

My prog-MS ezine

For those with progressive MS and other interested people

Issue 65, March-April 2026

Hello, and welcome to the 65th edition of my ezine about progressive multiple sclerosis and MS progression in general. My name is Ian Cook. I'm a non-active secondary progressive MSer (naSPMS) from Birmingham, UK. My EDSS is 6.5 and rising and my age is 67 and also rising.

In issue 65 there are eight pages of prog-MS news stories. There are also two features. The first, on page 5, looks at the problems of getting a good night's sleep when you have MS, a problem that gets worse the longer you have the condition, as I know only too well.

On page 10 another feature looks at the rather surprising decision made by the US drugs watchdog, the FDA, not to approve tolebrutinib for non-active secondary progressive MS (naSPMS) That's despite having given earlier signs that it would be almost certain to be approved.

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News

FDA says ‘no’ to tolebrutinib for non-active SPMS

In a major shock announcement The US Food and Drugs Administration (FDA) has said it won’t approve tolebrutinib for non-active secondary progressive MS (naSPMS). The EMA (European Medicines Agency) has still to make a decision.

Bio-pharma Sanofi, which has developed tolebrutinib, says it will work to find a path forward with the FDA for the experimental MS therapy despite a ‘complete response letter’ from the FDA indicating the new drug application would not be approved in its present form.

The reasons behind the decision were not disclosed although there are indications that a number of factors including the drug’s effect on the liver may have played a part. (See feature on pages 10 -11).

Sanofi has stated the company remains committed to finding a path forward, asking the FDA to consider scientific experts and patient perspectives alongside their data. So, while the announcement may not be the news American MSers wanted to hear, it may not be the end of the road for tolebrutinib in the US.

Elsewhere in the world regulatory authorities have taken a more positive approach to tolebrutinib and the drug was approved in the United Arab Emirates in July 2025 marking its first worldwide approval. Tolebrutinib is still being considered for approval in Europe by the EMA (European Medicines Agency), with a decision expected in the coming months.

Tolebrutinib is a once-daily pill aimed at modulating the activity of immune cells such as B-cells and microglia (a term for a number of different brain cells which survey the brain, respond to threats, and clear debris).

By inhibiting the BTK (Bruton’s Tyrosine Kinase) protein in these cells, the therapy is thought to reduce the chronic inflammatory processes within the brain and spinal cord that contribute to progressive neurodegeneration and long-term disability in MS.

According to Sanofi this distinguishes tolebrutinib from other disease-modifying MS therapies, which are designed to target acute MS inflammation. Sanofi says tolebrutinib may be the first therapy to target smouldering (chronic) neuroinflammation, a key driver of disability accumulation in MS and had sought approval for the therapy specifically for nonrelapsing SPMS to slow disability accumulation independent of relapse activity.

For details of sources for all news stories go to page 14. Also see feature on 10 which looks at the thinking behind the FDA decision

News

Foralumab nose spray shows improvement in naSPMS

A group of 14 patients with non-active secondary progressive MS improved or remained stable within six months of starting treatment with Tiziana Life Sciences' nose spray drug foralumab.

That's according to an annual report filed with the U.S. Food and Drug Administration (FDA) by bio-pharma Tiziana. The 14 patients with nonactive secondary progressive MS (naSPMS) received foralumab nasal spray for periods ranging from about six months to 3.5 years in an open-label expanded access program (NCT06802328).

This programme provided access to foralumab nasal spray outside clinical trials, and participants were found to have remained stable or improved within six months of starting treatment. There were no drug-related serious side effects reported after long-term use.

Foralumab is an antibody therapy designed to target the CD3 protein on immune T-cells. This is believed to activate regulatory T-cells, which help calm mistaken immune responses, while suppressing the pro-inflammatory T-cells that cause damage in MS.

The therapy has also been shown to reduce the activity of microglia, the brain's resident immune cells that contribute to MS progression in nonactive SPMS and other progressive forms of MS.

In addition to the expanded access programme, Tiziana is running a larger Phase 2a clinical study (NCT06292923) testing foralumab in up to 54 adults with nonactive SPMS. The study, will report results later tis year.

Participants in the trial are being randomly assigned to receive foralumab at either of two dose levels or a placebo for about three months. The main goal is to see if foralumab is safe and confirm that it can reduce the activity of microglia. After the placebo-controlled phase, all patients may receive foralumab for an additional six months to study its longer-term effects.

For details of all news stories go to page 14

Japanese Patent for new drug masitinib in progressive MS

Bio-pharma AB Science has been granted a patent in Japan covering the use of investigational drug masitinib for both primary progressive MS (PPMS) and nonactive secondary progressive MS (naSPMS)

AB Science plans to seek similar protection in other regions for drug's use in MS. The new patent provides protection for this use of the drug until February 2041. This makes Japan the first country to formally grant patent protection specifically for this treatment approach in progressive MS, according to a press release from AB Science. The company said it plans to seek similar protection in other regions.

Masitinib is already covered by worldwide patents for motor neurone disease, a condition often known as amyotrophic lateral sclerosis (ALS) through to 2037, and AB Science also holds patents for its use in other conditions, such as Alzheimer's disease, sickle cell disease, and certain cancers and inflammatory disorders.

Previously known as AB1010, Masitinib, is a small molecule designed to inhibit enzymes involved in the activation and survival of immune cells called mast cells and microglia. It also blocks the activity of tyrosine kinases, which play a key role in mast cell and microglial activation and growth. By reducing their numbers and activity, the therapy aims to reduce inflammation in the brain and spinal cord and slow disease progression.

This approach differs from most currently available MS treatments, which mainly target inflammatory processes outside the brain and spinal cord that drive relapses and active inflammation. These therapies are effective in relapsing forms of the disease but have limited impact in progressive forms.

According to AB Science, masitinib's development in progressive forms of MS is supported by results from a now-completed Phase 2b/3 clinical trial and an ongoing confirmatory Phase 3 study. That first clinical trial, called AB07002 (NCT01433497), enrolled 656 adults with PPMS or nonactive SPMS who were randomly assigned to receive masitinib at daily doses of 4.5 mg/kg or 6 mg/kg, or a placebo, for about two years. The final results showed that the lower dose significantly slowed disability progression compared with the placebo, as measured by changes in the Expanded Disability Status Scale (EDSS), meeting the study's main goal.

Based on those findings, the U.S. Food and Drug Administration cleared AB Science in 2023 to begin the MAXIMS Phase 3 trial (NCT05441488), also known as AB20009. The ongoing study is evaluating masitinib at a daily dose of 4.5 mg/kg versus a placebo in about 800 adults with PPMS or nonactive SPMS. The study is expected to conclude in 2028.

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MS in the 'land of nod'

One of the most tiresome problems of my MS is insomnia and I use the term 'tiresome' literally. There have been times when I have slept as little as four hours a night and sleep isn't just a night time problem. I often nod off during the day, falling asleep almost everywhere, anytime, except in bed at night.

Over the years insomnia has had a such a major impact on my life that I count it as one of my main MS problems. So, as the nights are still longer than the days, it seems a good time to look at sleep or lack of it and share some of my tips for dealing with this problem.

When I mentioned insomnia to my neurologist, he said sleep is one of the most poorly managed and hidden symptoms that affects his MS patients. And neurologist Prof. Gavin Giovannoni agrees. In one of his 'MS Selfie' posts, he said 40-50% of people with MS have insomnia, adding the following: "In my MS practice, insomnia is the most common sleep disorder I encounter, and it generally goes untreated. The reason is simply because most people with MS accept it as part of living with MS, and it is not prioritised by healthcare professionals who tend to focus on other MS-related problems." So, if you are having MS related sleep problems, it may be some consolation to know that you are not alone, far from it.

Sadly, we MSers often have other night time problems which confound insomnia. I am talking here about neurogenic bladder, spasticity, and restless legs, all of which I suffer from, so lack of sleep is far from being a straightforward problem.

To talk about my problem in a little more detail I go to bed late, after midnight, following taking anti-spasm drug baclofen and anti-spasticity drug clonazepam. Then, as I lie in bed I can feel something like an electric current moving up and down my legs. This is probably due to the 'over excitability' of the nerves.

If this feeling is too strong my legs will spasm and I probably won't fall asleep for an hour or so. If the 'excitability' is not too strong I can fall asleep more quickly. Then a few hours later I will wake up because I need to self -catheterise or because of a severe leg spasm. Falling back to sleep can take another hour or so. This cycle goes on all night.

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Continued from page 5 . Of course, I have tried all the obvious things like avoiding stimulants such as caffeine, giving up alcohol and avoiding heavy meals. I also ensure I get adequate exposure to natural light during the day to help maintain a regular sleep-wake cycle. But, a few months ago I wondered whether there was anything else I could do.

Most of us fail to realise how blue daylight actually, is. During the day about 25% of daylight is blue light, or light on the blue end of the visible light spectrum. The brain operates its own internal 'white balance' and shows us a world that is whiter than it really is. It does this by filtering out blue light. Although our eyes may not see this blue light it is still flooding into the brain, stopping the pineal gland releasing melatonin, the sleep hormone.

One of the things that has always interested me is the advice to avoid watching TV or using smartphones before bed. The reason is that these devices emit blue light and this 'tricks' the brain into thinking that it's still day time, something that stops the brain releasing melatonin. So, in terms of solutions, the first thing to do is block out all blue light at least one hour before going to bed. You don't need to turn off the TV or your smartphone. The easiest way to 'block the blue' is to buy a pair of yellow or red lensed glasses. These block blue light, and when wearing my yellow tinted 'sleep glasses' I feel slightly drowsy an hour after starting to wear them and I start to yawn, always a good sign of sleepiness.

The other useful thing I have read about falling asleep is that your body temperature falls as you nod off. When I thought how this might be relevant to MSers like me I wondered if it might help to raise my body temperature before bed time. It is said that taking a hot bath before bed can improve sleep quality and help you fall asleep faster. The science behind this is that a rise in body temperature during the bath, followed by a natural cooling-down afterwards, triggers a biological response that tells your body it's time to sleep, basically mimicking your body's natural 'falling asleep' mechanism.

Baths aren't always practical for us MSers who have mobility issues. Showers too aren't always very practical if you struggle to stand, so the most practical way to raise your body temperature is to get an electric blanket. As the heat from the blanket lessens over time, your body cools, mimicking once again the body's natural sleep process. Doing these two things (the glasses and the electric blanket) have improved my sleep quality enormously. The other thing I do is to wear a 'sleep mask' while sleeping in order to cut out every last bit of light.

Of course, I still have the neurogenic bladder and the restless legs to deal with but I take Hiprex and baclofen/ clonazepam to deal with these. Sleep is still a 'work in progress' but I tell myself that as I have already adopted a couple of successful strategies, I am heading in the right direction, heading towards the land of nod. So, on that note I will sign off. It's late so I will take off my yellow glasses switch off my electric blanket and go to bed. Sweet dreams everyone. **Picture: p1 and 5 Benjamin Voros on unsplash.**

News

Fenebrutinib matches Ocrevus in PPMS trial

Fenebrutinib, an investigational BTK inhibitor was as effective as Ocrevus (ocrelizumab) at reducing the risk of disability progression in primary progressive MS (PPMS), meeting the main goal of a Phase 3 trial.

That's according to fenebrutinib's developer, Genentech, who also say the therapy is the first in more than 10 years to slow disability progression in this MS group. Currently, only Ocrevus and its under-the-skin formulation Ocrevus Zunovo (ocrelizumab and hyaluronidase-ocsq) are approved for PPMS.

The fenebrutinib PPMS data came from the Phase 3 FENtrepid clinical trial (NCT04544449) and were shared at the Americas Committee for Treatment and Research in Multiple Sclerosis (ACTRIMS) Forum 2026, held Feb. 5-7 in San Diego. The presentation was titled, "Efficacy and Safety of Fenebrutinib vs Ocrelizumab in Primary Progressive Multiple Sclerosis: Primary Results of the Phase III FENtrepid Study,"

Fenebrutinib is designed to block BTK, (Briton's tyrosine kinase) an enzyme involved in the activation of immune cells that contribute to MS-related inflammation and nervous system damage. Not only does it target circulating immune cells that drive MS relapses and new MRI activity, but it can also get into the brain and spinal cord to target cells involved in progressive disease.

FENtrepid involved 985 adults with PPMS who were randomly assigned to receive oral fenebrutinib (twice daily) or into-the-vein Ocrevus (once every six months) for at least 120 weeks, or a little under 2.5 years. The study's main goal was to show that fenebrutinib is at least as effective as Ocrevus in delaying the onset of confirmed disability progression.

The trial met its main goal of showing that fenebrutinib was as good as Ocrevus at delaying cCDP12. (12-week Composite Confirmed Disability Progression). The data demonstrated that fenebrutinib numerically reduced the risk of cCDP12 by 12% compared with Ocrevus. The difference, seen as early as six months, persisted throughout the treatment duration.

The side effect profiles of fenebrutinib and Ocrevus were similar, although temporary and reversible elevations in liver enzymes were more common with fenebrutinib. More deaths occurred in the fenebrutinib group, but they were all considered unrelated to treatment.

An open-label extension part of the trial, in which all participants are receiving fenebrutinib, is ongoing**. For further details of all news and features go to page 14**

News

EBV can trick immune system to attack myelin in 'at-risk' group

The Epstein-Barr virus (EBV) appears to alter the immune systems of people with specific genetic traits, causing their own cells to mistakenly flag brain proteins as dangerous threats, according to a new study.

The study, "EBV infection and HLA-DR15 jointly drive multiple sclerosis by myelin peptide presentation," was published in the prestigious journal 'Cell'. This discovery offers insight into how these risk factors — viral infection and genetics — can interact to contribute to the development of MS.

In MS, the immune system launches an inflammatory attack in the brain and spinal cord that damages healthy myelin. The causes of this self-targeting immune attack aren't fully understood, but several risk factors have been identified. One of the strongest risk factors for MS is EBV infection. Genetics is also thought to play a role.

EBV is best known as the cause of glandular fever (infectious mononucleosis,) but it also causes a lot of non-specific childhood illnesses. Almost every adult has been infected with EBV at some point. A large body of data has shown that EBV infection is a key risk factor for MS. In fact, many scientists think MS can develop only in people who have been infected with EBV. However, exactly how the infection leads to MS is poorly understood.

In this study, scientists found that EBV infection can alter the activity of a B-cell protein called the human leukocyte antigen (HLA). The HLA protein acts like a display case for the immune system: loading pieces of infectious viruses and bacteria onto the surface of cells, allowing the immune system to identify threats. Depending on a person's genetics, there are many different variants of HLA, known as haplotypes. Previous research has shown that people with a specific HLA haplotype known as HLA-DR15 are at increased risk of developing MS.

In the study, the researchers found that when B-cells with the HLA-DR15 haplotype are infected with EBV, their HLA molecules begin to display different molecules, including pieces of myelin basic protein (MBP), a key component of myelin. Healthy B-cells that aren't infected with EBV, by contrast, did not display this myelin protein on their HLA molecules.

In cell experiments using immune cells from MS patients, the researchers demonstrated that T-cells — one of the primary types of cells that recognize the proteins displayed by HLA — can respond to the myelin protein presented by EBV-infected B-cells. Collectively, these data suggest that, in individuals with the HLA-DR15 haplotype, EBV may prime B-cells to display healthy myelin proteins erroneously, signalling other immune cells to launch an attack on myelin. This mechanism could help explain how both genetics and viral infection interact to affect MS risk, the researchers said. **For details of all news stories go to page 14**.

GA 'depot shot' keeps disability stable for most PPMS patients

A 3-year trial of GA Depot, a monthly injection of glatiramer acetate saw disability being stabilised in most primary progressive MS (PPMS) patients, with some even showing mobility improvements.

These interim results from a Phase 2a clinical trial were delivered by developer Mapi Pharma at the Americas Committee for Treatment and Research in Multiple Sclerosis (ACTRIMS) Forum 2026. The data presented at the conference suggest that the experimental long-acting formulation of glatiramer acetate (the active ingredient in Copaxone) may offer a new lifeline to a patient population with very few treatment options.

About 15% of the 'MS population' is diagnosed with PPMS, which is marked by a gradual worsening of symptoms independent of relapse activity right from the onset of disease. Like SPMS, PPMS is much harder to treat than relapsing MS. Whereas more than 20 disease-modifying therapies are approved in the US for relapsing MS only Ocrevus (ocrelizumab) and Ocrevus Zunovo (ocrelizumab and hyaluronidase-ocsq) are used to treat PPMS.

An open-label Phase 2 clinical trial (NCT03362294) is investigating this GA Depot formulation in 30 adults with PPMS. Among them, 10 are being treated at a low dose (25 mg), while the other 20 are getting a higher dose (40 mg). Treatment will be given for up to 148 weeks, or nearly three years. The trial's main goal is to evaluate the safety of GA Depot in people with PPMS. The interim analysis covered different treatment durations due to study termination, but some patients were treated for up to three years.

Data indicated that disability remained largely stable over three years. One of the 30 patients experienced 12-week confirmed disability progression — defined as an increase in Expanded Disability Status Scale (EDSS) scores sustained for at least 12 weeks — since starting treatment. Average EDSS scores decreased from 5.1 at the trial's start to 4.5 at three years, which suggests disability eased slightly, a notable contrast from the typical progression of PPMS, where disability worsens over time. Safety data have been generally positive. Other disability measures have also remained generally stable.

Mapi Pharma say this data provides support for evaluating this potential PPMS treatment in a Phase 3 clinical trial. Mapi plans to launch that trial this year, though the company has not shared details. Mapi is also developing GA Depot as a potential treatment for relapsing forms of MS. The company sought U.S. approval of the therapy for this indication in 2023, but the FDA decided not to approve it for reasons that have not been made public.

For further information go to page 14



Why did the FDA say 'no' to tolebrutinib?

Late last year, the US Food and Drugs Administration (FDA) said it wouldn't approve tolebrutinib for non-relapsing secondary progressive MS.

This shock announcement was a huge blow to those of us like me with this type of MS, for which there are no treatments.

What makes things worse is that the FDA had originally given tolebrutinib a 'fast track' designation' ie: a 'green light' so the FDA 'U-turn' was a complete surprise and shock.

All of which begs the question: what explains this U-turn and what exactly is going on? I had heard rumours that the new 'America First' regime that has characterised Trump's America, might mean an application, coming from a French bio-pharma called Sanofi, would be looked on less favourably than if it had come from a US biopharma.

Not so, say the FDA. In a recently published 'complete response letter 'the FDA says its rejection is all do with the science, nothing to do with politics. The letter points to serious safety risks with tolebrutinib, as well as uncertainties regarding its efficacy.

The FDA identified four main issues with tolebrutinib. The first was the drug's "serious risk of severe" drug-induced liver injury (DILI). Six such cases were detected among about 2,700 patients taking the drug in trials. That's about 0.2%. In one patient, the liver injury needed liver transplant and ultimately led to death. To me 0.2% doesn't sound too bad when you're talking about a disease which has a far higher mortality rate. The FDA disagreed, and it's the FDA which makes the decisions, not me. **Continued on page 11**

Continued from p10: This safety 'red flag', the FDA claimed, "indicates a high level of hepatotoxic (liver) risk with tolebrutinib." While it conceded that liver toxicities are known side effects of drugs in the same class as tolebrutinib, (drugs called (BTK inhibitors), the FDA said "the risk of fatal DILI associated with tolebrutinib appears to be among the highest in the class."

And the FDA 'hatchet job' didn't stop with liver toxicities. It was also questioned how well tolebrutinb actually worked, and its therapeutic benefit in certain patient groups. Even Sanofi's record keeping was queried. Sanofi was said to be unable to distinguish between patients with active or non-active SPMS because it failed to collect historical MRI data.

For its part, Sanofi said it tried to provide such data, but the MRI information was said to be subject to "selection bias", as the data were only able to be collected from subjects who elected to enrol in and stayed in an open-label extension study so there was a 'selection bias'.

An analysis of Sanofi's follow-on submission suggested that tolebrutinib's treatment effect was not seen so much in non-active SPMS patients but largely by patients with active SPMS. This is how the FDA wordsmiths put it: "The analyses raise substantial uncertainties about the SPMS population that is more likely to benefit from tolebrutinib. Given the serious and unusually high risk of severe DILI, it is critical to have certainty about efficacy in a population in whom this level of DILI risk could be considered acceptable."

Another critical issue for the FDA was the lack of conclusive evidence demonstrating tolebrutinib's effectiveness at slowing the accumulation of disability in patients, independent of its ability to prevent relapses. Here, Sanofi was a victim of the system of measuring PIRA (Progression Independent of Relapse Activity). There is a lack of established standards for this particular measure, as the FDA noted. "There are no widely accepted criteria for defining disability accumulation independent of relapse activity," the FDA wrote, adding that this endpoint "remains an emerging construct with multiple limitations."

Taken together, these three shortcomings in the tolebrutinib package led to the FDA's final hammer blow, which is that tolebrutinib did not have a favourable risk-benefit profile in any tested patient subpopulation. In patients with active SPMS, the agency argued, tolebrutinib's "benefits would not be anticipated to outweigh the risk of DILI"

Despite the rejection, the FDA has left the door open to Sanofi, giving the pharma the opportunity to "identify a population for whom the potential benefits of your drug may outweigh the serious risk of severe DILI." Over to you Sanofi. But please be quick as progressive MS is always progressing and time is not on the side of people like me with progressive MS **For details of all features go to page 14**

News

Gut microbiota transplant led to rapid improvement in prog-MS

A woman with progressive MS saw a rapid improvement in her condition after 'faecal microbiota transplantation' followed by taking capsules which contained frozen microbiota.

The case, reported in the journal 'Frontiers of Immunology' is the latest in the growing field of cases of faecal microbiota transplantation being used to successfully treat MS.

It has been known for some time that although MS symptoms are neurological, the cause may involve the patient's intestinal bacteria, known as their 'gut Microbiota' (GM), which has been shown to play a role in autoimmune and inflammatory processes which underlie the condition.

When gut microbiota becomes 'unbalanced' it is believed it may ultimately lead to MS. Faecal Microbiota Transplantation (FMT) is therefore emerging as a potential strategy to restore a balance of gut microbiota, which would stop inflammation and in theory stop MS.

Faecal microbiota transplantation (FMT) transfers processed, screened stool from a healthy donor into a patient's intestines to restore gut flora. It can be delivered via colonoscopy, enema, or oral capsules.

In the paper Italian researchers treated a 42-year-old woman with severe MS disability by FMT via colonoscopic infusion followed by a 3-month treatment of daily oral capsules, containing frozen microbiota.

Clinical follow-up was conducted at 30, 60, and 90 days. Microbiota profiling (16S rRNA sequencing) and intestinal permeability testing were performed at several time points before and post infusion. Post-FMT, there were improvements in limb spasticity, trunk stability, and fine motor skills. Microbiota analysis revealed a marked reduction in the abundance of Akkermansia muciniphila (22.5% vs 6.6%). At phylum level, Actinomycetota remained elevated (22%), while Bacteroidota consistently showed low abundance (14%).

The most favourable microbiota profile was observed at 90 days, which coincided with the peak of clinical improvement. Intestinal permeability also improved over time, despite the patient's microbiota profile remaining distinct from the donor. This is the first report about combined FMT in MS.

Researchers say that the procedure was safe, well tolerated, and associated with neurological improvements.

" These findings support further exploration of FMT as a therapeutic adjunct in MS through controlled clinical trials.", they add. **For details of all stories go to page 14**

News

‘Backward walking’ may boost mobility and reduce falls

Training in ‘backward walking’ led to measurable improvements in postural stability and gait speed in a majority of those participating in the study.

That’s the finding of a group of US researchers from Wayne State University’s Eugene Applebaum College of Pharmacy and Health Sciences, School of Medicine, College of Liberal Arts and Sciences and Institute of Gerontology .

The study, “Structural and Functional Changes With Eight Weeks of Backward Walking Training in Multiple Sclerosis: A Case Series,” was published in the January issue of the Journal of Neurologic Physical Therapy.

The team led by Dr. Nora Fritz, aimed to determine if backward walking training could improve mobility, balance and brain structure, and reduce the risk of falling. Participants were involved in eight weeks of treadmill and overground backward walking therapies.

Dr. Nora Fritz said: “The results of this pilot trial indicated that backward walking training led to measurable improvements in postural stability and gait speed in a majority of those participating in the study. This suggests that backward walking may trigger positive physical adaptations.”

The authors indicated additional larger clinical trials are needed to confirm the benefits.

“This novel physical therapy intervention was designed to combat the progressive movement challenges associated with MS,” said Fritz. “We measured structural changes in the brain’s white matter in three brain regions – the body of the corpus callosum, the superior cerebellar peduncle, and the corticospinal tract.

“The results of this small trial suggest that this type of therapy may promote neuroplasticity in brain areas related to balance. Our next step is to conduct a larger trial to determine the potential impact this type of therapy may have on all MS patients.” **For details of all news stories see page 14**

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Pages 5-6 Sleep and MS

For a good overview of the problem and possible solutions visit the website https://www.nationalmssociety.org/news-and-magazine/momentum-magazine/research-and-science/understanding-ms-and-sleep-issues#:~:text=A%20study%20of%20136%20people,at%20risk%20of%20sleep%20apnea

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To view the study, visit https://journals.lww.com/jnpt/fulltext/2026/01000/structural_and_functional_changes_with_8_weeks_of.7.aspx. For more information about the study, visit https://cdnlinks.lww.com/permalink/jnpt/a/jnpt_50_1_2025_12_22_fritz_jnpt-d-24-00138r2_sdc1.mp4

To Contact me email iancookjournalist@yahoo.co.uk

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