BBME

Support Group

"Breaking the Isolation"

Welcome to Our September 2012 Newsletter

A Note from Caroline: Unfortunately, our regular newsletter editor is not well at the moment, so I've stepped in as interim editor for our September issue! Hopefully, Alex, who always does a great job keeping us up to date with the latest news, will be back at the helm next month.

Don't forget, you can keep in touch with other members on our private Facebook page: https://www.facebook.com/groups/buryboltonmecfs/ We have 41 members now and lately, we've been discussing alternative therapies, benefit appeals, the Olympics, the recent documentaries about benefits, and Bolton Council's "Handyperson" service.

Try concentrating on something you'd really like to do and I'm sure you'll feel much better!

With thanks to Invest in ME (www.investinme.org) for their kind permission to reprint this cartoon from the calendar available to download from their website.

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DISCLAIMER: Anything expressed within this newsletter may not necessarily represent the views of the editor, the Committee, nor the Bury/Bolton ME/CFS Support Group. Any products, treatments, or therapies featured are for information only and their inclusion should not be considered an endorsement.

Bury/Bolton ME/CFS Support Group & Sponsors

www.mesupportgroup.co.uk

The Bury/Bolton ME/CFS Support Group was founded in September 1990 and is managed by a committee of six members: Pam Turner, Alex Wootton, Carole Senior, Maria Sale, Lynda Marney & Phil Seddon

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Dates For Your Diary

Longsight Meetings: Our main meetings, often with guest speakers, are held at Longsight Methodist Church, Longsight Lane. Harwood, Bolton, BL2 3HX, on the third Thursday of each month from 7.30pm until 9pm (except in April, August and December). Entrance is £1, tea, coffee, water, biscuits, etc provided.

Thursday 20th September – Social evening with Alex selling her cards, (Alex has been busy making new cards for Christmas as she always has to be a few months ahead, other cards such as Birthday, Thank You and blank cards will also be available). All of the profit goes to our group, so an easy way to get organised in time for Christmas and support us at the same time!

Thursday 18th October – Bridget Fox from BEST (Bury Employment Support and Training). It is an agency run by Bury council which can help people with disabilities to stay in work, but will also support and advise people not in work. Some of our members have used them for general advice and found them very helpful.

Thursday 15th November – Pre-Xmas Bring and Share Supper. Always popular, make sure you arrive hungry!

Radcliffe Socials: We meet informally on the first Monday of each month, our next will be on **Monday 1st October** at **the new time of 2pm,** at **The Sparking Clog, Radcliffe Moor Road, Radcliffe, M26 3WY**. It has plenty of parking, good accessibility, comfortable seating and is relatively quiet. They serve very tempting chips that we just can't resist! For anyone who does fancy a snack, these start at just £1.50, with main courses from £3.50. We usually meet at the oval table next to the bar.

Yoga Classes: Are **3:00pm-4:15pm on Tuesdays** at the **Jubilee Centre, Darley Street (off Eskrick St), Bolton, BL1 3DX**. Designed to cater for the average ME sufferer, classes are free and yoga mats are provided. Please wear loose, comfortable clothing. Contact Olivia on 07746 197511, or olivia@oliviayoga.co.uk for more information.

Neuro Support Groups: These groups, run by Greater Manchester Neurological Alliance, provide information, advice and support for people with any type of neurological condition and/or their carers. Call 0161 743 3701 or visit www.gmneuro.org.uk for information about meeting times and locations.

If you are thinking of attending any of our socials, whether you are a new member or a member who hasn't been able to attend for a while, please remember that you can bring along your carer or a friend. We don't bite, but we understand that meeting new people or if you have been house bound for a while, it can be quite daunting going out by yourself and we look forward to seeing you.

PLEASE DO NOT WEAR STRONGLY SCENTED TOILETRIES WHEN YOU ATTEND OUR MEETINGS, AS SOME MEMBERS ARE VERY SENSITIVE TO THESE PRODUCTS, THANK-YOU.

Don't forget that when you shop on **www.amazon.co.uk**, go to our website www.mesupportgroup.co.uk first, and follow the Amazon picture links. We will then get 5% of the value your purchase. This is a very easy way to support the group and get us much needed funds.

Understanding Cellular Energy Production

By Sarah Wragg:

Article first published in the Nutrition I-Mag www.nutritionimag.com, reprinted with kind permission.

Ongoing fatigue can severely affect the quality of life for many individuals. Sarah Wragg explores the underlying metabolic defects associated with energy production:

Fatigue can come about as a side effect of a high-octane lifestyle and is also a common symptom associated with many 21st century health conditions such as multiple sclerosis (MS), Myalgic Encephalomyelitis (ME), Chronic Fatigue Syndrome (CFS), Fibromyalgia, bowel conditions, adrenal insufficiency and Type 2 diabetes, not to mention various types of infections and cancer.
Sales of energy drinks loaded with sugar and caffeine are exploding in popularity as an increasing number of people turn to short-term solutions to help boost their vitality. Fortunately, nutritional therapy can offer a healthier, long-term approach to improve energy levels and overall health.

Energy:

The body needs a constant supply of energy to build new molecules, enable movement and transport substances whilst creating new cells. A whole series of metabolic reactions are required to 'capture' the energy from our food and trap it in a form that the body can use. Cellular structures called mitochondria are the most important source of cellular energy and it's estimated that mitochondria make up 10% of body weight, approximately 10 million billion per adult.
Understanding how mitochondria generate cellular energy can also help us determine what can go wrong, which is crucial for supporting clients with any form of chronic tiredness. The mitochondria generate energy by a process of oxidative phosphorylation involving molecules of ATP (adenosine triphosphate), which is hydrolysed to ADP (adenosine diphosphate), releasing energy. ADP is quickly phosphorylated back into ATP and so the cycle continues. This cycle continues approximately every 10 seconds in a normal healthy body and is capable of producing more than 90% of cellular energy.
However, if the mitochondria are not efficient at recycling ATP, then the cell quickly runs out of energy, causing symptoms of poor stamina. The cell literally has to hibernate, waiting until more ATP has been manufactured. If the cell is pushed (i.e. stressed) when there is no ATP, then it will start to use ADP instead, converting the ADP to AMP (adenosine monophosphate). The problem here is that AMP is lost in the urine which means that ADP is unable to be recycled and must therefore be rebuilt from raw ingredients such as D-ribose, a process which takes days, resulting in 'delayed' fatigue.)

Dysfunction:

There is considerable evidence that mitochondrial dysfunction is present in individuals suffering from CFS. In essence, the pathology involved with CFS is that of slow recycling of ATP to ADP and back to ATP again. However, there is another problem. During times when the cells are extremely short of ATP very small amounts of ATP can be made from glucose via conversion into lactic acid. This is very common with CFS sufferers and leads them to switch into anaerobic metabolism. However the resulting lactic acid build-up which particularly occurs in the muscles causes a sensation of heaviness and aching. When mitochondria function well, lactic acid is quickly converted back to glucose through ATP and the lactic burn disappears. But if there is no ATP available – for example, during times of mitochondrial dysfunction, then the lactic acid persists, causing great pain and tiredness. Dr Myhill has demonstrated the power of the ATP Profile Test in confirming and pinpointing biochemical dysfunctions in people with CFS.

Insulin link:
Mitochondrial dysfunction has also been linked to insulin resistance (IR). According to research, individuals with IR have unusual fatty acid-stimulated changes in mitochondrial uncoupling proteins (UCPs). This results in an increased production of reactive oxygen species (ROS) and ultimately fatigue. But why does this happen? When mitochondrial respiration functions properly, the amount of ROS produced as a consequence of electron transport activity is effectively neutralised by antioxidants. Individuals with IR have been found to have approximately half the normal levels of UCP3 in their skeletal muscles. UCP3 prevents the build-up of excessive concentrations of ROS and removes fatty acids (formed by oxidative reactions) that can build up in the mitochondria. Fatty acids are particularly sensitive to ROS oxidation and result in damage to mitochondrial components if not controlled. The link between IR and serious fatigue may indicate that mitochondria dysfunction is also present.

Damage:
Damage to cellular mitochondria can impair the ability of cells to produce high-energy molecules, such as ATP. ROS damage generally accumulates because antioxidants cannot restore or replace ROS-damaged molecules fast enough. This can happen through ageing or because of an excess ROS production by the mitochondria, or lack of antioxidants. At the molecular level, damage to DNA, phospholipids and other lipids within the mitochondrial membranes caused by ROS can affect mitochondrial functioning and energy production. Ultimately fatigue follows.

There are ways to prevent this damage and reduce energy loss, however. Reducing cellular and mitochondrial membrane and DNA damage plus loss of membrane integrity are a starting point. This can be achieved, in part, by neutralising ROS with various antioxidants. However, antioxidants alone may not be the answer as they may not completely eliminate or reverse ROS damage. Some fascinating research conducted by Prof Nicolson and Dr Rita Ellithorpe provides further information. Glycophospholipids help replace damaged mitochondrial membrane phospholipids and other lipids. Glycophospholipids can therefore provide direct impact on production and maintenance of energy in fatigued clients. The clinical application of what is being commercially known as 'Lipid Replacement Therapy' which contain glycophospholipids amongst other lipids, antioxidants, nutrients, probiotics, vitamins, minerals and plant extracts can help strengthen the battle against fatigue.

Nutrients:
Minerals such as iron, copper, zinc and vitamins such as biotin, B6, B12 and B5 support mitochondrial function. If dietary intake of these vitamins and minerals is inadequate, electrons derived from food cannot move efficiently through the energy-producing steps. Instead of contributing to energy production, the electrons help increase production of ROS, increasing the rate of mutations in mitochondrial genes. It is at this point, the crossroads between mitochondrial energy production and ROS production, that diet could influence energy levels.

Re-charge:
1. Have rest days – These allow the mitochondria to recover, this is extremely important if the individual has a high exercise regime.
2. Acetyl-L-Carnitine – acts as a cargo train, transporting the fatty acids into the mitochondria whilst removing any metabolic rubbish (through its antioxidant support in controlling ROS damage). Protein foods provide a rich source of acetyl-L-carnitine.
3. Sports – Consider acetyl-L-carnitine and creatine for athletes or those committed to extreme exercise. The muscles typically have five seconds of ATP in their stores. This means that ATP reserves can be exhausted and depleted very quickly. Supplementing creatine helps to provide an immediate ATP loan, like an ATP overdraft facility, to exercising muscles.
4. Feed the mitochondria – ensure the diet supplies a good supply of the vitamins and minerals associated with mitochondrial needs.

5. Antioxidants – these help to mop up ROS. Include vitamins C, E, zinc, coenzyme Q10, N-acetyl cysteine, alpha-lipoic acid. CoQ10 is an important co-factor for the synthesis of ATP.
6. Glycophospholipids – these membrane lipids are obtained through foods such as meat, egg yolks, fish, turkey, chicken and beef.
7. Support – other nutrients to consider include D-ribose, NAD, magnesium and B12 plus essential fatty acids to further support the mitochondria. These help to restore normal function and support level.

Mitochondria are the most important source of cellular energy and if their function is impaired, available energy is limited. Restoration of mitochondrial membrane integrity and fluidity are essential for optimal functioning. Ultimately we need to address the underlying causes as to why there is mitochondrial damage and put in place a strategy to prevent ongoing damage. Factors that need to be considered include diet and nutrient deficiencies, toxic load, digestion, blood sugar and insulin balance, thyroid function, adrenal activity, presence of allergies and over exercising. Nor should emotional state and stress be forgotten. Together these factors could play a major role in the ageing process and prevent degenerative diseases – not just simply helping to give us energy.

Sarah Wragg gained a BSc (Hon) degree in Nutritional Therapy from the Centre for Nutrition Education & Lifestyle Management CNELM). She is a certified NLP pracitoner with particular interests in digestive complaints, fatigue, weight and children's health. Sarah has clinics in the Richmond/Wimbledon area. info@mattersforhealth.co.uk, telephone: 07702 492302

Dr Sarah Myhill's Latest Research

In June 2012, Dr Sarah Myhill published her second paper on the connection between mitochondrial dysfunction and ME/CFS, along with co-authors Dr. Norman E. Booth and Dr. John McLaren Howard. The full scientific paper can be found here: http://www.ijcem.com/files/IJCEM1204005.pdf.
Below is their press release explaining the paper and its findings. It is quite complicated and scientific in parts, but it's an important study, hopefully the above article by Sarah Wragg helps you understand it!

"In 2009 we published our first paper looking at mitochondrial function in ME/CFS patients. What we found is that those patients with the worst mitochondrial function had the worst levels of fatigue and vice versa. There was a very clear relationship between the two. The importance of this paper was that it gave backing to certain treatment interventions and also that it clearly established ME/CFS as a physical condition with physical causes. The mitochondrial function can be used as an objective assessment of fatigue and of course this has obvious practical implications.

Hitherto any assessment of the level of disability had to be subjective and this created great difficulties for patients in cases where their physicians disbelieved the serious nature of their symptoms. For a detailed explanation of the clinical issues please see http://www.drmyhill.co.uk/wiki/CFS_-_The_Central_Cause:_Mitochondrial_Failure

This second paper further explores the above ideas. In this second paper the size of the patient group is much larger with 138 ME/CFS patients involved. Their mitochondrial function tests were compared with 53 normal healthy controls.

The findings of the first paper were repeated and confirmed, but the analysis of this second paper was carried out slightly differently.
It was found on careful inspection of the biochemistry that there were various sub-groups of ME/CFS patients with their own characteristic biochemical pattern. In particular, one of the five parameters measured, namely translocator protein function IN (TL-in), can be higher as well as lower for patients as compared with controls.

(**Editors note:** This translocator protein transports ADP into the mitochondria and transports ATP out. TL-in is the amount of ADP entering the mitochondria).

This second paper also attempts to explain what is happening at the biochemical level to result in such an abnormality.
To this end, Dr. Booth provides an alternative method of assessing mitochondrial function. He noticed that the percentage inhibition of ATP closely correlates with TL-in factor – this is probably because the biochemistry of these two measured quantities is so closely associated.

So instead of using TL-in to calculate the mitochondrial energy score, he used percentage ATP inhibited – this provided a solution to the problem of translocator protein IN being higher in some patients than in controls, a factor which in itself is abnormal.
Dr. Booth then went on to plot the relationship between mitochondrial energy score and the number of factors within the normal region to achieve an extremely close correlation.

Importantly this test identifies a clean separation between the ME/CFS cases and the healthy controls.
So this first part of the paper very much confirms the work of the first paper published in 2009 which is that those patients with the worst ME/CFS had the worst mitochondrial function and vice versa.

It must be remembered that patients attending a clinic for ME/CFS are usually the most severely fatigued – no mildly ill patients were tested. Within these limitations the ATP profile is an exclusive and sensitive test for ME/CFS. However, we cannot claim that it is specific to ME/CFS because there are many other neurological illnesses and metabolic syndrome also associated with mitochondrial dysfunction.

Dr. Booth went on to analyze sub-groups within the main group.
When mitochondria are stressed, i.e., energy demand exceeds energy delivery, in the short term they can switch into an alternative means of making ATP, of which there are 2 possibilities identified. Dr. Booth called these patients cohort 1 and cohort 2.
• **In cohort 1**, the mitochondria switch into anaerobic metabolism with increased glycolosis (where 1 molecule of glucose is catabolised to 2 molecules of pyruvic acid, generating 2 molecules of ATP) in order to produce ATP.
• **In cohort 2** there was an alternative process to supply additional ATP. This alternative process involves the adenylate kinase reaction in which two molecules of ADP combine to make one of ATP and one of AMP. The problem with this reaction is that for every molecule of ATP generated, so is one of AMP. This is not recycled, but mainly lost in the urine. So there may be short term metabolic benefits here, but in the longer term metabolic disaster ensues as the energy molecules literally leak away. It takes time to replace these leaked molecules of ADP (leaked in the form of a 'lost' AMP molecule) and so this may explain one of the clinical features of ME/CFS, namely delayed fatigue.

A vital feature of ATP studies is that they identify the mechanisms by which mitochondria 'go slow'. Essentially they can 'go slow' for one of three common reasons:

• Either there is substrate deficiency, i.e. lack of essential co-factors for mitochondria to work such as Co-enzyme Q10, magnesium, vitamin B3, or acetyl-L-carnitine,
• Or secondly, because mitochondria are blocked by toxins. Typically the blockage can be of oxidative phosphorylation and/or translocator protein function. Dr. John McLaren Howard has developed several further tests to look at the nature of these blockages. These tests include microrespirometry studies, TL protein function studies, intracellular calcium studies and so on.

• The third possible mechanism for mitochondria malfunctioning has to do with membrane function. The membranes of mitochondria need to be of just the right consistency in order to hold the bundle of enzymes in the correct 3D configuration to allow efficient movement of substrate from one enzyme complex to another. To this end, again Dr. John McLaren Howard has developed cardiolipin studies which look in more detail at mitochondrial membrane structure and function.

Many of the above tests have been available in research laboratories, some John has developed through his own brilliance and initiative. What is so wonderful is how he has given these cutting edge research tests a clinical application.
This is extremely helpful for patients and clinicians because we can see exactly why mitochondria are 'going slow' and thereby correct deficiencies using both nutritional supplements, correct gut function, as well as being able to tailor detoxification regimes to individual patients.

This second paper also goes on to look at cell free DNA in ME/CFS patients.
Cell free DNA is a measure of DNA in the bloodstream that is not bound up within cell membranes. It can only, therefore, come from damaged cells and therefore is a measure of cell damage within the body.
What we found is a strong negative correlation with mitochondrial energy scores, ATP levels and the rate of oxidative phosphorylation. What this means is that those patients with mitochondria that perform extremely poorly have the highest level of cell damage and vice versa. This makes perfect biochemical sense – if mitochondria 'go slow' one can expect there to be the production of free radicals which have the potential to damage tissues.

Therefore addressing these issues of poor antioxidant status is an essential part of the package of treatment for ME/CFS patients.
These abnormal results clearly show that the effect on mitochondria is a systemic effect, not just confined to the neutrophils that are being tested. Very often we see levels of cell free DNA of a similar magnitude to those in patients who are experiencing a serious illness such as cancer, stroke, autoimmunity, or severe viral infection. Again this underpins the fact that ME/CFS is a physical condition with clear indications of marked cell damage.

This puts ME/CFS firmly in the realm of major organic pathology.

IMPLICATIONS FOR THE TREATMENT OF ME/CFS

These bio-medical tests have been extremely helpful in the diagnosis and management of ME/CFS patients. This is because they clearly identify the biochemical lesions that underpin the cause of this illness. Furthermore, identification of these lesions has clear implications for management using the standard methods of nutritional and environmental medicine.

We are currently preparing a third paper which looks at the efficacy of these interventions in patients by measuring mitochondrial function tests before and after such interventions and correlating these with the clinical picture."

For further information, see her book, available online:
http://www.drmyhill.co.uk/drmyhill/images/7/76/Cfs_book_27.pdf.
Alternatively, to get a printed copy of the 110 page book, please send a cheque for £12 (incl. p+p) made payable to *Sarah Myhill Limited* plus a note with your own UK postal address to: Dr Sarah Myhill, Upper Weston, Llangunllo, Knighton, Powys LD7 1SL.

Twitter

For those of you with a smart-phone, or access to www.twitter.com, did you know that many of the big ME/CFS charities are on **Twitter**? Twitter is a free social media service, where people, organisations or charities can send out "Tweets", which are short 140 character-long messages, to anyone who "follows", or subscribes to them. Look out for these charities and organisations usernames to keep up to date on their news: @Actionforme, @JaneCColby (who runs the Tymes Trust for children), @AYMEUK, @Invest_in_ME, @fibroaction, @AYMEUK, @MEAssociation, @ME_CFS_Support, @CitizensAdvice, @CarersTweets, @Directgov, @Boltoncouncil, @BuryCouncil, @TheBoltonNews , @BuryTimes, @MENnewsdesk, @dwppressoffice, @DHgovuk

Late Edition

Here's a brief roundup of some of the recent articles covering CFS/ME during August.

Exercise and behavioural therapies are the most cost-effective and successful ways to treat CFS/ME. The study was conducted on a sample of 640 patients and published in the journal PLoS ONE. The new findings show that, given in addition to medical care, they are also cost effective to provide. Another treatment option, adaptive pacing therapy (APT), which was not judged to be good value. **Chronic fatigue syndrome: Brain training is most cost-effective treatment – BBC – 1 August 2012**

President Obama recently followed up on a pledge he made in 2011 to a woman in Reno, Nevada to investigate funding into chronic fatigue research. "In an unprecedented step, President Obama has asked the National Institutes of Health and the Department of Health and Human Services to elevate Chronic Fatigue Syndrome in priority, assigning his Deputy Chief of Staff to follow their efforts." **The "Obama Promise" Fulfilled: Obama Requests NIH Elevate Priority of Chronic Fatigue Syndrome – Phoenix rising blog – August 17th 2012**

New research has identified the true extent of inequalities faced by adults who require access to specialist CFS/ME services in England. The findings, published in the journal BMJ Open, reveal a 'postcode lottery' whereby patients from more affluent postcode districts are more likely to be referred to specialist services than those from more deprived areas. **Chronically fatigued patients face huge inequalities in accessing specialist services – medicalexpress.com - August 17, 2012**

The QT interval is part of the heart's electrical system, and helps to maintain a normal rhythm. A faster heart rate means the QT interval is shorter than normal. New research published in the Bulletin of IACFS/ME has shown that CFS patients had significantly shorter QT intervals, than non-CFS and control populations. The researchers believe this can be a useful indicator when diagnosing CFS/ME. **Heart rhythm typical of ME/CFS confirmed in large UK study – prohealth.com – August 13 2012**

The US Food and Drug Administration has accepted and pronounced complete Hemispherx Biopharma Inc.s 'response submission' regarding its New Drug Application for Ampligen® as a therapy for Chronic Fatigue Syndrome (ME/CFS). The drug hasn't been fully approved yet, and the company will need to conduct a lot more work before Ampligen can be prescribed by health professionals in the US. **Ampligen New Drug Application Process Clears Another Hurdle – prohealth.com – August 13 2012**

The recent research at Manchester University which Pam and Yvonne have been involved in, regarding GPs being able to accurately diagnose ME/CFS, will soon be published in the esteemed journal "Family Practice". This is a huge accomplishment and well done to all involved!