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Introduction to Cell Symbiosis Therapy

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Editorial

Is it time for Naturopaths to join the Mitochondrial Revolution?

By David Potterton

WHAT DO we really know about the mitochondria? Well, most of us, not being cell biologists, probably know them as powerhouses of the cell. They are tiny bodies, generally termed organelles, that have the ability to convert oxygen and various nutrients into ATP (adenosine triphosphate), which is used in the body to store and release energy as required.

Strange intracellular structures were visualised as long ago as the 1840s. Then in 1894 Richard Altmann identified them as organelles, which he called 'bioblasts'.

The term mitochondria was coined by Carl Benda in 1898, while today's popular "powerhouse of the cell" description was introduced by Philip Siekevitz in 1957.

Today the mitochondria are demanding even greater scientific attention with the realisation that they are descendants of a once separate life form – a bacterium with its own genome that somehow became absorbed into what was to become the human cell, which also has a nuclear genome.

Vital functions

According to the *New Scientist* (see "The Micromanagers", by Garry Hamilton, September 20, 2014), the latest thinking seems to be that the mitochondria are not just powerhouses, but influence vital bodily functions from memory and ageing to combatting stress and disease.

As Gilian Crowther explains in this *BNJ Supplement,* Cell Symbiosis Therapy (CST) sees the mitochondria as the ultimate orchestrators of our cellular health.

The mitochondria play a crucial role in apoptosis, and thus mitochondrial dysfunction has been linked with an increased risk of cancer. Mitochondria require oxygen to produce ATP, whereas cancer cells thrive in a low oxygen or anaerobic environment. Instead of utilising oxygen, cancer cells derive their energy from the breakdown of glucose.

It was Nobel prize winner Dr Otto Warburg who first suggested that cancer is associated with a lack of oxygen at the cellular level.

Coenzyme CoQ10, one of the nutrients involved in mitochondrial production of ATP, has also been linked with improving cognitive function.

Mitochondrial dysfunction is generally associated with intense fatigue: however, nutritional therapy using nutrients that improve mitochondrial activity have been shown to improve patients' energy and quality of life.

Many conditions presented by patients seen by Naturopaths, including obesity, diabetes and atherosclerosis, have mitochondrial connections, and thus perhaps education in this field should be seen as a desirable activity.

Original paper

Mitochondria still possess many features of their bacterial ancestors, including a double membrane, a circular genome and the ability to replicate independently of the cell nucleus.[4] The existence of the mitochondria's separate genome is considered to be a large factor in our health and disease.

Introduction to Cell Symbiosis Therapy

Mitochondria as the ultimate drivers of health and disease

By Gilian Crowther
MA (Oxon) ND/NT

Abstract
Cell Symbiosis Therapy (CST) sees the mitochondria as the ultimate orchestrators of our cellular health. It is based on a unique understanding of the evolutionary origin of our cells, including their hybrid nature stemming from a dual genome. The therapy is targeted at restoring full mitochondrial function, and prides itself in always striving to be both verifiable and replicable.

THE ORIGIN of the term *Cell Symbiosis* lies far back in history – 2.1 billion years to be exact – when the first Ice Age occurred. Before this, the Earth's atmosphere was devoid of molecular oxygen, but dominated by carbon dioxide (CO_2) and methane gas (CH_4). The CO_2 was a result of volcanic activities in the Earth's crust, while the CH_4 was produced by the ubiquitous archaea – a largely anaerobic life form – which converted CO_2 into CH_4[1].

When the global ice sheet melted, the O_2 concentration in the atmosphere began to rise exponentially, most likely as a result of cyanobacteria metabolising CO_2 into oxygen via photosynthesis. This is clearly evidenced in oxide bands in iron sediments from that time[2].

The existence of the archaea was now threatened. Evolutionary biology has established that the most likely explanation for the development of eukaryotes – cells with a nucleus and organelles – was that the archaea engulfed α-proteobacteria, which already at that time had a functioning electron transport chain, so could metabolise oxygen[3]. These bacteria enabled the archaea to survive in areas where oxygen levels were high, and were the predecessors of our mitochondria.

Mitochondria still possess many morphological and biochemical features of their bacterial ancestors, including a double membrane, a circular genome, the absence of histones, and the ability to replicate independently of the nucleus[4]. Our cell's nucleus with its DNA stems from the archaea, while the mitochondria have their own separate genome. This dual genome is a large factor in our health and disease.

Evolutionary advantage

To understand why, we first need to look at what the evolutionary advantage was for these bacteria of living within an anaerobic host? Certainly one can imagine that this would at that time have ensured their survival in areas where oxygen levels were still low, but there is another fascinating reason.

The generation of ATP (adenosine triphosphate) using oxygen produces large numbers of free radicals. These would damage the DNA of the cell nucleus when it divides, as it is not covered by a protective envelope during replication. Cyclin D1 – one of a family of proteins that are active at different times in the cell cycle – appears to inhibit the mitochondria during the S phase when DNA is replicated[5].

At this point the archaea's anaerobic energy generation – glycolysis – is used instead as no oxygen is required, and therefore no oxygen radicals are produced.

This switching from one form of energy generation to the other is an ancient programme that our cells still carry within them: the "a genome" (archaeal genome) is responsible for replication and repair, but our cells are intended to switch back to high-intensity energy production (the "b genome" – b

for bacterial, i.e. mitochondrial) the moment cell replication is over[6].

Before thinking about how this switching process can go wrong – which lies at the heart of mitochondrial dysfunction – we should look at the two very different forms of energy generation, because this is also key: *intramitochondrial*, and *cytosolic*.

When ATP is produced in the intracellular fluid (cytosol) via glycolysis, one mole of glucose generates a net yield of 2 ATP, whereas inside the mitochondria, the net yield via the electron transport chain (ETC) is 34 ATP[7] from one mole of glucose (under optimal conditions), but much higher from fats, e.g. one mole of oleic acid generates about 146 ATP.

But it's not just 2 versus 34 (or 146). We have at least 1,500 mitochondria in each cell on average (except in erythrocytes which have none), and thousands of electron transport chains within each mitochondrion[8]. So the energy generation our cells are capable of, if oxidative phosphorylation (energy production along the ETC) is fully functional, is infinitely more efficient than cytosolic energy generation.

This is why the symbiosis of these two organisms – archaea and proteobacteria – was so fundamental to evolution: the huge energy production possible along the ETC enabled the development of multicellular life.

However, if constant cellular repair processes are necessary, the archaeal genome is upregulated, as the genes exposed during cellular repair and replication would be damaged by the oxygen radicals being fired off continually along the ETC.

Mitochondrial dysfunction involves much more than just an energy deficit: cells also lose their ability to orchestrate our cellular metabolic processes.

An additional reason is that glycolysis produces building blocks for the synthesis of new macromolecules essential to cellular repair processes (such as nucleic acids, lipids and proteins)[9].

Chronic inflammation mirrors exactly this scenario: cells are constantly being sloughed off and need replacement. Thus, it is part of our natural programming for the "a genome" to be upregulated and the "b genome" to be downregulated during chronic illness that involves any kind of cellular replacement and repair.

Light sensor molecules

When the mitochondria shut down there is clearly going to be a major energy production problem. But it is not just the energy perspective that is so critical in mitochondrial dysfunction.

Electron transfer along the ETC takes place at highly accelerated rates, and when an electron accelerates it emits photons characterised by wavelength and frequency: a genuine electromagnetic field (EMF).

Gilian Crowther MA (Oxon), mBANT, mNNA, CNHC

Gilian Crowther is a naturopath and nutritional therapist who studied complementary therapy in Germany for many years before completing her training in the UK. She encountered Cell Symbiosis Therapy (CST) while there, and found it to be a unique and highly effective approach that traces the origins of much chronic disease to mitochondrial dysfunction. Gilian sat the exam as a certified CST practitioner and now practices CST within a large network of practitioners, with a clinic in London. She also holds seminars for the CST Academy and translates literature on the subject, including books.

Dr Heinrich Kremer, the doctor and researcher who has spent more than two decades analysing the science behind CST, suggests that these photons enable a unique form of information transfer.

He postulates that ATP stores, bundles and emits targeted photon-modulated information in the EMF arising from the freely-moving electrons in the alternating double bonds of the adenine molecule[10]. This would mean that mitochondrial ATP has an additional function – perhaps its prime function – transmitting information.

Interestingly, even NASA is now looking into the potential of the light-sensor pathway involved in mitochondrial ATP production: "We have developed an experimental system for demonstrating transfer of information and energy from the ambient electromagnetic field to the universal electron transport chain in living cells"[11].

This would mean, then – as Professor Otto Warburg, who won a Nobel Prize himself for his work on the mitochondria, said long ago in the 1930s – that mitochondrial ATP is indeed different in structure and function to cytosolic ("archaeal") ATP. And this is a central precept of Cell Symbiotic Therapy: that mitochondrial dysfunction involves much more than just an energy deficit: cells also lose their ability to orchestrate our cellular metabolic processes.

What can cause mitochondria to shut down or cease their normal function? They may lack some of the enzymes or co-factors required for the different complexes in the electron transport chain. They may lack sufficient antioxidants – especially glutathione – to quench either endogenous or exogenous free radicals. Heavy metals or other xenobiotics may be adducted to the cellular or mitochondrial membrane. Defects in detoxification may prevent toxins from being

Nutritional therapy is a vital first pillar, supported by an extremely sensitive IgG 1-4 food antigen test that has proved effective in quelling hyperimmunity in the gut. This can sidetrack so much ATP (immune reactions all require ATP to fuel them), as well as be a severe barrier to uptake of nutrients.

eliminated, causing blockages in normal cellular function. And as mentioned at the outset, chronic inflammation of any kind – due to the cellular turnover and therefore repair that it involves – leads to a switch to the a-genome, shutting down the mitochondria[12].

The mitochondria are also responsible for releasing cytochrome c whenever cells need to apoptose. This will not happen if this switch has occurred and the a-genome is running on automatic: cell proliferation (at some point later in the process) is a possible consequence due to the inability of cells to undergo apoptosis – obviously a highly deleterious aspect of this shift, quite apart from all the other issues related to mitochondrial dysfunction.

The mitochondria have such myriad functions. Apart from the energy they produce and the cell-cell communication just touched on, they are intimately interlinked to the T-helper 1 and T-helper 2 cells (Th1/Th2 arms) of our immune system, and the nitric oxide that is our primary intracellular defence against pathogens[13].

Mitochondria appear to have their own mitochondrial nitric oxide synthase that acts as a metabolic regulator, but if our antioxidant systems are overwhelmed, nitric oxide gas (NO) is downregulated within the cells, and the Th2 arm of the immune system is upregulated to compensate. This is a pattern that often accompanies mitochondrial dysfunction, and means that intracellular pathogens cannot be fought off successfully.

The first step of haem synthesis also takes place in the mitochondria using aminolevulinic acid synthase, so if the mitochondria are down, haem cannot be properly synthesised[14]. This does not just affect our haemoglobin production. Haem is rate-limiting in numerous proteins in the body: myoglobin, neuroglobin, cytochrome P450, the cytochromes of the electron transport chain, and multiple others[15]. Our mitochondria are also key to cellular detoxification. Beta oxidation takes place in the mitochondria, so fatty acids will not be broken down efficiently; the first step of the urea cycle is in the mitochondria, so the excretion of protein will be disrupted[16]. And monoamine oxidase is located on the outer mitochondrial membrane, which catalyses the breakdown of the monoamines, whether dopamine, serotonin, noradrenaline or adrenaline, so these neurotransmitters will be dysregulated[17].

Even excess calorie intake can throttle oxidative phosphorylation, as a high proton-motive force (which high calorie intake produces), leads – if unabated – to the generation of more reactive oxygen species than we have antioxidants to quench. The mitochondria downregulate as a result, to protect the cell from destruction[18].

Mitochondrial markers

CST has a number of tests that it uses as a marker for mitochondrial dysfunction. The LDH-isoenzymes are one. Lactate dehydrogenase is a cytoplasmic enzyme in all tissues, and catalyses the oxidation of lactate to pyruvate.

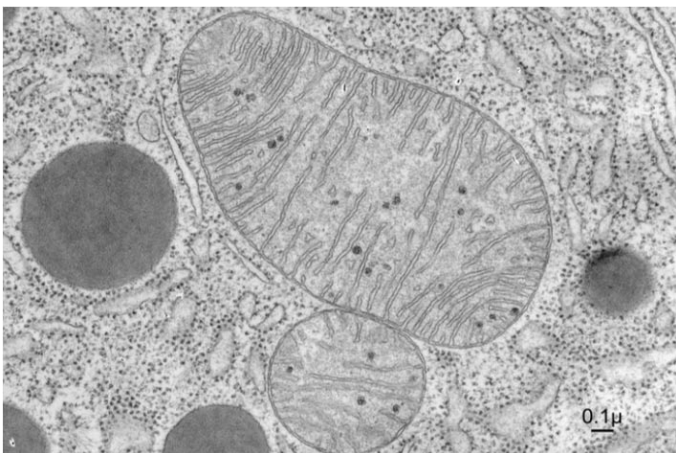
Isoenzymes 4 and 5 tend to be raised if ATP production in the cytosol is upregulated. M2PK is also used as a blood test. Pyruvate kinase isoenzyme type M2 is a systemic blood marker for the switch to glycolysis, and is a reliable marker for monitoring mitochondrial recovery. Multiple other tests are used depending on the patient's presentation, many of them from a specific mitochondrial perspective that helps to tailor therapy specifically to mitochondrial support.

CST therapy

CST therapy is aimed above all at remedying defects in mitochondrial function as swiftly as possible, and encouraging mitobiogenesis (the production of new mitochondria). If one accepts as posited that our mitochondria are programmed to work with light-sensor molecules, the most natural and efficient way of doing this is to feed them appropriate polyphenols, flavonoids and isoprenes. Therapy includes an entire spectrum of approaches, ranging from nutritional support, chelating out toxins suppressing the patient's normal mitochondrial function, to supplementation with macro- and micronutrients.

Nutritional therapy is a vital first pillar, supported by an extremely sensitive IgG 1 - 4 food antigen test that has proved effective in quelling hyperimmunity in the gut. This can sidetrack so much ATP (immune reactions all require ATP to fuel them), as well as be a severe barrier to uptake of

Our mitochondria: originally bacteria



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nutrients. It is said that 90 per cent of our cells are bacteria,[19] so dysbiosis can have huge systemic reactions, including on neurotransmitters and the brain (gut/brain axis).

The mitochondrial remedies used in the core CST "Mitochondrial Triad" consist of substrates and co-factors for the electron transport chain. One is specifically aimed at helping to repair the electron transport chain at Complex 4, cytochrome c. Another primarily targets the generation of new mitochondria, working on the sirtuin pathway using resveratrol from Japanese knotweed[20] and another 11 of the strongest and most effective polyphenols, including quercetin, wheatgrass, Rhodiola rosea and amla berry extract.

To speed recovery, CST therapists also suggest the use of IV therapy with specific amino acids, vitamins and trace elements tailored to the patient's needs, to support existing mitochondria and activate mitobiogenesis.

Some 4,000 therapists are now working with CST (www.cst-academy.co.uk) across Europe, and finding it a very successful therapeutic approach, particularly in chronic disease. The CST Academy has now also started training in the UK: courses are held regularly by the Academy of Nutritional Medicine (AONM), which has joined forces with the Academy in Germany. For more information on CST please contact the author at gilian_crowther@btinternet.com

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