Our newly funded projects
editorial

When people ask what we do, we answer that ME Research UK exists to fund biomedical research projects – as simple as that. As the main independent funder of biomedical research in Europe, our major focus is to give grants to researchers, and we’ve already invested over £1 million on 38 distinct studies in the UK and overseas. The spur to all our efforts is the seriousness of the disease and the significant impairment and disability it causes, something highlighted in the new report from the Institute of Medicine in the USA (see page 12).

However, we have other functions too. Research into ME/CFS is small-scale and funding difficult to obtain compared with other chronic illnesses, so researchers with fresh, novel ideas have to be recruited and encouraged to stay in the field. This is the most challenging task of all, not least because central government funding is difficult to access.

It is at this leading edge that ME Research UK sees its role: we suggest projects; give help to biomedical scientists for novel, seed-corn research projects that would otherwise not be funded; and support research groups in a variety of ways, including providing formal support letters for applications to larger agencies, such as the UK’s Medical Research Council.

The strategy has worked well, and several university departments – Leicester, Newcastle, Dundee, Vrije Universiteit Brussel – have benefited from it in the past. As Prof. Julia Newton has said, her success in obtaining major funding shows what can be achieved by biomedical researchers working closely with charities, such as ME Research UK, in a collaborative way.

But, because ME/CFS is an ‘orphandisease’ in terms of clinical recognition, public perception and scientific research, we need to do more than just fund studies – we need to raise awareness. That is why we produce this magazine, Breakthrough, host a website with a wealth of information about ME/CFS, and have a Facebook page for our most up-to-date research news.

Funding research studies is an urgent challenge, but studies can only get off the ground with your support. Please help if you can.

Dr Neil Abbot
Research & Operations Director

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this issue

Our newly funded projects ......................... 4–5
Studies on oxidative stress and reading difficulties

UK ME/CFS Biobank established ..................... 6
The biobank at the London School comes of age

Vitamin D and arterial stiffness ..................... 7
Results of the University of Dundee’s vitamin D trial

Going for a SNIP ........................................... 8
Gene SNP analysis at the University of London

Epigenetics and immune dysfunction .............. 9
A new report on epigenetic changes in ME/CFS patients

Brain abnormalities in ME/CFS .................... 10
Evidence of white matter and brain tract abnormalities

Research Collaborative Conference ............... 11
Our report of the CMRC conference in Bristol

Research bites ............................................ 12–15
Immunity and exercise, diagnosis in children, faecal transplants, gynaecological problems, IOM report, midbrain conduction, Canadian definition, and more

Tea for ME .................................................. 16–19
Fundraising by friends of ME Research UK: cup of tea, marathon run, barefoot scientist, Roamer visits Bonanza, Sue’s Graticast, and more
Combating oxidative stress

Over the past decade, ME Research UK-funded researchers at the University of Dundee have uncovered a range of biological abnormalities in ME/CFS patients (see Box on opposite page), including high levels of apoptotic (dying) white blood cells and increased arterial stiffness. Their main finding, however, has been that people with ME/CFS have high levels of reactive oxygen molecules, which can harm blood vessels and muscles. These molecules are formed in the body during biological processes that use oxygen, such as exercise. In healthy people they are counter-balanced by antioxidants that detoxify the oxygen molecules to prevent damage, but sometimes an imbalance can lead to increased ‘oxidative stress’ and an increased risk of cardiovascular disease.

It is important to discover the origin of these molecules, so that ways of counteracting oxidative stress-related cardiovascular damage can be developed. For this reason, the research team in Dundee has received funding from ME Research UK to investigate the role of ‘nuclear factor erythroid-derived 2’ (Nrf2). This is an extremely important regulatory protein in the body, and is now believed to be a master activator of the body’s natural defence against oxidative stress. When reactive oxygen species are generated, Nrf2 is activated, stimulating the body’s antioxidant pathways and thereby providing a buffer against oxidative stress.

The researchers’ aim is to test whether Nrf2 activity is low (in quantity and in gene expression) in blood samples from ME/CFS patients, and whether Nrf2 levels are related to levels of oxidative stress. Importantly, the team will also examine whether the Nrf2 antioxidant system of ME/CFS patients can be activated by certain foodstuffs and by some therapeutic drugs. At present, several drugs that stimulate the Nrf2 pathway are being assessed as treatments for other diseases, including multiple sclerosis in which oxidative stress is involved, but this is the first time ME/CFS patients have been studied.

During the 18-month study, blood samples will be taken from 40 ME/CFS patients in the unit headed by Prof. Julia Newton in Newcastle, and from age and sex-matched control subjects recruited from the general population. A range of assays will be undertaken in the laboratories at the University of Dundee, including for oxidative stress and Nrf2 protein levels (by Western blotting), and for the expression of Nrf2-target genes (by real-time PCR).

An important part of the experiment is to examine whether Nrf2 target genes can be induced in white blood cells by dietary components and/or therapeutic drugs, and whether oxidative stress is affected. Accordingly, the researchers will treat white blood cells from patients and controls with various doses of dietary compounds that activate Nrf2, including sulforaphane (from cruciferous vegetables), carnosol (from rosemary), curcumin (from turmeric) and quercetin (from apples). Also, white blood cells will be treated with therapeutic agents such as NAPQI (a metabolite of paracetamol) and parthenolide (from feverfew).

If low Nrf2 levels are found to play a central role in the increased oxidative stress found in ME/CFS patients, stimulation of Nrf2 could become an important treatment strategy, as there are currently no specifically effective treatments for the illness. The findings may also have broader implications for studies of Nrf2-targeted treatments in other conditions characterised by elevated oxidative stress, such as cancer, diabetes and liver disease.

We are delighted to feature two new investigations funded by ME Research UK: one looking at Nrf2 activity and its link to oxidative stress, the other investigating the impact of visual problems on reading.

We actively encourage researchers to contact us with their ideas for new studies. In fact, we recently issued a call for applications for ME/CFS research funding from active research groups across the world.

To date, our research has taken place at institutions in the UK and overseas and has involved many of the systems of the body, and we aim to build on this success by expanding our portfolio of funded projects.

Our priority is novel clinical and biomedical studies that help to unravel the biology of ME/CFS, and studies that may point towards therapeutic strategies. We particularly welcome applications for pilot studies or seed-corn projects. In general, projects should cost less than £50,000, though this limit may be exceeded in exceptional circumstances.

We encourage researchers to send us a summary of their research proposal on our one-page Outline Application Form, which allows us to give an initial opinion about its suitability and feasibility. Applications can be submitted at any time, and can include requests for items of medical equipment, consumables associated with laboratory tests, or salaries. All Full Applications are reviewed by external referees and the members of our Scientific Advisory Panel.

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Problems with reading

People with ME/CFS report a range of eye and vision-related symptoms, including hypersensitivity to light, difficulty focusing on images, and slow eye movements. These problems often become a pervasive part of their condition, exacerbate other symptoms, and interfere with everyday tasks such as reading and driving. Despite this, there was no solid empirical evidence of visual difficulties in ME/CFS until, in 2011, ME Research UK funded Dr Claire Hutchinson, Lecturer in Visual Neuroscience at the University of Leicester, to identify and quantify them.

Three robust scientific papers have now been published from this project, and these have shown that problems with eyes and vision are indeed a common feature of the illness (see our report in Breakthrough, Autumn 2014). The most important findings are that most ME/CFS patients experience eye pain (which is severe or very severe in one third of cases); that eye movement dysfunction is prominent; and that visual processing in the brain may also be impaired.

Since these findings were published, two ophthalmic charities have come forward with additional support for the blossoming research programme to include retinal imaging, and topics such as distance focusing and the perception of colour.

The research team now wishes to explore the effect of these vision problems on everyday activities, and ME Research UK has supplied further funding to investigate visual discomfort during reading, a common activity which is important for optimal quality of life. The researchers will examine visual stress (discomfort experienced when looking at complex, repetitive patterns such as those present in text) and abnormal eye movements while focusing on text. Importantly, they will examine possible interventions to improve the reading experience, such as coloured overlays which may reduce visual discomfort and improve the experience of reading.

During the study, fifty patients and fifty healthy control subjects matched for age, gender, and education will be recruited. Each participant will complete a series of standard outcome measures to assess diagnostic criteria, symptoms and quality of life. The researchers will examine visual stress (discomfort experienced when looking at complex, repetitive patterns such as those present in text) and abnormal eye movements while focusing on text. Importantly, they will examine possible interventions to improve the reading experience, such as coloured overlays which may reduce visual discomfort and improve the experience of reading.

Research in Dundee

In modern science, real breakthroughs come at the end of a programme of painstaking work by a specialist group of researchers. One of the few examples of such a programme on ME/CFS, anywhere in the world, is the work at the Vascular Diseases Research Unit, University of Dundee. This group has received a number of grants from ME Research UK in the past 13 years. In a step-by-step progression involving both adults and young people with the illness, the group has discovered:

- Unusual sensitivity of blood flow to acetylcholine (a neurotransmitter).
- Increased levels of isoprostanes (a gold standard marker of oxidative stress in the bloodstream).
- An unexpected increase in dying (apoptotic) white blood cells, consistent with activated inflammation or persistent infection.
- Increased cardiovascular risk factors with arterial stiffness in patients.
- Biochemical anomalies in children mirroring those found in adults with the illness.
- Dysfunction of the vascular endothelium (the lining of blood vessels).
- An association between vitamin D and markers of cardiovascular risk.

During the study, fifty patients and fifty healthy control subjects matched for age, gender, and education will be recruited. Each participant will complete a series of standard outcome measures to assess diagnostic criteria, symptoms and quality of life. In the first series of experiments, non-invasive, infra-red eye-tracking methods will be used to estimate eye movements to and from targets while reading (see photo above). The accuracy of eye targeting during reading (by manipulating word length), word frequency effect, perceptual span and binocular coordination will also be assessed.

Further experiments will test reading performance by measuring reading acuity, critical print size and maximum reading speed from MNREAD Acuity Charts, and the effect of visual stress/glare patterns on reading ability. The final experiments will determine whether coloured filters overlaid on text have beneficial effects, with the effectiveness of each filter determined by the change in reading rate.

Problems with eyes and vision certainly seem to be a clinical feature of ME/CFS. Experimental evidence of difficulties with reading-related eye movements and/or glare may help raise awareness among healthcare professionals, including ophthalmologists, of the significant impact these symptoms have on patients’ quality of life.
For the past three years, a consortium of charities – ME Research UK, Action for ME and the ME Association, with the help of a private donor – has provided funds to create the infrastructure of a UK ME/CFS Biobank. The aim was to create a ‘disease-specific’ resource consisting initially of blood samples from well-characterised cases of ME/CFS and from healthy control subjects (see the Box below).

Situated at London’s Royal Free Hospital, where it can link with the extensive research facilities at University College London, the biobank project has been driven forward by principal researchers Dr Eliana Lacerda and Dr Luis Nacul from the London School of Hygiene and Tropical Medicine. They recently announced the end of the establishment phase, to the delight of all the stakeholders (pictured above).

In providing initial funding, the consortium’s hope was that a major funder would contribute funding for a project to allow analysis of the samples, and help with the continuation and expansion of the biobank resource. So it was very welcome news when the prestigious National Institutes of Health (NIH) in the USA stepped up to the plate, under a funding call issued by its Department of Health and Human Services, and awarded £1,029,411 ($1,588,225) over three years for research and the expansion of samples. The biobank now holds approximately 16,000 samples from over 350 participants. As its primary purpose is to make samples available to external researchers for specific biomedical studies, the team should be ready to consider research applications in 2015.

The researchers at the London School are continuing to recruit participants and will be initiating virological, immunological and gene expression analyses on the collected samples using the grant from the NIH.

The aim in the longer term is the creation of a pan-European network of ME/CFS biobanks with harmonised and, in some cases, standardised protocols for clinical assessments, sample and data collection, research questionnaires, and laboratory processing procedures. Such a step would allow outside researchers access to larger sample sizes and the comparison of cohorts from different geographic regions in Europe.

The UK ME/CFS Biobank is a valuable resource, but its long-term sustainability is dependent on securing continuous financial support to allow it to reach its full potential. This will involve the expansion of participant recruitment with long-term follow-up, and the processing and analysis of samples and data to test a range of study hypotheses, as well as the ongoing sharing of samples and data with the research community.

As Dr Neil Abbot of ME Research UK said, “We are delighted to see the biobank established, and proud to have been one of the co-funders. The whole CURE-ME research team in London deserves congratulations on this achievement. The UK now has a unique ME/CFS research resource linking bio-specimens with clinical data, and we look forward to seeing it made available to scientific researchers across the world.”

What are biobanks?

Biobanks are large collections of biological specimens (blood, tissue, cell or DNA samples) obtained from donors – patients or healthy people – who have volunteered their tissues for research. Crucially, each sample is linked with comprehensive clinical information about the donor (clinically ‘well-characterised’ in research parlance).

From the patients’ perspective, the information can be used in many research studies over many years, even though samples and information are donated once only. From the perspective of the scientist, there exists a valuable database of ‘well-characterised’ samples, with individual privacy and confidentially maintained, that can be accessed for approved research projects.

Over the past decade, a large number of ‘disease specific’ biobanks have been formed, for illnesses such as cancer, schizophrenia, heart disease and MS. In the same period, two biobanks have been created to house samples from ME/CFS patients: the “SolveCFS BioBank” run by the Solve ME/CFS Initiative in Los Angeles; and the Whittemore Peterson Institute repository of more than 8,000 samples and clinical information in Nevada. Both ME/CFS-specific repositories are located in the USA, however, and their existence highlighted the need for a similar biobank in the UK.
Vitamin D and arterial stiffness

Low levels of circulating vitamin D in the blood are associated with increased blood pressure and poorer vascular health, and they are known to increase the risk of future cardiovascular events such as heart attack and stroke.

For this reason, several large trials are now underway across the world to explore the effects of vitamin D supplementation: giving high-dose vitamin D by mouth to increase levels in the body. A variety of diseases are being studied, including the disease, osteomalacia, which involves softening of the bones and (like ME/CFS) comes with fatigue, muscle pain and problems with skeletal muscle.

In 2009, a study by London-based researchers found vitamin D levels to be considerably lower in ME/CFS patients than in healthy people. Shortly thereafter, an investigation funded by ME Research UK at the University of Dundee discovered an association between lower vitamin D levels and arterial stiffness, dysfunction of the endothelium (the lining of blood vessels) and inflammation.

These results suggested that low vitamin D might well be contributing to the burden of illness in ME/CFS patients, and were the reason why ME Research UK decided to fund a prospective randomized trial. The idea was to determine whether vitamin D supplementation could reverse the impairments in vascular health and alleviate some of the symptoms of the illness.

In the trial, 50 ME/CFS patients, diagnosed according to the Fukuda (1994) and Canadian (2003) criteria, were divided into two groups: one group received 100,000 units of vitamin D3 by mouth while the other received an indistinguishable placebo. Doses were given every 2 months for 6 months.

As the researchers were particularly interested in the effect on vascular function, the main outcome measure was arterial stiffness, measured using a technique called ‘pulse waveform analysis’ (see Box), which previous research had shown to be worse in ME/CFS patients than in healthy people. Other measurements included blood pressure, cholesterol, and markers of inflammation, as well as fatigue and other symptoms.

After 6 months, high-dose oral vitamin D3 levels in the blood had increased significantly (by 22 nmol/L) in the treatment group compared with the placebo group. However, the increase in vitamin D levels had no effect on arterial stiffness levels at 6 months, and no improvement was seen in other vascular and metabolic outcomes, or in fatigue or other symptoms.

The lack of effect of supplementary vitamin D on vascular or inflammatory markers in ME/CFS patients contrasts with its positive effects in other diseases. In type 2 diabetes mellitus patients, for example, vitamin D (at the same dose as the ME/CFS patients in Dundee) improved endothelial function and blood pressure, while in stroke patients improvements in endothelial function have been observed after supplementation.

The reasons for these negative results are unclear. Could low vitamin D levels be a result of ME/CFS itself – in which case, supplying more would not necessarily reverse the vascular dysfunction. Or might larger or more frequent dosing be needed in order to produce a clinically relevant effect in this specific group of patients?

This last possibility is in the researchers’ sights, and they are deciding on the next experimental steps. As principal investigator Dr Khan says, “Given the increased arterial stiffness we’d previously found in our ME/CFS patients, it was certainly worth investigating whether it could be reversed by vitamin D supplementation, which is a cheap and very convenient therapy. Our thanks go to the patients in Tayside who took part, some of whom had travelled long distances.”

Pulse wave analysis

When you place your fingers on your wrist, you can feel your pulse; that is, the regular increase in pressure as each pulse of blood travels down the radial artery. This pulse can also be detected by a pressure sensor applied to the wrist, and this is the technique used by Dr Khan and his colleagues to determine arterial stiffness.

The sensor produces a continuous recording of the fluctuations in pressure caused by each pulse wave, and their shape is analysed to determine how stiff the artery is. The pulse pressure wave is composed of a wave generated by the ejection of blood from the heart and a reflected wave from the periphery. As arteries get stiffer, the speed of both waves increases, causing the reflected wave to arrive earlier in the aorta and augment the size of the pulse. The measured ‘augmentation index’ is therefore related to blood vessel stiffness.
Going for a SNIP

The information inherited from our parents (usually in the form of a gene, a sequence of DNA) has to be translated into a product, such as an RNA molecule or a protein, before it can be used by the body. This process is called gene expression. In the past few years, a number of research groups worldwide have investigated gene expression in people with ME/CFS, including Dr Jonathan Kerr’s group at St George’s Hospital, University of London.

After initial pilot studies, the group published a paper in the Journal of Infectious Diseases in 2008 outlining its identification of a putative gene ‘signature’ for the illness, consisting of 88 human genes. When these genes were subdivided into categories based on diseases or functions, some were found to be associated with haematological (22 genes), immunological (14) or dermatological functions (3); with cancer (31); or with the endocrine system (9). Unfortunately, a subsequent blinded study showed this ‘signature’ to be less robust across populations, although it was able to classify roughly two-thirds of both ME/CFS and healthy samples successfully.

As these 88 genes had been linked to the pathogenesis of ME/CFS, the logical next step was to examine single nucleotide polymorphisms (SNPs, pronounced ‘snips’; see the Box below) within these genes. With funding from ME Research UK, the group at St George’s began work on identifying the key SNPs for each of the 88 genes.

As there are hundreds of SNPs within each gene and the cost of studying all of them would be prohibitive, the team focussed on ‘determinative’ SNPs (i.e. those which are known to predict all or most of the others within one gene); the number of determinative SNPs per gene typically varies between 3 and 7. Once these determinative SNPs had been identified for each gene, the researchers designed low density array cards to contain their respective assays, had these manufactured, and then used them to test genomic DNA samples of 108 patients and 85 people without ME/CFS, including some with endogenous depression.

The headline result, published in the Journal of Clinical Pathology in 2014, was that 21 SNP alleles had significantly different ‘frequency distributions’ in ME/CFS patients than in depression control or healthy control subjects – seven of these SNPs were within the BMP2K gene and two were within the IL6ST gene. A significantly different distribution between ME/CFS patients and healthy controls was observed for 10 ME/CFS-associated gene SNPs. In addition, the authors found 148 SNP alleles that were associated with one or more of the putative genomic ‘subtypes’ of ME/CFS, although the distribution of these alleles was not related to the gene expression data for each subtype.

Will SNP analysis based on ‘susceptibility genes’ eventually become useful for the diagnosis or clinic management of ME/CFS? Well, as the authors point out, a SNP-based test is certainly more robust and more easily reproducible than gene-expression–based testing (which is intricate and time-consuming). The first step, however, is to see these results replicated on other groups of ME/CFS patients and on patients with other, similar chronic diseases. Only after this is done can work begin on acquiring hard SNP-based evidence for the existence of ‘subtypes’ in the illness.

What are SNPs?

Single nucleotide polymorphisms (SNPs) are small genetic changes in DNA that vary between individuals. Humans are 99% identical as regards their gene sequences, and the 1% which remains is mostly accounted for by SNPs, of which there are approximately 10 million in the human genome. This makes SNPs very useful. They can serve as helpful landmarks for population genetic maps, but their greatest importance is in biomedical research, for comparing specific regions of the genome between groups or individuals with and without a disease.

While most SNPs are silent, some can have important consequences for individual susceptibility to disease and reactions to treatment. An example is the apoE gene which is associated with an increased risk of Alzheimer’s disease. It is also thought that certain SNP combinations can contribute to a predisposition to developing medical conditions.

At present, an enormous amount of literature exists reporting possible associations between SNPs and diseases; the SNP500Cancer project, for instance, is examining samples to locate important SNPs, and there are many such examples in a range of illnesses.

The challenge in ME/CFS is to identify SNPs which will ultimately allow patients to be quickly and simply ‘diagnosed’ from a sample, and possibly assigned to illness subgroups so that they can receive specific therapies.
Epigenetics and immune dysfunction

We have all become accustomed to the idea that diseases can have either environmental or heritable causes – in fact, this dichotomy is now hard-wired into our views of the world. However, a relatively new field of endeavour, epigenetics, is beginning to challenge these assumptions.

Conventional genetics is concerned with changes to sequences of DNA (the genotype) which are then inherited, but epigenetics postulates that changes in gene expression (the way information from a gene is used to make products, usually proteins) can be affected by other factors and processes. These include childhood development, drugs, diet, environmental chemicals and even the ageing process. In particular, epigenetic modifications (through DNA methylation, for example) can have effects on the function of genes over the long term, and may be involved in a range of illnesses, such as diabetes or cancer.

In ME/CFS, a variety of studies have examined gene regulation; these have shown alterations in gene function in the immune system of patients, supporting other evidence of immune system abnormalities (see pages 14 and 15). Since epigenetic modifications could well be involved, researchers at the University of Toronto decided to test for these in the DNA of 12 female ME/CFS patients (who all reported sudden infectious onset of their illness) and 12 matched healthy controls, all recruited through the SolveCFS BioBank in the USA.

There are many techniques for identifying epigenetic modifications of DNA, and the researchers chose to examine the occurrence of DNA methylation in white blood cells using a specific BeadChip array and complex analysis techniques, including gene ontology (GO) analysis, to identify the biological pathways in which changes in DNA methylation might be involved.

The results (published in PlosOne, 2014) make interesting, if complicated, reading. The researchers found a range of specific regions of DNA (methylated CpG dinucleotide sites) which differed between ME/CFS patients and controls. After GO analysis, four cluster groups, consisting of a total of 57 GO terms, were identified – a cellular processes group, a positive metabolic regulation group, an enzyme kinase activity group, and an immune cell regulation group.

Within the four cluster groups, 511 unique genes containing a total of 637 CpG sites were significantly ‘hypermethylated’ in ME/CFS patients compared with healthy people, and 184 unique genes containing 237 CpGs were significantly ‘hypomethylated’. The immune cell regulation cluster, with 22 GO terms, had the largest “enrichment of differentially methylated gene pathways” (indicating alterations in gene expression in ME/CFS patients compared with healthy people), and there were clear indications of a shift towards ‘hypomethylated’ immune genes in the patient group.

Overall, the epigenetic analysis found evidence that immune cell regulation differs between ME/CFS patients and controls, a result that accords with what we already know about functional changes in immune profiles in the illness. The other differences noted by the researchers in gene set-enrichment (linked to differences in cellular processes, enzyme kinase activity and positive metabolic activity) also support current understanding about the role of dysregulation of cellular metabolism and oxidative stress in ME/CFS.

The importance of these findings (at least at present, until further epigenetic investigations are done) is that they confirm, using very different methodologies from studies in the past, that multisystem dysregulation is a feature of ME/CFS. However, they also implicate the involvement of specific DNA modifications in the causes or consequences of the disease, a potentially important finding in itself. The reasons for these epigenetic modifications (environmental agents? infections?) in patients remain unknown. Nevertheless, the researchers point out that “epigenetic changes can exert long-term effects on gene expression and are potentially amenable to therapeutic intervention”, citing the example of cancer in which therapeutic interventions targeting epigenetic mechanisms have had some success in altering inflammatory pathways.

Although it is still too early to tell whether or not the new ‘epigenetic perspective’ will revolutionise the investigation and treatment of complex chronic diseases, this novel report from the University of Toronto has presented valuable evidence to an international audience that epigenetic alterations can have a role in the pathophysiology of ME/CFS.
Brain abnormalities in ME/CFS

Nervous system symptoms are as characteristic of ME/CFS as post-exercise malaise or muscle pain (myalgia). In historical publications on ‘epidemics’ of ME, symptoms consistent with central nervous system pathology were regularly reported, as Sir Donald Acheson pointed out in a review more than 50 years ago. And today, fatigue, non-refreshing sleep, short-term memory impairments, sensitivity to variable stimuli like bright light and chemicals, and widespread pain are all suggestive of central nervous system involvement.

In a previous issue (Breakthrough, Autumn 2014), we reported on research from Osaka City University, Japan, showing inflammation of nervous tissue in widespread brain areas in ME/CFS patients. Since then, Prof. Jose Montoya’s group at Stanford University School of Medicine has published a scientific report containing some striking results which complement the Japanese findings.

The US researchers’ aim was to see whether ME/CFS patients had differences in gross brain structure, microscopic structure or brain blood flow that could explain their symptoms, so they compared brain MRI images from 15 long-term ME/CFS patients with images obtained from 14 age and sex-matched healthy volunteers with no history of relevant symptoms.

They found abnormalities in a brain tract called the arcuate fasciculus, as well as reductions in the volume of white matter in the brain in patients compared with healthy controls. As lead author Dr Michael Zeineh, assistant Professor of Radiology, explains, “Using a trio of sophisticated imaging methodologies, we found that CFS patients’ brains diverge from those of healthy subjects in at least three distinct ways.”

Overall, the three key findings were, first, that white-matter content (which tends to carry information between different parts of the brain) was reduced by about 7% in the brains of ME/CFS patients compared with healthy people. Second, using an advanced imaging technique (diffusion-tensor imaging), a consistent abnormality in the right arcuate fasciculus (‘curved bundle’ in Latin) which is a long bundle of nerve fibres (axons) connecting the frontal and temporal lobes. The function of this structure remains something of a mystery, but it is generally thought to connect two brain areas (Broca’s area and Wernicke’s area) that are important for the use of language.

This Stanford study adds to the growing body of evidence that people with ME/CFS have real physical defects, especially in the central nervous system, and may point to an underlying mechanism in the disease process. Unusually for a ‘forgotten illness’ such as ME/CFS, the results were widely reported across the world, including the New York Times blog which pointed out that “many experts now believe that, in people with ME/CFS, a viral infection or some other physiological insult or exposure, or perhaps a combination of exposures, has kicked the immune system into permanent overdrive, leading to the cascade of symptoms. The new brain research appears at a timely moment.”

Prof. Montoya is planning a substantially larger study to explore the findings: “In addition to potentially providing the CFS-specific diagnostic biomarker we’ve been desperately seeking for decades, these findings hold the promise of identifying the area or areas of the brain where the disease has hijacked the central nervous system.” He makes the point, however, that these results, though quite robust, need to be confirmed by others since replication is the backbone of science.
The UK ME/CFS Research Collaborative (CMRC) hosted its very successful inaugural conference in Bristol in 2014. The aim of the CMRC – the first of its kind in the world – is to promote the highest quality of basic and applied evidenced-based and peer-reviewed research into ME/CFS by bringing together national agencies, ME/CFS charities, and active researchers from across the UK who wish to work towards that common goal.

The conference took place in Wills Hall, part of the University of Bristol, and the organisers had invited a range of international speakers as well as researchers from the UK. These included Prof. Robert Dantzer of the University of Texas, Prof. Andrew Lloyd of the University of New South Wales, Prof. Ian Lipkin of Columbia University, New York, and MRC-funded research project leaders, each giving a brief presentation followed by a panel discussion and question and answer session.

Conference numbers were impressive, with more than 70 professional delegates registered for the research conference, and more than 60 Associate Member delegates (mainly patients and families) registered for the workshop, which also included 20 researchers. ME Research UK was represented by Vice-Chair, Sue Waddle, and Research & Operations Director, Dr Neil Abbot.

Opening the event, Prof. Stephen Holgate, Chair of the CMRC, welcomed delegates, and introduced Prof. Robert Dantzer who has a long-standing interest in the relationship between cancer-related fatigue and inflammation, and gave a fascinating overview of the journey towards understanding this relationship over the past 25 years.

As well as lectures, there were shorter presentations on an ‘inflammatory’ theme by researchers from Brighton & Sussex University, Kings College London, St Barts London, and the University of Liverpool.

The key event was the main afternoon workshop, which brought together patients and their families, and researchers to discuss issues of concern to ME/CFS patients, and ways of working together to facilitate research. The workshop was run by experienced facilitator Sally Crowe (pictured below with our Dr Neil Abbot), and frank dialogues on specific topics took place at individual tables throughout the afternoon.

When the conclusions were collated, the most pressing themes were found to be the exclusion of severely affected patients from research studies; the importance of ‘expert patients’; the importance of research on sleep and alternative & complementary medicine; the need for more stratified clinical groups in research (phenotyping) since the heterogeneity among patients is a clinical challenge; and the low profile of the illness and the need to assess and highlight its economic impact.

The workshop was followed by a robust and wide-ranging discussion between the expert panel (Profs Dantzer, Lloyd and Lipkin, and MRC-funded researchers) and the expert-patient audience. At the end, two key elements were agreed by all: the need to bring high-profile people into advocacy for the illness (well-known spokespeople), and the need to entice bright and thrusting young biomedical researchers into the field.

The members of the CMRC are grateful to the Medical Research Council for subsidising the conference, and particularly for its sponsorship of the patient–researcher session. As the MRC has said, “We have been keen to bring together charities and researchers in this area, which is why we supported the setting up of the CMRC. The MRC hopes that the patients’ session at this conference will be beneficial to those trying to take the field further – both patients and researchers.”

Sally Crowe and Dr Neil Abbot
Research bites from around the world

WASHINGTON

Institute of Medicine report

The committee formed by the Institute of Medicine in the USA to examine ME/CFS released its 280-page report in February 2015. As the president of the Institute, Victor Dzau, said, “The diagnostic criteria offered in this report are intended to promote prompt diagnosis for patients and enhance treatment, as well as improve public understanding of the disease.”

Its message overall was that ME/CFS is a serious, chronic, complex and multisystem disease that frequently and dramatically limits the activities of affected patients. Accordingly, the committee recommended:

• New diagnostic criteria, more focused on the core symptoms of ME/CFS than other definitions. These include activity reductions plus fatigue for more than six months, post-exertional malaise, unrefreshing sleep, and either cognitive symptoms or orthostatic intolerance.
• A new name: ‘Systemic Exertion Intolerance Disease’ (SEID). The committee points out that the name ‘CFS’ perpetuates misunderstanding of the illness and dismissive attitudes from healthcare providers and the public, and it believes that “SEID captures the central characteristic of the disease – that exertion of any sort can adversely affect several organ systems and many aspects of patients’ lives, often seriously and for long periods.”
• A new disease classification in ICD-10, not linked with ‘chronic fatigue’ or ‘neurasthenia’ as at present.
• An official toolkit appropriate for screening and diagnosing patients, that could be developed centrally by the US Department of Health and Human Services.

There are many different opinions on the report. Some people think the new name cumbersome, and that ‘Ramsay’s disease’, in honour of Dr Melvin Ramsay, would have been preferable. Others think that the absence of a list of exclusionary conditions with similar symptoms is more likely to compromise diagnosis than clarify it.

Everyone agrees, however, that new criteria will only improve diagnosis and care if healthcare providers actually use them and find them to be an improvement. This will be the big challenge for the future.

Source: Institute of Medicine, National Academies Press, 2015

LONDON

Fear of exercise?

The Lancet Psychiatry recently published an analysis of data gathered during the PACE trial, funded by the Medical Research Council in 2011. This was the sixth sub-analysis in the series, others having dealt with cost-effectiveness, pain, ‘recovery’, adverse effects and statistical methods. However, these findings were the most difficult to understand, as they deal with the ‘mechanisms’ underpinning the effect of cognitive behavioural therapies (CBT) or graded exercise.

Using very sophisticated statistical analysis, the researchers eventually conclude that ‘fear avoidance beliefs’ (the attitude that exercise will make symptoms worse) by ME/CFS patients is a major factor in the success or otherwise of these therapies.

While this report may be fascinating to professional cognitive-behavioural theorists, the central fact remains – as the original PACE trial data showed – that the effects of these psychosocial approaches are modest, benefiting only around 10 to 15% of ME/CFS patients over and above the benefit of standard medical care. As the Editorial accompanying this study said, most patients do not recover after these interventions, and “quite a few patients do not profit at all”.

The sad thing is that every time one of these sub-analyses of PACE trial data is published, a rash of media stories trumpet its arrival. For instance, the Daily Mail headlined their report with “Victims suffer fear of exercise”, while The Daily Telegraph decided to go with “ME: fear of exercise”.

These stories are presumably based on the press release, and are run off to satisfy the 24-hour news cycle. However, they have little or no relevance to the real, lived experience of patients. We all know that people with ME/CFS are highly motivated to get well, so the notion of ‘fear avoidance’ seems inappropriate and absurd, however fascinating that concept may be to academic psychologists.

Source: Chalder et al., The Lancet Psychiatry, 2015
Faecal transplants

Faecal microbiota transplantation (FMT) is the infusion of faecal matter from a healthy person into the colon of an ill person, usually by enema, with the aim of re-establishing a balanced intestinal flora to the digestive system. It is most commonly used to treat Clostridium difficile infection, but a new review from Tianjin Medical University in China suggests broadening its application beyond intestinal disorders to other chronic diseases, including autoimmune conditions and ME/CFS.

They point out that one clinical trial has been conducted on FMT in metabolic syndrome (which can lead to diabetes and heart disease), showing that the infusion of microbiota from ‘lean’ donors increased sensitivity to insulin and raised levels of ‘good’ butyrate-producing intestinal microbiota.

They also describe case reports showing favourable outcomes in Parkinson’s disease and multiple sclerosis, and they mention one uncontrolled study on 60 long-term ME/CFS patients with gastrointestinal symptoms. After FMT, 70% of patients “responded to treatment” and 58% had “complete resolution” of symptoms during a 15 to 20-year follow-up period.

We can’t conclude much from one retrospective uncontrolled study, of course. Yet the idea behind FMT does chime with recent scientific discoveries about the importance of the human ‘microbiome’ (the hidden yet extensive world of the microbes that live in our bodies), and it may eventually come to be an important, if rather unpleasant, therapy.


Canadian definition is insensitive

One of the most animated debates surrounding ME/CFS concerns the name of the illness and how it is diagnosed. At present, a range of possible definitions exist, but each is different, and today the terms ME, CFS and their various combinations mean different things to different people. Does this matter? Absolutely – because the different ways of diagnosing patients capture different kinds of symptoms, and this influences clinical research, the treatment options that are offered, and the perception of the illness by employers, benefit agencies and the public generally.

One of the most commonly discussed definitions is the 2003 Canadian Criteria for ME/CFS. This was designed by consensus to help health professionals make a diagnosis effectively, but has never been formally validated, which was why researchers at the University of Oslo wanted to explore its clinical usefulness. They examined young people with ME/CFS and subgrouped them according to the whether or not they fulfilled the 2003 criteria.

The two groups were then compared on a wide range of tests, including clinical examination, blood sampling and testing for disease markers (of cognitive, HPA-axis and autonomic function, and inflammatory responses). They also completed an inventory of the frequency and severity of 24 common symptoms of illness during the preceding month.

Fascinatingly, disease markers and other outcomes were no different in youngsters who satisfied the criteria than in those who did not. Group differences were found on only one test of cognitive function, which was poorer in the group fulfilling the criteria. Also, activity levels measured by an activPAL accelerometer were the same in both groups over the 30 weeks of the study. These results question the usefulness of the 2003 Canadian Criteria in research as well as in clinical practice, so clarification of the case definitional morass surrounding ME and CFS remains an urgent challenge.


Gynaecological problems

A new study, reported in the journal Menopause, links ME/CFS with early menopause (most often following a hysterectomy), along with other gynaecological problems and pelvic pain.

The researchers examined 84 ME/CFS patients and 73 healthy women in the USA. The women with ME/CFS reported significantly more gynaecological conditions: menopause status (62 versus 37%), earlier average age at the onset of the menopause (38 versus 49 years, commonly because of hysterectomy), excessive menstrual bleeding (74 versus 42%), non-menstrual pelvic pain (26 versus 3%), and a range of other gynaecological difficulties. The patients had also undergone more surgical operations than the healthy women, most often hysterectomies (55 versus 19%) which occurred before the onset of ME/CFS symptoms in most cases.

As the North American Menopause Society explains, ME/CFS can take a tremendous toll on women’s lives at midlife, and clinicians should be aware of its associations with the reproductive system, particularly with earlier menopause. The higher prevalence of gynaecological conditions and surgery in women with the illness highlights the importance of regularly evaluating their gynaecological health.

Source: Boneva et al, Menopause, 2015
Diagnosis in children

Illness in youngsters has a particular poignancy. Estimates of the number of children with ME/CFS vary, but with prevalence figures of 60 to 70 cases per 100,000, it is likely that there are many thousands in developed countries. The Chief Medical Officer in 2002 was clear; the illness “represents a substantial problem in the young” and “potentially threatens physical, emotional, and intellectual development of children… at a particularly vulnerable time of life”. So it is important that clinicians and paediatricians know about ME/CFS, recognise the symptoms and diagnose it early.

Dr Sarah Knight and colleagues at the Murdoch Children’s Research Institute in Melbourne decided to explore the ways Australian paediatricians diagnose and manage ME/CFS in the children under their care. An online survey was sent to members of the Australian Paediatric Research Network, and 178 replies were received. Only 70 of the paediatricians (39%) said that they actually diagnosed and managed the illness, and those who reported seeing youngsters with ME/CFS were significantly more likely to be in private practice. Interestingly, 51% said that they did not use any of the published diagnostic criteria for ME/CFS to make a diagnosis.

Initial medical investigations varied, and most of the paediatricians undertook standard testing, such as a full blood count, blood biochemistry, electrolytes and creatinine, ESR and CRP, or viral serology. However, only 17% said that they routinely conducted the first-line investigations recommended in Australian guidelines, despite the fact that the guidelines were developed more than ten years ago. Management was multidisciplinary, and the paediatricians commonly engaged a school teacher, physiotherapist and/or psychologist as part of their approach.

Clearly, much more needs to be done, and clinical guidelines for paediatricians in Australia (and across the developed world) need to be revised, updated and actually implemented. After all, what’s the point of guidelines if they are left lying in a drawer?

Source: Knight et al., Journal of Paediatrics and Child Health, 2014

“Robust evidence” of biological illness

A dramatic scientific paper from Columbia University’s Mailman School of Public Health caught the attention of the world’s media this year. The researchers had measured 51 immune biomarkers in blood plasma samples collected from two large multicentre studies, representing a total of 298 ME/CFS patients and 348 healthy control subjects, and they found “distinct plasma immune signatures” in the early stages of ME/CFS.

The headline finding was that patients who had the disease for three years or less had a specific immune ‘signature’ that was not present in control subjects or in patients who had been ill for more than three years. Patients with a shorter duration of illness had increased amounts of many different types of immune molecules called cytokines. In particular, there was a marked association between early ME/CFS and the cytokine, interferon gamma, which has been linked to the fatigue that follows many viral infections, including Epstein-Barr virus which causes glandular fever. The patients who had been ill for the longest were older, of course, but the differences in immune function could not be explained simply by age. As the lead author, Prof. Mady Hornig, explains, ME/CFS patients seem to be flush with cytokines until around the three-year mark, at which point the immune system shows evidence of exhaustion and cytokine levels drop.

The results support the idea that ME/CFS may reflect an infectious ‘hit-and-run’ event. Patients often report getting sick, sometimes from something as common as glandular fever, and that they then never fully recover. These infections throw a wrench in the immune system’s ability to quieten itself after the acute infection; in effect, the immune response becomes like a car stuck in high gear. As Prof. Ian Lipkin said, “This study delivers what has eluded us for so long: unequivocal evidence of immunological dysfunction in ME/CFS.”

Source: Hornig et al., Science Advances, 2015
Pain, fatigue and malaise after exercise are hallmarks of ME/CFS, and many of these symptoms can be explained by the actions of the immune system in response to over-exertion. But is there good scientific evidence to back this up? Prof. Jo Nijs and his team at Vrije Universiteit Brussel, who have received ME Research UK funding for several studies in the past, have just completed a systematic review of the scientific literature on exercise-induced immunological changes in ME/CFS patients. In total, they found 23 relevant case–control studies addressing the issue using standardized exercise protocols in a laboratory setting.

Compared with healthy people, ME/CFS patients have more pronounced immune responses to exercise in three key areas. First, they respond to strenuous exercise with a slow but stronger increase in blood complement C4a levels; two independent studies have confirmed the relationship between an altered complement response and post-exercise malaise. Next, there is accumulating evidence that oxidative stress following exercise occurs earlier and lasts longer in ME/CFS patients, as studies at the University of Dundee have shown. Last, there is some evidence of post-exercise increases in immune cell gene expression. A biological signature or clinical thumbprint for ME is the holy grail, and post-exercise immune changes could well be a key component.

Source: Nijs et al., Exercise Immunology Review, 2014

Two recent studies have reported unusual findings in the brains of ME/CFS patients: increased inflammation and white matter reductions (see page 10). Critics sometimes claim, however, that psychological brain states, such as depression and anxiety, can account for such findings, and it can be difficult to refute this directly from the experimental evidence. A new investigation from Griffith University in Queensland has attempted to pre-empt its critics by controlling for these factors.

The researchers used voxel-based MRI to assess brain white matter in 25 people with ME/CFS and 25 healthy controls. This technique shows the size and shape of brain structures, but also gives other vital information in the form of T1 and T2-weighted signals. In the absence of brain lesions, the T1-weighted signal is mainly an indicator of myelin (which lines nerve fibres and helps conduct signals), so it can reveal subtle changes in white matter. By contrast, the T2-weighted signal is an indicator of brain blood volume. Crucially, the researchers also measured severity of illness, and depression and anxiety scores.

Their results showed that the T1-weighted signal increased with the severity of patients’ illness in three brain areas – the ventrolateral thalamus, internal capsule and prefrontal white matter – indicating impaired nerve conduction in the midbrain. Also, there were changes in the T2-weighted signal in the middle temporal lobe white matter, a site where nerve communication problems can affect brain function. Adjustment for depression and anxiety did not alter the main findings. As the researchers say, “impaired brain–body and brain–brain communication through the midbrain could explain many of the autonomic and cognitive symptoms” experienced by ME/CFS patients.

Source: Barnden et al., NMR in Biomedicine, 2015
Tea for ME
Recent fundraising for ME research

Have a cup of tea for us
As Father Ted’s housekeeper, Mrs Doyle, says, “There’s always time for a nice cup of tea.” And Stephanie Devlin from Dumbarton certainly agrees. She recently hosted a ‘Cup of Tea for ME’ event at her mum’s house. “We had loads of lovely, home-baked cakes, tea and coffee. All my family, friends, colleagues and neighbours gathered for a brilliant day. I was blown away with everyone’s generosity, and thank them all!” So we’re raising a cup of thanks to Stephanie for supporting us.

For ME Awareness Week 2015, we’re inviting you all to follow in Stephanie’s footsteps. Follow our Facebook page and website for details on how you can create your own event. For instance, we have invitations ready to be personalised and printed off, and much more information on how to have a little party for us.

Marathon run
Manny Virdi ran the Bristol half marathon for us recently, and what’s very special is that he has no personal experience of ME – he simply cares enough to support our cause. He was inspired to raise money after watching the film ‘Voices from the Shadows’, a moving account of life for people with severe ME.

Manny is pictured below with his friends Tom Whittingham, an ME Research UK Ambassador, and Tom’s sister, Beth, who both also completed the half marathon. Tom and Beth’s sister, Naomi, has had ME for more than 20 years, and this has impacted greatly on the life of the family. As Tom says, “Naomi was a happy 13 year-old whose life was taken away from her by ME.” Thanks to Manny, Beth and Tom for all they do to help the cause.

ME Support Northern Ireland
We have received another fabulous contribution to our research funds from our friends at ME Support Northern Ireland. The donation will be used to help fund our research project on severely affected ME patients, which Professor Julia Newton is conducting at the University of Newcastle.

There has been a chronic lack of research into severe ME, and there is a serious void in the scientific knowledge-base about this group of patients, so ME Support Northern Ireland’s donation will be put to good use.

Many thanks to the group and to its Chair, Antoinette Christie, for this generous contribution to the work of ME Research UK.
The Barefoot Scientist

Neuroscience PhD student Zoltan Derzsi has been raising awareness of ME by casting aside his shoes and walking the streets barefoot, raising funds for both ME North East and ME Research UK.

Zoltan (right) says, “I was inspired to start a campaign after a friend’s daughter was diagnosed. She has endured almost constant immobility and pain since then. I am walking without shoes and socks to shed light on the fact that in this area of science there needs to be far more research and help for patients.”

Barefoot, he has visited places from Durham Cathedral to his local Tesco, though eyebrows were raised in Optical Express, which had never had a customer without socks and shoes before. So he now wears a jumper with ‘Barefoot For Charity’ to explain what he is doing!

21st birthday celebration

Katharine Cheston (below) was 21 last year. She couldn’t run a marathon or sky-dive to raise funds for ME Research UK, but she had other plans. She asked for donations in lieu of presents, and held a fundraising tea party. Friends from across the globe joined her party via Facebook, including those unable to travel due to illness.

As she explains, “Celebrating my 21st birthday was a special moment because I was able to spend it with almost all of my closest friends. It was a privilege, and despite all the frustrations and losses ME brings, I consider myself truly blessed to have such great friends – alongside the most supportive family possible.”

Fighting for ME Research

After completing a course run by the ‘Ladies Charity Boxing’ organisation, Pink Collar Boxing, Caroline Whitaker stepped into the ring last November at The Waterfall, Derby. The course aims to turn women from pugilistic ‘Beginner to Winner in 8 Weeks’, while helping a range of charities at the same time.

Diagnosed with ME 4 years ago, Caroline is slowly getting back on her feet and, despite setbacks, she managed to complete the 8-week training period and entered the ring to raise money for ME Research UK.

Three cheers for Caroline and Pink Collar Boxing; as its motto says, “You don’t have to be great to start but you have to start to be great!”

Roamer visits Bonanza

Roamer, our roaming Ambassador to the World, has been travelling across the US. So he just had to drop into Incline Village at Lake Tahoe, Nevada, which was the site of the famous and well-documented cluster of outbreaks of ME between 1984 and 1986. But Incline Village is also where the 1960s TV western series ‘Bonanza’ was filmed latterly, and was home to the Cartwright family’s Ponderosa Ranch. Look out for more of Roamer’s travels in 2015.

The Great Cumbrian Run

The Great Cumbrian Run covers 13.1 miles around the historic city of Carlisle – and Anita & Julie Harry and Darren Hendren successfully completed the course at the end of last year, raising a fabulous total for research. Of course, they were helped by a dedicated ‘pit crew’.

The group also harnessed the power of the press, and the local Whitehaven News carried an article about the run, the illness and, more importantly, why funding research into ME is so close to their hearts. Julie’s daughter (and Darren’s girlfriend) Emma has had ME for the past decade. As Emma says, “I’m really proud of my family and Darren for doing the Cumbrian Run. I’ve had to sacrifice a lot growing up, but I don’t want sympathy. I just want people to understand.”
Help us by walking for ME in 2015

ME Research UK is delighted, once more, to have been chosen as one of Walk for ME’s featured charities for 2015. Now in its third year, Walk for ME is determined to build upon the success of last year’s event which raised almost £15,000. As well as the UK, supporters in Ireland, Spain, New Zealand and the US took part.

Distances varied depending on participants’ health. As there is no minimum distance set, no targets and no set dates (though ideally walks would happen during ME Awareness Week, 11th to 17th May 2015), everyone can take part. As the organisers say, “The whole idea is that a friend or family member is doing something that their loved one would love to be able to do but can’t.”

This year, Tracey Wilson, an ME Research UK Ambassador, will walk pushing her daughter’s wheelchair. You too can follow in the footsteps of Tracey and join the Walk for ME initiative. Our website has all the details and links to help you take the first step.

Sue’s Graticast sofa-cast!

As part of the Graticast series, Chip Colquhoun and his team (below) visited ME Research UK’s Vice-Chair, Sue Waddle, at her home for an interview on YouTube. Sue has been involved with ME for many years, and the chat covered her family’s own experiences of the illness, the difficulties patients face, and ME Research UK’s quest to fund research.

Chip and friends’ weekly humorous Graticasts aren’t specifically about ME, but the illness always gets a mention, and they aim to raise awareness by helping the general public look on the bright side of life – you can subscribe to Chip’s feeds for free at youtube.com/user/graticast.

Many thanks to the Graticast team – as Sue says at the end of the sofa-cast, “Everyone needs a Chip”, and it’s true!

Church Crookham

The beautiful village of Church Crookham in Hampshire dates back to the Domesday Book. The arrival of the railway brought day-trippers from London to admire the picturesque setting, though nowadays people are just as likely to shop before commuting.

That’s why Tracy Wilson, who lives there, decided to set up a stall outside the Sainsbury’s Local one Saturday afternoon. As she says, “Supermarket collections are one of the simplest and least energetic ways of raising money, so I thought I’d give it a go.”

On the day, she was joined by our Vice-Chair, Sue Waddle, who lives not too far away.

“One of the great advantages of the day was to be able to explain about ME and its consequences to shoppers as they milled around,” Sue explained.

Could you arrange a supermarket collection? If so, please contact us and we may be able to help with some promotional materials.

2015 miles in 2015

Chris Croshaw (below) aims to run 2015 miles in 2015 (the equivalent of Gateshead to the Kremlin in Moscow), and is raising funds at the same time. He took up the sport just 3 years ago, having never really done any running before, but once started he was hooked, always wanting to go faster and further.

Chris’ inspiration is his wife, Jocelyne, who was diagnosed with ME/CFS, fibromyalgia and osteoarthritis 3 years ago. As he explains on his Facebook page (2015forME), “Since that day our lives have changed in ways neither myself nor my family could have imagined. When I hear people say, ‘Oh yeah ME, that’s where you feel tired isn’t it?’, it makes you realise how little people know. ME/CFS is an invisible illness, and bringing attention to this and all the other invisible illnesses out there is part of what my challenge for 2015 is all about.” Thanks, Chris, and we all wish you well with the running and fundraising.
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