

# Dr. Maureen Hanson: From Plant Biology to ME/CFS Champion

**DR. MAUREEN HANSON** is the Liberty Hyde Bailey Professor in the Department of Molecular Biology & Genetics at Cornell University and a member of the Solve ME/CFS Initiative (SMCI) Research Advisory Council (RAC). Notably, she is directing a National Institutes of Health (NIH) funded myalgic encephalomyelitis/chronic fatigue syndrome (ME/CFS) Collaborative Research Center recently established at Cornell following a competitive grant process. Over the next five years, the Center will work on three primary research projects and work cooperatively with a network of three other ME/CFS centers. We recently sat down to speak with Dr. Hanson about her work.

***If you had a magic wand, what are the top three barriers you would remove first in order to accelerate the discovery process or improve the lives of patients?***

Despite recent increases in funds for ME/CFS at NIH, an important barrier is the relatively modest amount of NIH support devoted to grant proposals to study ME/CFS. The funding rates of investigator-initiated proposals at most NIH Institutes is less than 20%. If proposals to study HIV and AIDS had been this low during the AIDS epidemic, many more years would have been needed to develop life-saving drugs. We need the federal government, NIH, other government research agencies, and the general public to realize that there is a hidden disease raging that is taking away people's lives, even though most victims are not being buried in the ground. Requests For Applications (RFAs) for regular research grants are needed, with designated funding, so that a larger number of worthwhile proposals can be funded even if they don't rank

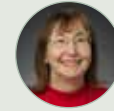


*Project Leads for the Cornell ME/CFS Collaborative Research Center at our "kickoff" meeting. Left to right: Dr. Dikoma Shungu (Radiology, Weill Cornell Medicine), Dr. Maureen Hanson (Molecular Biology and Genetics, Cornell University), Dr. Andrew Grimson (Molecular Biology and Genetics, Cornell University).*

## SOLVE ME/CFS INITIATIVE RESEARCH ADVISORY COUNCIL



Anthony Komaroff, MD  
Harvard Medical School



Maureen Hanson, PhD  
Cornell University



Susan Levine, MD  
Medical Office of Susan M. Levine, MD



Jose Montoya, MD  
Stanford University  
Medical Center



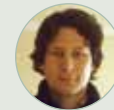
Peter Rowe, MD  
Johns Hopkins



Sheila Stewart, PhD  
Washington University School  
of Medicine



Tarek Absi, MD  
Vanderbilt University Medical  
Center

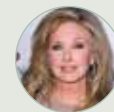


Daan Archer, MBA, MSc  
Massachusetts Institute of  
Technology Media Lab



Nathalie Bloch, MD, MPA  
Mount Auburn

### AD HOC MEMBERS



Morgan Fairchild  
Actress, Patient and Activist



Cindy Bateman, MD  
Bateman Home Center



Andreas Kogelnik, MD, PhD  
Open Medicine Institute



John Nicols, MBA  
Codexis, Inc.

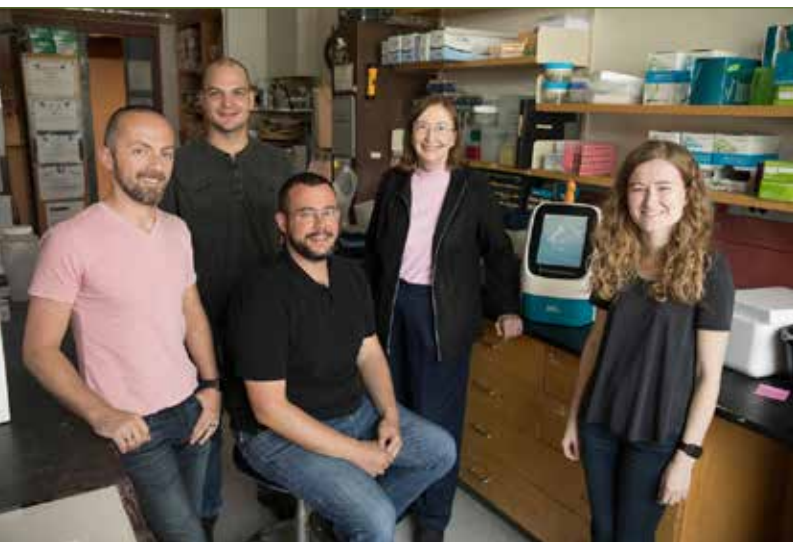
## Dr. Maureen Hanson: From Plant Biology to ME/CFS Champion (cont'd)

in the top 20% of all proposals submitted to NIH. A proposal that ranks in the top 25% is not a “bad” proposal that cannot accelerate the discovery process for this neglected disease. But without a targeted RFA with a designated funding level, such a proposal will be rejected.

Second, my wand would wave a readily performed diagnostic test into existence. If patients could arrive at their doctor’s office complaining of ME/CFS symptoms, and a simple blood or urine test could be ordered that would reveal whether or not the patient has ME/CFS, then no longer would patients be told they are lazy, have psychosomatic illness, and are not truly physically ill. Also, such a test would likely stimulate interest from the pharmaceutical industry in drug development. Once patients can be objectively measured, and the difference between an ill and a healthy

individual can be observed, drugs can be tested and the patients’ responses to the drug can be objectively measured to determine whether the drug induces a healthy profile.

Third, I would have the FDA approve a drug for the treatment of ME/CFS. The best candidate for such a drug is Ampligen, which is a miracle for those patients who respond, though only a subset does respond. The approval of a drug in the US that can restore some patients to health has great value symbolically. It will demonstrate to the medical profession and the general public that the disease can be treated and that more drugs that help additional subsets of patients can and should be developed. The drug industry will take notice. And at least some fortunate Ampligen responders will get their lives back.



The ME/CFS researchers in the Hanson lab. Left to right: Dr. Arnaud Germain, Mr. Adam O’Neal (graduate student), Dr. Ludovic Giloteaux, Dr. Maureen Hanson, Ms. Alexandra Mandarano (graduate student).

### **What policies can be advanced by advocates and government officials that will support progress in ME/CFS? What are some of the barriers/strategies to enacting these policies?**

Advocates need to pressure the federal government for additional support for ME/CFS for relevant agencies, including NIH, CDC, and in the Department of Defense. I would wager that a large number of service personnel have been disabled by ME/CFS. They need the same attention and research support given to their disease as has been provided to study Gulf War illness.

I have heard it said repeatedly at CFSAC [the Federal Chronic Fatigue Syndrome Advisory Committee] meetings by various governmental agency representatives that there are not enough researchers who are interested in ME/CFS, so no more funds for ME/CFS research grants should be allocated. I disagree. Researchers will come into the field once funds are available. How many individuals were studying retroviruses when HIV was initially discovered? The answer is very few. But funding for AIDS resulted in many researchers from other fields changing their research em-

phasis to work on HIV/AIDS. Because of the hypercompetitive nature of NIH grant funding these days, many new researchers would be attracted into the field were RFAs for ME/CFS proposals available.

***How could the NIH-sponsored center that you lead affect change and improve our understanding of ME/CFS?***

Our Center is focused on one of the most disabling symptoms of ME/CFS: post-exertional malaise (PEM). We want to understand why it occurs and we are using the phenomenon to probe what goes wrong in victim's bodies when it is happening. Our research projects will gather data about patients in their baseline state and after PEM has been induced. Learning what has changed at the molecular, biochemical, neurological, and physiological levels may reveal to us what fundamental disruptions are occurring in the disease. At Weill Cornell Medicine, Dr. Dikoma Shungu is leading a project to assess neuroinflammation and oxidative stress in the brain. At Cornell-Ithaca, I am supervising a project to examine the role of extracellular vesicles, signaling molecules, and metabolism. Functioning of the immune system will be studied through single-cell RNA sequencing in a project led by Dr. Andrew Grimson. All of these projects also have the potential to lead to diagnostic tests for the disease. Our website [www.neuroimmune.cornell.edu](http://www.neuroimmune.cornell.edu) provides more information about these projects and the collaborators and patient advocates who are associated with the Center.

***Why are you so dedicated to the MECFS population and what sparked your interest in this disease in the first place?***

I am very aware of the seriousness of the disease and how it affects patients' lives since my adult son has now been ill for 20 years and has missed many of the milestones of adolescence and young adulthood. Fortunately, he is not bedbound, though he is largely housebound and must spend much of the day horizontal. After he was diagnosed, I attended the IACFS [International Association for Chronic Fatigue Syndrome] meetings in 2004 and 2007, where I realized that very few molecular geneticists were studying the disease. All my previous scientific research had been in plant genetics and I had never previously studied human genes. But, to paraphrase Gertrude Stein: a gene is a gene is a gene. I knew that I could contribute to knowledge of ME/CFS and thus 10 years ago, I began seeking grant support to initiate biomedical projects.

***Where do you think the major finding will come from? Do you have a favorite theory?***

It is my theory that a single fundamental biological disruption underlies the disease, despite the differences seen between the symptom constellation in patients, the existence of subsets of treatment responders, and variation in the illness-inciting events that patients report. Real progress will be made in producing therapies once the identity of this damaging culprit is revealed. There may be existing, FDA-approved drugs that could benefit patients, if we understood the cause of the disease and therefore knew that the medications should be administered. Such existing drugs give the best chance to improve the lives of patients without long delay. ■