Dr Ron Davis updates on current research, May 2018

https://www.youtube.com/watch?v=ZQTURNzSbVY

Hello. I'd like to give a brief update of where we are in our research on ME/CFS.

We've been working hard, trying to finish everything with the Severely Ill patient study. Most of the data has been collected, and analyzed. And we are now trying to get it up on a website where people can access all of the data. And also, we are writing a paper, which we will submit soon. I'm hoping that that will help a lot of researchers, by which they will have access to all of this data. And it can be used for them to prepare hypotheses, which then could be possibly funded by NIH.

Other work that's going on in the lab is carrying out another analysis, a big data study, but on a range of severities. And, that hopefully will complement this Severely Ill patient data.

We're also doing a study on families where we look at affected people, sometimes more than one, and also the patients in the family that are unaffected. That will also help us, I believe, tease out some of the genetic components of this disease.

The other activity that I'm excited about is the fact that we are really working hard now on the T-cell activation. The nice thing about that project is that we're getting involved with Mark Davis, who is a superb immunologist, and he's helping us in that as well as students from his lab. And also Lars Steinmetz has joined that effort, because he used to develop a number of technologies for the sequencing. So that allows us to expand our efforts and our expertise. That looks very encouraging in the sense we do see T-cell activation. Of course, the real problem is: what's activating the T-cells? And that's what we will take on after we collect enough data.

Also, to do that study we have to do the HLA region, and we have worked out a method for that sequencing, which has now been commercialized. It's used at Stanford and throughout the world for transplant matching, and it looks like it's been very very successful. I'm told that rejections are now at a lower frequency.

Also, we will do the KIR sequence. It's very much like the HLA. The HLA is a major locus that determines self, and it's very involved in autoimmunity. The KIR locus seems to be more involved in interacting with viruses. From the Severely Ill patient study, the KIR locus is showing up to be a very important locus. It's hard to sequence. We're going to be working with the clinical HLA lab to carry out that sequencing.

I'm also excited about the analysis of the metabolomics, combining it with our sequencing, especially from the Severely Ill patient data. And, pathway analysis. So, this combination of data is allowing us to look for what we call a 'metabolic trap'. The reason we're excited about it is that the behaviour of this disease – in terms of patients, which frequently have rapid onset – seems like a switch has been thrown. A metabolic trap would look like a switch. The other part why that's so important is the fact that if we can find a trap that is really causing this disease, it will be easy to reverse. And that would be a cure, not just a treatment. I'm hoping that's the case. But we always have to test the hypotheses that we have, and that's what we're now actively doing.

The other thing that I'm concerned about is the fact that things are going slower than I would like, and that's because we don't have enough people. Every project has just one person. We need to ramp that up, to get through things faster. And so, one of the things we're going to do with this big $5 000 dollar donation [*Dr Davis apologizes, the correct amount was $5 Million*], is hiring more people. The other thing we have to do is to make certain we have salary support for people beyond just one year, because it's very difficult to recruit people if you only hire them for one year. And so, part of the money will be used to guarantee salary support for everybody that's operating here, in a second year.

Also, we're making good progress on the biomarker. We have four biomarkers, I've talked about those in the past. One of them, we have prepared a paper which will go out soon. And, we're writing a second paper on the nanoneedle. Those all look pretty good as a biomarker for ME/CFS. So far in the nanoneedle every patient shows a clear signal, and none of the healthy controls do. So, that could be a biomarker that says something is wrong, that you are not a healthy individual. And that's what we are looking for first. The second problem would be, can we distinguish it from other diseases? I think that's less important, because the other diseases that could be confused with ME/CFS can be diagnosed with other methodology. And it's most important, I think, to show that the patients are not faking it, and it's not all in their head, and it's not psychosomatic. And I think that these tests that we are doing will absolutely show that.

I need to recruit some other expertise, and we also need to go out and find other expertise that could help in this project. It's easiest to do it if it's at Stanford because we can more easily collaborate. But we're looking also anywhere in the world for people who have expertise that we don't have, and we'd like to have them work on certain types of projects.

So, we're ever increasing and expanding the things that we work on, and we have to look basically everywhere to figure out what is causing this disease. If we can really figure out the cause, that's great hope for figuring out how to cure it. Or, at least treat it. I'm open and pushing for a cure. And although that sometimes can be quite difficult, but that's my major goal. We collect data and we look at how we might use it for a treatment or cure.