Learn about new clinical trials in MOGAD

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Dr. Benjamin Greenberg: [00:00:04] Your calendar for today says Dr. Levy will be talking about experimental treatments for MOGAD and NMOSD, he's not. He's only talking about MOGAD because he didn't read the instructions completely. And you can give him all the feedback you want. He thought I was gonna keep that quiet, I didn't. And secondly, he thought I was gonna keep quiet that his preferred name is Mikey and not Mike. So, feel free throughout the conference. Mikey take it away.

Dr. Michael Levy: [00:00:35] Thank you Benji. Thank you. So, I teased Ben about calling him Benji and since then he's taken it out on me calling me Mikey. Which is fine, I really don't mind if brother calls me Mikey. But it is true I am gonna focus today's morning presentation on the research trials in MOG antibody disease. There are still a couple ongoing in aquaporin-4 NMO, and I'm happy to take those offline with anybody who's interested. So, Anastasia did a great job explaining the difference between a preventive therapy and an acute therapy. I wanna mention that there's research being done in both. And I'm gonna present some new data that's never been shown before. So, you are among the first to see this. And this is an acute therapy for MOG antibody disease.

[00:01:18] So just to remind you what acute therapy is. So, you have an optic neuritis attack, and you suddenly lose vision, and you come into the hospital. And an MRI or by other means we can tell that there's inflammation in the optic nerve. The goal of treatment at that point is to get rid of the inflammation in the optic nerve. We wanna suppress it, we wanna get rid of it, we want to get to the point where you're starting to heal. And there are many ways of doing this. Historically, since the 80s people have been using corticosteroids for multiple sclerosis. This is the methylprednisolone. Sometimes we give it in pills. You might be taking 25 pills at home, that kind of thing. All of that history comes from multiple sclerosis. And as you can imagine when MOG rolled around and people had an optic neuritis associated with MOG as opposed to multiple sclerosis, we just tried the same thing and steroids work really well in MOG.

[00:02:26] But I think if you're my patient and you're on steroids, I can understand why you're not happy with me because steroids have so many side effects, so many toxicities, especially in the long term. I really have been trying to avoid it. But not just that, in MOG antibody disease specifically, when you use steroids and then you try to come off of the steroids, that often will trigger a relapse. In fact, in my experience, it's one of the most potent triggers for MOG relapses. Put somebody on steroids and then suddenly stop it, and then

almost certainly in the next few days or weeks you're gonna get a relapse. So, for all those reasons the whole field has really kind of shied away from steroids trying to find better ways of treating MOG antibody disease. And you saw some of the preventive data Anastasia presented for MOG antibody disease. That's to prevent relapses. But IVIG is also good for treating acute relapses. At least that's the data I'm gonna show you today.

[00:03:31] This was a multicenter study. We had seven centers around the world. I think two or three in the US, and Germany, and Israel, and I can't remember the rest. And it only amounted to 39 patients. But that's pretty good if -- this is how observational studies start. You just put your experiences together, you put a story out there, and then it builds momentum. So, these 39 patients were mostly -- they were all MOG and they mostly had optic neuritis when they came in, but also Some kids with ADEM.

[00:04:10] The treatment course was fairly similar. Everyone got this high dose. It's two grams of IVIG for every kilogram of body weight. And that seemed to be the treatment of choice for most of these people. A lot of people more than half still had steroids on board in some form or another, because it's really hard to deny anybody steroids. They work so well, and you can't see in one eye, and here I am, I'm gonna tell you that I'm not gonna give you a treatment that could restore your vision. That's a really hard thing to do. So often times people would get the steroids and either use IVIG at the same time, or they'd wanna see if the steroids worked and if not, then use IVIG. 'Cause this is all still very experimental stuff. MOG is still such a new disease, and all of these cases were taken from the past couple of years.

[00:05:07] So this is still kind of new stuff. But here are the results. And let me just walk you through this. There's two graphs here, the one on the left, it's called the EDSS score. It's a measure of disability. The higher the disability the worse. And just to give you a sense of what a four is, you could see a four was the average disability when patients came in. That's no vision in one eye, essentially is a score of four. But it's also a measure of mobility. So, if you had a transverse myelitis, a score of four might be a limitation in strength in one or two legs, for example or something like that. And this graph on the right is visual acuity. VA is visual acuity. And these numbers are converted, you go into the doctor's office and that big E is 20/400. That means that you have to stand at 20 ft to see the E, whereas someone with normal vision can stand at 800 ft away. But all of these numbers can be converted into a linear scale and that's the Y axis here. And the point of these two graphs is to show you that after IVIG therapy scores go down and that's good. They all improve. They improve in EDSS score, they improve in visual acuity. These are statistically significant. Not just at the time the patient is discharged, they're already doing better with IVIG, but also when they come in for their follow up clinic visit about three months later on average.

[00:06:39] So these were really encouraging data and we're sending it in for publication. I hope it gets the field thinking about better treatment strategies in the acute stage for MOG. Anastasia already presented the data for preventive, but this is something that we're still sorting out because one of the things we noticed with prevention therapy is there is a difference in dose. The lower dose or one gram of IVIG per kilogram of body weight has more failures and the higher dose has fewer failures. But I can tell you the number of side effects from the higher dose is much higher than from the lower dose as you might expect. So, this is a balance of risks. You have to think about what the risk of failure is and what the side effect profile is gonna be when you make these decisions.

[00:07:35] I think there was a question about how long you have to treat, and Anastasia said about two years. I think that's kind of what we usually do, but there's no data behind that. It's just kind of something that we've started doing because it feels right. That's when MOG antibody levels sometimes disappear and that's always encouraging because that suggests the immune system isn't angry at MOG anymore and stop making an antibody to it. That's a good sign that maybe the disease is kind of going away. My colleague at the Mayo Clinic who you met yesterday, Eoin Flanagan. His number is about four years. He likes to treat for

four years and feel more confident that the disease is not relapsing. And maybe a lot of this comes down to biomarkers. Maybe the MOG antibody level, maybe something else that will tell us, okay, the disease is finally calming down and you can start either maybe going down on the dose of treatment or stopping it altogether.

[00:08:36] So there are new MOG trials that have launched and I'm gonna talk about two of them in more detail because they have already launched and they are public, and then one that is planned to launch. The two that have launched already: one is rozanolixizumab, I'll call it rozumab, and satralizumab. Satralizumab you've already heard about from Anastasia. This is the same IL6 that's already approved in NMO in the aquaporin-4 that's now being tested in MOG. And then tolebrutinib, which is a drug being developed for MS, is something that we're looking at from MOG as well.

[00:09:17] These slides come from the pharmaceutical company, so they have a different format and it's kind of there. I'm going to give them credit for making these slides. They're a lot prettier than the ones I made. The first study is rozumab. This is a drug that is thought to work very similar to IVIG. And a lot of the diseases where IVIG is helpful, drugs like rozanolixizumab are being developed for those diseases and many of them have already been approved. So IVIG Is helpful in myasthenia gravis and a drug like this has already been approved, and this one is also being tested in myasthenia gravis. Same with CIPD you heard yesterday a case of chronic inflammatory demyelinating polyneuropathy. That disease is treated with IVIG, and this drug has been approved to treat CIDP. Maybe not this brand but drugs that work like this. And they work on a mechanism that recycles your own antibodies. So, when you take this drug your antibody levels drop a lot. It's kind of like plasma exchange. When you get your antibodies removed, many of you have had plasma exchange, but this one is removed internally. Your own blood vessels take up the antibody and recycle them. So that's how the drug works. And it's a once-a-week dosing and it goes under your skin. But unlike a tire formation that might form with IVIG, this is very small volume. It's less than a teaspoon. And it's too big for the FDA to allow in an injection. It goes through this little pump it goes into your skin through a patch. That's the mechanism.

[00:11:02] Now the study design is intended to prove that it works and in order to do that you need a placebo arm. This was controversial at first. Some people felt like MOG patients should not have to enroll in the study with a placebo arm because what if there's a relapse that occurs and you lose vision. So, we debated that for a long time and the consensus was that most MOG attacks if you treat them quickly don't lose vision permanently, and that if you treat them quickly you often will recover vision pretty well. And so, for that reason, the consensus was that scientifically this is the first trial that's launched we need to know. We need to know if the drug works and therefore you have to have a placebo group.

[00:11:49] Here's the trial design. If you enroll, you have a 50/50 chance of being in the treatment arm. But if you do have a relapse in either arm, if you're in placebo or treatment, we never know it's blinded. But if you do have a relapse, you're automatically in a new arm called the open label phase where you definitely get drug and it's not blinded anymore. And the idea there is, we really only want you to have one relapse because we don't want you to relapse over and over and over again if this treatment is not working for you. After a certain number of relapses has occurred altogether, then the trial will end, and the statisticians will take the data and tell us statistically whether or not this drug can prevent relapses.

[00:12:29] The inclusion criteria are pretty straight forward. You have to have MOG disease, MOG antibody disease. The drug launched before there was widespread consensus, but it fits most people's understanding of what MOG is, which is a relapsing disease, causes optic neuritis or transverse myelitis associated with the MOG antibody. In this trial, we do not want people who have just one MOG attack because a lot of people like that will never relapse. If we put you on a drug, how do we know if it's the drug or just the natural course of your disease? So, we want you to have a second attack at some point in your life, because then we'll know

you have a relapsing form. And then we don't want you to go 10 years in between attacks. So, we want you to have one attack in the past year as proof that you have like an active disease process. And that way if you don't relapse on the drug in the next year or so, that gives us some confidence that maybe the drug is actually working. So those are the inclusion criteria.

[00:13:50] There's a lot of activities involved in being in a study. I think it's kind of fun. Patients come in, they get treated like VIPs, I hope. We give them free parking, you get a bagel, you get coffee, and then you do about an hour and a half worth of surveys. Everything from, do you want to kill yourself? Which I hope is no to, how much pain do you have? And what limitations do you have? Things like that. Lots of different surveys because we're interested in quality of life. And we do EKGs and blood tests and everything else and then you get the drug. And it's kind of anticlimactic because there's not a lot of -- we're not expecting much, we just wanna prevent the next attack. We're not trying to fix anything permanently or see a change or make your legs start working or your eyes start working. That's not the point. We just wanna prevent the next attack. So, you get the drug and then you go home, and I hope not to hear from you because if I hear from you, I get worried. So far, the trial has launched. We have about maybe six people who have enrolled worldwide and we need about 100 something. It probably won't be at next year's SRNA that will be presenting results. It'll probably be sometime around 2025. That's the expectation.

[00:15:14] Okay. And then the second study is with satralizumab. This trial is called meteoroid, and it's also blinded placebo-controlled phase three, meaning we really, really, really want to know if this works. We don't just wanna know if it's safe. We think it's safe because MOG patients use satralizumab as Anastasia mentioned and it seems to be very safe in NMO patients. So, safety is lower on our list of concerns. Now we're running this phase three trial to just see beyond a shadow of a doubt, does this drug work to prevent relapses? It has a lot of rationale. There have been many MOG mouse models where IL-6 is very high and blocking IL-6 works really well. There also studies in people where IL6 is really high. At this MOG symposium that we hosted a couple weeks ago, a case from UCSF in MOG had super sky-high levels of IL-6 in the spinal fluid. And the thought is, if you block that inflammation, block that pathway that IL-6 pushes, could you treat the inflammation and MOG? Now this is a preventive approach. The drug is injected once a month and it lasts all through that month. And so, we're hoping that by doing that we could prevent MOG attacks.

[00:16:43] Trial design is very, very similar. This was also launched before we had any drugs approved. So, there was again the same debate about placebo but the same conclusion. Which is, that if you're in the study and we know right away whether you're having an attack, we treat it very quickly and hopefully there's no damage as a result. And then at the end of it we get a very clean scientific answer, does the drug work or not? And one thing I should mention, there is one difference between these two studies. In this trial with satralizumab, the way they did it with aquaporin-4 NMO in Europe is they allowed people to stay on mycophenolate and azathioprine if they were already on it, and that they were stable on that dose. There was a concern at the time again about placebo. You can be on placebo in this trial, but you could also be on one of these two drugs. Now if you failed one of these two drugs, you have to wonder about whether you should stay on it. But for some people having this on board gives them a little bit of comfort, that maybe if they end up in the placebo arm, they're on something. And that would hopefully prevent attacks for them.

[00:18:02] Now when the FDA evaluated the NMO results in aquaporin-4 where people were on background therapy, there was a conclusive enough picture overall to make an approval for aquaporin-4 NMO. So, they're hoping for the same thing here. So, you are allowed on background therapy. Very much the same patient population. We want relapsing disease. There's a little bit more flexibility in your last attack. It doesn't have to be in the last year, it could be two in the last two years. But we still don't want people who've been in remission forever because then we don't know how to interpret remission for another year.

[00:18:43] The end points are the same. We wanna prevent attacks. Again, you're only allowed to have one attack and then you're out of the blinded phase. Okay, so two very similar trial designs and they'll both read out probably around the same time I'm guessing. The third trial is with a drug called tolebrutinib. Now this one has not launched yet, we don't have FDA approval, and this is not a sponsored trial meaning the pharmaceutical company is not running this. This is something that we approached them about and said the good rationale for this which I'll run through real quick. And they gave us a little bit of money to run this study. But it's not their study, this is really our study. And the drug is called tolebrutinib. It is being developed for multiple sclerosis. And the rationale is based on these complicated pathways here. These are immune pathways. I'm showing you three of them. They all have BTK. You can see in yellow. BTK is involved in signaling. So, when your immune system gets activated, BTK is important in at least three of these activation pathways. One of them is when your antibodies bind to cell, that's the one in part C. There's another one in part A where B cell gets activated through its B cell receptor. BTK is involved there. And in part B those pathways where there's no antibody but there's something in the environment that triggers the immune system. BTK is involved there. And BTK is always inflammatory. And so, the idea was -- and this is not new for MOG but even in MS and in other diseases that are autoimmune. The idea was, well if you block the BTK pathway, this Bruton's tyrosine kinase pathway and stop inflammation through these pathways, can you improve outcomes?

[00:20:45] In multiple sclerosis this went through a phase two trial and the outcomes were primarily by MRI. So, these are the two graphs that I'm gonna show you. MRI lesions with the 60 mg dose was -- what is it? 85% reduction in number of new lesions in MS, enhancing lesions and then regular lesions 89%. Yeah, 89%. So dramatic reduction in the number of MRI lesions with this drug and MS. And for that reason, they have launched phase three trials in MS. Four of them different forms of MS. And we approached them and said, based on this mechanism of BTK, we think it could be involved in MOG as well. There are lots of mouse studies using MOG where this drug is helpful. And based on the mechanism of BTK involvement in B cell receptors and in cells that take up antibodies and also in the brain, we think that this might be helpful.

[00:21:55] There are a lot of BTK drugs and there are a lot of them being developed for MS. There are four of them listed here and they're all competing with each other. When you go to MS meetings, you can hear them all. They have very similar side effect, they have class effects, things that we have to look out for example arrhythmias. So, part of this study patients have to take a little EKG machine home and they put their fingers on it. You've seen the Apple devices where you put your finger on this device and tells you if you're in an arrhythmia or not. We're using that device for this trial. So, there are a lot of different issues that we have to consider, but the drug class in general is they're very helpful in MS and we think also in MOG. And there's one extra little perk to this drug from MOG and that is that that BTK pathway is also involved in immune cells in the brain. So, we're really hoping to calm the immune system down, not just in the periphery and in lymph nodes but also in the brain in specific cells called microglia.

[00:23:05] This is a Phase two trial design. There will not be a placebo arm. And the reason for that is that we're just not even sure that this drug works in MOG. I can't find a single MOG person in the world who's ever tried this. This is really just rationale. We don't think it would be unsafe, but we just don't know if this would work or not. So, we're trialing it in 25 people. Again, we're targeting recurrent MOG disease, we're trying to prevent relapses, we wanna see if it's safe at the 60 mg dose, we wanna see if we can prevent relapses or if relapses just progress as usual then we'll know it's probably not much better than maybe other MS drugs. We're also gonna look at MOG antibody titers, see if those change.

[00:23:55] The cool thing about this trial is gonna be entirely remote. We pitched this trial to the company when COVID was in full swing, and everything was shut down. We thought, how we're gonna do a clinical trial? And we thought, we could really do all of the assessments over Zoom. We're doing it anyway for our patients

through telemedicine. So, let's see if we can put the whole thing together with the arrhythmia evaluations and relapse evaluations and everything. And we managed to do it, so this is gonna be conducted entirely by Zoom. So, although the center for the trial will be in Boston, the patient can be anywhere in the US, and they just need to have an iPhone or an iPad or a laptop. So, we hope to launch coming year and I'm hopeful that we'll present around the same time as the other two trials, but we'll see.

[00:24:50] And last, I just wanna leave you with this idea of a phenomenon called tolerization. Tolerization is the approach to treating autoimmune disease that it's really -- if you think about how your immune system got turned on to something. In the case of MOG your immune system got turned on to attack MOG, in NMO it got turned on to attack aquaporin-4. In other autoimmune diseases, there are other targets. But just in the same way that the immune system gets flipped on, we have technology now to flip that off, and that technology is called tolerization. There are many, many different approaches to doing it. And MOG is one of the most exciting proof of concept models for this approach of tolerization because it happens naturally, people get over their MOG disease sometimes. And so, if the immune system can reset itself and can turn off the response to MOG, then all we need to do is just push it in that direction. We need to figure out how to do that.

[00:25:54] We had a great symposium a week ago in Boston where we invited a bunch of companies. And I can tell you that there are at least 10 companies that are interested in MOG specifically. You might be wondering like, why MOG? Who all has heard of MOG? It's a rare disease but it's got a wonderful mouse model that's been around for decades, and it has a very clean outcome. We have MOG antibodies, we have yes or no, did you relapse? We have really clean outcomes. And if it works for MOG, these companies are not gonna make all their money on MOG. They're gonna take it to other autoimmune diseases that are much more common. But MOG is thankfully a very nice proof of concept model. So that's why all these companies are interested and we're happy about that. We're working with anyone. We don't have any money on any particular company or race here. We just wanna get something that works. So, thanks for your time. I'm happy to take questions.

Dr. Benjamin Greenberg: [00:27:01] And while people are thinking of questions, we do have multiple from online that I'll pepper in, and some of them are gonna be for Anastasia. So, I asked her to join up at the table. So, on the research side, somebody asked, "Do you have an idea of when the first FDA-approved treatment could be made available for MOGAD?" If you had to think of a timeline, what would you expect that first possible date?

Dr. Michael Levy: [00:27:27] I think 2025 is a reasonable target. Based on the recruitment so far, we've had for MOG trials, I think that that's a realistic outcome. 2025.

Dr. Benjamin Greenberg: [00:27:38] And then the other question which you partially actually already addressed for the non tolebrutinib studies that you mentioned, somebody asked, "Do you have to travel for these studies?" And you brought up the comment that yours is completely remote, which is incredible. For the others, are the traditional studies where you have to go to a site that's enrolling?

Dr. Michael Levy: [00:27:55] Yeah. The other two do require travel. Companies are happy to pay for it because you can imagine it's much more cost effective to pay for someone's travel than to keep the trial running indefinitely. So, they will pay for travel for a reasonable budget and it's not a constant trip. So, for one of the trials, it's a drug every week, but after a certain period of time you can get a home care company to do them. For the satralizumab trial, I think you have to come for the first 12 weeks to the site and then after that you can do the injections at home. So, there are ways around the travel issue, but you do have to at least show up a few times.

Dr. Anastasia Vishnevetsky: [00:28:39] There are many sites involved.

Dr. Michael Levy: [00:28:40] Oh, and that's a good point. Anastasia mentioned there are many sites involved in those two phase three trials. For rozumab I think they're at least eight in the US and for satralizumab I don't remember but they're gonna be several. Washington D. C. has already launched, we're gonna be [a site], San Francisco and so on.

Dr. Benjamin Greenberg: [00:29:02] Okay. So still from online -- Anastasia I think this one's for you. Somebody indicated that they're 22, they were diagnosed with MOGAD in August. Titers are 1 to 1000. They don't have any other diagnoses and have only had one attack so far in February which presented as optic neuritis. And the question was, "Do you think I should start on IVIG?"

Dr. Anastasia Vishnevetsky: [00:29:26] Yeah, I think this is a great question and always a hard conversation. I think that the consensus I would say within the field right now and likely what we would recommend as well is not to start on a long-term preventative after that one attack. But I think there's a lot of other things that go into that decision. So, if the recovery has been really, really tough especially despite acute treatment and essentially one of your eyes is not really working then I think that's a conversation that might be something that pushes in a different direction. But I think in general we don't typically recommend treatment after a first attack.

Dr. Benjamin Greenberg: [00:30:14] And this next question is still for you since you have the mic. Is an interesting one. They say, "Hi, I am wondering, what is the risk of getting PML on rituximab? I understand it is low, but I'm still concerned." This person is writing us from Canada and they're not sure if they can get the blood test done to look for antibodies to the JC virus. And they had a second part to the question which is about managing rituximab and pregnancy. That they're getting different advice on how to pursue pregnancy under rituximab. So, let's do the PML question first, and does everybody here know what PML is?

Dr. Anastasia Vishnevetsky: [00:30:50] So, yeah.

Dr. Benjamin Greenberg: [00:30:50] You might want to brief –

Dr. Anastasia Vishnevetsky: [00:30:52] So two great and tough questions. So PML is progressive multifocal leukoencephalopathy. It is a potentially fatal but always very serious brain infection, and it's due to a virus, the JC virus that most of us actually have or many of us have latent in our body. So, we were infected at some point, it's not really causing any symptoms or any problems, we almost certainly don't know about it, but in the context of certain immunosuppressive medications, that virus can be reactivated and cause this really serious devastating brain infection. The context a little bit if we're thinking about rituximab and PML is there's a few particular MS medications that can really increase that risk of PML significantly. Natalizumab or tysabri was one that increases simply. And so, before starting patients on many of these medications, we'll test for the JC virus antibody. And in general, if you don't have the JC virus antibody, then you almost certainly don't have that risk of PML. Obviously, you can get JC virus and that can kind of transition, but if you don't have JC virus, you're not gonna develop PML.

[00:31:38] Rituximab, there have been quite a few cases associated with rituximab with PML, but the caveat is that the majority of those cases were in the setting of other immunosuppression. So, rituximab was initially used in cancer patients with lymphoma and leukemia, or it's also used in transplant patients who have heavily immunosuppressed. So, a lot of the PML cases in the context of the rituximab occurred like that.

And then in tocilizumab which is a medication very similar to rituximab in many ways. And rituximab has both had a couple of several cases of PML, but in many cases it was not clear whether those were related to old immunosuppression. And there were other potential risk factors or other -- patients might have been on a medication that increased the risk of PML.

[00:32:36] So I think it's tough to give an exact estimate in patients who are not otherwise immunosuppressed, like NMOSD or MOGAD patients. I'd typically it would be significantly less than one in 10,000. For rituximab, and it's not one of the complications in again, otherwise non immunosuppressed NMOSD patients that I'm most fearful of. There's other infections that can occur, blood account suppression and things like that. But I think it's tough to give a precise estimate of just rituximab alone.

Dr. Benjamin Greenberg: [00:33:47] And then briefly because we're going to give people a break, the pregnancy issue.

Dr. Anastasia Vishnevetsky: [00:33:51] Yes. So, for pregnancy also a complicated issue because originally the guidelines were that, or the guidance was that you should not use rituximab if you are pregnant. That was guidance in multiple sclerosis for quite some time. However, in neuromyelitis optica the relapses can be very severe and having a patient not on any kind of therapy while pregnant can lead to really devastating attacks. So, the thing that we try to do most commonly, and this is not something that everyone has consensus about, what we'll try to do is treat before a patient is trying to conceive and starting to think about pregnancy. So that they have uh protection and immunosuppression during their pregnancy, and then retreat after delivery so that they're protected in the postpartum period which can be associated with a higher relapse risk. So, they are essentially getting immunosuppressed -- they have low B cells during their pregnancy. Sometimes rituximab can often last longer or the effect of rituximab can last longer than six months, sometimes up to 12 months, but often eight, something like that. So, we'll try to get them essentially to be covered and try to avoid retreatment during pregnancy. But if that's done, it doesn't look like there's a huge -- there's not a ton of data but it doesn't seem like there's an obvious safety risk.

Dr. Benjamin Greenberg: [00:35:32] Great. And while we can't get to all the questions that are online, I apologize. The last one that will get to is for Mike. It's an interesting one. They said, "What is considered "very high" MOG titer?" When we use that term, very high, what does that mean?

Dr. Michael Levy: [00:35:48] We usually use very high as 1,000, 10,000 or I think even 100,000 I've seen a couple of times. And the reason that we categorize them that way is because there are people who have lower titers, 20 and 40 who have a high risk of having a false positive, of having maybe multiple sclerosis with an antibody that's not meaningful. And then 100 is kind of in between. It's about 80, 82% specific but still some false positives. But once you get to 1000 and higher then it's like a perfect test for MOG antibody disease.

Dr. Benjamin Greenberg: [00:36:30] All right. So, thank you both. We are gonna move to the break which we ate into a little bit, but we still have 20 minutes. It's taking place in the [Carmel] room which is your exit is to your right. There are refreshments and the exhibitors are up, and we encourage everyone to take the time to network. And we will start back here at 11:00 with Dr. Becker. Thank you, guys. Thank you.