CFS PHOENIX

PHOENIX RISING: A CFS/ FMS NEWSLETTER by Cort Johnson	SPECIAL EDITION: LAYMEN'S GUIDE TO THE 8th IACFS CONFERENCE, January 2007
Subscribe to PHOENIX RISING!	PART V: Defining CFS, Economic Costs, Epidemiology, Behavior, Takeaway Points

These overviews do not follow the conferences agenda (fatigue, pain, gender, sleep, etc.). Several of those sessions were undersubscribed and had papers on different subjects shoehorned in to fill them out. In order to obtain a more orderly presentation some new sections (cardiovascular/ vascular, Exercise and CFS) are added in this overview while others are retained (Brain, Immune, etc.). Papers that I found most interesting are highlighted. None of the posters got presentations; the information from them is from the syllabus. Some overviews are found under more than one category.

CFS and War

Han Kang, Clare Mahan, Paymon Hasehemi, Michael Lyons, Seth Eisen, Charles Engel. Change in Chronic Fatigue Symptom-complex: 10-year interval Post Gulf War Deployment.

There appear to be many ways to get CFS; infection, stress, chemical exposure¼ this study shows us a new one – war. This study examined how many Gulf War veterans meet the criteria for CFS over 10 years. It found that five years after the war about 5% had CFS. Over time most improved but ten years later about 30% still had CFS. (Seventy percent were in remission or had been cured – we don't know what remission means). It was clear that being in the Gulf war increased one's risk of coming down with CFS but oddly enough there was some suggestion that just being in the military did as well. Only about 1% of non-Gulf veterans had CFS in 1995 but almost 3% did ten years later. The ill Gulf War vets looked very much like CFS patients; they did the same things that CFS patients do to avoid getting worse – they avoided exercise, cut down on work and social activities, ate well, stayed away from chemicals and alcohol, etc.

Researchers have started to track down the cause of post-infective fatigue by following people who fail to recover from various infections. They have been frustrated, however, in their ability to follow people whose CFS was not triggered by an infection. This and some of the presentations following it suggest that one way to do so is to follow a cohort of people who have been through a very stressful experience, whether it be engaging in war, going through a natural disaster such as a hurricane, being exposed to a chemical spill, etc. and parsing those out who don't recover. Dr. Reeves, I believe, stated that he expected that a large number of CFS cases will show up in post-Katrina New Orleans residents .

DEFINING CFS

Snow to the Eskimo, Fatigue to the CFS Patient: Distinguishing the Types of Fatigue in CFS.

Examining and distinguishing types of fatigue – Nicole Porter, Ph.D. Dr. Porter started off this interesting discussion by displaying how many terms Eskimos have to describe snow. She noted that 'snow' describes as little of the world of the Eskimo as does the word 'fatigue' to the CFS patient. Dr. Porter set out to remedy this situation by defining just what kinds of fatigue are present in CFS. She found five different types of fatigue occurring in the CFS patients but only one in the healthy controls. This study indicates that CFS patients do experience a very different type of fatigue than is found in the general population and should help to dispel the idea that CFS patients are 'just tired'.

The Five Kinds of Fatigue Found in CFS:

- Post-exertional fatigue – extreme fatigue after exertion
- Wired fatigue – overstimulated with extremely low energy
- Brain fog – difficulty with words, etc.
- Molasses fatigue – feeling of heaviness in limbs
- Flu fatigue – muscle aches and pains, nausea, sleepiness – this was the only type of fatigue experienced by the healthy controls.

THE JASON PAPERS - Thank God for Lenny Jason. Dr. Jason lead the Pediatric definition group, produced the first complete economic costs estimate of CFS, was instrumental in coming up with the correct prevalence estimates in CFS, and has highlighted the central role post-exertional and cognitive problems play in CFS among other contributions. He is now, and has been, something of a gadfly regarding questions of CFS epidemiology and defining CFS. He had three papers in the conference, each of which dealt with important aspects of CFS.

CONFERENCE HIGHLIGHT

Is Anyone Out There Listening? The Economic Costs of CFS. S

Leonard Jason, L. Valentine, S. Torres-Harding, A. Johnson, M. Benton, N. Porter. The economic impact of Chronic Fatigue Syndrome in a community based versus a tertiary sample.

We already have an assessment of the indirect costs of CFS – those caused by unemployment, disability, etc. – to the U.S. economy. They came out to a hefty 9.1 billion, yes BILLION dollars a year. Now Dr. Jason, with his study on the direct costs of CFS – medical costs – is able to give us, for the first time, an estimate of the total annual losses to the U.S. economy from CFS.

These costs are, of course, heavily affected by estimates of CFS prevalence. Dr. Jason's history of prevalence estimates was quite instructive; they started out absurdly low with early estimates suggesting about 20,000 CFS patients in the U.S. Jason's small early 1990s community based study bumped that up significantly (400,000), and his replication of that study doubled it again (800,000). The late 1990s CDC Wichita study confirmed Jason's estimates (800,000-1,000,000) and Jason used 880,000 as the basis for economic cost calculations.

With the direct economic costs added, Jason estimates that CFS costs the U.S. economy a whopping 19-25 billion dollars a year. This is about double the amount that prompted the Japanese government to sponsor a large CFS research program. These numbers suggest that if the Department of Health and Human Services woke up it would at the very least realize that investing in CFS research makes economic sense and would start funding CFS like the substantial health issue it is. Somebody in the DHHS, after all,

wants to know how damaging this disease is – they sponsored the first economic loss estimate (via the CDC) and must have paid for this one too – now if only they would act on it. With the CDC program winding down and the NIH research program essentially moribund, DHHS spending on CFS could be as low as $10,000,000 – or about what it commits to several very (very) rare genetic diseases.

CONFERENCE HIGHLIGHT

The First Pediatric Definition of CFS

IACFS Pediatric Case Definition Working Group (Jason, L., Bell, D., Rowe, K., De Meirleir, K., Jordan, K., Lapp, C., Gurwitt, A., Miike, T., Torres-Harding, S and E. Van Hoof. A pediatric case definition for CFS.

The first pediatric definition of CFS put together not by the CDC or by another governmental research group but by an International CFS Pediatric Case Definition Work Group convened by the IACFS. It is almost identical to the Canadian Consensus Definition and is much more rigorous than the Fukuda definition; besides requiring that a specific type of chronic fatigue be present (not substantially alleviated by bed rest, not due to ongoing exertion, etc.), it states that the pediatric CFS patient must exhibit five factors; post-exertional malaise or fatigue, unrefreshing sleep or sleep disturbance, certain types of pain (without edema), at least two neurocognitive problems and at least one symptom from two of three categories (autonomic/neuroendocrine/ manifestations).

This new Pediatric Definition could substantially alter the kind of patients deemed to have CFS. It, for instance, requires that post-exertional fatigue - an unusual symptom not associated with mood disorders - be present. This requirement in itself should shut out a good number of people with mood disorders who might otherwise qualify for CFS.

The new Pediatric Definition is another reason why we need to give the IACFS as much support as we can. The CAA takes the lead on patient advocacy, education and other issues but only a professional group such as the IACFS has the clout to move CFS forward on important research issues such as the definition. Please support the IACFS (click here).

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Defining CFS....Or Something Else?

Jason, L., Porter, N and N. Najar. Evaluating the CDC criteria for an empirical case definition.

The CDC believes that the Fukuda definition is so vague *'that it is essentially impossible to compare results between studies critically*" and that this definition has contributed to difficulty CFS researchers have in replicating study findings.

Two years ago the International CFS Working Group (ICWG) convened by the CDC recommended that the Fukuda definition be revised to better characterize the fatigue, disability and symptom severity found in CFS. The ICWG suggested that several different tests be used; the CDC took two of them (SF-36, MFI) and added one of its own (Symptom inventory) and then came up with criteria it proposed would differentiate CFS from CFS-like patients. These tests consist of a series of questionnaires that examine different aspects of fatigue (mental, physical, etc.), disability (mental, physical, emotional, etc.), etc. People scoring at the 25th percentile or lower on one of the

questionnaires from each of the tests are deemed to have CFS. The CDC now uses this definition to determine which patients it will include in its CFS studies.

Except for the symptom index, the tests in the new definition are standardized tests used to measure fatigue and disability in diseases for many years. Dr. Reeves is proposing that if you are fatigued and disabled to some extent and have a certain symptom profile and don't have any other diseases, then by definition you have CFS.

This new definition is a radical departure from past definitions. Other attempts at a CFS definition have focused solely on finding the right symptom profile for CFS and a consensus has emerged in North America regarding how CFS presents itself symptomatically; the Canadian Consensus and the IACFS Pediatric definitions are remarkably similar. The CDC clearly believes that symptoms cannot be used to differentiate CFS patients from CFS-like patients and it has some evidence for this - a large study that attempted to do so failed.

Dr. Jason, however, pointed out that several of the questionnaires in the SF-36 and MFI concentrate solely on emotional or mental aspects of fatigue and disability. *Two SF-36 questionnaires focus on emotional problems and mental health and one MFI questionnaire focuses on reduced motivation.* This suggests that under the new CDC criteria fatigued people with emotional problems but not necessarily with the physical components of CFS could meet the criteria for CFS and participate in CFS studies. In a small study Dr. Jason found that 40% of patients with major depression qualified for CFS.

This, of course, suggests that future CFS studies by the CDC could have an increased component of patients with depression or other mood disorders. This may already have happened; the recent Heim study employing the empirical definition found that 62% of the sample had evidence of early childhood abuse of one type or another. This contrasted with a study employing the Fukuda criteria that did not find high levels of childhood abuse. It bears noting, however, that people diagnosed with major depression (with melancholia or psychosis) in the last five years are excluded from participating from CFS studies under the new definition.

Dr. Jason also argued that these criteria were developed more or less arbitrarily; that they were derived using judgments rather than scientific analysis. *This was rather ironic given the fact that arbitrariness was one of the arguments Dr. Reeves used against the Fukuda definition. Although the components of the empirical definition were vetted by another international group, the definition itself was not. Indeed it appears to have been developed much like the Fukuda definition, as Dr. Reeves put it, by a small group of people (Dr. Reeves and his research team) in a 'smoke-filled room'. In the definition paper Dr. Reeves and his team acknowledged that "*one could debate our choice of specific subscales¼ .and the specific cutoff values we chose*".*

The definition of CFS ought to be a biomarker and Dr. Jason suggests that it be identified and undergo rigorous testing to ensure that a) it includes those that have CFS and b) it does not include those that do not have it. The CDC has shown that empirical definition does a better job than the Fukuda definition at the first, and it has been involved in testing different aspects of it, but it has not checked if people with other diseases might also meet this new definition for CFS. In most cases it doesn't have to – most are excluded from taking part in CFS studies anyway.

Dr. Reeves' analysis did find that the empirical definition did successfully differentiate CFS from CFS-like and healthy people – something neither the Fukuda definition nor symptoms have been able to do. Interestingly the three subjects in which the CFS patients did not differ from the CFS-like population had a psychological or mental basis; they were motivation, role emotional and mental health. The empirical definition was able to target a group of CFS patients in which a category called 'role physical' was, in contrast to the CFS-like patients, strikingly important. This suggests the definition did single out a distinct group of patients.

This is an ongoing battle to define CFS and it is a very important one. If a new group of patients that differs from the type of CFS patient now studied is selected for new studies, then their presence will alter both the findings and direction of CFS research. Whether or not these CFS-like patients are similar or not to 'CFS patients' it is clear that there are a lot more CFS-like than 'CFS' patients out there; this year for a short period the CDC boosted their estimates of CFS prevalence 4-fold to 4 million.

Two things at least are clear; the CFS *research community needs to a) come up with a good definition and b) the entire research community needs to use it. No matter how good a definition is, if it is not used by the majority of the CFS research community, then its introduction will only further muddy the waters.*

Toward a (Real?) Empirical Definition of CFS?

Leonard Jason, Karina Corradi, Susan Torres-Harding. Toward an empirical case definition of CFS. (Poster)

Whether or not the new CDC empirical definition needs to be changed, Dr. Jason is bringing up questions that need to be asked. In this study he outlines an empirical way to come up with an empirical definition.

In this study he gave 114 CFS patients questionnaires regarding symptoms associated with a range of different systems including the vascular, inflammatory, muscle/joints and others. If I understand this correctly he found that using these symptoms provided a better biological interpretation than did using those associated with the Fukuda definition. He was able to identify four clusters of patients (subsets) with distinct symptom presentations. He noted two of the clusters identified could not have been assessed using the Fukuda definition. This means that a much broader range of symptoms need to be assessed in CFS than the seven or eight present in the Fukuda definition and that important symptoms in CFS have been missed.

This study leapfrogged over the question of how to define CFS in general and went to what may be an even more important question; Identifying coherent subsets in CFS. We often think of a biomarker as a kind of holy grail in CFS research but defining coherent subsets would, if anything, have even more importance. Being able to do would constitute an evolutionary leap in our understanding of CFS, lead to more significant and replicable study findings and initiate the breakup of the CFS label. Given the worries about the vague CFS definition and the subsets, it is unfortunate that it took 15 years to get this type of study accomplished.

To its credit the CDC has already looked at that question of symptom presentation in CFS and CFS-like patients; they found that, even using an expanded symptom list, the two groups looked very similar symptomatically and they could not differentiate them. They, of course, also attempted to derive subsets using the voluminous data set generated by the Wichita studies.

We end this section with a question asked by Mary Schweitzer at the conference that bears on this issue, "*How do we get homogenous sample groups for CFS studies?*"

Dr. De Meirleir stated that the current definition was created before researchers had any idea of possible biomarkers in CFS. He thought the definitions will hold for only a few more years. Once researchers identify the different mechanisms present in CFS, the present definitions will fade away.

Myalgic Encephalomyelitis vs. CFS.

Byron Hyde - Myalgic Encephalomyelitis (M.E). The Definition

There is another question roiling the 'CFS' community right now. The IACFS changed its name to IACFS/M.E. and a name change committee convened by Rich Carson endorsed CFS/M.E. as a new name for 'CFS'. These changes have prompted fierce outbursts by some M.E.

advocates who object to having the more general term CFS associated with Myalgic Encephalomyelitis – a term they believe denotes a discrete disease process. Here we look at a definition of M.E. by one of M.E.'s chief proponents, Dr. Byron Hyde, a physician who may have longest track record in covering M.E/CFS.

What is M.E.? Dr. Hyde informs us that *"M.E. is essentially an acute onset post viral illness" "caused by a diffuse...injury of the Central Nervous System's (CNS) vascular system".* He believes this CNS injury is the defining injury in ME and that it alters neuro-endocrine and probably neuron-chemical interactions, and these, in turn, cause problems throughout the body.

M.E.'s Clinical Presentation – M.E. begins with an infection usually (but not always) with an incubation period of between 4-7 days that is followed closely by a chronic phase in which SPECT, PET or QEEG scans can detect diffuse changes in the CNS. In order of increasing severity these changes involve one side of the cortex, both sides of the cortex and both sides of the cortex with subcortical structure involvement (e.g. pons, cerebellum and/or brainstem) Patients with the less severe CNS damage can improve but those with the most severe CNS damage generally show little improvement over time.

Pain - Early on, the M.E. patient suffers from severe headaches of a type never before experienced that are often associated with increased neck rigidity, pain at the back of the head, eye pain, migratory muscle and joint pain, hypersensitive skin and eventually a fibromyalgia-like pain. Over time the pain symptoms tend to abate.

Neuropsychological Problems – Neuropsychological changes including short-term memory loss and other cognitive problems, irritability, confusion, etc. that are exacerbated by physical and/or mental activity are present. These too may improve over time.

Sleep – Sleep dysfunction including reduced daytime alertness is present.

Muscle dysfunction – including muscle pain and rapid loss of muscle strength after exercise is present.

Vascular dysfunction – Dr. Hyde has posited for many years that a vascular dysfunction plays a major role in M.E. and causes many of the symptoms. This vascular dysfunction is present in all M.E. patients but is most obvious in M.E. patients with postural tachycardia syndrome (POTS) and other heart rate changes, Raynaud's disease (temperature regulation problems) and bowel dysfunction.

Endocrine dysfunction – shows up later in the disease.

An Interpretation: It's beyond the scope of this overview to compare the differences between the M.E. definition and the various CFS definitions. Comparing the two is, in fact, like comparing apples and oranges; the M.E. definition elucidates a cause, describes a process and then characterizes a condition; the CFS definitions generally simply try to characterize the condition of the patient.

Very few studies have examined patients that meet the definition for M.E. Except for anecdotal reports from physicians such as Dr. Hyde, people who identify themselves as M.E. patients can only access 'M.E. research' via studies on CFS. This cohort is just beginning to be singled out in CFS research studies.

The question asked here is whether the CFS research findings presented at this conference are consonant with the post-infection CNS illness process elucidated by Dr. Hyde. Setting aside the issue of what triggered their diseases, we ask if CFS patients in general look like M.E. patients.

It appears that they do. Dr. Lange indicated that abnormalities are commonly seen in areas of the brain targeted by Dr. Hyde and these abnormalities, intriguingly, are more evident in CFS patients without mood disorders. Spinal fluid abnormalities suggest CNS infection, inflammation and blood vessel vasoconstriction. MRI studies suggest damage in one area of the brain causes CFS patients to utilize different parts of the brain than normal. Dr. Kuratsune charted a model of CFS pathophysiology that began, as did Dr. Hyde's scenario, with central nervous dysfunction which then affected other parts of the body. Dr. Spence's study on arterial stiffness singled out, as does Dr. Hyde, the vasculature in CFS. Dr. Kerr's gene expression studies suggest that genes involved in nerve demyelination, inflammation, and viral activity are involved in CFS.

Dr. Albright's heredity study indicated that relatives of CFS patients had increased rates of migraine (headache?), Raynaud's disease, irritable bowel syndrome and in particular, myalgia, all of which are mentioned in the M.E. definition.

The closest application to M.E. comes from the Dubbo studies examining the pathophysiology of patients who do not recover from a variety of viral and bacterial diseases. These studies suggest that because their immune systems have trouble recognizing these pathogens they are hit harder by them. Dr. Whistler's study indicated that infected cells in these patients have trouble killing themselves off before the viruses replicate inside them. This project, which began in the periphery is now focused on the central nervous system. Dr. Ablashi's and Dr. Levine's study suggests that the activity of a common CNS pathogen, HHV-6, is increased in a significant number of 'CFS patients'. Dr. Glaser's study suggests that another CNS pathogen, EBV, is as well.

Several of the findings presented at the IACFS conference appear then to be consonant with an M.E.-like disease process. At least two explanations could explain how a heterogeneous CFS population could appear similar to a more discretely defined M.E. population; M.E. patients make up the majority of CFS patients tested or CFS/M.E. patients in general share a similar pathophysiology.

There is evidence for both. Acute onset CFS patients are generally believed to be most prevalent in clinic-derived studies. Some researchers also believe that neural insults of the kind that may be present in CFS are not necessarily pathogen derived but could be triggered by other kinds of physical stressors such as toxin exposure or psychological stresses.

The identification of subsets in CFS is long overdue and CFS/M.E. patients still await the kind of large-scale studies that can identify coherent subsets. The post-infectious illness subset in CFS (aka M.E.) is, however, the most readily distinguishable subset and is the only subset that is currently receiving special attention. The CDC-sponsored Dubbo studies and Taylor's NIH- sponsored post-infectious mononucleosis study are sophisticated efforts using laboratory and gene and protein expression tests that attempt to detect the pathophysiological changes that occur when individuals fail to recover from a variety of viral and bacterial infections. These types of studies are long, long overdue but they give hope that the post-infectious subset of CFS/M.E. will be the first to be elucidated and treated.

An Early Epidemic of CFS or Something Else?

Eirkuir Lindal, Jon Stefansson, Sverrir Bergmann. Chronic Fatigue Syndrome in Iceland

What does CFS look like in Iceland? These CFS patients were mostly middle-aged females who worked full time and believed their disease was stress-related. About 2.2% of the population had CFS.

The researchers then did an interesting thing; they used a genealogical data base to determine if these individuals were more likely than would be expected to be related to people who came down with what some researchers believe was one of the first documented epidemic outbreaks of CFS, the Iceland Disease illness of 1947. *If the Iceland Disease was caused by the same infectious agent that causes CFS then it presumably would have been passed down from generation to generation. In the Underhill studies reviewed in the Gene Overview of this conference we saw evidence that a multi-generational infectious process is present in CFS. On the other hand if there is genetic*

predisposition to CFS, as studies in this conference have suggested, then the present day CFS patients in Iceland should have an increased relatedness to earlier outbreaks of CFS.

The current day CFS patients were not more related than expected to the victims of the 1947 Iceland Disease. *This appears to suggest that the Iceland outbreak of 1947 was a unique event perhaps caused by a pathogen that does not figure in the CFS now found in Iceland. The lack of genetic relatedness between present day CFS patients and the victims of the Iceland Illness appears to suggest that the Iceland Illness was not a precursor of CFS.*

BEHAVIORAL SESSION

Ellie Stein – Introductory Overview (from the conference syllabus)

I missed the behavioral session but it's worthwhile given this very controversial subject to summarize Ellie Stein's presentation. Dr. Stein first gave us some reasons why cognitive behavioral therapy or CBT caught on with some; the increased levels of mood disorder in CFS, findings suggesting that attribution (what the patient thought was wrong with them) played a role in illness severity, the high rates of inactivity, the inability to explain CFS scientifically, the inability to find good treatments, etc. all suggested to some that CFS was more a behavioral disorder than a biological one. At its most extreme this lead CBT practitioners to attempt to radically alter the way a CFS patient felt, thought and acted. They did this by encouraging CFS patients to disregard their symptoms and engage in activities such as exercise that they felt were harmful.

Dr. Stein reported that CBT study results have been lackluster with 4/7 studies showing improvement. Two of these used the less restrictive Oxford criteria which is believed to select patients with increased rates of mood disorder. Only one study using the Fukuda criteria on adolescents was positive. Two exercise studies using the Fukuda criteria resulted in reduced fatigue. A CBT/exercise study showed improved quality of life but little change in the ability to exercise. The long term effects of this therapy appear poor.

In conclusion, Dr. Stein reported that these programs are moderately effective at best and suggested that CBT's basic premises - that attributing CFS to a physical cause has negative effects, that poor coping mechanisms are very important in the course of this (or other) illness(es), and that exercise is an benign activity in CFS - were wrong. She noted that attempts by CBT practitioners to tout CBT as a cure for CFS were contrary to its usage in other diseases such as multiple sclerosis and rheumatoid arthritis in which it has positive effects. She noted that 'CBT' is a fluid field with different approaches and different protocols. More recent CBT programs in fibromyalgia appear to be dropping some of the damaging premises.

She then turned to a different behavioral model, not focused on CFS but on chronic illnesses in general, called the Stanford Model. This model states that patients with chronic illnesses know the best about the consequences of their illness and attempts to improve it but can be assisted in finding beneficial health practices and in continuously using them. A wide variety of possible interventions are used in this model including exercise, nutrition, energy and sleep management, medication, managing emotions and symptom management. The Stanford Model had long term beneficial results in one group.

She suggested that in CFS/ME, behavioral treatments should focus on self observation/data collection (instead of ignoring one's symptoms rigorously monitoring them!), sleep and activity management, diet, stress, emotions and environmental toxin. She believes Fennell's four-phase coping model provides a framework which CFS patients can use to adapt to their disease.

She concluded by stating that CBT/GET have not significantly altered the course of CFS over time and many do not benefit and that alternative approaches, such as mentioned above, should tried.

As time goes on, it appears that at least some behavioral therapists are radically altering their approach to CFS and FMS. Instead of telling CFS patients how they should think and act they are rigorously listening to them and then using their expertise with chronic illnesses to assist them in improving their health and quality of life within the confines of their illness.

CONFERENCE LOWLIGHT

Reality Check - Progress in CFS Research?

Fred Friedberg, Brett Schmeizer, BA and Stephanie Sohl. Stagnation in CFS Publishing? Comparisons with Fibromyalgia and Fatigue: 1995-2004. (Poster).

Leave it to Dr. Friedberg to provide one of the few conference lowlights. Dr. Friedberg, a current member of the CFSAC, is decidedly not happy with the status quo in CFS research.

Yes, it was a good conference. The results of research studies are beginning, in some cases, to converge, and new and interesting fields are continuing to emerge. But let's not get carried away. CFS is a very complex disease that is still very, very significantly underfunded; funding levels at the NIH are lower than they have been for over a decade and they are due to drop significantly at the CDC. Dr. Klimas several times noted the need for new researchers in the field.

CFS has been a subject of inquiry for almost 20 years – long enough, one would think, for it to have embedded itself in the research community and built a following that should be able - given the increasing estimates of prevalence rates and economic costs – to grow substantially over time. Here Dr. Friedberg here asks a very good question: have CFS, fibromyalgia and/or non-CFS fatigue publication rates significantly increased over the past decade?

There's some good news and bad news here. The good news is that the fibromyalgia and non-CFS fatigue publication rates very significantly increased over the past decade. The bad news is that the CFS publication rate did not. *It appears that at some point in the last five years or so fibromyalgia has 'made it' as a research topic but CFS has not. This 'breakthrough', if that's not too strong a term, is good news for CFS; no disease is so closely allied with CFS and it was not so long ago that FM, then referred to as fibromyalgia Syndrome or FMS, was a very controversial disease; if FM can make it then so can CFS.*

The other good news is the increased interest in fatigue in general. *While there's more to CFS than fatigue, fatigue in its physical and mental forms, is still a defining characteristic of the disease. Researchers are becoming more and more interested in the fatiguing aspects of diseases such as cancer, overtraining syndrome, multiple sclerosis and liver disease. Insights into these diseases may help us understand CFS. Liver disease researchers, for instance, have recently presented evidence indicating that pro-inflammatory cytokine production in the central nervous system contributes to the severe fatigue found in some patients.*

Dr. Friedberg's study also demonstrated that two countries, the U.S. (38%) and the United Kingdom (32%) dominate CFS research with just three others (Netherlands 5%, Australia – 3%, Germany (3%) getting mention. Surprisingly, despite the supposed dominance of the 'Wessely School' of thought in the U.K., psychologically oriented publications were not ascendant there (18%) or in the U.S. (21%).

Dr. Friedberg's study indicated that while CFS researchers are getting better at understanding CFS the rest of the medical research community still responds to CFS research as it always has; as something of an oddity or a fringe topic, something they're not sure deserves extensive research and something they're certainly not interested in investing their time or money not to mention their careers, studying.

On that happy note let's focus our attention on the researchers and physicians who have decided that CFS is worth studying and have been willing to stick with us through thick and thin. These are courageous and committed people; some are new to the field, others came to CFS after long and successful careers elsewhere. We are lucky to have each of them.

The AWARDS

You can find out more on each of these awardees by clicking here.

Govenor Rudy Perpich Memorial Award – Daram Ablashi D.V.M., M.S., Dip. Bact. – Dr. Ablashi has quite a resume. He worked at the NIH for 22 years, was a co-discoverer of the HHV-6 virus, co-founded the IACFS (then the AACFS) and is now the Scientific Director of the HHV-6 Foundation. Read Dr. Ablashi's engaging talk about Rudy Perpich and the history of the IACFS by clicking here.

Junior Investigator Award – Elke L.S. Van Hoof, Ph.D – A forthright and articulate speaker, Dr. Hoof cut a dynamic figure at the conference. A colleague of Dr. De Meirleir's, she has been heavily involved in epidemiological and behavioral research. In his introduction Dr. De Meirleir joked that he had two similar assistants; one of whom got sick and one which couldn't handle the CFS patients! Dr. Hoof seems to be doing fine.

Nelson Gantz Clinician Award – Daniel Peterson, M.D. – Dr. Peterson was in Europe during the award presentation. Along with Dr. Cheney, Dr. Peterson diagnosed and treated the Incline Village Cohort in Nevada that really sparked interest in CFS in the mid-1980s. A strong advocate for CFS Dr. Peterson has been instrumental in helping elucidate the post-viral subset of CFS.

SUMMING UP: My Takeaway Points From the Conference

Everybody who attended the conference probably got something different from it. Most of the conference reports do, however, mention one striking feature – the degree to which the research findings are beginning to come together. CFS is a multi-systemic disorder; once research findings from different parts of the body begin to cohere we can get the feeling that the researchers are on the right track.

Disability Can Be Reliably Measured in CFS – The inability to find an accurate and replicable test of disability has left many CFS patients in dire straits financially and medically. Three studies indicated that simply extending an exercise test to two or three sessions results in declines in widely accepted measures of aerobic and cognitive function. This protocol makes sense with regard to CFS patients' symptoms and will hopefully fulfill government standards for disability - this could be a real breakthrough in a much needed arena.

Mitochondrial Dysfunction Plays a Role in CFS - Evidence of poor energy production in the brain (high lactate levels), poor performance of the energy intensive phase (diastolic) of the heart beat, altered mitochondrial gene expression in three studies, findings of impaired mitochondrial activity in fatigued animals, and the effectiveness of the two mitochondrial enhancers in CFS, D-ribose and NT factor, all suggest that mitochondrial dysfunction, whether a direct or indirect aspect of CFS, is present.

Inflammation is a Key Component of CFS – the Spence arterial stiffness study, the Maloney metabolic syndrome study, the Gurbaxani IL-6 study, Hurwitz's 'failed' low blood volume treatments, the efficacy of Dr. Parks and Dr. Shoemaker's treatments and the proteins in Baraniuk's cerebral spinal fluid all suggest that inflammation – a complex immune response to tissue injury that involves the blood vessels – plays a role in CFS.

There Is A Hereditary Predisposition To CFS – When independent research groups confirm each other's findings we can begin to trust their results. Several gene mutation studies have found increased mutation rates in the same stress response genes (corticotropin-releasing factor receptors, serotonin transporters, glucocorticoid and dopamine receptors and trytophan metabolism). These indications of a heritable component in CFS were backed up by two studies indicating increased rates of CFS and CFS-like illnesses in the close relatives of CFS patients.

Circulatory and Blood Flow Problems Are Present in CFS – The reduced cerebral blood flow levels reported by Dr. Lange, the Baraniuk study suggesting CNS protein aggregations, blood vessel inflammation and vasoconstriction, Dr. Park's finding of decreased kidney microvascular blood flows, Dr. Spence's findings of increased levels of arterial stiffness, Dr. Hurwitz's findings of low red blood cell volume all suggest that blood vessel problems play a role in CFS.

Subset Identification is Critical To The Understanding And Treatment of CFS - Levine's HHV-6 studies and the Montoya and Lerner antiviral studies suggest that, if properly identified, a subset of CFS patients can be effectively treated. Gurbaxani's intriguing analysis of IL-6 cytokine data, Maloney's findings of a subset of metabolic syndrome patients, Jason's empirical definition study and Whistler's distinctive gene expression findings in a post-infectious subset all indicate that identifying subsets is critical in CFS.

Gene Expression Results In CFS Can Be Replicated (!) – Dr. Kerr's report that his large-scale, rigorously planned gene expression study is finding the same pattern of gene expression as his smaller one suggests that this important technology can be harnessed to give us a better understanding of CFS and how to treat it.

The First Federally Approved Drug for CFS Appears to be on the Horizon – the success of the phase III clinical trials for Ampligen appears to signal that the first federally approved drug for CFS in the U.S. is on its way. It may be too expensive for many (if not most) CFS patients but its advent is a breakthrough for CFS in a larger sense and it should give CFS increased credibility in medical and research community and increased focus on the immune system.

Behavioral Therapies, if Done Correctly, Can Improve the Quality of CFS patients Lives – CFS physicians seemed to agree that while behavioral therapies are not a cure, if done correctly, they can as in other chronic diseases help improve CFS patients' quality of life. Dr. Stein's presentation suggested that more informed 'CBT' therapists are dropping some of the damaging premises of the past and beginning to seriously listen to CFS patients and use their experiences with other chronic illnesses to help them manage their disease better. Dr. Fennell, a most impressive speaker and the originator of the 'Fennell Stages" of illness has written a book describing effective ways to deal with chronic illnesses.

CFS Researchers Need More Support! - Too many studies at the conference were preliminary or methodologically impaired in one way or another (no control groups, small numbers, etc.). This probably reflects cash-strapped researchers doing the best with what they have but it is hard to trust results from these studies. On the other hand too few studies at this conference employed the kind of rigor that makes their results impossible to ignore. Similarly too many studies are simply trying to characterize CFS while too few are trying to actually explain it. Dr. Friedberg's study indicated that despite the increasingly promising results we are seeing, CFS, unlike fibromyalgia and other fatigue producing diseases, has still not 'caught on' as a research topic; it's publication rate has been flat over the past ten years.