When the autonomic nervous system goes wrong, the effects can be severe. For instance, one of the main consequences is orthostatic intolerance; that is, the inability to remain standing for long without suffering ill effects. Standing is one of the key difficulties faced by ME/CFS patients, particularly standing still without experiencing symptoms such as dizziness, altered vision, nausea and fatigue. It is therefore very possible that a dysfunction of the autonomic nervous system could be involved in the illness.

A scientific article in the current issue of the Quarterly Journal of Medicine reports the findings of Dr Julia Newton and colleagues at the University of Newcastle who, with a grant from ME Research UK and the support of the regional ME/CFS service, have been examining people with ME/CFS using a well-validated battery of autonomic function tests. The researchers report a clear and significant association between ME/CFS and the symptoms of autonomic dysfunction. Indeed, autonomic dysfunction occurred in three-quarters of the patients investigated, and a particularly strong association was seen with symptoms of orthostatic intolerance, suggesting that an abnormality of dynamic blood pressure regulation is particularly associated with fatigue severity in ME/CFS.

These are important findings because they confirm some previous scientific reports, and suggest that the assessment of autonomic function can be a robust, reproducible and objective diagnostic tool for identifying physical symptoms in a significant sub-population of people with ME/CFS. Read the full story on page 6. •
The ‘New Horizons’ conference on ME/CFS biomedical research — co-sponsored jointly by ME Research UK and the Irish ME Trust — was held at Edinburgh Conference Centre, part of the leafy campus of Heriot-Watt University, Edinburgh, on 25th May 2007. The aim of the day was to bring together researchers working towards understanding the biomedical basis of ME/CFS, and to raise awareness of the need for biomedical investigation. Many of the 130 attendees were biomedical researchers, some funded by ME Research UK, but there were also representatives from a variety of healthcare professions, and delegates from local ME/CFS support groups and from most ME/CFS charities in the UK. The full day’s program consisted of invited keynote lectures and shorter research presentations from scientists from Scotland, England, USA, Canada, Belgium, Spain and Japan.

Dr Vance Spence of ME Research UK chaired all the sessions, and the conference was opened by Alex Fergusson MSP, Presiding Officer (Speaker) of the Scottish Parliament, and former Chair of the Cross-Party Group on ME at the Parliament (pictured below). Alex spoke of his own family’s experience of ME/CFS, stressing the need for research to move beyond psychosocial aspects and towards the elucidation of the pathophysiology of the physical illness.

The first keynote lecture was given by Jonathan Kerr, from St George’s University of London, who is principal investigator of a research group on gene expression in ME/CFS (see pages 4 and 5 of this issue). The genetics theme was continued in the next keynote lecture by Estibaliz Olano, a senior scientist in Bilbao, Spain (pictured far right), who described her work on genetic profiles in fibromyalgia and ME/CFS, centering on distinguishing these two illnesses with somewhat overlapping symptoms, which are difficult to distinguish and diagnose properly. Using single nucleotide polymorphism (SNP) analysis, her group has identified 15 SNPs able to discriminate between the illnesses, and between milder and more severe cases in both illnesses.

Dr Akikazu Sakudo of Osaka University, Japan described his use of Vis–NIR spectroscopy to examine blood sera from ME/CFS patients and healthy donors. His results so far have been spectacular: using 45 patients and 54 donors, he has been able to correctly identify 100% healthy donors and 93.3% of the ME/CFS patients from masked serum samples, suggesting that he might have found a promising tool for the objective diagnosis of the illness.

The team from the University Department of Medicine, Dundee gave three presentations. The keynote lecture was given by Prof. Jill Belch, head of the Vascular Diseases Research Unit. Prof. Belch described the range of potentially important findings reported by her group.
in scientific papers from 2003 to 2007. These include increased oxidative stress, abnormally sensitive acetylcholine metabolism, and increased neutrophil apoptosis, consistent with an activated inflammatory process. While these tests are not yet diagnostic markers, they reveal biological anomalies that might well help to explain many of the clinical features associated with the illness. Her colleague Dr Faisel Khan described his experiments on arterial stiffness (greater in ME/CFS patients than controls), while Dr Gwen Kennedy discussed her current work (funded by ME Research UK, the Young ME Sufferers Trust and Search ME) on inflammatory markers in children with the illness. A keynote lecture of the day was by Dr Eleanor Stein from Calgary, Canada, who discussed behavioural interventions in ME/CFS, and gave suggestions for future directions. Dr Stein concluded that behavioural interventions can lead to an apparent short term subjective improvement, but they alone do not lead to measurable changes nor lasting symptomatic changes. Nor is there evidence that behavioural interventions address the pathophysiology of ME/CFS.

Presentations were also given by Dr Gregor Purdie (Dumfries and Galloway Health Board) on moves towards setting up a Scottish Clinical Network on ME/CFS, Joan Crawford (Liverpool Hope University) on attitudes of nurses towards people with the illness, Dr Les Wood (Glasgow Caledonian University) on motoneuronal excitability, Mark Robinson (University of Strathclyde) on a pilot study on interleukin-6 and exercise, and Rebecca Marshall (Glasgow Caledonian University) on a cross-sectional study of the pain experience in ME/CFS.

Order your copy of the 2-DVD set of the presentations by contacting our headquarters. The cost is £6 (which includes P&P) for the two-DVD set, and please make cheques payable to “ME Research UK”.

Recognising that much of the existing research into ME has concentrated on psychological interventions designed to “manage” the illness, ME Research UK believes that biomedical research is urgently required and is what most patients and carers want to see. For this, researchers with fresh, novel ideas have to be recruited and encouraged to undertake research in this field. This is the most difficult task of all, and ME Research UK sees its role at this leading edge: to give help to biomedical scientists for novel research projects that would otherwise not be funded, and to support research groups to the stage where they can apply to major funding agencies for further support based on their initial data.

With your help — and building on our close working relationships with other ME/CFS organisations around the world — ME Research UK can be a force for change, and a source of real hope for the thousands of people with this debilitating illness.
Gene expression is the way in which the information inherited from our parents (usually recorded as a gene, a sequence of DNA) is translated into a product, such as a protein or an RNA molecule, that can be used by the body. There are now a number of worldwide research groups investigating gene expression in people with ME/CFS, and over the past few years the number of published scientific reports in this field has been steadily increasing (see Table opposite). Some of these studies have not, unfortunately, confirmed their microarray results with real-time polymerase chain reaction (PCR), a flaw which makes interpretation of the results extremely difficult. However, when PCR-confirmed studies are examined, the genes identified in ME/CFS seem related to immunity and defence, supporting what is already known about the role of the immune system in the illness.

The group led by Dr Jonathan Kerr at St George’s hospital, University of London, has over the past five years made advances in defining the molecular basis of ME/CFS. Initially, they performed a pilot study of gene expression in patients compared with controls, and have demonstrated marked human gene dysregulation, principally affecting the immune system. After confirming their findings using a large microarray and real-time PCR, they undertook a pilot study looking for protein biomarkers and have identified several molecules which seem to be specific to ME/CFS. Protein biomarkers are important since they are the backbone of a diagnostic test, and the group is almost at the stage of announcing a “gene signature” for the illness. As regards a treatment for those people who are currently ill, Dr Kerr explains, “On the basis of the results of gene expression studies, funded by the CFS Research Foundation, a clinical trial of interferon-alpha is currently underway at St George’s University of London. We envisage that this will be the first of several clinical trials that are based on our gene expression findings, using the novel gene approach.”

Science moves methodically, however, and it is important to know whether the “signature” found in “sporadic” ME/CFS patients (see box ME/CFS: the Clinical Conundrum) is specific to this group, or can also be found in other groups of patients who share similar symptoms, and who, in fact, fulfill the diagnosis of ME/CFS. For this reason, ME Research UK (in conjunction with the Irish ME Trust) has just granted “seed-corn” funding for Dr Kerr’s group at St George’s University of London to perform a confirmatory study of the putative ME/CFS gene signature in a group of 25 patients who became ill after service in the Gulf War 1990–91, and who report the standard ME/CFS symptoms of fatigue, joint and muscle pain, cognitive complaints, sleep disturbances, and gastrointestinal problems. After full clinical assessment and characterisation of gene expression and protein analysis, it will be possible to tell if the gene signature is similar (suggesting a common pathogenesis) or different (suggesting an entirely different aetiology) to the illness known as ME/CFS.

Science moves step by step, and the journey can be long and complex. Experience from the use of genome-wide scanning technologies for cancer screening has shown that discovery and validation of biomarkers requires multiple phases of research over many years. Nevertheless, the work on gene expression is one of the most exciting recent developments in ME/CFS in the past decade, and could open the door to the development of pharmacological interventions. As Dr Russell Lane, a neurologist at Charing Cross Hospital in London, has said of the work on genes, if the researchers succeed and identify “clear physical changes in people with CFS, the lingering opinion that it is all in the mind could finally be laid to rest”. •
*How does it work?*

A key component of this gene research is the use of microarray technology to analyse the genetic material of a person with CFS. Researchers take a sample of blood or tissue, and apply it to a glass slide called a microarray which contains more than 20,000 gene identifiers. From these, the researchers are able to determine which genes in the sample are being “expressed”, that is, turned on or off, or turned up or down. This gene expression profile provides a window into the disease process. Since there can be tens of thousands of distinct probes on an array, expression levels for thousands of genes can be monitored simultaneously. Arrays have therefore dramatically accelerated many types of investigations.

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### ME/CFS: the clinical conundrum

As clinicians often point out, patients can have very different illnesses, yet share the same symptoms. So, “syndrome” diagnoses like ME/CFS that are based solely on a collection of vague symptoms shared with other conditions have a real problem. In formal terms, the problem is one of specificity since the diagnosis ME/CFS does not, in practice, completely exclude patients with other biomedical conditions or, indeed, those with a primary psychiatric disorder. In truth, the diagnostic label is no more than a “black box” at present, and the problem is made even worse by the failure of many doctors to examine patients properly before giving them the label and closing the lid. Who knows the secret of the magic black box, and who cares to look inside?

In the 1990s, work at the University of Dundee examined a large general group of patients, all fulfilling the criteria for ME/CFS: the groups comprised people who had developed ME/CFS-like symptoms sporadically (corresponding to “classic” ME/CFS patients); people with similar symptoms which began after military service in the 1991 Gulf War (a group also known as “veterans with CFS” in the USA); and people with ME/CFS-like symptoms which began after exposure to organophosphate insecticides. The three groups appeared very similar in regards to duration of illness and number of symptoms reported, showing that it was perfectly possible for the broad ME/CFS diagnosis to contain (and subsume) a number of possibly distinct groups of patients all with similar self-reported symptoms.

To know what’s inside the ME/CFS black box, we have to unpack it, and microarray technology offers a way of doing this. Illnesses are most easily accepted when they have a specific clinical or scientific “signature” — a biochemical test, a cluster of specific symptoms or signs, a cluster of activated genes, etc. — which confers legitimacy in the eyes of healthcare professionals. The discovery of such a signature specific for ME/CFS could transform the outlook for patients.

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### Gene expression studies in ME/CFS

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<th>Principal Institution</th>
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**Dr Jonathan Kerr**
Autonomic dysfunction is prevalent in ME/CFS

The autonomic nervous system controls cardiovascular, digestive and respiratory functions, as well as having a range of other important roles. When it goes wrong, the consequences can be severe; for instance, one of the main consequences is orthostatic intolerance, which is the inability to remain standing for long without suffering ill effects. Since one of the key difficulties that ME/CFS patients face is standing, especially standing still, without experiencing symptoms such as dizziness, altered vision, nausea and fatigue, it has been speculated that a thorough assessment of autonomic function might be a way to identify a specific, definable subset of patients, or might even be diagnostic if the underlying mechanisms could be understood.

Dr Julia Newton (pictured above between nurses Katharine Wilton and Jessie Pairman) of the School of Clinical Medical Sciences, University of Newcastle, has been investigating fatigue in people with the autoimmune liver disease primary biliary cirrhosis. In this group of patients, she has discovered that abnormalities of the autonomic nervous system contribute to their fatigue, which is itself related to low blood pressure and abnormalities of sleep. In addition, the fatigue in these patients is associated with excess mortality, which could also be linked with autonomic abnormalities. Could, she wondered, these abnormalities also be found in ME/CFS patients who experience many similar symptoms?

With a grant from ME Research UK, and the support of the regional ME/CFS service and ME North East, Julia has been testing a large group of people with ME/CFS using a well-validated battery of autonomic function tests. These test cardiovascular reflexes by assessing heart rate and blood pressure responses to a variety of manoeuvres. The intention is to examine 100 ME/CFS patients initially, and, depending on the findings, to monitor their progress over time using further tests. The cardiovascular laboratory in which the tests are being done is one of the largest autonomic testing labs in Europe, with all the necessary equipment and expertise for the testing that is being done.

Julia explains, “While there have been a few investigations of dysautonomia in ME/CFS patients in the past, they have been limited by the lack of sensitivity of the assessment methods used, and by the tendency to carry out small-scale observational studies with limited control groups. With our battery of well-validated, sophisticated tests, and our large and well-characterised patient group matched to normal controls, we hope to see how prevalent autonomic problems really are in people with ME/CFS, and whether they can be used to assist standard diagnosis.”

What do the results show?

A scientific paper reporting Dr Newton’s findings — her first on ME/CFS — has just been published in the Quarterly Journal of Medicine (August 2007). It reports on the group’s comprehensive assessment of symptoms of autonomic dysfunction in a large and well characterized group of ME/CFS patients, and essentially combines two distinct study phases in one report. Phase
(derivation) involved 40 ME/CFS patients and 40 age and sex-matched controls, and phase 2 (validation) attempted to replicate and confirm the phase 1 results in a mixed ME/CFS population of 30 patients, 37 normal controls and 60 patients with primary biliary cirrhosis (in whom there is a well-recognised association between autonomic dysfunction and fatigue). All were assessed using the Composite Autonomic Symptom Scale (COMPASS) which consists of 73 questions, grouped into domains relating to individual aspects of the autonomic nervous system, such as orthostatic intolerance (generalised adrenergic function), vasomotor function (peripheral adrenergic), gastrointestinal function, bladder and syncope. Importantly, in 15 representative ME/CFS patients, COMPASS scores were compared with an objective measurement of autonomic function (such as baroreflex sensitivity and heart rate variability, using continuous digital photoplethysmography) to test the validity of assessing autonomic function from patients’ reports.

The researchers found a clear and significant association between ME/CFS and the symptoms of autonomic dysfunction. In three-quarters of the patients, autonomic dysfunction was present, and it was found that a COMPASS score greater than 32.5 (defined in phase 1 and confirmed in phase 2) appears to be a robust, reproducible and objective diagnostic tool for identifying autonomic dysfunction. The researchers stress, however, that a minority of patients did not have elevated COMPASS scores, suggesting that they may have identified subgroups of CFS patients with potentially different origins for their illness.

A particularly strong association was seen between ME/CFS and symptoms of orthostatic intolerance (see the chart below), suggesting that an abnormality in dynamic blood pressure regulation is particularly associated with fatigue severity in ME/CFS, and confirming the conclusions of a previous review by Spence and Stewart (Biologist 2004, available from the ME Research UK website).

Two key questions arise, however. First, is the apparent autonomic dysfunction in ME/CFS a part of the illness or a consequence of being ill, such as deconditioning? Well, the researchers say that if deconditioning was involved, a more prolonged experience of fatigue should be associated with increased autonomic dysfunction, and this was not the case. Second, is there a recognised treatment for dysautonomia? Again, the researchers say that treatments for dysautonomia used in previous small studies have proved disappointing, and it is therefore likely that treatment of orthostatic intolerance in ME/CFS will not be possible until the mechanisms underlying the problem are unravelled and quantified.

The researchers confirmatory study of gene expression in peripheral blood of patients with Gulf War Syndrome
Dr Jonathan Kerr, St George’s University of London

Focal and global endothelial function and their association with arterial stiffness
Dr Faisel Khan, Prof. Jill Belch, Prof. Chim Lang, University of Dundee

Post-exertional malaise in ME/CFS: the role of intracellular immunity and sensory processing
Dr Jo Nijs, University College Antwerp

An investigation into biochemical and blood flow aspects of ME/CFS in children
Dr Gwen Kennedy, University of Dundee

Physiological cost of walking at self selected and matched speeds in ME/CFS
Dr Lorna Paul, Glasgow Caledonian University

Our primary aim is to fund high quality projects to investigate the causes, mechanisms and symptoms of ME/CFS, and ultimately to develop effective treatments. At present, we fund the work of a growing number of scientists. Some of these are listed below, while others are awaiting announcement or going through our in-house assessment procedure.

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Dr Lorna Paul, Glasgow Caledonian University
Acetyl L-carnitine treatment

Researchers in Catania, Italy (Archives of Gerontology and Geriatrics 2007) supplied acetyl L-carnitine (ALC) to 96 elderly people (older than 70 years) meeting more than four of the Holmes major criteria or at least six of Fukuda minor criteria for CFS. ALC is an amino acid that can be purchased as an individual supplement, and is thought to have a range of effects, including facilitating the uptake of acetyl-coenzyme-A into the mitochondria.

Subjects were randomised into two groups (48 ALC, 48 placebo), and were given ALC as tablets by mouth, 2 g twice-a-day, for 180 days. By the end of the treatment, significant differences between the two groups were found for physical and mental fatigue (both p<0.001), muscle pain (decrease of 27% versus 3%, p<0.02), prolonged fatigue after exercise (p<0.0001), sleep disorders (p<0.05), fatigue severity scale (p<0.0001), functional status (p<0.0001), and mini mental state examination improvements (3.4 versus 0.5, p<0.0001).

Given that there have been reports of the usefulness of ALC in other conditions, such as cognitive impairment or mild Alzheimer’s, these findings are very interesting, though ALC alone is unlikely to be curative, and these results need to be reproduced by other groups in other settings.

Recent research from

CBT on the rack

A cloud of dust has been raised by an essay in the March 2007 issue of the British Association for Behavioural and Cognitive Psychotherapies magazine, in which Professor David Richards addresses “what’s wrong with cognitive behavioural therapy (CBT)” His major point is that the CBT establishment has become “arrogant, inflexible, remote”. But it is his critique of the rickety evidence-base for CBT that sounds familiar: “It is hardly surprising that our detractors are suspicious. They are right to accuse us of a selective use of the evidence, our prized and cherished weapon of choice. They have many other objections: we ourselves write the research questions which now get funded… most CBT trials are small and poorly executed; quality thresholds for RCTs in NICE guidelines are notoriously low, allowing the results of meta-analyses of small poor quality studies to direct policy.”

Prof. Richards’ criticisms match exactly ME Research UK’s, most recently about the draft NICE guidelines of 2006 which flagged up as “treatments” for ME/CFS non-curative psychosocial strategies, like CBT, that at best are adjunctive and symptom-managing. He could also have quoted the famous BMJ editorial of 2002: “Many patients… report that thinking differently does not make their disease go away… Rigorous biochemical research… would be preferable in their eyes to promulgating an… incomplete paradigm as though it were a cure.”

Sudden illness onset and neurocognition

There is an ongoing debate about the meaning of the ever-expanding literature on neuropsychological deficits (such as impairment of short-term memory, information processing and word retrieval) in people with ME/CFS.

Researchers at the University of Hawaii (Neuropsychology 2007) decided to overcome some of the limitations of earlier studies with a co-twin control study of 22 pairs of monozygotic twins (one with and one without the illness) in which the effects of comorbid depression and mode of illness onset were taken into account. The results showed that twin groups had similar intellectual and visual memory functioning, but twins with ME/CFS exhibited significant decreases in motor functions, speed of information processing, verbal memory and executive functioning. Major depression did not affect neuropsychological functioning among ill twins, but twins with sudden illness onset demonstrated slowed information processing compared with those with gradual onset (p<0.01).

An association between sudden illness onset and slower information processing has been reported before, and the authors speculate that this may reflect an infectious trigger and involvement of the central nervous system. Furthermore, the results point to the need to subgroup patients by mode of onset (sudden or gradual) in future investigations.
Vaccinations and ME/CFS?

One of the models proposed to explain ME/CFS is that an initial infection by an unknown agent leads to a “chronic immune activation” clinically expressed as the symptoms characteristic of ME. Since vaccinations are used to stimulate the immune system, it has been suggested that vaccinations might induce the aberrant immune response in ME patients. Indeed, support for this possibility came in 2000 from a cross-sectional study of the vaccination records of 923 UK Gulf War veterans, which found an association between multiple vaccinations given during the conflict and later evolution of Gulf War Syndrome, a condition symptomatically similar to ME/CFS.

A recent review from Tel-Aviv University, Israel (Appel et al, Autoimmunity 2007), however, could find no good “published” evidence that vaccinations are associated with the illness, citing negative studies in 1992, 2003 and 2006, mainly on the aftermath of the hepatitis B vaccine. However, the review took no account of the 2002 study by De Becker (published in the relatively inaccessible Journal of Chronic Fatigue Syndrome) which — from data on over 1,500 CFS patients — found an association between multiple vaccinations given during the conflict and later evolution of Gulf War Syndrome, a condition symptomatically similar to ME/CFS.

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Our Friends scheme provides the core support needed for our work to continue. There are three categories: Individual Friends, Corporate Friends and ME Group Friends, all sharing our aim of a biomedical breakthrough in ME/CFS, and representing many thousands of patients and carers across the globe.

Individual Friends can give their support in a variety of ways, such as fundraising, regular donation by standing order, taking a collection box, or by just spreading the word — word-of-mouth is one of the most efficient ways of getting our work known.

The Group Friends scheme is for local ME support groups, and there are currently 25 groups informally signed-up. The Groups range from Castleford to Solihull & Birmingham, and from Aberdeen to Warwickshire, and the full list can be found on the Friends of MERUK section of our website.

Corporate Friends is designed for larger independent organisations — corporations, larger registered charities, companies, businesses — that share our aims, and the scheme brings collective power to the drive to energise ME research.

With the help of all our Friends, we can continue to be a force for change, and a source of real hope for the thousands of people with this debilitating illness.

Sunday 25th March 2007 was the day of the Bath Half Marathon in which Tom Whittingham and his friend Paul Lannon were running for our research programme. They did marvellously well, coming in at 104 and 114 minutes, respectively, cheered along by all the support they’d received from individual sponsors and through their Justgiving websites.

Tom’s sister, Naomi, has had ME for over 17 years, and it has had a great impact on the life of the family. As he says, “ME has completely taken over Naomi’s life; she was a happy 13 year-old, whose life was literally taken away from her. She is housebound and disabled, and relies completely on full time care from my mother.”

The photo shows Paul and Tom after the race, and, after matching company sponsorship was taken into account, the total raised was £2,300, a spectacular result for which we send our grateful thanks!

The medieval Persian poet Saadi described gardens as a “a delight to the eye and a solace for the soul”. And so it was for the summer garden party which the family of Alex Milopoulos’s boyfriend held in their garden in May 2007 in aid of both the 25% ME Group (which supports housebound and bedbound people with the illness) and ME Research UK.

Alex has suffered from ME for nearly nine years with three periods of severe relapse, so the family have seen at first hand how serious this neurological illness is, and they wanted to raise money for support and research. Visitors enjoyed tea and cake in the beautiful, picturesque garden, and as Alex said, “We sold plants, bric-a-brac, handmade cards, held a raffle, and there was a human fruit machine at which people could try their luck.”

Alex was delighted that her health was good enough to be able to attend (which would have been completely impossible at the same time last year), and a big thank you must go to Kathy and Ken, and everyone who made the day such a success.
**A MESSAGE FROM OUR PATRONS**

“ME is a substantial medical and social problem, yet relatively little research has been conducted into its causes and consequences.

Roger Jefcoate, CBE

“A recent report to the Chief Medical Officer said that a programme of research on all aspects of the illness is urgently needed, and that improvement of health and social care is an urgent challenge.

The Countess of Mar

“Given the recent sea change in the public perception of ME, and the possibility that ME patients will now be encouraged and supported rather than derided and scorned, we hope that ME Research UK’s scientific and policy research will lead the way towards a treatment and cure for people with ME. Please help us to make a real difference to the lives of people with ME.”

Roger Jefcoate, CBE

**The Great Glen Way**

Catriona and Jim Marshall and a group of friends went walking the Great Glen Way in May 2007, and had a rare old time yomping the 73-mile trek across heather, moorland and mountain sides.

Catriona’s nephew, Andrew, has had ME for some time, and as she explains, “Andrew is a fine young man of 29 who was full of zest for life, but developed ME two years ago and at present is more or less bedbound. In the longer term, the only answer to this problem is biomedical research — hence our support for ME Research UK through this walk.”

In total, Catriona and Jim raised £3,650, and the photo shows them (on either side of the cheque) making the presentation to Dr Vance Spence, Bob and Betty McRae, and Dr Neil Abbot during an interval at our New Horizons Biomedical Conference at Heriot Watt University on 25th May 2007.

**Laura’s marathon**

On Sunday 20th May 2007, Laura Duerden did something remarkable: she completed the Great Manchester Run in 1 hour, 15 minutes, 3 seconds! “In January, I literally couldn’t run to the end of the road,” she explains, “so training as been very hard indeed — four times a week — but in the end the adrenalin pulled me along on the day!”

Laura’s best friend Amy has been ill with ME for some time, but for the past 18 months she has been exceptionally poorly and has been cared for by her wonderful parents. As Laura lives hundreds of miles away, she doesn’t get to see her anywhere near as much as she would like to, hence the decision to raise money for ME research as a way of doing something to help.

Laura raised over £1,300, which shows, she explains, how much Amy means to so many people. The photo on the left shows Laura in her custom-made ME Research UK t-shirt (showing pictures of Amy and herself), and running partners Helen and Jen (in white vest).
To allow us to press ahead with our mission to Energise ME Research, please consider responding to our Standing Order appeal.

ME Research UK receives no public money and relies entirely on donations from ordinary people. It is vitally important that all our supporters understand that we are one of the very few charities in the world funding biomedical research into ME/CFS, and raising awareness of the issues in a truly professional manner.

Help us to make the breakthrough that patients need and deserve by completing the standing order form on this page, or by donating through the online giving facility via our website.

Please send this form to:

**ME Research UK**  
The Gateway  
North Methven Street  
Perth PH1 5PP, UK  

Tel: 01738 451234  
Email: meruk@pkavs.org.uk  
www.meresearch.org.uk

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For office use only:

Clydesdale Bank  
23 South Methven Street, Perth (82-67-09) for the credit of ME Research UK, a/c no. 50419466

Bank reference number:

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<td><strong>6</strong> If you are a UK taxpayer, under the Government’s Gift Aid scheme ME Research UK can reclaim the tax you have already paid on your gift. This means that your donation can increase by nearly a third at no extra cost to you. It doesn’t matter what tax rate you pay as long as you pay an amount of income or capital gains tax equal to the tax we reclaim on your donations in that financial year. Please inform us of changes in your tax status. Please indicate below if you would like ME Research UK to reclaim the tax on your gift.</td>
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Thank you for your support.