

## Topic index

*[With thanks to Dr. Vallings for giving permission to post her summary on this website. Ed.]*

[Virology research](#)  
[Treatment advances](#)  
[Fibromyalgia \(FM\)](#)  
[Case definitions](#)  
[Exercise challenge](#)  
[Immunology](#)  
[Paediatrics](#)  
[Epidemiology](#)  
[Genomics and genetics](#)  
[Brain and neuro-endocrine functioning](#)  
[Conference summary](#)

Read the [abstracts of all papers](#) presented at the conference.

## Summary of IACFS/ME conference, September 22-25,2011, Ottawa, Canada

*by Rosamund Vallings MB BS*

The conference opened with the keynote speaker, **Christine Kozak** (Bethesda,USA), who discussed and clarified issues relating to “Gammaretroviruses of mice and their links to Prostate cancer and CFS/ME”. She described how MLVs cause leukaemias in mice, and penetrate right into the cell nucleus, and can be transmitted genomically. There are 3 categories:ectotropic, xenotropic and polytropic. The latter two are distributed widely in house mice species. Xenotropic viruses have a wide range of hosts, and there are 5 functional variants of receptors. XMRV can replicate in some species of mice. Various host factors restrict XMRVs in mice, such as serum factors, receptor block, receptor variation, Apobec 3 and Fv1 (both of which can block viral replication at the reverse transcriptase stage) and tetherin at the budding stage. Humans do not have Fv1, but have TRIM5 $\alpha$  retrovirus restriction factor. The conclusion was that MLVs and ERVs are found in house-mice worldwide. Receptor variants have evolved in mice carrying XMRVs. XMRV has a distinct host range. XMRV contributes to induced neoplastic diseases. Multiple host restriction factors limit virus transmission. Contamination may be the reason we see this virus in humans.

## Virology research

**G.Simmons** (San Francisco,USA) discussed multi-laboratory evaluations of XMRV detection assays. He presented work investigating the prevalence of XMRV in blood donors. Published work on 22/9/11 in Science Express showed a failure to detect XMRV in any sample. Only one lab found clinical samples to be nucleic acid test (NAT) positive. These positives were not reliable among replicates. Using a number of techniques, there was very little correlation between positives from the original WPI study and other assays. The conclusion was that routine screening of blood donors for XMRV is not warranted.

**K.de Meirleir** (Brussels, Belgium) detected anti-XMRV antibodies in the serum of patients and healthy blood donors. Of 84 Belgian CFS patients, 21 had developed CFS after receiving a blood transfusion. Controls were 44 healthy blood donors. 57% of CFS patients and 16% of controls tested antibody positive. 10 of the 21 CFS patients who had had a blood transfusion tested positive. PCR was not used. Western blot was used to confirm the serology data. These results were statistically significant. Samples were blinded and analysed at the WPI.

**M.Hansen** (NY,USA) presented work looking for MLV-like gag sequences in blood and cell lines incubated with plasma from CFS patients and controls. 30 were patients from D.Bell, 24 from S.Levine and 12 controls from Ithaca, NY. No XMRV was detected in the Bell group. Cells were cultured for 30 days – some gag sequences were found but no other retroviral sequences could be found. There were no statistical differences between patients and controls. Everything possible was done to avoid contamination. The Levine samples were all negative for gag sequences. This research is ongoing.

Post-SARS Syndrome was outlined by **H.Moldofsky** (Toronto, Canada). SARS results from infection by a coronavirus A. It creeps into the brain via the olfactory bulb in mice, and possibly via this route in humans. 250 cases occurred in Toronto, transmitted by one person who had been in Hong Kong. There were 44 deaths and 50 cases who remained ill post-SARS. These correlated with a diagnosis of CFS. Sleep was disordered and this was similar to that seen in fibromyalgia syndrome (FMS), but there was a lower rating of the alpha EEG sleep anomaly in post-SARS as compared to FMS. The myalgia was also less severe.

**J.Montoya** (Stanford,USA) considered the role of the immune response in CFS. Typical pathogens in CFS are involved, and are mostly intracellular. There is initial tropism (e.g. respiratory or GI) followed by involvement of target organs (eg lymphatics)). Different pathogens

take similar pathways. CFS may be sustained for years, and pathogens reactivate periodically. Reactivation tends to be at low levels. This leads to an immune response, but this is not strong enough to kill the organisms, so the bugs remain latent, and then reactivate again leading to symptoms. CFS is a multi-system disease with phases of immune response.

Peripheral blood studies are useful and convenient but imperfect. Immune abnormalities have been inconsistent across labs, although some are consistent. Inconsistency may be due to host variables, multiple triggers, fluctuating nature of the disease, duration and severity. There are also other non-CFS variables such as methodological variables and statistical issues. There is a need to involve all available data, apply new technology and coordinate research.

**J.Mikovits** (Reno,USA) and **J.Coffin** (Boston,USA) discussed the case for and against human gammaretroviruses in CFS. Mikovits outlined her earlier work involving detection of XMRV. As well as identifying the virus in a significant number of patients, their lab have identified an inflammatory cytokine and chemokine signature that distinguishes XMRV-infected patients from controls with 94% sensitivity and specificity. Further tests are being developed for detection and characterisation of XMRV. Coffin's lab had looked hard for XMRV in mice, and did not find it in any mouse strain tested, but found an XMRV ancestor in the mouse genome. A detection assay has been developed. He pointed out that XMRV is a virus and MLV is not a virus, but fragments.

Mice are extremely widespread, and mouse DNA can be found on laboratory surfaces and can contaminate common reagents and materials. Most virologists now consider XMRV to be a consequence of a collection of artefacts originating from endogenous MLVs prevalent in the laboratory. It is likely to be an accidental laboratory creation from the 1990s. It is yet to be worked out how it has got into clinical samples from CFS patients.

[Back to top](#)

## Treatment advances

**D.Strayer** (Philadelphia, USA) gave an update on the use of Rintatolimod (previously known as Ampligen). They had studied the use of the drug in XMRV/pMRV antibody positive patients. Of the patients selected, 33.7% were antibody positive. The antibody negative group had lower activity for daily living scores. Those who were antibody-positive showed a significantly greater increase in exercise treadmill tolerance when treated with the drug than those treated with

placebo and those who were antibody negative. The responding patients also showed a decrease in use of other medication.

The use of Rifampicin was found to augment the effects of oxymatrine (Equilibrant) in ME/CFS patients by **J.Chia** (Torrance,USA). Those with chronic enterovirus infection had previously been shown to benefit from oxymatrine (Equilibrant). 46 ME/CFS patients were treated with Rifampicin 300mg bd for 7 days while taking oxymatrine and compared with patients taking just oxymatrine, and a control group. Initially flu-like symptoms occurred in those taking the rifampicin plus oxymatrine, but subsequent symptomatic improvement was observed in 60%. Short courses of rifampicin may therefore be beneficial in oxymatrine responders. Rifampicin induces nitric oxide from human alveolar macrophages causing the initial flu-like symptoms. 2nd or longer courses of rifampicin did not appear to help.

**F.Friedberg** (Stonybrook,NY) tested a brief self-management protocol for unexplained chronic fatigue and ME/CFS in primary care. Two self-management sessions focussing on CBT were undertaken in 3 study conditions: 1) standard medical care alone, 2) standard medical care plus nurse-delivered attention control condition of symptom monitoring and 3) standard medical care plus nurse-delivered self-management CBT. There was modest improvement in fatigue severity and patient global impression of change (PGIC) ratings in the self-management programme. Ratings tended to reflect different attitudes to the illness and/or differential exposures to negative major life events. Improved patients reported increased awareness of behaviour and affirmative steps to pursue more healthy activities. Self-management can generate improved outcomes.

[Back to top](#)

## Fibromyalgia (FM)

A lively debate followed between **R.Staud** (Florida,USA) and **D.Claauw** (Ann Arbor,USA) entitled: "Are tender points necessary?" Staud outlined the American College of Rheumatology criteria for FM (1990) which includes widespread body pain of 3 months duration and presence of 11 out of 18 tender points. The tenderness should be with 4kg of thumb pressure. In trials this has not been found to be reliable and a more accurate diagnosis can be made using the 2010 provisional FM criteria: 3/12 duration of pain with a widespread pain index in 19 areas with a severity scale of at least 9. This scoring system is quite different, and additional symptoms include fatigue, unrefreshing sleep, cognitive symptoms and somatic symptoms. (There is overlap with CFS). Many different ways have been looked at for triggering pain for measurement. Emotional "windup" could be useful, but is not reliable. Tonic heat and

mechanical stimulation can be applied to painful and non-painful areas. This can be used for assessment of pain or for stimulating pain. Tenderness does correlate with pain and can be measured by quantitative measurement of pain sensation (QST). For clinical purposes, tender points provide little mechanistic information about an individual's pain and associated symptoms.

Clauw feels that tender points in diagnosis are unnecessary, and outlined 10 reasons why:

1. Convey inappropriate message about FM
2. Excludes males
3. Practitioners do not know how to do it, and often do not want to learn
4. Very few chronic pain states have a specific examination to diagnose pain
5. Tender points are an inadequate measure to assess experimental pain threshold
6. There are better ways to assess pain threshold
7. Tender points are not normally distributed
8. Tender point count was never meant to be a "physical exam" and should not replace routine clinical examination
9. No evidence that they are necessary in diagnosis
10. Is the horse dead yet?!!

[Back to top](#)

## Case definitions

**B.Carruthers** (Canada) outlined the New International Criteria for ME. The 2003 Canadian definition has been further defined. The 6 month period is no longer required, but left to clinical judgement at 3 months. Post-Exertional Neuro-immune Exhaustion (PENE) was kept criterial and further articulated. Modifications for paediatric cases have been included. The illness is called ME rather than CFS. The Canadian definition clearly separated out genuine cases, and this new definition can be used clinically and in research and epidemiology.

**L.Jason** presented work contrasting case definitions. The Fukuda definition has only 4 core symptoms, which do not include post-exertional malaise. But this definition has been used by researchers for over 15 years. He compared this definition with the 2003 ME/CFS Canadian criteria and the older Dowsett ME criteria. His study suggests that the more recent criteria and the ME criteria could be used to identify patients with more homogenous and severe symptomatology and functional impairment.

His second paper describes Data Mining as being a useful tool in aiding the diagnosis of ME/CFS. An objective computer-driven decision is combined with a physician's medically influenced decision. The Canadian criteria (compared to the Reeves 2005 criteria) were found to have more construct validity and were more accurate, identifying 87% of cases. Post-exertional malaise, neurocognitive symptoms and sleep disorders were not identified as discriminating symptoms with the Reeves criteria.

**E.Unger** (CDC Atlanta, USA) continued discussions about case definitions. She explained that definitions are not specific to CFS. They are used for epidemiological studies, clinical diagnosis and to determine the biological basis of disease. She stressed the importance of standardization. She asked the question "Will refining lead to a homogenous population?", and pointed out that heterogeneity is challenging. Phenotypes are imperfect indicators of biology. Case definitions may not be sufficient to discover pathways to pathogenesis. Standardized measures will allow stratification and subtyping.

[Back to top](#)

## Exercise challenge

**B.Keller** (New York, USA) studied the effects of fatigue on functional capacity in patients with CFS. There is a need to quantify impairment. The cardiopulmonary exercise test (CPET) measures functional impairment and is an objective measure of energy expenditure and physical work. It is validated and reliable in health and disease, but she questioned whether it was useful in CFS. The purpose of the study was to measure the effects of post-exertional malaise in CFS. She concluded that patients' disability went from mild to moderate on first test and from moderate to severe on second 24 hours later. Looking at maximum exercise tests, it was shown that those with CFS will exacerbate symptoms associated with post-exertional malaise simply by completing normal daily activities.

**C.Snell** (Stockton, USA) then covered the importance of exercise challenge, and the need for a standardised measured approach in diagnosis and management. He described fatigue as a reduced efficiency as a result of doing "work". Some measures can be indirect (e.g. heart rate) or direct (e.g. gas exchange). Field tests are easy to administer and require minimal equipment but are unmonitored and therefore less likely to be accurate. Motivation and pacing both play a big role in the results. He discussed the PACE trial and showed that the results actually equate to 1.94 – 2.35 mph and at 2 METS this equates to 7ml/min/Kg oxygen. The NY Heart

Association would classify this as “severely disabled”. Anything greater than 3mph is the anaerobic threshold for most CFS patients. Direct assessment of aerobic capacity should be the gold standard. CPET is uniquely able to quantify efficiency with measures of workload and the metabolic cost of the work. Healthy people do better on a second test, but in CFS there is a massive drop. Post-exertional malaise (PEM) is an exacerbation of symptoms after exertion. Most healthy people will recover in 48 hours, but in the group studied, only one patient with CFS recovered in 48 hours. The respiratory exchange rate (RER) is the most reliable gauge of subject effort – it encompasses an analysis of expired gases.

[Back to top](#)

## Immunology

Natural killer cell (NK) function in a prospective cohort of adolescents with CFS compared to controls following infectious mononucleosis (IM) was discussed by **B.Katz** (Chicago,USA). He felt the study was important because NK function has been much studied and results are not always consistent. 9 with CFS and 9 matched controls were studied. Blood was taken at 6, 12 and 24 months following IM. NK quantification and function was measured. There was no difference in NK cell numbers at each of the 3 points of analysis compared to controls. However NK cell function was higher in cases than controls at 6 months, with less differential at 12 and 24 months. The conclusion was that there was no decrease in NK cell function in this group.

However, **E.Brenu** (Gold Coast,Australia) looked at cytotoxic function of NK cells and CD8+T cells in CFS, and her findings showed significant decreases in cytotoxic activity compared to controls at baseline, at 6 and 12 months. NK CD56 bright cells remained decreased in those with CFS. The study confirmed reduced immune function in CFS, and she highlighted the possibility that NK cell cytotoxic function could be a potential biomarker.

Her second study assessed proteins and receptors secreted and expressed by CD4+T lymphocytes over time. At baseline IL-10, TNF $\alpha$  and IFN $\gamma$  were increased in the CFS group. At 6 months, IL-2 was increased and IL-10 and IL-17a were significantly decreased in the CFS group, and at 12 months only IL-2 was significantly increased in the CFS group. The results suggest that the cytokine profile in CFS changes over time during disease progression. Experimental findings need to be matched with data on clinical disease progression.

The objectives of a study presented by **V.Falkenberg** (Atlanta, USA) were to determine the

pattern of perforin gene methylation in conjunction with gene expression, and whether these features were altered in CFS. Increased promoter DNA methylation correlated with reduced perforin expression in the non-fatigued group, the relationship was not seen in CFS. Small but significant differences in methylation were detected over the day and there were differences between both groups. Further studies are needed to help explain and understand these differences.

**N.Klimas** (chairing this session) pointed out the importance of these papers in helping us understand the nature of the immune response and the variations over time.

[Back to top](#)

## Paediatrics

**G.Broderick** (Edmonton, Canada) looked at the links between lymphocyte metabolites and the clinical course of post-infectious fatigue in a group of adolescents following infectious mononucleosis (IM). They were followed over 2 years and 3 clinical courses were distinguished. 1) sustained increase in fatigue after early partial remission 2) a monotonic decrease in fatigue and 3) slow decrease in fatigue after a peak at 12 months. They surveyed lymphocyte gene expression. 107 genes were differentially expressed. 40% were linked to immune metabolism and 20% to immune signalling and cell functioning. Gene expression supports directed functional interaction. Processes are linked to the biochemistry of the stress response. Phenylalanine metabolic activity supported the separation of the fatigue sub-groups. High activity was linked to a more favourable prognosis. Results correlated with the clinical course over 2 years.

**T.Miike** (Hyogo, Japan) presented a fascinating overview of the daily life of children in Japan, with emphasis on their vulnerability for developing CFS. These children are subject to sleep deprivation as a result of modern daily life in Japan, and developed abnormal sleep rhythms. He discussed the importance of reducing risk of developing CFS by attention to children's daily life and lifestyle in Japan.

**S.Tajema** (Kobe, Japan) confirmed the relationship in Japan between the abnormal biological clock system and childhood chronic fatigue. At their newly-formed centre, they are now treating this disorder. In the study, the treatment of children with CFS with bright light therapy, thermal therapy (20 minutes of 60C to the head), medication (melatonin, clonidine and sedative



psychotropics), CBT and lifestyle training over 8 weeks was presented. Circadian rhythm and sleep disorders were much improved, but other symptoms of CFS were not significantly improved at this time. Recovery from the sleep disturbances is looked on as the first stage of improvement for these patients.

**K.Rowe** (Melbourne,Australia) had seen 788 paediatric patients (aged 6-18) between 1991 and 2009, and she presented follow-up to look at the natural history of the illness. The average duration of the illness was 5 years, with a range of 1-15 years. By 5 years, 60% reported recovery. By 12 years 88% reported recovery, but in approximately 1/3 of these they reported conscious monitoring of their workload. Less than 5% were not working or studying, often due to factors other than CFS, such as marrying or having children. 90% completed or intended to complete post-secondary training. Treatments used were studied and the only alternative practitioners who were deemed helpful were those providing relief of muscle pain with massage or who provided good dietary advice. Restrictive diets and supplements did not reach placebo levels of response. The important issues were balancing life to include social contact, physical activity, educational input and a commitment to attend at least one activity each week. Ability to engage in education was the best predictor of functional outcome. She concluded that the outcomes for young people in Australia with this illness are generally positive although prolonged.

[Back to top](#)

## Epidemiology

**L.Jason** (Chicago,USA) had looked at the natural history of the illness over 10 years. His study's major finding was that rates of CFS appear to have been relatively stable over the past decade. 67% of those with CFS continued to have CFS over time. Some of those initially diagnosed with Idiopathic Chronic Fatigue (ICF) had progressed to CFS, suggesting that ICF is a group at higher risk of developing CFS. Of those in remission, 50% went from a diagnosis of CFS to ICF, indicating that while they no longer fitted the CFS criteria, they did still suffer from fatigue. Post-exertional malaise is the cardinal symptom. Of interest 29.4% of the CFS patients had had a blood transfusion.

CFS knowledge and illness management among US healthcare providers was reviewed by **E.U nger**

(Atlanta,USA). When looking at results for health practitioners, 94% of doctors had heard of CFS, 71% believed it was a medical and psychological illness, 14% believed it was a psychiatric illness, 37% had made a diagnosis. Studies of public knowledge indicated that 57% had heard

of CFS, 27% considered it a medical condition and 2% believed it was a psychological illness. Nearly 10% of the public knew of someone with CFS. The top 3 ways in which health care providers manage CFS were: referral to a medical specialist (35%), medication (29%), and referral to a psychologist/prescribing graded exercise therapy (26%). The public sought information by talking to family doctor (72%), searching the internet (54%) and talking to a medical specialist (25%). Only 7% would join a support group.

**J.Allegre** (Barcelona,Spain) presented results of a study to determine the sociodemographic, clinical and therapeutic characteristics of CFS patients in Spain. The condition was found to affect mainly middle-aged, educated women. Onset most often occurs following an identifiable trigger, such as infection, delivery or stress, and was sudden in 20%. At the time of diagnosis 62.5% were not working. Treatments were: symptomatic medication (analgesics, antidepressants, anxiolytics) in 78.3%, alternative treatments in 3% and physical exercise and/or CBT in 5%.

[Back to top](#)

## Genomics and genetics

Expression patterns of genes relevant to immune function were discussed by **E. Brenu** (Gold Coast, Australia). Her study confirmed changes in microRNA expression in cytotoxic cells that may be related to the poor function of these cells in CFS patients.

**M.Rajeevan** (Atlanta,USA) had looked at the immune and inflammatory alterations in CFS to determine if genetic variants in inflammation and immune pathways could be linked to CFS, as well as to quantitative measures of functional impairment, fatigue and symptom inventory. Compared to controls, CFS was associated with 34 functionally relevant single nucleotide polymorphisms (SNPs). 12 of these are in pathways related to complement cascade, chemokines and cytokines/cytokine signalling and Toll-like receptor signalling. Differences in these associations found for subjects with exclusionary conditions otherwise meeting criteria for CFS, suggests important differences between these groups.

**L.Bateman** (Salt lake City,USA) presented work to determine whether baseline and/or post-exercise expression of genes involved in signalling and modulating sensory fatigue and muscle pain are potential biomarkers for distinguishing those with CFS and FM from healthy controls. At least 2 sub-groups of patients were identified by gene expression following

exercise. The larger subgroup showed increases in mRNA for sensory ion channels and adrenergic receptors and a cytokine. Symptom severity was associated with greater post-exercise increases in these genes. The smaller subgroup were mainly patients with orthostatic intolerance and there was no post-exercise increase in any gene, and was defined by decreases in mRNA for  $\alpha 2A$  adrenergic receptor. The FM only patients were identified by baseline increases in 3 genes. Post-exercise increase in 4 genes distinguished CFS from controls, and could be an objective biomarker for CFS. Diagnosis based on gene expression may eventually be possible.

Following work with Gulf War veterans, **L.Steele** (Waco,USA) investigated, with a small sample whether exposure to neurotoxicants are risk factors for developing Gulf War Illness (GWI). Some troops who were exposed however did not develop illness, so genetic differences may have been implicated. Findings are supportive that GWI may be associated with the PON1 genotype. PON1 is a detoxifying enzyme. GW veterans whose PON1 genotype is known to provide slower hydrolysis of some organophosphate pesticides are at greater risk of GWI in relation to reported use of pesticides and prolonged use of pyridostigmine. And GW veterans who carry the R allele PON1192, which is known to provide inefficient hydrolysis of sarin were at increased risk of GWI if they had heard chemical alarms, which indicated potential exposure.

**L.Garcia** (Miami,USA) compared gene expression patterns in CFS and GWI. Study was based on Jonathan Kerr's work which had identified 79 genes associated with CFS with defined subgroups. Her group used gene activation patterns in CFS and GWI during and after exercise challenge to better understand the mediators of persistence and relapse. Kerr's earlier findings were confirmed in the CFS group. There were significant differences when compared to controls. There were important overlaps with GWI. EB12 (an EBV induced gene) was 6-fold higher in CFS than in controls, and 2-fold higher in GWI. ETS1 was upregulated in both groups. Transcription factor 3 was markedly elevated in GWI and less so in CFS, though was significant. Apoptosis genes were markedly elevated in both groups though 400 fold higher in GWI. The overall trend however was that most of the gene regulation activities associated with CFS were not significantly different between GWI and controls. Additional genes specific to GWI have however been identified by the group. With exercise challenge, there were changes in genes at peak of exercise which were unique to CFS. This was accompanied by altered immune signalling pathways.

[Back to top](#)

## Brain and neuro-endocrine functioning

**I.Treasaden** (London,UK) looked at volumetric changes in regional grey and white matter in CFS using Voxel 3D MRI techniques. He compared 26 CFS patients with controls. In the CFS patients there was reduced grey matter in the occipital lobes (associated with visual processing), in the right angular gyrus (involved in perceptual sequence learning, conscious awareness of actions) and in the posterior division of the left parahippocampalgyrus (associated with memory function and retrieval). There was also reduction of white matter in the left occipital lobe. This data helps to confirm a neurological diagnosis, and needs to be correlated with biochemical changes.

That there is evidence for reduced aldosterone in those with CFS was presented by **R.Boneva** (Atlanta,USA). Many of the symptoms in some CFS patients do overlap with those of Addison's disease. These include orthostatic intolerance, orthostatic tachycardia and heat/cold intolerance. Maintenance of blood volume may be poor and this is dependent on regulation by aldosterone and mineralocorticoid receptors in the brain. In this study of 70 CFS patients and 212 controls, those with CFS had comparatively lower aldosterone levels. A previous study (Wichita) had reported higher plasma renin levels. Further studies should measure aldosterone response to salt restriction and postural changes.

**A.Miller** (Atlanta,USA) presented 2 papers. The first was on behalf of J.Jones (Atlanta,USA) using functional MRI (fMRI). It has been suggested that CFS symptoms may be linked to altered cognitive or pre-cognitive processing in the CNS. This study investigated the sense of "self" and "illness-related semantic information". The focus was on the right anterior insula, an area associated with awareness and self-related processing. Conclusions were that there is a real alteration of body physiology in CFS. The interoceptive landscape is acquired cognitively and precognitively in an altered way, enhancing the prominence of symptoms related to fatigue.

The second paper demonstrated decreased basal ganglia activity in CFS associated with fatigue by fMRI. Results compared to controls, showed decreased activation in the right caudate and right globus pallidus. The decreased activation in the right globus pallidus was significantly correlated with increased mental fatigue, general fatigue and reduced activity. Dopamine has a central role in basal ganglia regulation, so alterations in dopamine metabolism may be involved. Dopamine transmission and metabolism in these areas may be due to activated immune pathways. Pharmacologic strategies targeting dopamine and the basal ganglia may be therapeutic possibilities.

**J.Dyke** (New York,USA) discussed a new brain imaging technique known as arterial spin labelling MRI was used to compare regional cerebral blood flow (rCBF) in CFS, patients with major depression (MDD) and healthy controls. The 2 patient groups were

psychotropic-medication free for 1 week prior to scanning. rCBF was significantly decreased in CFS in the left anterior cingulate cortex and the right lingual region compared to controls, while those with MDD had a trend towards significantly lower rCBF in the left anterior cingulate cortex. rCBF for CFS and MDD did not differ significantly. It is unclear whether the hypoperfusion in rCBF in CFS would account for previous observation of increased ventricular lactate. This increase could be due to oxidative stress, mitochondrial dysfunction or decreased rCBF.

[Back to top](#)

## Conference summary

The conference ended with an excellent over view by **Anthony Komaroff** (Boston,USA). He mentioned an informal meeting which had taken place to discuss multicentre research initiatives. He stressed the importance of a variety of initiatives pulling clinicians and researchers together. Forms, laboratory tests etc. need to be standardised. He pointed out that “Research Needs Money”.

[Back to top](#)