

My prog-MS ezine

For those with progressive MS and other interested people

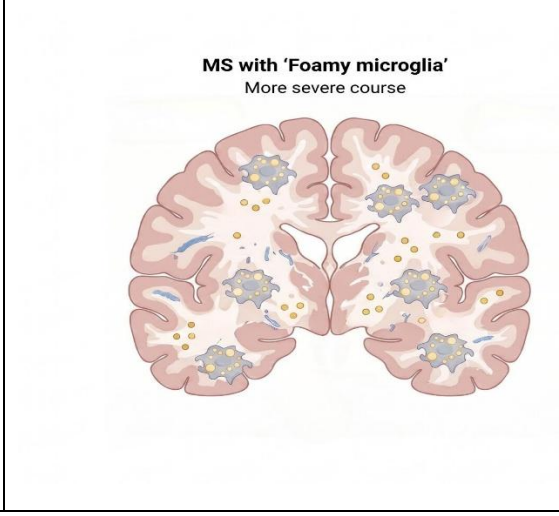
Issue 67, July-August 2026

Hello, and welcome to the 67th 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 (68 in August).

In issue 67 there are eight pages of prog-MS news stories. There are also two features. The first, on page 5, looks at the brain changes that Dutch researchers have found in 'advanced' MS. These changes may explain MS progression? Go to page 5, you may be surprised.

On page 9 another feature looks at a recent debate sparked by the 'enfant terrible' of the MS world – Prof. Gavin Giovannoni. He recently suggested that MS could be seen as a 'preventable dementia'. This upset a lot of people. But does Prof G have a point? Is MS with its associated 'brain shrink' a dementia? It's certainly a subject worth discussing so I have set out the argument and its pros and cons on page 9. Make your own judgment.

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News

Foralumab shows promise in non-active SPMS, new data suggest

Foralumab nasal spray shows promise for nonactive secondary progressive MS, slowing disability progression and significantly easing fatigue in patients. That's according to new data from an expanded access program (EAP), where patients use experimental therapies outside clinical trials.

In trials Tiziana Life Sciences' experimental nasal spray foralumab continues to show signs that it may help slow disability progression and ease fatigue in people with nonactive secondary progressive MS (naSPMS).

Fourteen people with nonactive SPMS have been using the spray in one trial (NCT06802328). After about three years of follow-up, just one participant experienced a sustained worsening in disability scores.

"These longer-term results from the Expanded Access SPMS Programme continue to support the potential of intranasal foralumab as a novel, immunomodulatory therapy for patients with non-active SPMS," Howard L. Weiner, MD, chair of Tiziana's scientific advisory board, said in a company press release. The data also continue to suggest that foralumab may help ease fatigue, one of the most common and debilitating symptoms of MS.

Safety findings also remained consistent with prior reports. Tiziana said no new safety signals have emerged, and the therapy remains generally well tolerated.

While some people with SPMS continue to experience occasional relapses and/or show signs of disease activity on MRI scans, having what's known as active SPMS for which there are treatments, there are few available treatments available for people with nonactive SPMS (naSPMS).

Foralumab is an antibody-based therapy that targets CD3, a protein produced by T-cells, a type of immune cell. The therapy aims to modulate the activity of T-cells and other immune cells to dampen MS-driving inflammation.

Tiziana is currently sponsoring a Phase 2a clinical trial (NCT06292923) that aims to test foralumab against a placebo in about 54 adults with nonactive SPMS who have experienced disability worsening despite at least two years of treatment with available MS treatments. The main goal is to assess the safety of the experimental therapy, its effect on nasal symptoms, and changes in the activity of brain immune cells called microglia. Researchers are examining changes in disability progression and fatigue as secondary endpoints.

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CAR T-cell therapy has big role in treating prog-MS

A recently published academic paper says that Chimeric antigen receptor T-cell (CAR-T) therapy can lead to a significant decrease in disease progression, a recovery of impairment, and an immense reduction in inflammatory markers in progressive MS.

The paper published in the journal 'Inflammopharmacology' goes on to say: 'the therapy has the potential to transform the therapeutic paradigm toward a sustainable immunological reset and prolonged remission in clinical neuroimmunology' The paper is titled 'Reprogramming immunity: the promise and challenges of CAR T cell therapy in multiple sclerosis.' and adds to the growing body of evidence that CAR T cell therapy has a role to play in treating progressive MS. The paper concludes 'CAR-T treatment represents a revolutionary approach for individuals with refractory MS.' Refractory MS' is MS that continues to worsen, or show lesion progression on MRIs, in other words the progressive side of MS.

CAR T cell therapy aims to reset the immune system by targeting the harmful B cells responsible for nerve damage. A patient's own T cells are collected and reprogrammed in a lab to target a specific protein (like CD19) found on B cells before being re-infused back into their body. These reprogrammed cells can cross the blood-brain barrier for lasting effects.

Clinical trials are already taking place in the US. A Phase 1 trial of azer-cel, a CAR T-cell therapy, for progressive MS is now recruiting participants at eight sites across the U.S. Results are expected in 2029 but early indications may be made public later this year (2026). The study (NCT06680037) is expected to enrol up to 32 adults with primary progressive MS or secondary progressive MS to determine whether genetically engineered immune T-cells from healthy donors can safely and effectively improve patient outcomes. The first participant was dosed last year.

Another trial, in London UK called AUTO1-MS1 is also evaluating CAR T-cell therapy in MS. The study is spearheaded by Dr. Wallace Brownlee at University College London Hospitals (UCLH) and the National Hospital for Neurology and Neurosurgery, with clinical trials also taking place at Barts Health NHS Trust (Royal London Hospital). The trial aims to recruit up to 18 patients globally by early 2027.

Other groundbreaking clinical trials in London are investigating CAR T-cell therapy as a treatment for another autoimmune disease –severe, treatment-resistant lupus. The pioneering CARLYSLE trial has shown promising early results, successfully putting patients into remission further raising hopes that the therapy my work in MS.

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Remibrutinib in phase III trials for active SPMS

While prog-MSers with non active secondary progressive MS digest the breaking news that Tolebrutinib is moving closer as a treatment for this type of MS, (See below) bio-pharma Novartis has announced it is currently developing a similar drug to treat active SPMS.

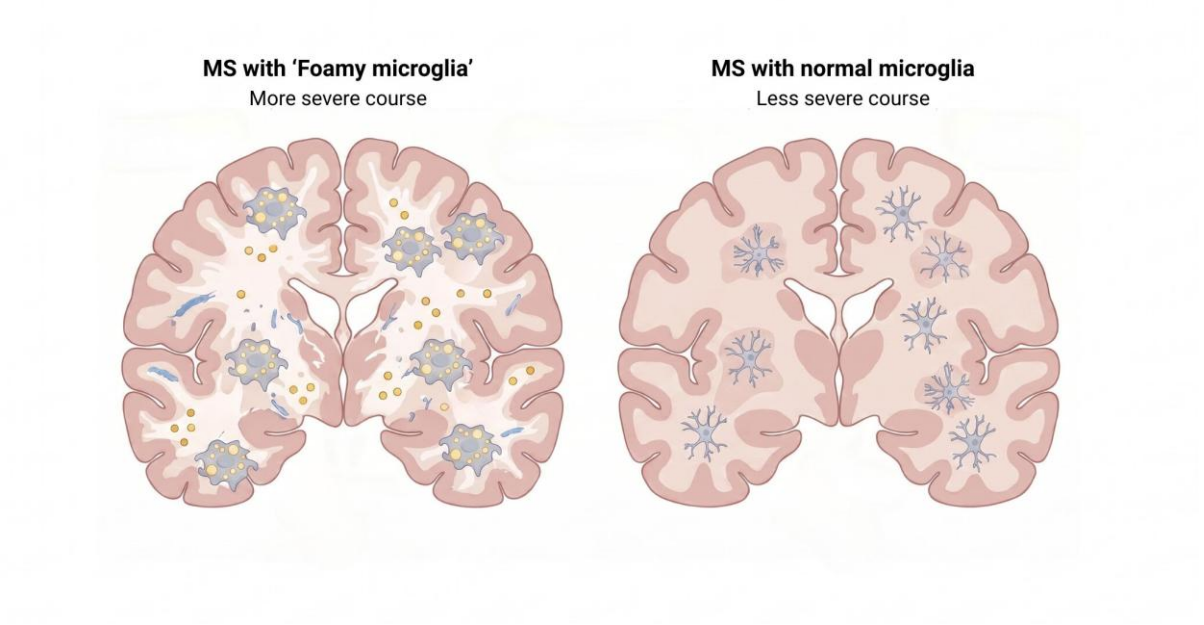
The active SPMS drug, called Remibrutinib, also known as LOU064, is an experimental oral medicine (film coated capsule) which is also being investigated for use this type of SPMS as well as another inflammatory disorder called chronic spontaneous urticaria, characterized by itchy hives and swelling under the skin.

Like Tolebrutinib the new drug is a BTK inhibitor. This class of drugs inhibit Bruton's tyrosine kinase (BTK) enzyme, which plays a critical role in the inflammatory activity of certain immune cells such as B-cells and microglia. By blocking this enzyme, remibrutinib dampens the inflammatory activity behind the blood-brain barrier that drives MS progression. Two identically designed Phase 3 clinical trials are now recruiting participants in the U.S. and several in Spain. One trial (NCT05156281) also includes locations in Slovakia and France, while another study (NCT05147220) has sites in Poland. Each study aims to enrol 800 patients ages 18 to 55 including patients with active secondary progressive MS. Those completing the study may then join an open-label extension study, in which all participants will receive remibrutinib for up to five years.

The main goal is to compare the effect of the treatments on disability progression, MRI measures of disease activity, and quality of life. Top-line data from the trials are expected as soon as October 2025. Remibrutinib appears to have fewer and less serious side effects than tolebrutinib.

Meanwhile, in late breaking news, naSPMS drug Tolebrutinib is now licensed in the European Union. In June 2026 it was granted marketing authorization by the European Commission for adult patients with secondary progressive multiple sclerosis (SPMS) without relapses in the last two years. In Europe, the drug is marketed under the brand name Cenrifki. Makers Sanofi plans to roll out Cenrifki commercially in Germany first, later this year, with subsequent availability across other EU countries and patient support programmes being managed in coordination with local MS specialists. It will also be available in Australia and has been available in the United Arab Emirates since August 2025. In the UK as of mid-2026, tolebrutinib is still under assessment for formal licensing by the UKHRA and the NHS (NICE) where it is hoped it will be licensed and soon made freely available.

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Does 'foamy microglia' explain progression?

An important mechanism which may explain MS progression has been uncovered by Dutch researchers, and it may offer a revolutionary new approach treating progressive MS.

Researcher Daan van der Vliet, together with colleagues from the Netherlands Institute for Neuroscience, Leiden University, and Utrecht University, have discovered that in brain tissue of patients with progressive MS, there are large numbers of abnormal immune cells overloaded with fat droplets. The work is published in the journal Nature Neuroscience.

In MS, the fatty insulating layer surrounding nerve fibres (myelin) is broken down in the brain and spinal cord. This can lead to neurological symptoms such as difficulties with walking and vision. MS progresses differently in every patient. Some people live for decades with relatively mild symptoms, while others become severely disabled at a young age. Researchers have long tried to understand what causes these differences.

In the new study, the scientists focused on microglia: a class of immune cells in the brain that clear waste and help repair damaged tissue. In MS patients these cells change shape. They become filled with fat droplets, giving them a 'foamy' appearance. Perhaps unsurprisingly researchers call these cells "foamy microglia."

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Continued from page 5 .. Under normal circumstances, microglia help clean up damage in the brain. In MS, however, this task may sometimes become too big. The researchers believe the cells absorb so much damaged myelin that they eventually become overwhelmed by their own waste-processing system and become 'foamy' in appearance literally bulging with fat. The researchers also discovered that brain inflammations containing foamy microglia behave very differently at the molecular level from MS inflammations without these cells. For example, these 'foamy' cells contain specific fats which trigger chronic inflammatory responses.

For a long time, inflammation was thought to be the driving force behind disease progression, but the study also shows that MS may be more complex than that alone. According to the researchers, the new work points to a more subtle process. "It does not appear to be simply about the inflammatory response alone," says Van der Vliet. "These cells are probably attempting to clear damage and promote repair, but that process fails, worsens inflammation, and counteracts recovery."

For the study, scientists analysed brain tissue from 28 deceased MS patients who had donated their brains to the Netherlands Brain Bank. The team combined several advanced techniques that simultaneously examined gene activity, proteins, and fats within the same MS inflammatory lesions.

According to the researchers, the combination of modern technology and detailed knowledge of brain pathology was especially crucial. Van der Vliet says: "Today we have incredibly sophisticated techniques that can map the brain in great detail. The technologies are fantastic, but they tell you relatively little if you cannot connect them to pathology in brain tissue. Precisely because brain tissue has been carefully studied and classified for years by the Netherlands Brain Bank, we were able to recognize these abnormal patterns."

In the long term, the scientists hope their discovery may help improve predictions of MS disease progression. The researchers found signs that certain fats associated with foamy microglia can also be measured in patients' cerebrospinal fluid. "That opens the possibility of developing biomarkers in the future that could help doctors identify earlier which patients are at risk of rapid decline—and which treatment would suit them best," Van der Vliet says. In addition, the findings align with ongoing interest in new medicines targeting fat metabolism and chronic lesion expansion in MS. Some of these drugs are already being investigated in clinical studies in collaboration with Roche.

Given the current interest in weight loss drugs such as metformin, currently being investigated in the Octopus trial and the interest inGLP-1 (Glucagon-Like Peptide-1) receptor agonists like Ozempic as MS treatments, it could be argued the 'foamy microglia' investigations are following a well-trod path and one that may lead to promising outcomes.

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Ocrevus reduces disability progression in PPMS

A study published in the medical journal 'The Lancet' says that Ocrelizumab (Ocrevus) has a major impact on disease progression in patients with Primary Progressive MS (PPMS) when EDSS, the nine hole peg test and MRI activity are measured

The study, called ORATORIO-HAND, was a phase 3b trial of ocrelizumab in PPMS. It was found ocrelizumab reduced the risk of 12-week composite confirmed disability progression (cCDP) on the EDSS or the nine-hole peg test (9HPT) by 30% Baseline MRI activity, was lower too.

When looked at in more detail, Ocrelizumab treated patients showed a significant reduction in the risk of disability progression with a 41% risk reduction in 12-week disability progression in 9HPT, corresponding to progression rates of 17% with ocrelizumab and 25% with placebo (Table 1); and 33% risk reduction (p=0·0013) in 12-week CDP in EDSS, corresponding to progression rates of 23% with ocrelizumab and 31% with placebo.

Among pwMS who were still walking, the risk of requiring a wheelchair fell by 52%. One of the paper's authors Prof. Gavin Giovannoni said: "I suspect ocrelizumab will be a cost-effective treatment for pwPPMS who are using a wheelchair. Don't forget that once you lose your hand function, you lose your independence and need 24-hour-a-day care. Being EDSS 8.5 and above is very expensive.

"Please note that the safety profile of ocrelizumab in ORATORIO-HAND was similar to that observed in other pivotal phase 3 studies and in a decade of post-marketing experience and surveillance.

"Importantly, the ORATORIO-HAND study is not merely a confirmatory trial of the original phase 3 ORATORIO PPMS study. It is the first randomised, placebo-controlled evidence in MS that a disease-modifying therapy (DMT) can preserve hand function in people with advanced PPMS, i.e. in subjects aged up to 65 years of age, with an EDSS score between 6.5 and 8.0, who are wheelchair-dependent, using a composite endpoint of confirmed disability progression on either the EDSS or the 9HPT.

"This treatment effect in the more advanced subjects would have been missed if the EDSS alone had been used. Each of these findings overturns the MS orthodoxy that has, for too long, restricted who gets recruited into clinical trials and, subsequently, who gets treated, when, and on what evidence," he added.

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Frexalimab to be tested in two new SPMS trials

A Phase 3 trial is testing frexalimab in patients with non-active secondary progressive MS. The trial called FREVIVA (NCT06141486) is testing frexalimab in an intravenous (In vein) formulation of frexalimab. French bio-pharma Sanofi is still working on the subcutaneous (under the skin) version.

In a paper, "Subcutaneous Administration of Frexalimab in Relapsing Multiple Sclerosis and Nonrelapsing Secondary Progressive Multiple Sclerosis: Design of the Phase 3 Frexcite Trial," researchers described the design of the Phase 3 FREXCITE trial (NCT07325292), which is testing whether the subcutaneous formulation works similarly to the intravenous version.

About 160 people with relapsing MS or non-relapsing SPMS will receive monthly frexalimab, either subcutaneously (1,800 mg) or intravenously (1,200 mg) for about six months, after which all will be switched to subcutaneous dosing.

Subcutaneous doses will be self-administered using single-use, wearable injection devices placed on the abdomen. These injections take six to 30 minutes, while intravenous infusions take about an hour.

The main goal is to demonstrate that subcutaneous dosing is just as good as intravenous dosing for achieving therapeutic levels of the medication in the bloodstream. Other pharmacological and safety properties will also be evaluated, along with patient preferences and other patient-reported outcome measures. The trial completion date is late 2028.

Frexalimab targets and blocks the CD40/CD40L pathway, a crucial molecular route that regulates the activation of both the adaptive and innate immune systems. By preventing CD40L from binding to its receptor, the drug dampens the inflammation and nerve damage that drive autoimmune diseases. By contrast Sanofi's other MS drug tolebrutinib inhibits the BTK enzyme, which is critical for the activation of B cells, macrophages, and microglia

Those who are interested in the EBV theory of MS may be interested to know frexalimab targets the specific communication pathway that EBV uses to trigger immune activity in MS — blocking the CD40/CD40 ligand connection between B-cells and T-cells without causing general immunosuppression. In Phase 2 trials it showed an 89% reduction in new lesions in the high-dose arm. 96% of high-dose participants were free of new lesions at week 48.

If the Phase 3 results hold, this could be the first MS drug that actually addresses a root cause mechanism rather than broadly suppressing immune function.
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Phase 2 weight loss drug trial begins dosing in progressive MS

A Phase 2 trial has begun testing a drug called pegsebrenatide in progressive MS. The drug is designed to activate GLP-1 receptors and modulate neuroinflammation, although it also leads to weight loss. The study will track brain volume changes as a measure of neurodegeneration.

Pegsebrenatide, also known as NLY01, is designed to modulate neuroinflammatory pathways and protect neurons, with the goal of addressing processes thought to drive ongoing nerve damage and disability worsening in progressive MS. The medication is designed to activate the glucagon-like peptide-1 (GLP-1) receptor. GLP-1 receptor agonists are best known to many people as medications used for diabetes or weight loss but GLP-1 receptor agonists have recently attracted interest as potential MS treatments.

Pegsebrenatide is intended to modulate the activity of microglia and astrocytes — support cells in the brain and spinal cord that can become activated and fuel neuroinflammation. Such inflammation is thought to contribute to neurodegeneration, or the gradual damage and loss of nerve cells. Preclinical research has suggested that the drug can reduce inflammatory immune activity and ease disease severity in a mouse model of MS.

The first participant has been dosed in a Phase 2 clinical trial testing pegsebrenatide as a potential treatment. The trial will test whether pegsebrenatide can provide benefits for people with progressive MS. Participants will be randomly assigned to receive weekly subcutaneous, or under-the-skin injections of pegsebrenatide or a placebo for 96 weeks. Treatment will begin at a lower 5 mg dose for the first four weeks, then increase to 10 mg, with dose adjustments allowed if needed due to side effects.

Called TAG-MS (NCT07497399), the study is being led by Ellen Mowry, MD, at Johns Hopkins University in Maryland, with additional clinical sites expected to participate. The plan is to eventually include about 120 adults with progressive MS who are clinically stable on MS therapy for at least one year, with no relapses or new brain MRI lesions during that time.

The trial's main goal is to measure changes in normalized brain parenchymal volume, an MRI-based measure of brain tissue volume. Slower rates of brain atrophy could suggest the therapy is helping to slow neurodegeneration.

Researchers will also assess changes in the volume of specific brain regions as secondary measures, as well as changes in the retina, the light-sensitive layer at the back of the eye. Such retinal changes may reflect nerve damage in MS**.**

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Is MS really a dementia?

It's the question very few MSers want to talk about, particularly those of us in the 'progressive' camp. The question is whether MS is a form of dementia or, to re-frame the question slightly, can MS lead to dementia?

Although many MSers are reluctant to talk about this subject, a small group of academics talk quite a lot about it. They also write papers about it, and their words sometimes make uncomfortable reading. One paper published in January 2026 said MS increases your dementia risk by up to five times, others say the risk is far lower, but there is a general consensus that MS does increase your dementia risk. If true, the question is: by how much?

Last year a paper was published in the journal 'Brain and Behaviour'. The paper was titled 'Dementia in People With Multiple Sclerosis,' and was a 'meta-analysis' that's means it's a big study. The key finding was that that there is a 67% higher chance of MS patients being diagnosed with dementia compared to the general population. Older MSers like me (68 in August) were found to be particularly at risk.

The reasons for this increased risk are said to be three-fold. First, MS-related neurodegeneration, although initially affecting the brain's 'white matter', can spread from white to grey matter, causing 'collateral damage' as the illness progresses and leading to the 'brain shrink' that characterises progression. (My last MRI said I had brain shrink) Second, oxidative stress and mitochondrial damage make neuronal injury worse. Finally, MS lesions may play a role. Previous studies have shown that cognitive impairment in MS is linked to the number of lesions sustained during the course of the illness and where they are, whether in the brain or spinal cord.

One academic who has made great efforts to explore the relationship between MS and dementia is Prof. Gavin Giovannoni of London's Barts Hospital.

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Continued from p10: Professor Giovannoni's interest in the subject goes back more than 10 years. In 2013, the European Medicines Agency invited him to give a talk on the treatment of MS. He takes up the story: 'To shift the debate, what I call "reframing", I decided to define MS as a "preventable dementia". My talk triggered several altercations with different members of the audience. One very senior Italian academic accused me of being irresponsible and that MS was not a dementia.

'Another Canadian academic said to me after the meeting that I was being reckless. He had looked after many patients with MS who were blissfully ignorant of their cognitive impairment and were fine without the knowledge of having dementia. A senior pharmaceutical medic congratulated me on being brave enough to highlight 'the elephant in the room.'

So, is 'MS dementia' really the elephant in the room? Is it something we MSers are aware of, but avoid discussing? Based on my own experience and following conversations with family members I would agree that cognitive decline is something we MSers don't talk about often enough. That said, I feel that calling MS a 'dementia' is probably overstating the case a little. MS may lead to dementia but I don't see MS as being the same as dementia or being a type of dementia.

The biggest difference between MS and dementia I believe is that as an MSer I can still do most things I used to do, although physical and cognitive tasks become an increasingly difficult and hence I tend to do fewer things and stick to things I have done before and find relatively easy. This, I believe, is because the decline in my physical condition means everything becomes a huge effort. That applies to 'brain work' as much as to physical work.

I would, therefore, say MS does share similarities with dementia. However, the differences are more significant than the similarities. For example, dementia typically shortens life expectancy, with the average survival time after diagnosis ranging from 3 to 13 years. By contrast, most people with MS are diagnosed in early adulthood and live for decades

I would further argue that although MS and dementia do share some and the idea of 're-framing' MS as a preventable dementia may grab the readers' attention, I would say it is largely a 'catchy' headline, and that the real story is far more complicated and nuanced than that. . So, sorry Prof G, while I think you are right about so many other things, I think you are wrong about this one. I wouldn't say you are irresponsible or reckless I just think you are wrong. MS is, in my opinion, not a dementia.

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News

Exercise Hormone Protects Neurons in Preclinical Model of MS

A new study offers clues as to why exercise can improve neurological symptoms in people with MS. A hormone produced by muscles during exercise called Irisin has been shown to reduce clinical symptoms and neuron loss in a mouse model of the disease.

That's according to a team of investigators from Harvard Medical School and University Medical Center Hamburg-Eppendorf (UKE) in Germany. Additionally, when the team deleted the gene encoding irisin, the protective effects of exercise disappeared.

The findings suggest that irisin can protect neurons from inflammation-driven neurodegeneration and that it does this by acting directly on neurons rather than by suppressing the immune system. This uncovers a new way that exercise acts on MS and makes irisin a potential target for future MS therapies. The results were published May 21 in Nature Metabolism.

Current therapies for MS reduce inflammation but do not prevent neurodegeneration, and no approved treatments directly target neurons to halt nerve cell loss. Research has shown that aerobic exercise can improve MS symptoms, but the exact mechanisms have been unknown.

Mice that exercised on running wheels before developing 'mouse MS' and during its progression showed less neuronal loss and milder symptoms than sedentary animals. These benefits vanished entirely in mice with the gene encoding irisin deleted.

Irisin reduced neuronal loss in three parts of the animals' central nervous system — spinal cord, hippocampus, and retina — and reduced synapse loss and restored a neuroprotective gene programme in spinal cord neurons.

Notably, irisin appeared to act directly on neurons rather than by dampening the immune response. No significant differences in immune cell activity were observed between irisin-treated and control animals. This neuronal pathway complements earlier work from HMS researchers showing that some benefits of exercise arise from changes in the immune system, underscoring that multiple biological mechanisms likely contribute to exercise's protective effects in the brain.

Future work will determine whether the findings translate to humans. The team plans to continue investigating the hormone's effects and mechanisms in future studies

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News

Late-onset MS linked to relapse-free disability progression

People who develop MS at age 50 or later are more likely to accumulate disability through mechanisms that are not directly linked to relapses or MRI activity than those with earlier-onset disease, according to an Italian registry study.

The study, "Late-onset multiple sclerosis is associated with relapse-independent disability accrual: a propensity score-weighted recurrent-event study," was published in the Journal of Neurology.

MS usually begins in early or middle adulthood, but about 5% to 12% of patients have late-onset MS (LOMS), usually defined as onset at or after 50 years of age. Compared with adult-onset MS (AOMS), LOMS usually has less inflammatory features and is associated with faster disability accumulation.

Because differences in inflammation cannot fully explain the more severe disease course in these patients, researchers believe that ageing-related mechanisms may contribute to faster disease progression.

To validate this, researchers in Italy investigated whether age at onset influences how disability accrues in people with MS. The researchers focused on three disability outcomes: confirmed disability worsening (CDW), progression independent of relapse activity (PIRA), and MRI activity (PIRMA).

Results showed that all three disability outcomes occurred in a greater proportion of people with LOMS. Notably, the association between LOMS and worse disability outcomes remained evident even in patients taking high-efficacy disease-modifying therapies.

"This observation is consistent with recent evidence suggesting that anti-inflammatory treatments, although highly effective in reducing relapse activity and MRI inflammation, may have limited impact on progression independent of relapse activity," the researchers wrote. "These findings highlight the need to consider age at onset as a key prognostic factor and suggest that current monitoring strategies based predominantly on inflammatory activity may be insufficient in this subgroup."

For details of all news stories see page 14

References further details, etc

Page 2 Foralumab shows promise in na-SPMS

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Page 3 CAR-T cell therapy being trialled in London UK

https://www.uclh.nhs.uk/news/first-ms-patient-uk-trialling-car-t-cell-therapy-uclh

Page 4 Remibrutinib being trialled in active SPMS

https://multiplesclerosisnewstoday.com/remibrutinib-multiple-sclerosis/

Pages 5-6 'Foamy' microglia

Daan van der Vliet et al, Foamy microglia link oxylipins to disease progression in multiple sclerosis, Nature Neuroscience (2026). DOI: 10.1038/s41593-026-02302-3

Page 7 Ocrevus reduces disability progression in PPMS

Giovannoni et al. Efficacy and safety of ocrelizumab in primary progressive multiple sclerosis, including older patients and those with more advanced disease (ORATORIO-HAND): a multicentre, double-blind, randomised, placebo-controlled, phase 3b study. The Lancet, 407(10544):2195-2207.

Page 8: Frexelimab to be tested in two new prog-MS trials

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Page 9: Weight loss drug trialled in prog-MS in US

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Page 10-11 Should we re-brand MS as a dementia?

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Page 12 Exercise hormone protects neurons in 'mouse model' of MS

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Page 13: Late onset MS is less inflammatory, more progressive

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