

My prog-MS ezine

For people with progressive MS and other interested people

Issue 51, November/December 2023

Hello and welcome to the 51st edition of my ezine about progressive Multiple Sclerosis and MS progression in general. My name is Ian Cook. I'm a secondary progressive MSer from Birmingham, UK. I'm 65.

In this 51st issue, there are as usual eight pages of prog-MS news stories. There are also two features. One on page 5 is a feature I never thought I would write. It's about a comparison between MS and leprosy. The comparison is made by an eminent Professor of Neurology. Read more and be surprised.

There's another feature asking if all MS is, in reality, progressive right from the start. I think it is. See page 10 for further details. Picture below thanks to vitor-camilo-7NoYTT9OmiI-unsplash

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News

New EBV drug data for nearly 100 prog-MS patients due in weeks

As we went to press Atara Biotherapeutics was planning to release the one-year data from its EMBOLD Phase 1/2 study, which is testing the company's experimental treatment ATA188 for progressive MS which focuses on EBV (Epstein Barr Virus) as the cause of MS.

ATA188 is an experimental therapy that uses immune T-cells to fight off EBV-infected immune cells in MS patients. It contains allogenic T-cells obtained from healthy donors who have previously been infected with EBV; the cells are, therefore, primed to recognize certain EBV proteins and launch an attack against the infected cells.

Atara has been testing the therapy in people with progressive MS in its two-part EMBOLD trial (NCT03283826) launched in 2017. The trial specifically examines how safe and well tolerated ATA188 is, and how well it improves clinical disability in people with nonactive primary progressive or nonactive secondary progressive MS.

Data from the trial's Phase 1 part and its four-year extension portion showed that ATA188 may help patients achieve confirmed disability improvement, or a lessening of disability, defined as a reduction in EDSS (disability) scores recorded in at least two consecutive study visits. Of the trial's initial 24 patients, seven had disability improvements and 13 had maintained stable disability scores over about three years of follow-up. This benefit was maintained over time, with patients who experienced improvements continuing to have them at least after an average of more than two years.

In the Phase 2 part, patients were randomly assigned to receive ATA188 or a placebo, given in two treatment cycles, for one year. Then, in the second year, patients in the placebo group also received two cycles of ATA188, while the other group were given one cycle of ATA188 and one cycle of a placebo. After completing the two-year trial, patients may enter an open-label extension and receive ATA188 for up to three more years.

So far, data have come from an initial group of 34 patients observed for at least six months and 15 patients who were followed for at least one year. However, that early data did not provide enough evidence to make definite conclusions about the benefits of ATA188. In addition to announcing results from the primary outcome measure of confirmed disability improvement, the company anticipates sharing data for patients who have been observed for longer than one year.

The results to be published in November will be based on EDSS scores and the findings of other measures obtained from more than 90 patients who have completed at least one year of follow-up in the study. ***For details of sources for all news stories go to page 14***

Foralumab nose spray led to less disability in SPMS, trial shows

Six months of treatment with the nasal spray Foralumab, led to decreases in microglial activity in five of six people with nonactive secondary progressive MS (SPMS) involved in an expanded access program trial There was evidence of lower disability too.

Microglia are resident immune cells in the brain believed to play a role in driving inflammation and nerve damage in MS. The new data were shared at the European Committee for Treatment and Research in Multiple Sclerosis (ECTRIMS) and the Americas Committee for Treatment and Research in Multiple Sclerosis (ACTRIMS) joint meeting, held Oct. 11-13 in Milan and online. The poster was titled "Treatment of six non-active secondary progressive MS patients with nasal anti-CD3 monoclonal antibody (Foralumab): safety, biomarker, and disability outcomes." Tiziana Life Sciences, the company developing Foralumab, funded the work.

Foralumab is an antibody-based therapy delivered through the nose. It's designed to inhibit the activation of T-cells. The activation of this type of immune cells is thought to play a central role in MS-driving inflammation. Foralumab specifically targets a T-cell protein called CD3.

The poster presented at the ECTRIMS-ACTRIMS meeting shared data from six people with nonactive SPMS who received Foralumab through expanded access programs. All of the patients had experienced clinical disease progression despite treatment with available disease-modifying therapies. Each was given Foralumab in three-week cycles The first two patients now have been on Foralumab for more than a year, and the other four have been followed for at least six months. The experimental therapy has thus far been well tolerated, with no severe side effects reported.

Data from PET scans of the brain showed that, after six months of treatment, five of the six patients showed a reduction in activity of microglia, which are a type of inflammatory brain cell. The sixth patient showed an increase in microglial activity at six months. Overall disability scores, decreased in two patients, after six months on Foralumab. In the other four patients, EDSS scores were stable over the six months. Three patients experienced improvements in a test of walking and four reported less severe fatigue after six months on Foralumab. These values were largely stable for the remaining patients.

Tiziana now is planning a Phase 2a clinical trial to continue investigating Foralumab in nonactive SPMS patients. The company is looking for about 6-10 clinical sites to conduct the study, and patient screening is expected to start some time in November**. For details of all news stories go to page 14**

New evidence further links EBV to MS, explaining auto-immunity

Infection with the Epstein-Barr virus (EBV) may prime the immune system to accidentally attack a brain protein called ANO2, new research shows. The findings may further explain how EBV infection can lead to MS developing.

The new research adds to existing evidence of "cross reactivity" as a driver of auto-immunity in MS where it is already believed that EBV transcription factor EBV nuclear antigen 1 (EBNA1) primes the body to react with central nervous system protein glial cell adhesion molecule (GlialCAM) .

In the new research Daniel Jons, PhD, a scientist at the University of Gothenburg, Sweden, presented his findings in a paper titled, "Epstein-Barr virus seroreactivity, putative autoimmunity and axonal injury in pre-symptomatic multiple sclerosis."

The immune system normally defends against infectious invaders like EBV, but in MS, it attacks healthy brain tissue. While it's not known exactly what causes this self-targeting immune attack, but infection with EBV has been identified as a strong risk factor for MS, with virtually all MS patients having a history of EBV infection.

EBV is best known for causing infectious mononucleosis ("mono"), also called glandular fever, and at least three cancers, but it also can cause unspecific mild illness or no noticeable symptoms at all. Most people have been infected with EBV by the time they reach adulthood. It's thought EBV may set the stage for MS due to an immune phenomenon called cross-reactivity. The basic idea being that certain pieces of the EBV virus are structurally similar to molecules normally found in healthy brain cells.

When the immune system attacks EBV, it creates antibodies and other molecular weapons that can stick to specific pieces of the virus, but these may also stick to brain proteins that have a similar shape, causing the immune system to mistakenly attack the brain. Research has specifically suggested that an EBV protein called EBNA1 has a similar structure to a brain protein called GlialCAM. MS patients are frequently positive for antibodies against EBNA1, studies have shown, and data suggest anti-EBNA1 antibodies are detectable before the earliest biological signs of nerve damage in people who go on to develop MS.

The new research now shows that some EBV positive MS patients have antibodies that target Anoctamin2 (ANO2), a protein on nerve cells and other supporting cells in the brain. Some data have suggested an immune attack against certain EBV proteins may prompt an attack against ANO2 through cross-reactivity, similar to how the attack on the EBNA1 protein prompts an attack on GlialCAM**.**

For further details see page 14.



Will the real MS say 'hello'? Or why MS is like leprosy

When I was diagnosed back in 1993, I was told my MS was relapsing. I was also told that after several years this relapsing phase would likely move on to secondary progressive MS where existing symptoms just get worse and worse.

However, according to an eminent British professor of neurology, this is not what the "real MS" is like, and his view chimes with my own experience. Prof. Gavin Giovannoni of Barts Hospital in London has also made the rather startling comparison of MS with leprosy, but more of that later.

Back to my "MS journey". Yes, there were early relapses in my own MS but it did not "jump" neatly from the relapsing phase to secondary progressive. Progression or to be accurate Progression Independent of Relapse Activity, (PIRA) was there right from the start. My MS was always progressive.

My first brush with MS, was back in 1987, aged 29. I woke up one morning and found my left hand was useless. I couldn't button my shirt, or tie my shoe laces. By the time I finally got to see a neurologist a month later everything was pretty much back to normal. This was a classic relapse.

Around this time, I was also having problems urinating. It was difficult to pee and I didn't feel as though I was fully emptying my bladder. This problem gradually got worse over the years with me starting to use catheters around 2010. I would say my urinary problems which began in the 1980s were PIRA (progression independent of relapse activity).

In a paper published in 2022 titled: "Smouldering multiple sclerosis: the real MS." Professor Gavin Giovannoni said: "The 'real MS' is driven primarily by a smouldering pathological disease process. In addition, the progressive accumulation of disability in MS can occur independently of relapse activity from early in the disease course."

Continued on page 6

Continued from page 5 The reason why this matters is that while MS is regarded as an inflammatory disease of the central nervous system driven by autoimmunity it is easy to parcel it up with the other "auto-immune diseases", with which it does share several similarities. If "smouldering MS" is the real MS, as Prof. Giovannoni suggests, it means MS is something of an outlier because it is primarily a neurodegenerative disease and it is hard to find other similar diseases which "smoulder" for decades, gradually getting worse.

Strangely, and you will be surprised when I say this, the other neurological disease that fits neatly into this "smouldering" category is leprosy, an infectious disorder affecting the peripheral rather than the central nervous system. And if you think this comparison is as crazy as the proverbial box of frogs then I invite you to read one of Professor Giovannoni's MS selfie blogs called: "Will the real MS say hello?". In the blog Prof Giovannoni says the following:

"Multiple sclerosis can be viewed as an inflammatory neurodegenerative disease with the clinical spectrum or phenotype determined by the presence or absence of focal inflammation, similar to that which occurs in infectious diseases, e.g. leprosy."

Obviously, MS is not in any other way connected with leprosy but Prof. Giovannoni's comparison is valid because other chronic demyelinating diseases are caused by infection, not just MS. If his observation leads us to viewing MS as an inflammatory neurodegenerative condition rather than an auto-immune disease it does us all a service. Recognising "the real MS", as Prof Giovannoni puts it, is the first step on the long road to finding a cure for it, something we have failed to do in the 150 years since MS was first identified.

Picture P5Leprosy deformities hands, approx. 2022, Rajahmundry, India https://commons.wikimedia.org/w/index.php?curid=27098744By B.jehle - Own work, CC BY-SA 3.0, https://commons.wikimedia.org/w/index.php?curid=27098744 **For further details see page 14**

Cows' milk proteins likely trigger auto-immune response in MS

A significantly higher immune response against proteins found in cows' milk is seen in people with MS, likely because of similarities between milk proteins and proteins in the human central nervous system (CNS, the brain and spinal cord), a study has reported.

No such differences were seen between patients and healthy adults with almond and other plant-based milks or with sheep's milk, but the researchers cautioned against assuming that plant-based milk alternatives are "automatically a better choice." Rather, they "suggest screening of blood from MS patients for antibodies against different types of milk and milk antigens [proteins that trigger an immune response] in order to establish a personalized diet regimen." The study, "The prevalence of IgG antibodies against milk and milk antigens in patients with multiple sclerosis," was published in the journal Frontiers in Immunology.

Diet has been suggested as a potential environmental factor that modulates the immune response in people with MS. In particular, cows' milk and dairy products may enhance the inflammatory responses seen in the disease. Researchers believe this is due, to similarities between proteins in cows' milk and those in an MSer's central nervous system. When the immune system reacts against milk proteins, some antibodies also react to CNS proteins and damage nerve cells. MS is mostly seen in parts of the world where cows' milk is drunk.

In the latest research carried out in in Germany blood samples from 35 MS patients and 20 healthy adult controls were analysed. Animal milks tested were cow, goat, sheep, and A2 milk (a variety of cows' milk that mostly lacks a form of the beta-casein protein called A1). Plant-based sources were almond, coconut, cashew, hazelnut, and oat milk.

MS patients had significantly higher levels of antibodies against cows' milk, followed by goat milk, when compared with controls. Healthy adults had a significantly higher immune response against A2 milk than did patients. No differences between these groups were seen regarding sheep milk or the various plant-based milks.

Further analyses focused on determining which proteins caused the exacerbated immune reaction to cows' milk. Results found high levels of antibodies against beta-casein, one of the most abundant proteins in cows' milk, in people with MS. A statistical analysis also suggested a potential correlation between high antibody levels against beta-casein and more severe disability, as measured with the Expanded Disability Status Scale (EDSS).

While present at low levels in blood, anti-alpha-lactalbumin antibodies drove the greatest reactivity against cows' milk. However, antibodies targeting beta-lactalbumin, another cows' milk protein, showed the greatest reactivity against brain tissue samples, the researchers reported. **For details of all news stories go to page 14**

News

High hopes for BTK inhibitor Tolebrutinib in prog-MS

Tolebrutinib, a new "BTK inhibitor" drug to treat all types of MS including progressive MS is showing promise in trials for people with relapsing MS. There are hopes it will also work in prog-MS trials which are due to report next year.

Results just published from a Phase 2b trial have shown low relapse rates, stable disability, and few new brain lesions among people with relapsing MS (RRMS). Phase III RRMS trial results are expected in the next few months. Tolebrutinib is also being trialled in primary progressive and secondary progressive MS with these trial results expected in August 2024

Tolebrutinib has the potential to target smouldering neuroinflammation, a type of chronic inflammation that's been linked to MS progression and which many believe is the main driver of disease activity in progressive MS. It is one of a number of MS therapies designed to inhibit a protein called Bruton's tyrosine kinase, or BTK.

Bruton's tyrosine kinase (BTK) is an enzyme essential for the survival and activation of B-cells. B-cells are a type of white blood cell (lymphocyte) thought to be involved in the inflammation caused by MS which causes lesions in the brain and spinal cord and can lead to relapses. BTK also regulates immune cells in the brain called macrophages and microglia which have been linked to MS progression. Tolebrutinib blocks the actions of this enzyme and it is thought that this will prevent the MS activity in the long-term damage to nerve cells which causes progression.

Tolebrutinib is in trials for all types of MS. The trial for people with non-relapsing secondary progressive MS is called HERCULES (NCT04411641), HERCULES is a phase III study recruiting 1,290 participants with non-relapsing (non-active) secondary progressive MS. One half are taking Tolebrutinib and the other half a placebo for up to four years. The main aim of HERCULES is to assess the effect of Tolebrutinib on disability progression, measured as worsening of disability which persists for six months. Other measures include walking speed, manual dexterity, cognitive function, MS activity on MRI scans, and quality of life. Estimated completion date is August 2024.

The other prog-MS trial PERSEUS (NCT04458051), involves adults, ages 18-55 with primary progressive MS (PPMS). This phase III study is recruiting 990 participants. One half are taking Tolebrutinib and the other half a placebo for up to four years. The main aim of the study is to assess the effect of Tolebrutinib on disability progression, measured as worsening of disability which persists for six months. Other measures include walking speed, manual dexterity, cognitive function, MS activity on MRI scans, and quality of life. Estimated completion date is August 2024.**For details of all news stories go to page 14**

News

New Drug Could Be Breakthrough for Late-Stage MS after positive mice tests

A new re-myelination development in a lab at Georgetown University in the US could revolutionize the treatment of patients with advanced MS and slow progression of the disease.

Jeffrey Huang, a provost's distinguished associate professor in the College of Arts & Sciences' Department of Biology, and his team have developed a new drug that reversed the effects of late-stage MS in mice. The drug is now being planned for a clinical trial on people with MS.

"Our team is ecstatic to move to clinical trials," said Huang. "Based on our success in animal trials we believe our drug will help stabilize patients with the disease, reducing not only the number of relapses, but their severity as well."

Jeffrey Huang is a provost's distinguished associate professor in the College of Arts & Sciences' Department of Biology. His new research could lead to a breakthrough in treatment for multiple sclerosis. Huang's approach to myelin regeneration begins by looking at the root mechanics of remyelination. That process starts with the resident immune cells in the brain and spine called microglia. Microglia are like an all-star pit crew for the brain: They clean up damage after it occurs and ensure the brain is ready to keep racing. In late-stage MS, microglia become inflamed and aggressive and have trouble communicating with the cells that spur remyelination.

"Following injury, these cells go from Bruce Banner to The Incredible Hulk, and continue to cause damage to myelin and nerve fibres in the brain and spinal cord," said Huang. "Our drug is able to reverse its phenotype back into Bruce Banner, who can return to doing nice things in the brain."

Huang and his team's revolutionary new drug turn microglia back into their healthy, homeostatic state – and it's already seen success. They successfully achieved remyelination in mice by stimulating the microglia, which then starts a chain reaction in the brain and spinal cord that MS impedes. For people with late-state MS, then, this new drug could revolutionize their treatment, giving patients more healthy years.

The next steps for the drug are clinical trials in humans, which will be conducted by a pharmaceutical company working alongside the Huang team. In addition to the Huang lab, Georgetown is also home to the Center for Brain Plasticity and Recovery, which is dedicated to investigating novel approaches to restore brain function following neurological damage and disease. "The goal is to be able to stop the disease from progressing using our drug," said Huang. **For further information go to page 14**



Immune tolerance drugs– the new "big idea" in fighting MS

Although the debate about what causes MS is seemingly endless, the question of how to tackle it usually focusses on one thing - finding drugs that work on the immune system, making it less active.

Over the years "disease modifying drugs" (DMDs) have aimed to tame an immune system that is said to be "over-active". DMDs, at least one of which is based on a chemotherapy drug, have become so good at lowering the activity of the immune system that some of those taking these drugs have acquired potentially fatal infections that are only seen in those whose immune systems have been wiped out – such as in illnesses like AIDS.

However, lowering the activity of the immune system is no longer the only "game in town". Researchers are asking: is it possible to generate "immune tolerance" so that the bodies of MSers on longer become overly sensitive to myelin developing auto-immunity as a consequence. Clemastine an anti-histamine drug sold over the counter as Tavegil, has been trialled in MS but more of those trials later. There are other things happening in immune tolerance as a stratgey to tackle MS and stop its progression.

Recently Anokion, a US bio-pharma, which describes itself as an "innovative immune tolerance company" finished enrolling patients in a Phase 1 (safety) trial of an experimental drug ANK-700 which it is hoped will induce immune tolerance and prevent the immune system from targeting and attacking myelin. The company expects to complete a one-year safety follow-up in the first half of 2024 and full data should be announced later next year.Safety data so far from the trial called MoveS-it trial (NCT04602390) has shown the drug can induce immune tolerance and prevent the immune system from targeting myelin.."**Continued on page 11**

Continued from p10 Of course, before a new drug can be licensed it has to go through clinical trials and it will be some years before ANK-700, if successful, is available to MSers. One immune tolerance drug that is a little further down the development track is Masitinib which is in phase III trials for both primary progressive MS (PPMS) and secondary progressive MS (SPMS). Masitinib targets mast cells, a type of immune cell involved in allergic reactions. Masitinib leads to a reduced auto-immune response.

A recent Masitinib trial saw 611 participants with primary and non-active secondary progressive MS taking one of two doses of masitinib or placebo for 96 weeks at various doses. The lower dose of masitinib (4.5 mg/kg/day) was statistically significantly better at slowing down disability progression measured by EDSS (Expanded Disability Status Scale) compared to placebo in both primary and secondary progressive MS. A new trial based on the success of the 4.5mg/kg/day dose is currently recruiting 800 participants with non-active primary and secondary progressive MS. The study will test the drug's effectiveness over 96 weeks. Estimated completion date for the study is December 2025.

Perhaps the research that is most interesting for UK based prog-MSers is an over-the-counter drug called Clemastine, sold at pharmacists under the brand name Tavegil. Clemastine is an antihistamine used for treating allergies, such as hay fever, but it is believed in prog MS it could to act on receptors, found on oligodendrocytes, (nerve cells which make myelin). A trial called ReCOVER aims to assess clemastine in MSers with optic neuritis. The placebo controlled study is of 90 participants within two weeks of developing visual signs of this condition. The main measure of the study will be the thickness of the retina at the back of the eye, which is an indicator of myelin repair. Estimated completion date of this trial is less than a year away in August 2024.

Clemastine is best known as an anti-histamine. Histamine is involved in local immune responses communication, as well as acting as a neurotransmitter for the brain, spinal cord, Whether Clemastine works in the same way against MS as it does against hay fever is unclear, but one thing is certain – the ReCover trial along with Masitinib and ANK-700 act in radically different ways in controlling an immune system in MS and represent a new approach to a disease where fresh thinking is desperately needed. Bring it on, I say.

For details of all news and features go to page 14

News

Neubie electrical stimulation device found to help in prog-MS

A course of neuromuscular electrical stimulation using a neuro-bio-electric stimulator, a device dubbed Neubie, led to improvements in strength and range of motion alongside physical therapy (PT) for seven people with progressive MS according to a new study.

Use of the Neubie device, made by Neufit, alongside physiotherapy was seen to help ease spasticity in these patients, all who had progressive MS. The findings were detailed in a new study titled: "A case series in individuals with multiple sclerosis using direct current electrical stimulation to inhibit spasticity and improve functional outcomes," published in the Multiple Sclerosis Journal – Experimental, Translational and Clinical.

The Neubie system is designed to identify areas of the body where nerve-muscle communication is disrupted while patients are physically active. It then delivers direct current electrical stimulation to the skin over that area. This stimulation essentially works to re-educate or reboot the neuromuscular system and restore more normal functioning. Neubie is cleared by the U.S. Food and Drug Administration (FDA) for such neuromuscular re-education, and increasing range of motion, boosting blood circulation, preventing atrophy, and helping in pain management.

In a three-day physical therapy bootcamp hosted by NeuFit in 2021 the device led to measurable gains in function and strength for six MS patients. Now, Courtney Ellerbusch, a physical therapist at Centura Home Health, in Colorado, spearheaded the new effort to further test the Neubie device in MS patients with mobility impairments. Ellerbusch says she was inspired by her patients' motivation to improve their movement and mobility.

Her study involved seven participants with progressive MS whose advanced disease required them to use a walking aid or a wheelchair to perform most of their activities. These individuals completed six weeks of home health-delivered physical therapy, for a total of 18 visits. During that time, they also underwent a four-part electrical stimulation intervention with the Neubie device. With Neubie and physiotherapy two patients now could stand without spasticity. Overall, the Neubie device appeared feasible to use and well tolerated. Only one person missed a single visit during the program, which was due to a urinary tract infection unrelated to Neubie treatment.

Trends toward improved strength and reduced spasticity were observed across various muscle groups in the seven patients. For two patients, that meant they could stand for the first time in more than three years without their usual spasticity. Range of motion and walking improvements also were observed for some patients**. For details of all stories go to page 14**

News

Treating SPMS with OCH shows promise in small study

Five of six people with secondary progressive multiple sclerosis (SPMS) treated with an experimental oral therapy called OCH saw no disease activity over six months in a small clinical trial. That's compared with 0% of the SPMS patients given a placebo, according to new data presented by Tomoko Okamoto, MD, PhD, of the National Center Hospital, Japan.

The findings were shared at this year's joint meeting of the European Committee for Treatment and Research in Multiple Sclerosis (ECTRIMS) and the Americas Committee for Treatment and Research in Multiple Sclerosis (ACTRIMS), held Oct. 11-13 in Milan, Italy and virtually. The talk was titled, "Phase Ⅱ Clinical Trial of NKT Cell-Targeting Glycolipid OCH-NCNP1 for Patients with Relapsing Multiple Sclerosis."

OCH (also called OCH-NCNP1) is a molecule that's thought to activate anti-inflammatory responses, particularly from natural killer T-cells, immune cells that can produce large amounts of both pro- and anti-inflammatory molecules that influence the function of other immune cells. By activating these cells and promoting their anti-inflammatory function rather than a pro-inflammatory one, the therapy would reduce MS-driving inflammation. "[OCH] is an oral drug with a unique mechanism that acts on immunoregulatory" processes, Okamoto said.

Data from a first-in-human study published earlier this year suggested OCH was generally well tolerated and activated anti-inflammatory immune cells as expected. The National Center of Neurology and Psychiatry in Japan, sponsored a Phase 2 clinical trial (NCT04211740) to further explore the safety and effectiveness of OCH in people with MS.

The study enrolled 30 patients, 12 with SPMS and 18 with relapsing-remitting MS (RRMS). Two-thirds of them were female and most had been living with MS for more than a decade. They were given granules of either 3 mg OCH or a placebo, taken once weekly for 24 weeks. The study's main goal was to test whether the treatment reduced the number of new or enlarging MS lesions visible on MRI scans of the brain and spinal cord.

Fewer OCH-treated patients developed new or enlarged lesions than patients given a placebo (6.7% vs. 20%). In the overall study population, the patients given OCH also had a trend toward higher rates of no evidence of disease activity (NEDA), meaning no relapses, no new MRI activity, and no new disability. Two-thirds of those given OCH had NEDA, compared with one-third given a placebo. Among the 12 patients with SPMS, five out of six given OCH achieved NEDA over six months. None of the six SPMS patients given a placebo had NEDA, a statistically significant difference. **For details of all news stories see page 14**

References further details, etc

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For good advice on the trial into ANK 700 go tohttps://multiplesclerosisnewstoday.com/news-posts/2023/09/20/patient-enrollment-phase-1-rrms-trial-ank-700-complete/

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