the women in the project been allowed to choose the size of the meal themselves, it's likely that they'd have consumed more food and thereby more calories on the day they were served bread and jam than on the day they were given skyr and oats", explains Hansen.

"We already have new data incoming from a trial where participants received either a high-protein breakfast or a low-protein breakfast. The objective was to study how the different types of breakfast affect body composition and other parameters such as microbiota and cholesterol levels", says Hansen.

• **IHCAN comment:** Prof Hansen is a PhD in Aarhus's Department of Public Health and has said the results of this research may lead to new, "targeted" dietary recommendations. Skyr is only "high" in protein compared to other yoghurt-type foods. A typical serving - such as a small tub of Siggis - provides a mere 16g of protein. A cup of oats adds about 11g of protein, but 55g of carbohydrate (and 307 calories). This is not a "high protein" breakfast in our book.



MOVEMENT THERAPY

Women get the same exercise benefits as men, but with less effort

Women can exercise less often than men, yet receive greater cardiovascular gains, according to a new study from the Smidt Heart Institute at Cedars-Sinai.

"Women have historically and statistically lagged behind men in engaging in meaningful exercise", said Martha Gulati, MD, director of Preventive Cardiology. "The beauty of this study is learning that women can get more out of each minute of moderate to vigorous activity than men do. It's an incentivising notion that we hope women will take to heart".

Investigators analysed data from 412,413 U.S. adults in the National Health Interview Survey database. Participants between the time frame of 1997 to 2019 - 55% of whom were female - provided survey data on leisure-time physical activity. The researchers examined gender-specific outcomes in relation to frequency, duration, intensity and type of physical activity.

"For all adults engaging in any regular physical activity, compared to being inactive, mortality risk was expectedly lower", said senior author Susan Cheng, MD, MPH. "Intriguingly, though, mortality risk was reduced by 24% in women and 15% in men".

The research team then studied moderate to vigorous aerobic physical activity, such as brisk walking or cycling, and found that men reached

their maximal survival benefit from doing this level of exercise for about five hours per week, whereas women achieved the same degree of survival benefit from exercising just under about 2½ hours per week.

Similarly, when it came to muscle-strengthening activity, such as weightlifting or core body exercises, men reached their peak benefit from doing three sessions per week and women gained the same amount of benefit from about one session per week.

Cheng said that women had even greater gains if they engaged in more than 2½ hours per week of moderate to vigorous aerobic activity, or in two or more sessions per week of muscle-strengthening activities.

The investigators note their findings help to translate a longstanding recognition of sex-specific physiology seen in the exercise lab to a now-expanded view of sex differences in exercise-related clinical outcomes.

With all types of exercise and variables accounted for, Gulati says there's power in recommendations based on the study's findings.

"Men get a maximal survival benefit when performing 300 minutes of moderate to vigorous activity per week, whereas women get the same benefit from 140 minutes per week", Gulati said. "Nonetheless, women continue to get further benefit for up to 300 minutes a week".

BIOMECHANICS

Diabetes and back pain explained: hyperglycaemia “stiffens” the discs

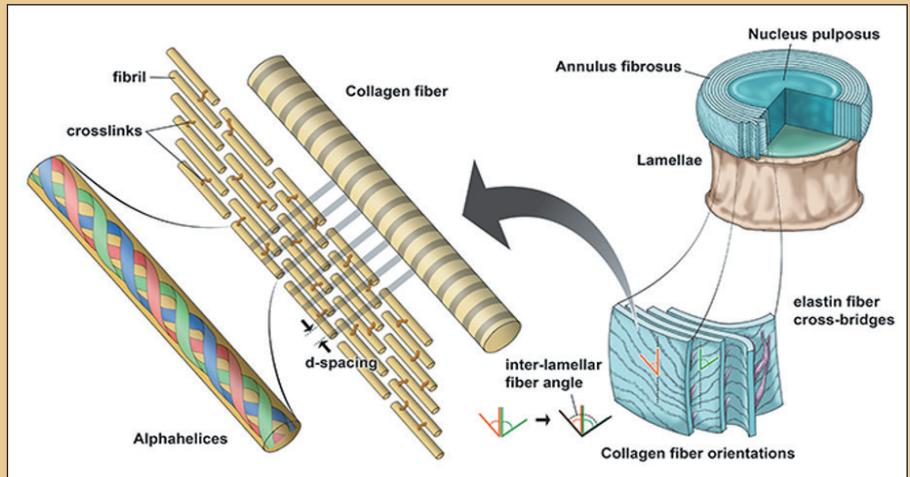
Type 2 diabetes alters the behaviour of discs in the vertebral column, making them stiffer, and also causes the discs to change shape earlier than normal. As a result, the disc’s ability to withstand pressure is compromised.

This is one of the findings of a new study in rodents from a team of engineers and physicians from the University of California San Diego, UC Davis, UCSF and the University of Utah.

Low back pain is a major cause of disability, often associated with intervertebral disc degeneration, and people with Type 2 face a higher risk of low back pain and disc-related issues, yet the precise mechanisms of disc degeneration remain unclear.

The research team was co-led by Claire Acevedo, a faculty member in the Department of Mechanical and Aerospace Engineering at UCSD and orthopaedic surgeon Aaron Fields, UC San Francisco. The researchers employed synchrotron small-angle x-ray scattering (SAXS), an experimental technique that looks at collagen fibril deformation and orientation at the nanoscale.

They compared discs from healthy rats to those from rats with Type 2 diabetes (UC Davis rat model). The healthy rats showed that



collagen fibrils rotate and stretch when discs are compressed, allowing the disc to dissipate energy effectively.

“In diabetic rats, the way vertebral discs dissipate energy under compression is significantly impaired: diabetes reduces the rotation and stretching of collagen fibrils, indicating a compromised ability to handle pressure”, say the researchers.

Further analysis showed that the discs from diabetic rats exhibited a stiffening of

collagen fibrils, with a higher concentration of non-enzymatic cross-links. This increase in collagen cross-linking, induced by hyperglycaemia, limited plastic deformations via fibrillar sliding. This sliding is one of the crucial mechanisms facilitating healthy whole-disc compression.

Type 2 diabetes disrupts these efficient deformation mechanisms, leading to altered whole-disc biomechanics and a more brittle (low-energy) behaviour.

IMMUNITY

Researchers discover new cell that remembers allergies

Researchers with McMaster University and Denmark-based pharmaceutical company ALK-Abello A/S have made a groundbreaking discovery: a new cell that remembers allergies.

The research, published in *Science Translational Medicine* last month, coins the brand-new cell as a type-2 memory B cell (MBC2).

“We’ve discovered a type of memory B cell that had unique characteristics and a unique gene signature that has not been described before”, says Josh Koenig, assistant professor with McMaster’s Department of Medicine. “We found allergic people had this memory B cell against their allergen, but non-allergic people had very few, if any”.

B cells are a type of immune cell that makes antibodies. These cells help fight off infections but can also cause allergies.

“Let’s say you’re allergic to peanuts. Your immune system, because of MBC2, remembers that you’re allergic to peanuts, and when you encounter them again, it creates more of the antibodies that make you allergic”, Koenig says.

To come to this discovery, researchers



McMaster University researchers Allyssa Phelps, left, and Josh Koenig, are part of a team that discovered a new cell that remembers allergies.

created tetramers - a type of fluorescent molecule - out of allergens like Birch pollen and peanuts to locate difficult-to-find memory B cells. Researchers further leveraged samples from ALK clinical trials with tablet sublingual immunotherapy, which allows for sequencing large amounts of IgE-producing B cells.

The “home” of allergy

Using cutting-edge technology, they were able to make direct connections between MBC2

and IgE, the type of antibody that triggers the allergic reaction. This provided necessary context, ultimately revealing the MBC2 as the home of allergy.

“Even though allergies are the most prevalent disease worldwide, it is still not fully understood how allergy occurs and evolves into a life-long condition. Finding the cells that hold IgE memory is a key step forward and a game-changer in our understanding of what causes allergy and how treatment, such as allergy immunotherapy, can modify the disease”, says Peter Sejer Andersen, head of research at ALK.

“The discovery really pinpoints two potential therapeutic approaches we might be able to take”, says Kelly Bruton, who co-led the research alongside Koenig when she was a PhD student at McMaster. “The first is targeting those MBC2s and eliminating them from an allergic person. The other option could involve changing their function and have them do something that’s not going to be ultimately harmful when the individual is exposed to the allergen”.

AGEING

England's oldest became frailer during austerity

The speed at which England's oldest adults became frailer accelerated during the UK Government's era of austerity politics, according to a new study.

Researchers say that the rate of frailty in people aged 85 and over in England increased 50% faster per year between 2012 and 2018 compared with the preceding eight years.

The impact of frailty - a decline in a person's mental and physical resilience to illness and injury - on the oldest in society must be considered should any new austerity measures be introduced, experts warn.

The study, led by researchers from the University of Edinburgh's Advanced Care Research Centre, analysed data from 16,410 people in the English Longitudinal Study of Ageing, a nationally representative sample of the English population aged at least 50 between 2002 and 2018. Researchers combined this with the frailty index, which captures broad age-related declines in functional ability and physical and mental health.

The sample had an average age of 67 years and an average frailty index score of 0.15 (on a scale of 0 to 1, with 1 being maximum frailty). Frailty index scores increased more rapidly across all genders and socio-economic groups during the study period, but it was particularly noticeable in the oldest people.

Frailty levels dropped in the 2000s but experienced a steep increase in the 2010s, when the UK government introduced a wave of public spending cuts in response to the 2008 global financial crisis, with all ages losing improvements that had been made.

For the oldest, the improvements were lost entirely, and they were frailer than those of the same age living in the prior decade, experts say.

The researchers did not examine why public sector cuts might cause these changes, but they say the findings correspond with the flattening of life expectancy seen in the 2010s, with higher mortality rates particularly seen in the eldest.

Dr Carys Pugh, Research Fellow at the University of Edinburgh, said: "A key implication of this research should be a recognition that public spending reductions likely have negative impacts on health and, in turn, mortality, particularly amongst the oldest in society. Frailty normally increases with age but as we emerge from the pandemic and into a cost of living crisis, any new austerity measures need careful consideration given their potential impact on long-term health, especially among the eldest who appear particularly vulnerable".



PHYSIOLOGY

Finger proportion linked to oxygen consumption

The efficiency of oxygen supply to tissues is a factor in the severity of important diseases such as COVID-19 and heart conditions. Scientists already know that the relationship between the length of a person's index and ring fingers, known as the 2D:4D ratio is correlated with performance in distance running, age at heart attack and severity of COVID-19.

Swansea University research has now found that footballers with a relatively longer ring finger compared to the index finger have more efficient oxygen metabolism.

Swansea University digit ratio expert Prof John Manning has been working with colleagues to look more closely at the subject, and has published findings from analysis of 133 professional football players. The footballers also completed an incremental cardiopulmonary test to exhaustion on a treadmill.

"With our partners from the Cyprus campus of the University of Central



Lancashire, we have clarified the relationship between 2D:4D and oxygen metabolism in a sample of well-trained athletes", said Prof Manning.

"The players with long ring digits (4D) relative to their index digits (2D) have efficient oxygen metabolism such that they reach very high maximal oxygen consumption in an incremental cardiopulmonary test to exhaustion on a treadmill".

Long ring digits relative to index digits are thought to be a marker of high testosterone levels in the womb. Testosterone has effects on oxygen metabolism through its influence on mitochondria.



“The world is very sick, and I fear for our future. I look at kids’ lifestyles, especially their diet, and it shocks me”

NAME: Keith Duffy.

WEBSITE: <https://www.keithduffyhealthcoach.com>.

QUALIFICATIONS: Health Coach.

TRAINING: Advanced Diploma in Nutrition and Health Coaching. Certificate in Sports Nutrition and Weight Loss. Both from IINH, the International Institute of Nutrition & Health (formerly the Irish Institute of Nutrition and Health): <https://www.iinh.net>.

How long did it take for you to qualify?

One year.

Tell us something about your training. What stands out for you?

Firstly, the tutors. Our main one was the director and founder of IINH, Richard Burton. The Institution was founded in 2002 and was the first in the world (as far I know) that offered a certificate in Health Coaching. His personal story and journey are very interesting. But what I liked about his teaching was the very relaxed manner it was done in. It was like story time with your grandad, even though he’s not that old at all! Although necessary, it never really felt like you were studying. It was like pull-up a pew and listen to what the man has to say - and he has a lot to say. He’s extremely approachable and extremely likable.

There was another tutor who has recently taken a step back from teaching I heard - Julia Sweetman. With her, things were different; she constantly tested us to make sure we weren’t falling behind, which was also really important as there is so much to take in. The contrasting methods were ideal I thought. Secondly, and what stands out for me in comparison to other Health Coaching courses, was that we had to do four real case studies, ie find a friend or family member who needed help with their health.



This alone was a game-changer for me, as we could immediately start using our newly-gained knowledge. All the case studies were monitored by Richard or other tutors.

Where do you practise? I live in Barcelona, so I do face-to face consultations but also online with clients from around the world.

How long have you been in practice? Since 2020.

What conditions or types of clients do you see most?

Clients who are overwhelmed with information. Digestive issues. Weight problems. In particular two types of clients:

- Clients who are lost in relation to their diet, what to eat and when.
- Clients who have weight issues, but especially those who are overweight. Why is this a feature

of my practice? I was a chubby kid, not so much a teenager, but they were years that scarred me, so I know what it’s like to feel like that and feel hopeless. I felt ugly and the odd one out among my slim friends.

Back then if you were overweight in any way, the reason was because you weren’t moving enough, and trust me, I was moving a lot! I played most sports and I was good at nearly all of them except football, to my Dad’s dismay. He managed the local football club for nearly 20 years. I used to go mark the pitches and put up the nets with him. Then I’d go off and play basketball!

My problem with my weight was my diet, but of course nobody really knew about that back then.

Why did you decide to become a practitioner?

Two reasons. I used to live in Brazil with my



Images: Elena Benedettini - <https://www.elenbvisual.com>

wife. While I was there, I practised some type of sport five days a week. Two days Brazilian jiu-jitsu, two days of Pilates (to put me back together post jiu-jitsu) and the other day I ran. As far as I was concerned, I was fit. In 2014, while on a bike ride coming from lunch with friends, I got a pain down my back. It went away once I got home. But the next day it was the same. The following Tuesday I was walking to work and I had to stop to catch my breath. That's when I knew something was wrong.

After doing various tests I found myself sitting in front of a specialist who rifled through all my test results until he came to the one he was looking for. He opened it, closed it. Asked me if my father was alive, and if not, how he had died. He's not and he died in his sleep - a heart attack. He was 68. He'd had a triple bypass done when he was coincidentally my age at the time, 45. My mum had died of ovarian cancer at the early age of 50 and that crushed him. He soon went back to smoking and drinking heavily. The specialist in front of me was a cardiologist, and right there, he stood up, picked up the phone and rang the hospital. He spoke to a doctor and informed him he was sending somebody in to see him. He put the phone down and told me to get a taxi to the hospital immediately. "It's Carnival here next week Keith, you know how it is, everything shuts down, so it's better we get this checked out ASAP".

In the end, I had three stents put in; one of the arteries was the main problem - it was 95%

"Just as people became a little more knowledgeable about money during the financial crisis of 2008, so happened the same with food during COVID. Everybody became an expert. News changed every day depending on whom or what you were listening to. But there is so much overwhelming misinformation out there that clients suspect everything".

blocked. My cardiologist told me later that the Carnival story was just a ruse to get me in. He said I was going to be one of those "fit" people you hear about who drops dead during a tennis match. I had assumed that because of my genes, it was only a matter of time. But it made me wonder, is that everybody's destiny? Sick parents lead to sick children?

A couple of years later, I turned vegan (I'm not anymore), but that's when I really started to look at food and food sources, labelling, ultra-processed food etc, and the more I read, the more I took heed and I started to "encourage" people to do the same.

When I say "encourage", I mean I became that back-seat driver, that reformed smoker - ie really really annoying!

A friend of mine sent me a cookbook, and under the profile of the author I saw that she had studied to be a Health Coach and it was the title that grabbed my attention. I have always been some type of coach throughout my life.

Qualified basketball and volleyball coach in school and clubs. I am an ESL teacher. I like being able to pass on what I know to others, and I enjoy speaking in front of people or even in public. This was the second reason I decided to become a Health Coach.

What do you find the easiest to work with?

Clients who are really willing to do the work necessary. My favourite types of clients are those that want to change not only for themselves, but for everyone around them.

I think we forget the impact our lifestyle choices have on the people around us. The decisions we make on a daily basis may mean someone in the future having to spoon-feed us, push our wheelchair or not.

What are the most challenging types of symptoms/illnesses/problems that you get presented with?

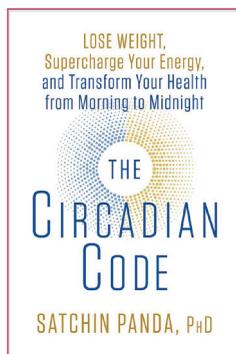


Skin issues. There can be so many factors involved. In the diet alone, there can be a multitude of reasons, including food sensitivities and allergies. Outside factors including work, family and friends issues, stress.

One interesting case I heard about was about a woman who had returned to her family home after being away in New Zealand for a year. She suddenly found that she was getting very bad

skin rashes. They seemed to get worse when she was around the family cats, even though she had grown up with cats and dogs from an early age. She happened to be studying nutrition at the time and talked to her lecturer about it. He spoke to her about her lifestyle and background and finally recommended that she cut out lactose for a couple of weeks. The rashes disappeared. She asked if she would not be able to eat milk again. He suggested she slowly re-introduce into her diet, which she did, and she was fine later and could also interact again with her cats.

The reason in the end wasn't the cats or lactose issues, but it was down to stress; she was finding the course a challenge and her body's reaction to it was to produce a skin rash.



What one thing is absolutely essential to you in your practice?

My wife. Apart from looking after my social media, as it's one part of my work I really have no time for, she supports me and believes in me unconditionally even knowing how challenging it can be to work in this field.

Which CAM book has helped or inspired you most, so far in your career?

Hmmm, there are so many, but to choose just one, I'd probably say *The Circadian Code*, by Satchin Panda, PhD.

Why do you do what you do?

Really want to help people. The world is very sick, and I fear for our future. I look at kids' lifestyles, especially their diet and it shocks me. Since COVID, everybody is more aware of the problems with ultra-processed food, sugar and movement. The enormous effort that is made in bringing up children, yet in most cases, they still eat "garbage" and then later on we permit them to drink alcohol and smoke cigarettes.

I live in Spain, and I find it most frustrating that people abroad go on about the Mediterranean diet. Come here and you'll see that nearly 50% of adults are overweight, one in

five being obese, 30% of teenagers, and - really worrying - 40% of children are overweight, with 10% of kids being obese. So what do these people do? They don't go to see Health Coaches or Nutritionists, they'll go to the hospital to talk to doctors who know very little about nutrition and in each one of these hospitals or clinics you'll find vending machines selling Coca-Cola and chocolate.

We all blame Food Inc for everything, but what about cafés and restaurants? I don't see anybody complaining about them. I'll only speak about here in Barcelona, where I live. But so many of these Spanish cafés or restaurants have menus that are just full of fried food, but yet we still sit there and order it along with beers and white bread because the café next door is serving the same rubbish. Eateries need to start taking responsibility for the food they serve, and offer healthier options and while they're at it, get rid of their fryers and replace them with grills.

I find it so frustrating eating out, yes, even in Spain, that the food on the menu can be just as bad as fast food, and that I should have stayed at home and cooked myself. The majority of times when my wife comes home from having dinner out, she says the same: "Your food is better". It shouldn't be like that. A lot of the time when I'm going to meet-ups with friends or my friend's kids' birthdays, or something, I'll fill up before I go and end up just drinking water, because I

know the options available are unhealthy and are not doing me any favours whatsoever.

Whether you're sitting in the sun in Barcelona, or hiding from the rain in Dublin, 'batatas bravas', white bread, fried fish and croquettes, (followed by beer and cigarettes) shouldn't be considered part of any healthy diet, regardless of what name you attach to it.

If money, time and effort were no object, what one thing would you change about your practice or integrative medicine in general?

It sounds a little bit out there, but if I had the money, I would set up a self-defence school, maybe a Brazilian jiu-jitsu school, for women, as the rates of domestic violence here in Spain are outrageous. But part of the school and compulsory would be nutrition lifestyle.

I would also like to do more public speaking, as I very much enjoy it and I like to educate and therefore empower people to look after their own health as well as those closest to them.

About integrative medicine, I would start introducing nutrition early as a subject in school or something to be spoken about regularly. Kids need to be told about the harmful effects, not only of smoking, but of alcohol too. The Government should set up free

programs to educate parents on nutrition, even organising cooking classes for adults.

Companies in general need to take a more hands-on approach to the wellness of their employees. In gyms, once you start, a sit-down with a nutrition expert should be compulsory and not an option.

What piece of advice would you give to newly-qualified practitioners who are just setting up a business?

Coach as much for free as possible. Start off with family and friends; it doesn't matter how little or unimportant they feel their issues might be. Forget about a website; if need be, have a presence on Instagram, Tik Tok and LinkedIn. Do one post a week - be consistent. I know people who don't have any site or social media platforms and work by word of mouth.

Keep learning, get as many qualifications as you can under your belt. And of course, subscribe to *IHCAN* magazine.

What is the biggest challenge you face as a practitioner?

Health Coaching is still relatively new, and so getting the news out there about us, especially here in Spain, and getting people to trust us is challenging. It's a slow burn, but we're getting there.

How did you adapt personally and professionally to the COVID-19 climate – how has it impacted your practice?

My work was definitely affected, especially financially, but I adapted - I offered discounts and did consultations online and I survived by having other sources of income.

Just as people became a little more knowledgeable about money during the financial crisis of 2008, so happened the same with food during COVID. Everybody became an expert. News changed every day depending on whom or what you were listening to. But there is so much overwhelming misinformation out there that clients suspect everything.

It's great that people are empowering themselves, but why are we still getting sicker? It's nice to be able to help.

What (or who) would you like to see covered in *IHCAN*?

A stand-alone issue on alcohol. I think it's the elephant in the room. Why? Because most people drink, including, Health Coaches, Nutritionists and doctors and maybe even worse, politicians...and heavily...in most cases.



Case study: Hashimoto's, fatigue, carpal tunnel and joint pain

A woman, 42 years old, originally from Sardinia, presented herself to me in February 2020. She had Hashimoto's as well as Carpal tunnel syndrome. She was gluten intolerant, had a nickel allergy and low blood sugar.

She slept badly, woke up a lot during the night, and was constantly tired. She had skin rashes. Her nails were brittle, and her hair was dry. She suffered from headaches and was sensitive to loud sounds. She had pain in the lower back if she sat down for too long. In the last couple of years, she had gained a lot of weight. She enjoyed her job, but it was stressful due to the workload.

Family background check revealed that her sister and mother also have Hashimoto's. Her brother has Hashimoto's as well as MS and diabetes.

She came to me wanting to clean up her diet, to learn to have a healthier lifestyle and to lose 20kg.

Assessment

With all my clients, before the main consultation I ask them to complete a Lifestyle Diary, which details everything from food, water, supplement and medication intake, to sleep and exercise routines. It's a seven-day diary and one I can also keep an eye on as they are completing it.

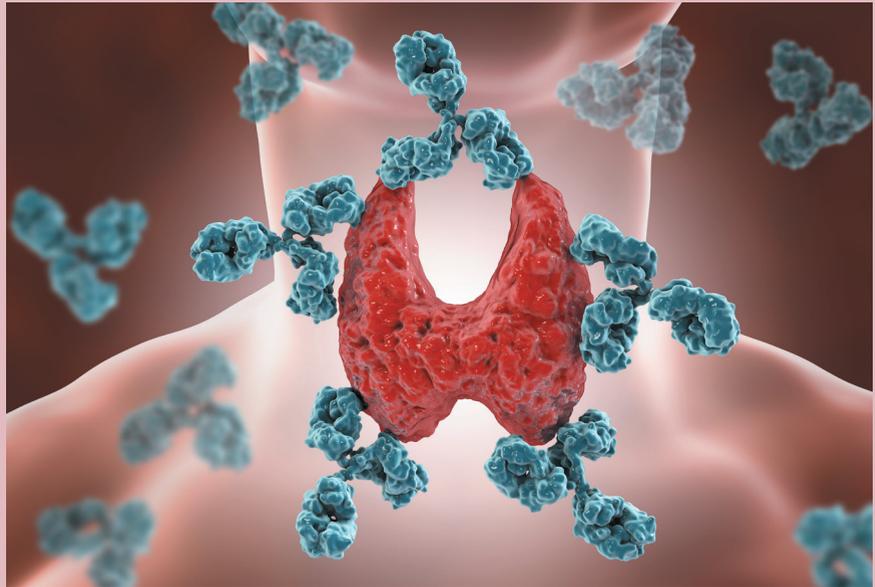
This in itself is work for the client. For nearly all of them it's like laying out all they consume on a table. They really get to see what they're eating. Most of my clients really appreciate this as part of the process.

Intervention

She was my very first client. We spoke about Hashimoto's during my course, but not a lot. I knew it was an autoimmune disease and it could be triggered by a food intolerance. I thought I would start there. She was already allergic to gluten, so the next obvious choice might be lactose, but something that was more prevalent in her diet were eggs - about 12 a week. I asked her to cut eggs out for two weeks and we'd speak then.

Outcome

It was ten days later when I sent her a text. It was a little less than the planned two weeks, but she was my first client and I was very curious as to how she was getting on. "You've changed my life", was her response. It must have been the eggs. She said it was like a switch. Her hair had started growing again and the shine had reappeared. Her nails were longer and stronger.



She was sleeping better. The pains were gone from her joints. She hadn't had any rashes. Her energy levels had skyrocketed and the weight was falling off her. (She later told me that she lost nearly five inches off her waistline).

When we did the online call a few days later, she was glowing. The change in her face alone was remarkable. She was so very happy. I still get goosebumps thinking about it.

Progress/Future

The client doesn't live here in Spain, but we have been in contact from time to time. As far as I'm aware now she's still doing well and I'm currently coaching her boyfriend.

Review/conclusion/learnings

What I've learned from this client and subsequent others was that you can get lucky - and also to expect people to be intolerant to anything!

Sometimes you can be quick to identify and sometimes not. But you just start with the most common one and work from there.

References and resources

From my course I learned to expect the wonderful and that intolerances can be anything. Moreover, they can also just arise and disappear without us knowing when or why or how. It's quite interesting actually.

We hadn't spoken much about Hashimoto's, but I researched about it and found Dr Izabella Wentz and her book *Hashimoto's Protocol*.

She grew up with Hashimoto's but not knowing what it was. It was crippling to her in her childhood; she had serious health issues and nobody knew

why. Her own journey is worth reading. She grew up in Poland and she thinks one of the reasons for her getting Hashimoto's was from the fallout of Chernobyl. It reminded me of my client from Sardinia telling me that Hashimoto's and MS were quite common on the island. When I asked her why, she felt it might have come from the NATO activity there. There's been a military base there since 1940! Curious to think that there is this on one part of the island, then on another, high in the mountains, there are people who live to be 100 years old or more. An area considered one of the few Blue Zones in the world.

HASHIMOTO'S PROTOCOL

A 90-Day Plan for
Reversing Thyroid Symptoms
and Getting Your Life Back



We know our practitioners are quietly getting on with changing people's lives, every day – and we want to celebrate and share the inspiration. In Practice is coordinated by regular contributor Rebecca Smith, who runs a successful practice of her own, established 20 years ago. Contact her direct to be part of the feature: rebecca@newportcomplementaryhealthclinic.co.uk, and follow her on Twitter: [@NCHHealthClinic](https://twitter.com/NCHHealthClinic).

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Treating Myopathy and Myalgia: lessons from athletes

Can we learn some things about neuromuscular pain and injury from sports physiology that we cannot from medical research? Athlete and research scientist **Dr CHANDLER MARRS**, PhD, slams the standard medical approach for presupposing “a certain futility”: the disease process is inevitable and cannot be halted or healed, just “managed”. This is not what the exercise physiology research says.

As a lifelong jock, athletics have been a part of my life for as long as I can remember, and though the sports I have participated in over the years have changed (my current passion is powerlifting), the lifestyle has remained the same. I train. I compete (even over 50), and I train some more. I enjoy training. I enjoy having goals to reach. For those of you who follow hormonesmatter.com, you know that I am also an avid researcher. Research is as much a part of who I am as is athletics, and so when the two sides meet, I am thrilled. Sometime back, those two worlds intersected. While reading an article (1) about the molecular mechanisms involved in hypertrophy - muscle gain - this sentence hit me like a load of bricks:

“The synthesis of muscle proteins must outpace the breakdown of muscle protein”.

Well, of course it must. Duh, right? This is so obvious in sports medicine and yet so underappreciated in conventional medicine. I’ll say it again. “The synthesis of muscle proteins must outpace the breakdown of muscle protein”. In other words, one has to “eat to perform”.

Think about it for a minute, what is required for muscle gain? Two things: food/fuel (2) and training (3). Sufficient fuel to promote protein synthesis and sufficient training that breaks it all down. It is a never-ending cycle. If you demand too much, if the breakdown outpaces the synthesis, survival mechanisms kick in, muscles eat (catabolise) themselves for fuel. Lactate builds up and that familiar fatigue and pain ensue.

The same thing happens if you train hard

but eat poorly. Without sufficient nutrients, protein synthesis cannot happen, at least not appropriate to the demands. No one doubts this chemistry in sports, but in medicine, we seemed to have missed the parts about providing the muscles fuel and activating them to process that fuel. We look for all manner of mechanisms to explain muscle loss in patients, except the most obvious: the absence of fuel and/or activity.

What do you need?

On the most basic level, it begs a question about what is required for protein synthesis in muscles. What are the core substrates, what is the fuel that powers this process? Well, firstly, it requires energy or ATP, perhaps consuming as much as 70% of the ATP produced (4). That means we need healthy mitochondria (5). Digging deeper, what do we need for healthy mitochondria? An abundant supply of macro- and micronutrients. In other words, we need to consume sufficient protein, fats and carbohydrates, along with sufficient vitamins and minerals to produce ATP, so that protein synthesis can occur at a rate greater than protein breakdown.

Can we learn some things about neuromuscular pain and injury from sports physiology that we cannot from medical research? I think we can.

Optimum performance versus symptom management

The research in sports physiology is useful for not only understanding athletic performance, but also for understanding the limits of health at the molecular level. In many cases, the work in sports physiology is far more detailed and I would dare say, hopeful, than similar topics covered from the medical perspective. In sports research, the objective is to understand and maximise optimum performance (3). Even in

the case of injury, the underlying goal is always to expedite recovery and how best to support the healing process. When athletes are injured or become ill, the question is not if they can train again, but when and how quickly they can return. **The prevailing assumption is that they will heal.**

In contrast, medical research involves understanding a disease process, and more often than not, any consideration of mitigating that disease process involves only the treatment of symptoms, which are surrogate markers of the illness at best. In medicine we get lost in in the



“On a daily basis, I interact with dozens of individuals who have been injured by the medical model and the ill-conceived notion that medications lead to health. Inevitably their illnesses involve some degree of mitochondrial dysfunction, and more often than not, core symptoms will include muscle pain and weakness”.

weeds, endlessly searching for this or that aberrant mechanism to pin the “disease” on. Rarely, does medicine ask why a particular mechanism might be aberrant, and even more rarely is there any consideration of how to correct the presumed disease process or maximise the health of the affected tissues, organs, or even the human experiencing that disease. Some would argue that approach emanates largely from of our predilection for pharmaceutical solutions, and to some extent I would agree. I think there is more to it, however.

In many ways, the medical model

presupposes a certain futility. It is as if the disease process, once initiated, can only be managed and never overcome. Health seems to have been written out of the equation entirely. Indeed, that health or illness evolves from a human organism and not just detached mechanisms seems absent as well, but that is a broader, more philosophical discussion (6) for another day.

Returning to our comparison of approaches, as an athlete and a research scientist I find the more hopeful perspective of sports research entirely more useful not only for athletics but for recovery in general.

Myopathy and Myalgia

What does any of this have to do with treating myopathy and myalgia?

On a daily basis, I interact with dozens of individuals who have been injured by the medical model and the ill-conceived notion that medications lead to health. Inevitably their illnesses involve some degree of mitochondrial dysfunction, and more often than not, core symptoms will include muscle pain and weakness, which in medical speak is myalgia and myopathy.

And yet, despite years of failed treatments and endless pain and suffering, when I ask

→ about diet and lifestyle, to say that I am met with chagrin is an understatement. “How could diet and exercise possibly be involved with the muscle wasting, with myalgia or myopathy? Those are legitimate illnesses”, I am sure they think to themselves. Well, let me repeat myself,

“The synthesis of muscle proteins must outpace the breakdown of muscle protein”.

This is something so simple (7) and utterly obvious to any athlete, but completely foreign to most in the medical field. Consider any discussion in the medical literature on muscle wasting - cachexia (8) or sarcopenia (9) - or disorders involving muscle pain and weakness: rarely is there any consideration given to the nutritional demands that drive these molecular changes.

Is the patient getting what he/she needs nutritionally to drive protein synthesis? Seems like an obvious first question, but it is not.

A second, but equally obvious question would include activity level, and barring conditions that restrict mobility, activity should be a core component of treatment and rehabilitation. If either or both of these factors are ignored, no amount of medicine will promote recovery, and in that regard, the model of disease futility is accurate, albeit self-perpetuating.

I would argue that the muscle pain and wasting we see with many an illness often represents no more or no less than a skewed balance between the synthesis and breakdown of protein. Sure, there are likely some individual genetic components that contribute, but no matter the genetics, if the substrates derived from diet are absent or lacking, muscle pain and weakness will develop and when there is muscle pain and weakness, activity is limited. Similarly, if mobility or activity is constricted or absent, protein synthesis will slow down, even if dietary considerations are met. Muscle requires adequate dietary proteins and adequate movement to avoid myopathy, myalgia, sarcopenia, cachexia. If either, but especially when both of those variables are lacking, the downward spiral of decreased protein synthesis and increased protein breakdown in muscles begins.

I hear the series of “buts” from clients now.

“But Chandler, you don’t understand, I cannot [fill in the blank] because of [fill in the blank]”.

In fact, every time I comment online regarding the need for either more protein in the diet or the requirement for more (or any) movement to recover, I am lambasted. How

“These disease processes are real, but within the current medical model the ‘realness’ of a disease is determined only by its connection to a pharmaceutical and the ability of the pharmaceutical to temper some surrogate marker of that disease process”.

dare I suggest diet and exercise are required to maintain health or recover from illness?

Admittedly, this flies in the face of modern medical treatments, with too many physicians looking to diet and exercise only as a last resort, and generally, with the very heavily biased assumption that if diet and exercise are involved, then the conditions for which the patient is seeking treatment must not be real.

Of course this is asinine. These disease processes are real, but within the current medical model the ‘realness’ of a disease is determined only by its connection to a pharmaceutical and the ability of the pharmaceutical to temper some surrogate marker of that disease process. When there are no pharmaceuticals that return the patient to health (because, given the chemistry, how could there be?), it is an easy jump to assume the patient is feigning their symptoms. They are not feigning their symptoms. The chemistry involved in the muscle wasting is real, but so too is the solution. The body needs certain things to function, period. When it does not get those things, compensatory reactions ensue and disease processes begin.

Severe muscle wasting

Returning to muscle pain and weakness and the more severe process of muscle wasting, we see littered throughout the research, the “recipe” for muscle health: protein and activity. For example, in the research involving muscle wasting in critical care patients where immobility is common (10), do you know what prevents muscle wasting? Movement. That is, the simple act of passive resistance - eg someone moving or exercising the muscles for the patient, reduces muscle wasting. This is because a whole slew of

mechanosensors in the musculature must be activated regularly so that the cycle of protein degradation and synthesis continues.

Immobility grinds this cycle to a halt. The muscles need to be activated to send the requisite signals that protein cycles should continue. Absent those signals, in other words, absent movement, atrophy and cachexia cycles begin, developing within only a few weeks of immobility.

Similarly, a review addressing various forms of muscle wasting (11) from sarcopenia (muscle loss in the elderly) to cachexia (muscle loss in cancer), found that both providing additional protein in diet and enabling resistance training, in whatever form possible, even passive resistance, decreased muscle wasting and increased function and quality of life.

This tells us that the answers are right in front of us. Dietary protein and activity are requisite to avoid muscle wasting. Absent one or both of these and problems develop. Absent one or both for a sufficient period of time and muscle wasting, pain, and other processes begin. Oh, and the brain needs protein too (12).

* References online at www.ihcan-mag.com/references.

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About the author

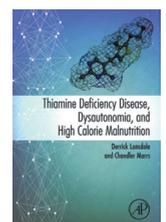
DR CHANDLER MARRS, MA, MS, PHD, is the founder and editor of Hormones Matter and has spent the last dozen years in women’s health research with a focus on

steroid neuroendocrinology and mental health. She has published and presented several articles on her findings.

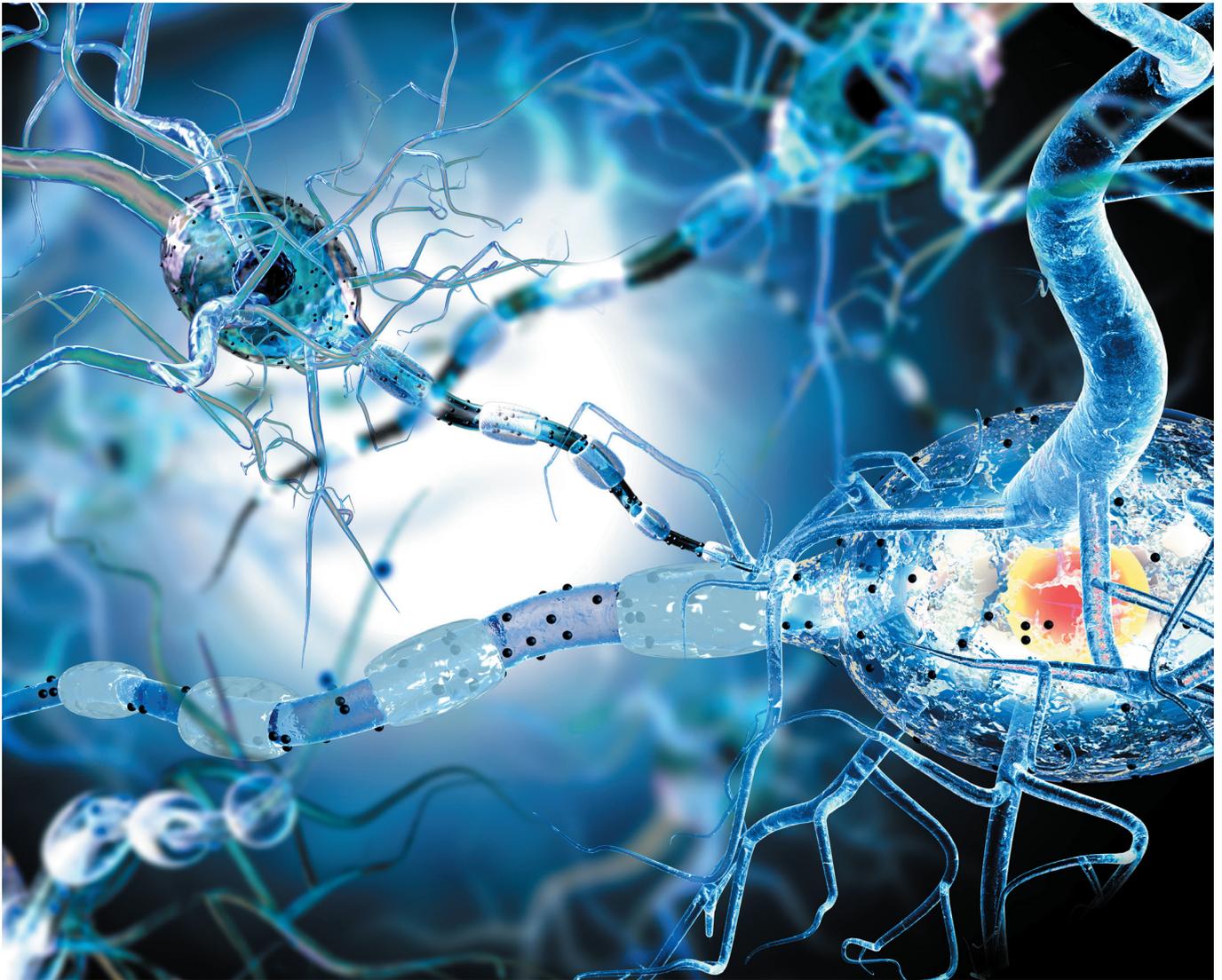
Hormones Matter™ is an online, community publication dedicated to advancing health research and knowledge.

As a graduate student, she founded and directed the University of Nevada, Las Vegas, UNLV Maternal Health Lab, mentoring dozens of students while directing clinical and Internet-based research. Post-graduate, she continued at UNLV as an adjunct faculty member, teaching advanced undergraduate psychopharmacology and health psychology (stress endocrinology).

Dr Marrs received her BA in philosophy from the University of Redlands; MS in Clinical Psychology from California Lutheran University; and her MA and PhD in Experimental Psychology/Neuroendocrinology from UNLV. She is co-author with Dr Derrick Lonsdale of the book *Thiamine Deficiency Disease, Dysautonomia, and High Calorie Malnutrition*.



• Follow Chandler on twitter @hormonesmatter.
• <https://www.hormonesmatter.com>.



A thiamine case report: post-COVID neurological symptoms

Hormonesmatter.com is world-famous as the main site for thiamine news and research, powered not only by Dr Chandler Marrs, but by the pioneer of vitamin B1 research, Dr Derrick Lonsdale, MD. The site is also becoming a repository for inspiring case reports. Chandler says: "At Hormones Matter, we give researchers and practitioners a forum for exploring complex health issues that would not otherwise be covered in more traditional journals and we give voice to patients with difficult to diagnose and treat conditions. This provides insight and interaction from health communities around the world.

"We publish three types of articles: research commentary and analysis, hypotheses, and first-person case stories. We are open to any health-related topic, the more complicated the better. Our mission is to build a health knowledge base, one research article, and one case study at a time". In that spirit, **ALEX ZODIAC** describes his recovery from a post-COVID syndrome that started with exercise intolerance and progressed into some kind of neurological disturbance for which doctors had no answers. →

→ After contracting COVID-19 in August 2020, I developed a post-COVID syndrome with gradually increasing neurological manifestations. It took several months to turn into something that could be called a serious neurological condition without an evident cause.

I had a relatively mild form of COVID-19. I never had any chronic diseases that I was aware of, and I was a happy and energetic man in my late 30s. However, COVID changed that abruptly. Shortly after I was discharged from the hospital, I began to experience shortness of breath. It wasn't happening all the time, but was noticeable during physical loads and periodically at night when I was trying to get some sleep. During these episodes, I measured my oxygen saturation and it always was 99%. So, no reason to worry, right?

Sore throat

After the shortness of breath, I developed a form of a sore throat that was aggravated during the episodes of shortness of breath. I did not draw a link between the two at first, but consequent observations proved they were linked somehow. I was also fatigued, and the fatigue lingered. I had an intellectually demanding job but could not work full-time anymore. Five hours out of eight were somewhat achievable, but anything longer

than that was problematic. I developed mild hypothermia as well. Instead of a normal temperature of 97.9 °F (36.6 °C) I always had 95.9 °F (35.5 °C). Finally, I also experienced intermittent episodes of slight tinnitus in the left ear.

I truly hoped these symptoms would be improved over time, but that did not happen. Moreover, in December 2020, four months after recovering from an initial infection, I began to experience what I can only describe as short and intermittent "halts" of consciousness. These episodes happened during work, especially when I was concentrating on a challenging task. The "halts" were like one-two seconds of fear that I was losing control over my body and was going to die. They happened every few minutes. If I relaxed a bit and moved away from the challenging task, the "halt" symptom weakened and disappeared.

In January 2021, I started to feel awkward. It was an unexplainable doom and gloom feeling of imminent death in the near future that was haunting me everywhere. I began to feel burning sensations in my right foot and right hand and my sore throat was becoming worse.

New onset panic attacks after walking

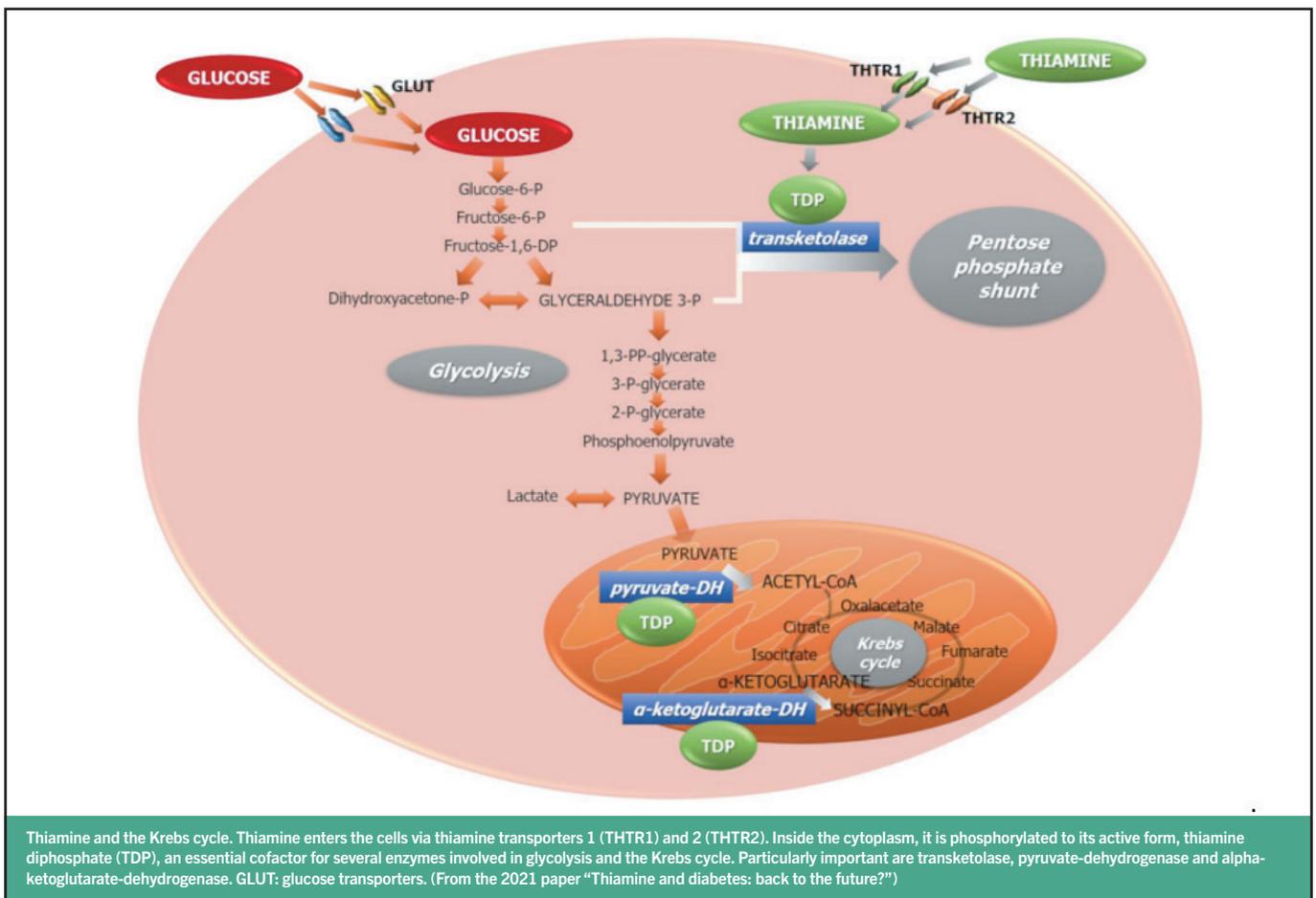
Meanwhile, I remained a big fan of walking, and walked up to eight miles (13 km) a day. After

one such walk in February 2021, I experienced the first panic attack in my life. My heart rate went crazy, and the shortness of breath reached its new maximum by turning into suffocation. I was thinking I was going to die and called the emergency.

I took every blood test I could imagine and visited every doctor I could reach. I went to a cardiologist, neurologist, endocrinologist and phlebologist (a venous circulation specialist). Every test was negative. I was declared healthy except for a few seemingly unrelated things. I had a mild form of Type 2 diabetes, with an HbA1c of 6.7 and insulin resistance with HOMA-IR of 7.8. My low-density lipoprotein levels were slightly elevated.

An interesting observation is that I had taken blood tests right before the acute phase of COVID, and I had no Type 2 back then. During a talk with an experienced endocrinologist, I was told that those relatively moderate levels of glucose in my blood could not wreak such havoc alone. It might be something else, but nobody knew what it was. A neurologist concluded that it was just severe depression and suggested taking some antidepressants, which I did without any positive results.

Meanwhile, my panic attacks intensified. The sore throat gradually transformed into dysphagia. The burning sensation in my right foot and hand took over the whole right side of



Thiamine and the Krebs cycle. Thiamine enters the cells via thiamine transporters 1 (THTR1) and 2 (THTR2). Inside the cytoplasm, it is phosphorylated to its active form, thiamine diphosphate (TDP), an essential cofactor for several enzymes involved in glycolysis and the Krebs cycle. Particularly important are transketolase, pyruvate-dehydrogenase and alpha-ketoglutarate-dehydrogenase. GLUT: glucose transporters. (From the 2021 paper "Thiamine and diabetes: back to the future?")

my body. I started to lose the ability to speak fluently, and I started to have problems with my gait and street navigation.

At the height of a panic attack, I felt that my intrinsic biological processes were stopping and that one day it may have an ultimate ending. I started to develop a condition called intestinal ataxia. My right hand now had neurological oedema. My peripheral neuropathy worsened significantly and became seriously painful. Everything went downhill very quickly, in a matter of weeks and days. At the beginning of April 2021, I faced an immediate risk of becoming an invalid and thought I might die.

The bitter part of the story is that nobody was going to help me. All the doctors I had met were helpless.

Improvement after specific foods

I started to observe strange things while I was suffering from the condition. First, my symptoms slightly improved after consuming specific foods. For instance, it happened every time I ate a burger with real beef. The positive effect persisted for two or three days and then vanished. The same improvement I've experienced from consuming a glass of milk, but the effect was way shorter lasting just one day.

Second, the condition was greatly improved on days when I consumed NSAIDs such as aspirin or ibuprofen for an unrelated reason.

Third, I felt almost healthy on the rare days when I ate those specific foods and consumed NSAIDs together by pure coincidence.

Unfortunately, I did not draw any specific conclusions back then. Such correlations just looked a bit weird to me, as I never experienced anything like that in my whole prior life.

Discovering poor brain glucose metabolism

While the condition was gradually worsening, I measured my blood glucose levels several times a day. One day I decided to conduct an experiment to find out how blood glucose levels are affected by walking. I started by measuring my base glucose level in the morning, which then was 126mg/dL (7mmol/L). Then, without eating anything, I decided to take an eight-mile (13 km) walk. To my amusement, the glucose level fell to 81mg/dL (4.5mmol/L) at the end of the route. Everything worked as expected; it seemed that I was not that insulin resistant after all.

But what about mental and intellectual activities? It would be a nice experiment to conduct, too. The next morning, without eating anything, I went to my job with a glucometer and measured the base glucose level, which was 117mg/dL (6.5mmol/L) at the time. Then I started to work, taking the most challenging



“Combining all these pieces and using the protocol for treating beriberi as the basis, I came up with an experimental therapy, which I first tested on myself. It consisted of B1, B3, B7, B2, multivitamin, magnesium, potassium, CoQ10, alpha-lipoic acid, resveratrol, l-carnitine, zinc, copper and aspirin in certain forms and proportions. Going beyond medicals, it also included dietary corrections (ketogenic non-vegetarian diet, no tea, no coffee and no alcohol) and mild physical activities (walking). To my surprise and amusement, it gave the desired results”.

tasks. After three hours of intensive intellectual work, I started to experience the aforementioned “halts” of consciousness. Time to measure the glucose level. It was 115mg/dL (6.4mmol/L). Wait, what? How is that possible after all these intellectual activities?

Yes, there is a process called gluconeogenesis that could raise the glucose level, but still, I did not expect such a high value after such a massive cognitive load on an empty stomach. Clearly, something was going wrong with my brain, as it had significant problems with glucose utilisation. This was the crucial moment. A simple scientific experiment allowed me to see the light at the end of the tunnel. The cells of my nervous system were unable to consume the usual levels of glucose as they were insulin resistant, and I just proved that with my measurements.

Was I deficient in thiamine?

A quick web search of such a metabolic condition suggested thiamine (vitamin B1) deficiency and beriberi as possible culprits. I knew about beriberi, but it never bugged my

mind to link it with my own condition. I always thought it could be only caused by extreme malnutrition, which was not the case with me.

Going deeper on that route, I found the work of Hans Krebs, who described the process of cellular respiration and received the Nobel Prize for it in 1953. I was amused. Not only did it explain everything I had experienced, but now I had some actionable plan to try and improve my health.

If the usual levels of glucose cannot be consumed by the cells due to their insulin resistance, does it mean that by artificially raising glucose concentrations in the blood we can stop a metabolic panic attack? I conducted the experiment on several panic attacks of mine and received a positive answer. Yes, a metabolic panic attack can be stopped or at least significantly decreased by consuming 15g of sugar. I immediately started to practise that to save my cells from further damage whenever a panic attack was mounting. That knowledge improved my condition a bit and gave me some time to find the appropriate therapeutic dose of thiamine.

→ Improving insulin resistance

I saw a paper in the *European Journal of Nutrition* titled “High-dose thiamine supplementation improves glucose tolerance in hyperglycaemic individuals: a randomised, double-blind cross-over trial” (2). Would thiamine help to reverse insulin resistance? I went to the pharmacy and bought benfotiamine [a synthetic, fat-soluble, highly-absorbable form of thiamine]. It was hard to find, but luckily it was provisioned in two pharmacies in my hometown, so I did not have to wait for too long. Being in serious neurological suffering and pain, I immediately consumed 150mg of benfotiamine. Instinctively, I expected some kind of reaction, so I consumed the dose gradually. No reactions developed, but I immediately felt better in just 15 minutes after taking the pill. After that experience, I understood that I will be able to survive.

Given this experience and research, I

wondered if the cellular damage would be reversed completely. It was an open question. Benfotiamine made me feel better, but I still experienced polyneuropathy and shortness of breath. The panic attacks went away completely, however.

Are vitamins the answer to post-COVID symptoms?

The new knowledge explained a lot of strange things that were happening with my body. Remember I talked about strange unexplained positive effects of consuming meat and milk? Now it became clear why: those foods contain nutrients that include a rich set of vitamins and minerals. Vitamins help the mitochondria to process the substrates (glucose and lipids) to produce the energy (ATP) that powers up the cells.

It also explained the strange positive effects of consuming NSAIDs. As it turned out, this

is relatively well known, too. The mitochondrial dysfunction causes an auto-immune reaction of the body to its own metabolically subpar cells (3). This inflammation does more damage than good in that situation: the vessels’ endothelium gets damaged, leading to various blood flow problems including micro-clotting (4). This further aggravates the tissue hypoxia and makes the mitochondrial dysfunction even more severe in the affected areas, leading to even more inflammation. This is a self-fuelling pathological process with a positive feedback loop.

Reversing mitochondrial dysfunction

An acquired mitochondrial dysfunction can be reversed to some degree by using supplementary vitamins, co-factors, minerals and antioxidants. Thiamine takes one of the instrumental roles in this process as it catalyses the reactions at the very start of the oxidative phosphorylation pathway, but thiamine alone may not do much unless a full spectrum of nutrients is supplied. Glucose is not the only fuel a mitochondrion can consume, and other pathways need attention as well.

Combining all these pieces and using the protocol for treating beriberi as the basis, I came up with an experimental therapy, which I first tested on myself. It consisted of B1, B3, B7, B2, multivitamin, magnesium, potassium, CoQ10, alpha-lipoic acid, resveratrol, L-carnitine, zinc, copper and aspirin in certain forms and proportions. Going beyond medicals, it also included dietary corrections (ketogenic non-vegetarian diet, no tea, no coffee and no alcohol) and mild physical activities (walking).

To my surprise and amusement, it gave the desired results.

I was able to get rid of the panic attacks, hypertension, tachycardia, neurological oedema, tinnitus, insulin resistance, dysphagia, cognitive impairment, fatigue and neuropathy. It took some time and effort. One part of that was my own body that needed the time to adapt and heal, another part was numerous therapy refinements.

At the beginning of the therapy, I took frequent blood tests to ensure the right therapeutic direction. All my outstanding markers were gradually normalising, proving that I was on the right track.

At the time, I was not aware of Drs. Derrick Lonsdale and Chandler Marrs’ work (their book *Thiamine Deficiency Disease, Dysautonomia, and High Calorie Malnutrition*) or Elliot Overton’s videos (<https://www.youtube.com/c/EONutrition>).

You can imagine the level of my sheer astonishment when I compared my humble findings to theirs.

Now, almost two years after the disease’s inception, I can call myself a healthy man, again.

IHCAN

- References online at www.ihcan-mag.com/references.
- Originally published at <https://www.hormonesmatter.com/recovering-from-post-covid-mitochondrial-dysfunction>. Used with permission.

Benfotiamine studied in Alzheimer’s

Fuelled by a \$45 million grant from the US National Institutes of Health and National Institute on Ageing, scientists led by the University of California San Diego are in the middle of a nationwide clinical trial to investigate the therapeutic potential of benfotiamine as a treatment for Alzheimer’s.

The trial addresses tissue deficiency of thiamine-regulated metabolic pathways linked to Alzheimer’s. Previous work by co-principal investigator Gary Gibson, PhD, professor of neuroscience at Brain and Mind Research Institute, has found that a reduction in glucose metabolism is linked to this deficiency in thiamine-dependent processes.

Using multiple experimental models, Gibson and others have shown that increasing thiamine to very high levels using benfotiamine supplementation appeared to be protective against Alzheimer’s-like symptoms. They set out to they enrol approximately 400 patients at up

to 50 US-based clinical trial sites, beginning in early 2023.

The study was due to monitor participants over 18 months, using several measures, including cognitive tests and blood markers.

“At the Burke Neurological Institute, we have been studying the effects of thiamine on neurodegenerative diseases for more than 40 years”, said Gibson. “This important grant will allow us to test the treatment with hundreds of Alzheimer’s disease patients across the US”.

High-dose thiamine and biotin treatments restore normal brain processes

A research team led by the University of California, Irvine, has linked the mutation that causes Huntington’s disease to developmental deficits in the brain’s oligodendrocyte cells that are caused by changes in metabolism. They found that high doses of thiamine and biotin

can restore normal processes.

OL cells generate the insulating coating around neurons, called myelin. The study, published in *Nature Communications*, provides detailed insight into the entire process of how these changes in the genes that regulate cell metabolism impair development of OLs, as well as the therapeutic value of treating HD with high doses of thiamine and biotin. Both B vitamins are involved in a wide range of metabolic processes that help keep the nervous system healthy.



Studying thiamine for 40 years: Prof Gary Gibson (right), director of the Laboratory for Mitochondrial Biology and Metabolic Dysfunction in Neurodegeneration, and professor at Weill Cornell Medicine.



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