

My prog-MS ezine

For those with progressive MS and other interested people

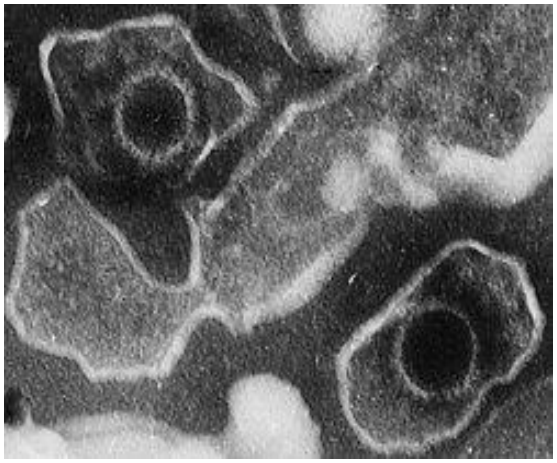
Issue 68, Sept-Oct 2026

Hello, and welcome to the 68th 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 now 68.

In issue 68 there are, as ever, eight pages of prog-MS news stories. There are also two features. The first, on page 5, asks whether we are now looking at a potential cure for MS. New research into the Epstein Barr virus and MS appears to show the way by which a seemingly mild glandular infection in childhood or adolescence could return as MS decades later. Anti-viral or anti-retroviral drugs may be the treatment we all need.

On page 9 another feature looks at a question that should be asked more in the MS world. It's about weight loss drugs, and whether weight loss drugs could be used to treat MS. It is well known that obesity and MS are linked, but new evidence is showing that anti-obesity drugs might have a role to play in treating MS. Research is currently taking place that might explain the process behind this intriguing fact.

IN THIS ISSUE



Is this proof that Epstein Barr virus causes MS? P5

Picture left EBV virus Wikimedia commons

Nasal MS trial for prog MS to announce results in October, p2

High dose vitamin D can ease MS disability - page 3

News

Nasal MS drug trial for prog MS to announce results in October

Top-line results of a Phase 2a trial for foralumab nasal spray in nonactive secondary progressive MS (naSPMS) are set to be presented at the joint ACTRIMS-ECTRIMS (American and European MS) meeting held in Toronto, Canada from October 21 to 23.

Foralumab aims to reduce inflammation and microglial activity in MS and the forthcoming conference will hear results of the INFORM-MS trial (NCT06292923) evaluating the safety, tolerability, and efficacy of foralumab versus a placebo in up to 48 adults. The trial is running at seven sites in the U.S.

The nasal spray is an antibody designed to target CD3, a protein found on the surface of T-cells, immune cells that contribute to MS-related inflammation and damage. By binding to CD3, foralumb is believed to suppress the activity of inflammatory T-cells while boosting regulatory T-cells, which help keep immune responses in check. The experimental therapy is being evaluated in an open-label expanded access program (NCT06802328), which gives some people with nonactive SPMS access to the medication outside of the clinical trials.

Early data from 14 patients showed that disability levels remained stable or improved after about three years of follow-up for most patients, with only one patient experiencing sustained worsening in disability scores. About two-thirds of patients also showed clinically meaningful improvements in fatigue.

The data suggests foralumab may reduce microglial activity, which is believed to contribute to disease progression in nonactive SPMS. Brain imaging data from 10 patients in an expanded access program showed that 80% experienced reductions in microglial activity after six months of treatment.

In the INFORM-MS trial participants were randomly assigned to receive one of two doses of intranasal foralumab — 50 or 100 micrograms per dose — or a placebo for about three months. Treatment was given in three-week cycles — three times a week for two weeks followed by one week off. The study's main goal is to see whether foralumab is safe and whether it can reduce microglial activity. Secondary and exploratory goals include changes in standard measures of disability and fatigue, as well as changes in inflammatory brain lesions and brain volume.

"Intranasal foralumab represents a promising therapeutic strategy for patients with non-active secondary progressive MS," said Tanuja Chitnis, MD, a neurologist at Mass General Brigham Neuroscience Institute and principal investigator in the trial. "We look forward to evaluating its impact on microglial activation, in this underserved patient population."

For details of all news stories go to page 14

High dose vitamin D can ease MS disability

Taking high-dose vitamin D supplements (more than 4,000 IU a day) for more than a year can significantly ease disability for people with MS according to a comprehensive review of more than 30 clinical trials. The supplements had to be taken long term to be effective. High-dose vitamin D taken for less than one year showed no significant differences.

A study "Vitamin D for multiple sclerosis as an adjuvant therapy: A network meta-analysis of randomized controlled trials," was published in the journal 'Clinical Nutrition'.

It is well established that vitamin D can influence disease onset and progression. Studies have found an association between low vitamin D blood levels and a higher risk of developing MS, while other research suggests that people with lower vitamin D levels experience more frequent relapses and faster disability progression. As a result, both the American Academy of Neurology and the European Academy of Neurology have incorporated vitamin D as an adjuvant (add-on) therapy in their MS treatment guidelines. However, those guidelines address vitamin D supplementation only in broad terms.

To address this, researchers pooled the results of published clinical trials to provide more detailed, evidence-based guidance on the optimal vitamin D dose for MS patients. The analysis focused on 32 clinical trials in which patients were randomly assigned to various doses of vitamin D or a placebo. These trials involved a total of 2,254 MS patients and, except for five that did not have any specific requirements on MS therapies, all others tested vitamin D as an add-on to standard disease-modifying therapies.

Data from all studies showed that vitamin D supplementation for more than one year was associated with a modest but significant improvement in disability scores (indicating less severe symptoms) compared with a placebo.

Long-term vitamin D supplementation was also associated with a 49% lower risk of having lesions with active inflammation and a significant reduction in total lesion volume. The team then compared different dosing and duration strategies. Interventions were classified by dose: low (less than 1,000 international units, or IU, per day), medium or mid-dose (1,000 to 4,000 IU/day), and high (more than 4,000 IU/day). They were also classified by duration: short (one year or less) or long (more than one year).

For outcomes measured beyond one year of follow-up, high-dose, long-duration supplementation showed the greatest benefit for disability scores.

In terms of safety, vitamin D supplementation did not increase the risk of serious adverse events, which included outcomes such as death, hospitalization, pregnancy complications, and kidney stones. **For details of all news stories go to page 1**4

Sheffield CAR-T cell trial for prog-MS - early results promising

A specialist team of doctors, nurses, researchers and pharmacists in Sheffield have successfully delivered CAR T cell therapy to a patient with progressive MS as part of an international prog-MS clinical trial.

The clinical work was filmed by the BBC at the NIHR (National Institute for Care and Health Research) Clinical Research Facility: Sheffield, and featured interviews with Professor Basil Sharrack and Professor John Snowden (Se link on p14). The BBC also interviewed the first Sheffield CAR T MS trial participant Numan Unees, who was diagnosed with MS six years ago and was left unable to walk unaided. The 40-year-old from Rotherham has become just the seventh person in the world to receive the pioneering new treatment at Sheffield's Royal Hallamshire Hospital.

Numan Unees was diagnosed with MS six years ago and, within months, lost his ability to walk. Unable to work or drive, the father of two says the groundbreaking CAR T-cell therapy has given him renewed hope for the future. Speaking just a month after receiving the immunotherapy, which was originally developed for some cancers, Numan told the BBC it would be “like starting a new life” if it works and he regains some mobility.

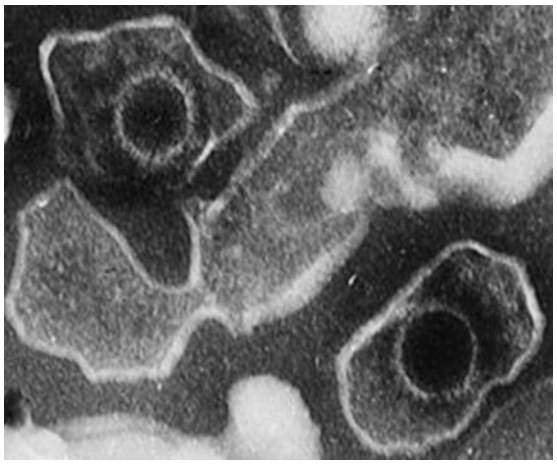
In MS, CAR T aims to reset the immune system by depleting B cells which are believed to be driving the autoimmune attacks causing the disease. Once infused back into the body, the patient's own modified T cells - which are given new instructions in the lab - act as a living drug to target and eliminate B cells.

It will take several years of careful study and research in clinical trials to work out if CAR T therapy is an effective and safe treatment for MS, but experts are describing it as a "significant moment" for MS patients. The trial is currently open to patients aged 18 to 60, diagnosed with progressive MS who are not responding well to existing medication and whose disability is worsening.

The complex, specialist treatment was delivered in Sheffield thanks to the work of pharmacy, nursing and research teams and the collaboration between haematology and neurology teams.

For the clinical trial researchers are recruiting up to 18 patients globally and the first phase is focused on testing the safety of the therapy. As this is research and not a treatment available yet, patients are asked not to call Sheffield as there is no further information at this stage. As this treatment is only available in clinical trials at the moment, the Shefield researchers ask you not to contact your GP or MS clinical team about CAR T as it is not something that they will be able to offer as part of current NHS care.

For details of all news stories go to page 14



Are we talking about a cure for MS?

The belief that EBV, the glandular fever virus, (pic left) causes MS has been around for decades. But over the past few years a growing body of research has further supported this view.

In a paper published in 2022 Prof. Alberto Ascherio of Harvard University said there was now 'compelling evidence that MS is a rare complication of infection with EBV, the Epstein Barr virus.'

And Prof. Ascherio produced some impressive figures to back up this 'rare complication' claim. In his paper published in the journal 'Science' Ascherio said that the MS risk is minimal or non-existent in individuals who are not infected with EBV but the risk increases over 30-fold following EBV infection. Sadly, despite Ascherio's remarkable findings there has been, until now, no hard evidence of the biological process by which EBV might cause MS. However, all that changed this Summer.

In July 2026 a group of US researchers led by Dr. Natalia Drosu, Postdoctoral Fellow, at Massachusetts General Hospital and a leading MS researcher published new research showing the mechanism by which EBV can 'trick' the immune system eventually leading to demyelination and MS. The results were published in the journal 'Science Translational Medicine'.

In her paper Dr Drosu revealed that people with MS have abnormally high numbers of CD4+ T cells. These cells are white blood cells that help coordinate the body's immune response. In MS, they specifically target proteins that EBV produces when copying itself.

People with MS have about twice as many of these CD4+ T cells as those who are infected with EBV but who don't develop MS. In the unlucky few of us who do carry the virus and do develop MS, Drosu believes a demyelinating disease process begins, driven by these CD4+ T cells, and goes something like this.

A set of memory B cells 'corrupted' by EBV act as instructors that mistakenly tell CD4+ T cells that healthy brain tissue or parts of it poses a threat.

Continued on page 14

Continued from page 5 .. This causes these T cells to react to the threat, enter the brain, and destroy myelin (the protective layer around nerves), a process probably caused by 'molecular mimicry', something that happens when a virus protein looks like a body protein. It has been noted that myelin and EBV share very similar proteins.

Of course, anyone who has followed the gradual acceptance of the views of researchers like Ascherio and Drosu will be heartened by the growing number of 'MSologists' who now agree that EBV is involved in some way in MS. However, there are still many 'nay sayers'. Such people ask how can a virus that infects 95% of the population only cause a serious neurological disease in less than 1%. It's a good question and probably the best person to answer it isn't a neurologist but a virologist, So, I asked a virologist, who incidentally believes EBV causes MS and this is what I was told.

The answer lies with the polio virus. While most people think of polio as universally disabling, the polio virus, which like EBV is highly contagious, generally results in mild flu-like symptoms. Fewer than 1% of all polio infections result in paralysis (paralytic poliomyelitis). This means that only 1 in 100 or even as few as 1 in 500 people who catch the polio virus will experience irreversible paralysis. To paraphrase Ascherio one might say that paralytic poliomyelitis is a rare complication of infection with the polio virus. You can see the similarities with MS.

Having explained the mechanism behind the MS disease process, Drosu is now looking to the future and the use of drugs which target EBV as MS treatments. Fortunately, there is an drug currently undergoing phase III trials both in progressive and relapsing remitting MS, The drug is given by infusion and is called frexalimab. There are a whole raft of HIV drugs that might work some of them are being looked at including tenofovir, now in clinical trials, but let's stick with Frexalimab for the time being.

Frexalimab targets the specific communication pathway that EBV uses to trigger immune activity in MS. In Phase II 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 III results hold, this could be the first MS drug that actually addresses the root cause of the illness rather than broadly suppressing immune function. If all goes well and the drug gets 'fast track' approval then frexelimab could be in clinic by 2028. Although it's presently given as a monthly infusion, it's also being looked at as a subcutaneous (under the skin) at home treatment. 2028 could be the year that changes everything.

I hate to use the 'C' word, but are we talking about a cure? Ask me in two years' time.

For further details of all news and features go to page 14

EBV virus pic P5 Wikimedia Commons

Early data from new prog MS drug trial expected by end of 2026

Early results from a Phase 3 clinical trial testing vidofludimus calcium in people with progressive MS are expected by the end of the year. If the trial results are positive, the developer says it plans to seek approval of the drug in the U.S.

Bio-pharma Immunic is pursuing vidofludimus calcium as a potential treatment for progressive forms of the disease. Its mid-stage CALLIPER trial enrolled 467 adults with primary progressive MS or secondary progressive MS who received 45 mg vidofludimus calcium or a placebo for up to 120 weeks, or nearly 2.5 years.

The treatment, taken as tablets, significantly reduced the risk of confirmed disability worsening compared with a placebo. Importantly, this risk was reduced even in patients without signs of active inflammation on MRI scans, supporting the idea that therapy may act on more than inflammation alone.

After completing the main CALLIPER trial, participants are offered the opportunity to enter an open-label extension and continue receiving vidofludimus calcium for as long as eight years. As of May 2026, 340 participants were still being treated in the extension part, according to the developer.

“We remain highly encouraged by the consistent and differentiated profile of vidofludimus calcium,” said Jason Tardio, president and chief operating officer of Immunic. “We believe this approach has the potential to deliver meaningful efficacy while maintaining the favourable safety and tolerability profile observed to date, positioning vidofludimus calcium as a potentially important new oral treatment option for people living with MS.”

As well as the CALLIPER rial Immunic is also testing vidofludimus calcium (IMU-838) in people with relapsing forms of MS in two Phase 3 trials, ENSURE-1 (NCT05134441) and ENSURE-2 (NCT05201638),

If any of the results are positive, the developer plans to submit a new drug application to the U.S. Food and Drug Administration in mid-2027, with a potential approval targeted for 2028, the company said in a press release providing financial results and a corporate update.

For further details of all news and features go to page 14

FDA approves first human trial of oral MS remyelination therapy

The US Food and Drugs Administration (FDA) has approved a first-in-human trial testing of PTD802, a remyelination therapy for MS. The treatment from Pheno Therapeutics is designed to boost repair of myelin, the protective coating surrounding nerve fibres and in such way control disease progression.

Pheno Therapeutics has been given the green light to begin clinical testing of PTD802, the company's oral (pill) treatment for boosting myelin repair in people with MS. The experimental remyelination therapy aims to potentially restore lost function in MS patients which is marked by loss of myelin, the protective coating surrounding nerve fibres.

With the FDA's clearance of its investigational new drug (IND) application, Pheno will now move forward with a first-in-human U.S. Phase 1 clinical trial. Such testing will first evaluate PTD802's safety and tolerability in healthy volunteers, according to a company press release announcing the regulatory agency's decision. The developer received a similar authorization from U.K. regulators early last year.

While existing disease-modifying therapies can reduce inflammation and prevent or slow further damage, no approved therapy can directly restore myelin that has already been damaged or lost.

Given this, researchers have long sought therapies capable of promoting remyelination — essentially, the repair of damaged myelin and production of new myelin — as a way to reverse damage and possibly restore lost function.

PTD802 aims to stimulate the body's own myelin repair processes. Developed by Pheno under an exclusive worldwide agreement with Belgium-based UCB, PTD802 is a selective small molecule antagonist, or inhibitor of the GPR17 receptor protein.

This receptor protein acts as a brake on the maturation of oligodendrocytes, the cells responsible for producing myelin in the brain and spinal cord. By releasing that brake, the therapy is designed to stimulate the body's natural myelin repair processes and promote remyelination. According to Pheno, PTD802 may ultimately be used alongside existing MS treatments to better control disease progression. Also, by reversing demyelination, it may help patients potentially regain some lost function.

Pheno called the FDA decision a "milestone." A company spokesman said: "As the first company to gain approval to begin clinical trials for a selective GPR17 antagonist, we are proud to be leading the way, and believe this approach has the potential to offer real patient benefit, in MS and beyond."

For details of all news stories go to page 14.

Stroke drug may ease MS damage, animal studies suggest

A stroke medication called ‘nimodipine’ could help preserve and repair myelin damage in MS. That’s according to the results of a ‘meta-analysis’ of existing studies.

Further research and early-phase human trials will be needed to determine if nimodipine can safely provide similar benefits in patients.

The ‘meta-analysis’ showed Nimodipine was also associated with reduced inflammation, nerve damage, and disease severity in several of the studies reviewed, suggesting it may be repurposed to treat MS.

The meta-analysis “Nimodipine in animal models of demyelination relevant to multiple sclerosis: a systematic review,” was published in the journal ‘Brain Research’.

Nimodipine is a prescription drug that is widely used to prevent blood vessel spasms following bleeding in the brain. It works by relaxing narrowed blood vessels to improve blood flow to injured brain tissue.

Research has suggested the treatment may also reduce inflammation and nerve damage while helping to rebuild myelin, and some studies have tested it in animal models of MS. A team of researchers in Iran reviewed the available evidence from these studies.

A total of five studies were included in the meta-analysis. In these studies, Nimodipine was associated with less myelin loss and fewer demyelinated nerve fibres. In one study, the treatment also increased the number of nerve fibres showing signs of remyelination. These findings were consistent with increased activity of myelin-related genes and higher numbers of cells actively involved in myelin regeneration.

Animals treated with nimodipine also exhibited fewer signs of inflammation in the brain and spinal cord, as well as lower blood levels of some inflammation-related proteins, which in one study resulted in less nerve damage.

Separately, the drug was linked to improved spinal cord blood flow and oxygenation, addressing a mechanism related to low oxygen levels proposed to contribute to nerve dysfunction in an animal model of MS. One study showed that nimodipine treatment reduced clinical scores and improved motor performance.

For further information go to page 14



Can Weight Loss Drugs treat MS?

You only have to hear the word ‘Ozempic’ and everyone knows what you are talking about. Several of my friends are taking the drug and you can literally see the weight falling off. It’s impressive. But what is more impressive is a story doing the rounds that Ozempic may also have the potential to treat MS and slow its progression. It’s a story that’s stranger than you might think.

The story starts with the simple observation that there’s a strong link between MS and obesity. Being overweight is associated with worse MS symptoms, greater disease severity and earlier disability. MS is more prevalent in developed or developing countries where obesity is a problem. But, how exactly might wight loss drugs work in treating MS?

Researchers have found that obesity makes MS worse in three ways: weight gain can cause chronic inflammation, cause immune system changes and worsen co-existing health conditions or ‘co-morbidities’.

Traditional approaches to controlling weight in people with MS focus on approaches like following healthy diets and taking regular exercise. In recent years more radical measures have been suggested. Intermittent fasting, has been proposed as a way of slowing progression and there is some suggestion this may be effective.

But what about weight loss dugs themselves? A 2024 study by a group of researchers led by Afsaneh Shirani head of MS/Neuroimmunology at the University of Texas Southwestern Medical Centre and Chief of Neurology at the Dallas VA Medical Centre, looked at this subject. The researchers’ findings were published in ‘Therapeutic Advances in Neurological Disorders’.

. **Continued on page 11**

Continued from p10: One of the weight loss drugs studied was metformin, which is used to treat type 2 diabetes, a condition commonly linked with weight gain. Metformin is currently being studied in the UK based Octopus trial looking for drugs which might slow progression in MS. The Octopus trial is expected to report its main findings by 2028 and there are high hopes that it will reproduce the findings of Shirani's 2024 study which showed some antidiabetic medications that also promote weight loss can help in MS, particularly in progressive MS.

But what about other weight loss drugs like the increasingly popular and hugely effective Ozempic and Wegovy? Although Shirani and her team of researchers agreed that weight management helps people living with MS, they warned it was too early to turn to GLP-1 (Glucagon-Like Peptide-1) medications such as Ozempic and Wegovy as treatments for MS itself.

"We are not recommending that patients use these drugs until scientific data from actual clinical trials in MS are available," said one of Shirani's co-authors Anne Cross. "We must wait to see what well-done studies in people with MS will show. We do not recommend that patients take these medications now unless they need them for another reason such as weight management," she added.

Those words were written in 2024. Fast forward to 2025 and a study published in April last year provided the first evidence that GLP-1 drugs could help in MS, working in a similar way to metformin. The 2025 study, published in the journal 'Neurology' titled 'Exploring the Effect of GLP-1 Agonists on Multiple Sclerosis Disease Progression', concluded that the use of these drugs correlated with reduced risk of MS disease progression as measured by Kurtzke Functional Systems Scores (FSS).

Sadly, we will have to wait until 2028 when the findings of the Octopus trial are published for a more reliable indication about metformin. Until then regular exercise and a balanced diet will continue to be recommended as the best way of maintaining good health.

But being of normal weight I have taken a different approach and I have started intermittent fasting, skipping breakfast daily for the past three months. Intermittent fasting may help a little and carries little or no risk. Given there are currently no treatments for my type of MS (non-active secondary progressive MS) I would argue I need all the help I can get. To quote the supermarket advert, every little helps.

Thanks to to i yunmai on Unsplash for pic on p10

For details of all features go to page 14

News

Blood protein marker may predict hidden MS progression

Higher levels of a blood protein called GFAP are associated with an increased risk of disease progression in the absence of MS relapse activity according to a new study.

The study also found that patients whose GFAP levels declined after starting MS treatment had a lower risk of future progression independent of relapse activity (PIRA), suggesting that changes in the biomarker may provide information about disease progression and treatment response. The study, "Serum Glial Fibrillary Acidic Protein Dynamics, Disease Progression, and Therapy Response in Multiple Sclerosis," was published in JAMA Neurology.

GFAP is found in astrocytes, cells that support and protect nerve cells in the brain and spinal cord. Increased GFAP levels can reflect astrocyte activation or injury, and they have been investigated as a marker of processes involved in MS progression.

"One of the key questions for us was whether progression leaves a different biological fingerprint than inflammation," Jens Kuhle, MD, PhD, study co-author and research group leader at the University of Basel, said in a university news story. "If these processes are biologically distinct, we also need biomarkers that reflect different aspects of the disease rather than expecting a single marker to capture everything."

To determine whether GFAP can serve as a biomarker of future PIRA (Progression Independent of Relapse Activity) risk, the researchers analysed long-term data from two studies following people with MS over time. One study included 1,709 patients followed for a median of 6.9 years, and the other 620 patients followed for a median of 13.1 years.

In line with previous studies, elevated NfL levels were associated with a 69%-100% increased risk of relapses in the following year. GFAP, on the other hand, was not associated with relapse risk, but it did show significant correlations with both long-term and short-term PIRA risk.

Specifically, elevated GFAP levels were associated with 22%-31% higher risk of PIRA over the entire follow-up period. In short-term analyses, the researchers found that patients with GFAP levels above the 84th percentile at one clinical visit were 36%-45% more likely to have evidence of PIRA at their next clinical appointment.

A researcher said: GFAP may capture more than a snapshot of disease activity and reflect biological processes that influence the future course of the disease**."**

For details of all stories go to page 14

News

Experimental compound boosts myelin repair in MS study

A small molecule, called CN045, shows promise for repairing myelin damage in MS. Preclinical studies show that CN045 can promote myelin-making cell maturation and remyelination. Further research is needed to determine whether CN045 could eventually help preserve nerve function in people with MS.

In a new 'preclinical study' researchers found that CN045 modestly increased remyelination, the process of repairing damaged myelin, in a mouse model of MS. While more research is needed before CN045 could be considered for testing in people, the findings support CN045 as an early lead candidate for developing a remyelination therapy for MS, the researchers said.

The study, "A novel small molecule remyelination therapy for multiple sclerosis," was published in the prestigious journal 'Nature'.

Within the brain and spinal cord, myelin is produced by cells called oligodendrocytes, which develop from immature oligodendrocyte progenitor cells (OPCs). Encouraging OPCs to mature into myelin-producing cells has long been considered a promising strategy for repairing damaged myelin in MS.

To identify compounds that could do this, a team led by researchers in the U.S. screened 20,000 molecules selected for properties favourable to use in the central nervous system for their ability to promote OPC maturation. CN045 emerged as the lead candidate for further study.

In lab experiments using human OPCs, treatment with CN045 increased the number of mature, myelin-producing oligodendrocytes. The newly formed cells also formed myelin-like coverings around nanofibers designed to mimic nerve fibres, showing that they could carry out a key function of mature oligodendrocytes. CN045 also promoted modest remyelination in a mouse model of myelin loss, indicating the compound can promote myelin repair in living animals.

The researchers also found several characteristics that could make CN045 a good drug candidate. The molecule was able to enter the brain, showed low cell toxicity in laboratory and animal testing, and had an intellectual-property path that the researchers said appeared open, potentially easing future development.

Overall, the team said CN045 represents a promising starting point to develop future remyelination therapies. **For details of all news stories see page 14**

References further details, etc

Page 2 Foralumab trial results forthcoming

multiplesclerosisnewstoday.com/news-posts/2026/06/30/nasal-ms-therapy-moves-closer-results-final-patient-dosed/?mc_euid=d11b71cbfa&utm_source=MS&utm_campaign=9ad6fd0c6e-Email_ENL_NON-US_MS&u

Page 3 Benefits of high dose vitamin D

https://multiplesclerosisnewstoday.com/news-posts/2026/06/25/adding-long-term-high-dose-daily-vitamin-d-reduces-ms-relapses/?utm_source=facebook&utm_medium=social&utm_campaign=news&utm_content=202607021000&fbclid=IwY2xjawS6FHxleHRuA2FlbQIxMABicmlkETFuWExmSDU3eUtRS3Nlazhjc3J0YwZhcHBfaWQQMjIyMDM5MTc4ODIwMDg5MgABHudSVS0lSZp4HhWbyiJSaDXC9yZMu-oY83Uzt_Xb4vcuyAJ8o21LWkiBe9ef_aem_9amsJxOzb9hbosHr4gR5YA

Page 4 Sheffield CART-cell trial

https://www.sth.nhs.uk/news/2026/08/03/pioneering-car-t-cell-therapy-for-ms-research-features-on-bbc-look-north/

https://www.bbc.co.uk/.../look-north-yorkshire-evening

https://www.bbc.co.uk/news/articles/c20jg8g57v1o

Pages 5-6 Research into EBV as the cause of MS

https://www.science.org/doi/10.1126/scitranslmed.adz6566

Page 7 Vidofludimus calcium trial into prog-MS

https://multiplesclerosisnewstoday.com/news-posts/2026/08/18/early-data-2-late-stage-ms-drug-trials-expected-years-end/?mc_euid=d11b71cbfa&utm_source=MS&utm_campaign=c977b811fc-

Email_ENL_NON-US_MS&utm_medium=email&utm_term=0_b5fb7a3dae-c977b811fc-74170629

Page 8 FDA approves re-myelination therapy

https://multiplesclerosisnewstoday.com/news-posts/2026/06/25/fda-clears-first-human-trial-testing-oral-remyelination-therapy-ptd802/?mc_euid=d11b71cbfa&utm_source=MS&utm_campaign=e1a67e6488-Email_ENL_NON-US_MS&utm_medium=email&utm_term=0_b5fb7a3dae-e1a67e6488-74170629

Page 9: Stroke drug may work as MS treatment

https://multiplesclerosisnewstoday.com/news-posts/2026/08/17/stroke-drug-ease-ms-damage-animal-studies-

Pages 10-11 Weight loss drug trialled in prog-MS in US

https://pmc.ncbi.nlm.nih.gov/articles/PMC11418341/
https://www.neurologylive.com/view/glp-1-receptor-agonists-linked-to-improved-physical-activity-multiple-sclerosis

Page 12 Blood protein predicts MS progression

https://multiplesclerosisnewstoday.com/news-posts/2026/08/11/blood-protein-marker-predict-hidden-ms-progression/?mc_euid=d11b71cbfa&utm_source=MS&utm_campaign=5e1475f3d3-Email_ENL_NON-US_MS&utm_medium=email&utm_term=0_b5fb7a3dae-5e1475f3d3-74170629

Page 13: Potential MS therapy improves myelin repair

https://multiplesclerosisnewstoday.com/news-posts/2026/07/28/potential-ms-therapy-improves-myelin-repair-cells-mice/?mc_euid=d11b71cbfa&utm_source=MS&utm_campaign=b35f28f2c5-Email_ENL_NON-US_MS&utm_medium=email&utm_term=0_b5fb7a3dae-b35f28f2c5-74170629

To Contact me email iancookjournalist@yahoo.co.uk

DISCLAIMER:

This ezine is strictly a news and information ezine/website about MS . It DOES NOT provide medical advice, diagnosis or treatment. This content is not intended to be a substitute for professional medical advice, diagnosis, or treatment. Always seek the advice of your doctor or other qualified health provider with any questions you may have regarding MS or any a medical condition. Don't disregard professional medical advice or delay in seeking it because of something you have read in this ezine. To Contact me email iancookjournalist@yahoo.co.uk