A research team from Glasgow University in Scotland has announced an altered pattern of gene activity in 50 patients with the Chronic FatigueSyndrome/Chronic Fatigue and Immune Dysfunction Syndrome/ Myalgic Encephalopathy (CFS/CFIDS/ME). Dr. John Gow, the senior researcher, said, "We have identified genes which were up-regulated compared with genes in normal healthy individuals... This means the genes are switched on or off an at inappropriate time... It looks like the immune system is working overtime when it shouldn't be..."

Dr. Gow and his team mapped the entire genome of 33,000 genes in the CFS/CFIDS/ME sufferers and then compared them with the genes of healthy people. Despite the initial results, Dr. Gow stressed that more testing of CFS/CFIDS/ME patients is needed to make sure that the unusual gene activity is specific for Chronic Fatigue Syndrome. He thinks this further testing would take about a year.

Diagnostic Test and Treatments:

The research team is hopeful that the potential "CFS gene signature" could lead to a specific diagnostic test, and has already patented the genes which would be involved in diagnostic testing. A prototype diagnostic testing kit has been developed which would give a yes or no answer as to whether CFS is present.

Even more exciting is the promise of medication to treat the abnormal gene/immune dysfunction. Dr. Gow stated, "Our work has given us clues as to which pathways are up- or down-regulated and we know which drugs activate different pathways, so we think we can find drug treatments that will be beneficial to patients."

These specific drugs are already on the market and therefore could be available to CFS/CFIDS/ME patients in the immediate future. Dr. Gow said, " ...it really needs to go through proper trials before these drugs become widely available."

This is important research since it includes a possible mechanism of action, a diagnostic test, and potential medication. Of course, we must wait and see.

Sources: *BBC News*, UK Edition, 28 May 2005; *The Scotsman*, 20 May 2005; Co-Cure; ME

Association.

Updated Information on Dr. Gow's Research

Since this article was written, two groups in Britain: MERGE (ME Research Group for Education and Support) and the ME Association have provided substantial funding to enable Dr. Gow and his associates to begin the second phase of their research. MERGE has provided an interim award of 8,000 pounds and the ME Association has granted 28,675 pounds (in addition to the nearly 9,000 pounds that the MEA has already provided).

The following update on the research is taken with permission from a Co-Cure post dated June 27, 2005:

"So the second phase of the study should now be able to commence in August.

Why is this type of genetic research so important in ME/CFS?

In very simple terms, the Glasgow University research group will be using a technique called DNA chip microassay analysis to map out what is happening to a vast amount of individual genetic information—over 33,000 gene sequences in each individual. The scientists will be carrying out this genetic analysis on a large group of people with ME/CFS, another large group of healthy matched controls, and a further large group of people with a range of other illnesses —
such a multiple sclerosis and depression
—
in which fatigue is a major clinical symptom. In particular, the scientists will be trying to identify whether there is a unique profile of genetic abnormalities in people with ME/CFS by looking for data which indicates that certain specific genes are either up-regulated or down-regulated —
roughly meaning that they are being over-active, under-active or 'switched-off'.

The activity of these genes—gene expression in medical jargon—can have very important consequences on the types of cellular activity, including crucial biochemical pathways, that they control in the nervous system, immune system, and all other parts of the body. So the ultimate

aim of the study is to identify specific gene abnormalities which may then lead to new avenues of research and the presence of a diagnostic biomarker or diagnostic biomakers which is/are only present in ME/CFS.

Preliminary results from phase one of this study already indicate that significant abnormalities in gene expression are present in the ME/CFS group, but this data now needs to be confirmed in a much larger trial.

This type of information on gene expression will also be highly relevant to new forms of treatment which are worth assessing. And as the data becomes clearer, a further phase of the research will hopefully then involve a clinical trial of drug treatment aimed at the underlying cause of ME/CFS."