

My prog-MS ezine

For those with progressive MS and other interested people

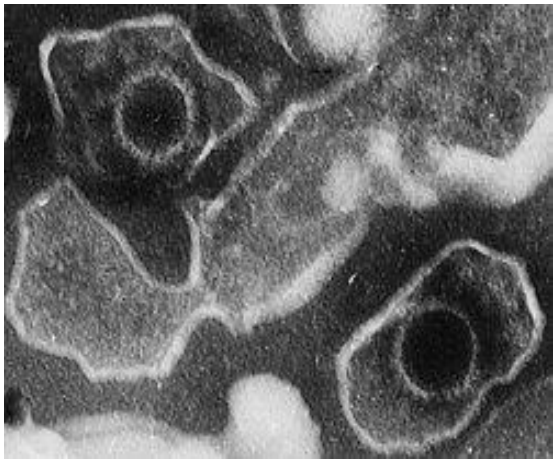
Issue 69, Nov-Dec 2026

Hello, and welcome to the 69th 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 still 6.5 and my age is still 68.

In issue 69 there are, as ever, eight pages of prog-MS news stories. There are also two features. The first, on page 5, looks again at the Epstein Barr virus (EBV) and MS, highlighting the fact that more than 60 papers or abstracts were devoted to this subject at the recent big ACTRIMS/ECTRIMS MS conference in Canada. EBV suddenly seems to be being taken seriously by the MS world. Everyone is now talking about EBV, not just me.

On page 9 another feature looks at another subject that seems to be talked about more and more in the MS world. I've even seen it on BBC TV news programmes. The subject is CAR T cell therapy which is a bit like a less invasive form of stem cell therapy for MS. There are several trials taking place across the world and some involve us progressive MS patients and of course no-one yet knows whether it will work, but hopes are high.

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Picture left EBV virus Wikimedia commons

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News

Alpha Lipoic Acid's shows promise as prog-MS treatment

Alpha-lipoic acid (also known as ALA) could become the first neuroprotective treatment in progressive MS. That's according to researchers from University College London.

But the researchers stress that people should not attempt to acquire ALA outside a clinical trial (ie: online) as further research is needed to fully understand its efficacy - and particularly its safety in MS.

The progressive MS trial, called Octopus is led by the University College London (UCL) Queen Square MS Centre through its Innovative Clinical Trials Unit (InCTU). The Octopus trial uses a multi-arm multi-stage (which means it can test more than one drug at the same time. As well as testing high dose alpha-lipoic acid the Octopus trial has been testing a diabetes drug, metformin, and comparing Alpha-lipoic acid and Metformin against a placebo.

Unlike Alpha-lipoic acid, Metformin did not show sufficient promise to be taken forward. Metformin will now be removed from the Octopus trial freeing up space for another drug.

More than 850 people have joined the Octopus trial at hospitals across the UK and Australia. Now the researchers are seeking hundreds more people with primary or secondary progressive MS to sign up. To find out more go to https://ukmsregister.org/octopus. In the coming years Octopus will become the world's largest ever trial in progressive MS. The trial design means other drugs could be added as soon as early 2027.

Octopus is being led by leading neurologist Professor Jeremy Chataway (UCL Queen Square Institute of Neurology). He said: "This is very exciting and proves not only that the Octopus trial design works, but that a new way of protecting nerves could be on the horizon. Of course, learning that metformin will be discontinued is disappointing. But finding treatments for progressive MS is extremely difficult, and we're so pleased to be taking alpha-lipoic acid into the second stage of the trial.

"We're now looking forward not only to seeing what the final results are, but also to adding in new 'arms' to Octopus next year. In the meantime, we do need to remind people with MS to not try to source unproven drugs outside the safety of a clinical trial."

Some versions of lipoic acid are available as dietary supplements, but they differ somewhat from the version used in research. Lipoic acid is approved in Germany to treat neuropathy, but not MS.

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HIV drug reverses paralysis and vision loss in ‘mouse MS’

An HIV ‘drug derivative’ reversed paralysis and vision loss in a mouse model of MS. Now, researchers hope that the drug may work in human MS as it is already in clinical trials for eye conditions in humans and early human results are promising.

The drug, called Kamuvudine K-9, is a derivative of HIV drugs already approved for use in humans. It was developed by Jayakrishna Ambati, MD of University of Virginia, US. In Ambati's research published in the journal ‘Science Translational Medicine’., K-9 preserved nerve fibres and their myelin insulation that MS destroys.

The drug also halted the rise in neurofilament light chain (NfL), a blood biomarker of nerve damage. In the same experiments, animals treated with an approved MS drug did not recover nearly as much function as those receiving K-9. The results are striking because existing MS treatments are designed to reduce future inflammatory attacks, not restore neurological function.

Ambati said the case for the therapeutic potential of K-9 is bolstered by the apparent MS protection offered by the HIV drugs K-9 is derived from, HIV drugs called nucleoside/nucleotide reverse transcriptase inhibitors, or NRTIs. NRTI use in patients, Ambati's team reports in a new paper, is associated with a 36% reduction in the annual relapse rate among patients with MS. The researchers drew this conclusion based on an analysis of health insurance data from more than 3 million people. K-9 has already entered clinical trials in eye diseases, including diabetic macular oedema and thyroid eye disease. That existing clinical experience could help accelerate evaluation of K-9 in MS.

The researchers emphasize that K-9 remains experimental for MS and that controlled clinical trials will be required to determine whether the recovery seen in mice can translate to people. They hope the work will also inform studies of other neurodegenerative diseases in which ‘inflammasome activation’ and axonal injury are implicated, including amyotrophic lateral sclerosis (ALS), a form of motor neurone disease in which K-9 is set to be tested."K-9 and the related K-8, which has already shown remarkable efficacy in macular degeneration, could also be beneficial in other diseases such as Alzheimer's disease and Parkinson's disease,” the researchers said.

Those interested in the use of HIV drugs in MS will know that certain HIV drugs like tenofovir (TDF or TAF) also inhibit the replication of EBV, a virus strongly linked to triggering MS and are currently being trialled in MS research.

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More nerve damage linked to less daily activity in prog-MS

Higher blood levels of neurofilament light (NfL), a marker of nerve damage, is linked to lower levels of daily activity in progressive MS, a study has found.

Study participants whose NfL levels increase more rapidly over time tended to be walking or exercising less. A 'reverse association' was also observed, where study participants whose NfL levels decreased more rapidly tended to be walking or exercising more. In very simple terms the more you exercise the less nerve damage you seem to get. In other words, exercise is good for prog-MS.

Higher blood levels of neurofilament light chain (NfL), a well-established marker of nerve damage, are associated with lower daily activity in people with progressive forms of multiple sclerosis (MS), the study found.

The study also demonstrated that patients in whom NfL levels increased more rapidly over time tended to experience greater declines in daily activity, as measured by the number of daily steps using a wearable device.

The findings suggest that "reduced daily physical activity is associated with greater ongoing tissue injury measured by NfL," researchers wrote.

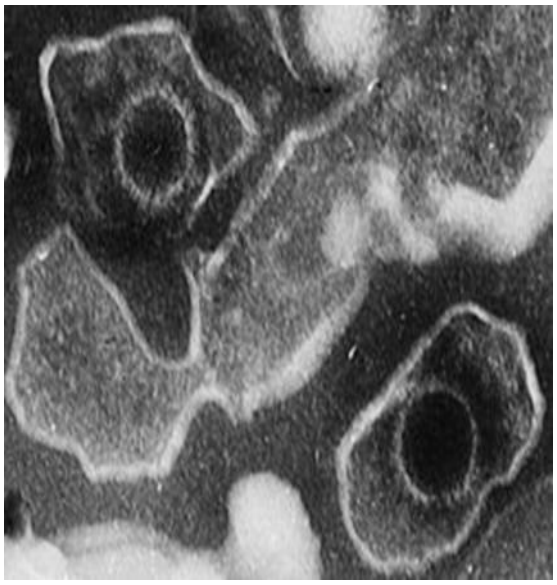
The study, "Association Between Neurofilament Light Chain and Real-World Ambulatory Function in Progressive MS," was published in Annals of Clinical and Translational Neurology.

As part of the trial, participants' daily step counts were assessed using a wearable device, and their NfL levels were also measured at multiple visits over time. The current analysis included 506 participants who had at least one blood NfL measurement paired with a daily step-count measurement during the study.

Over the 27-month study period, higher blood NfL scores were associated with lower daily step counts. Specifically, each 1-point increase in the NfL score was associated with approximately 3.4% fewer daily steps.

The reverse association was also observed, with a 10% increase in daily steps being linked to a 0.015-point lower NfL score. These associations remained consistent across sex, disability level, trial treatment, and use of disease-modifying therapies.

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The ‘MS virus’ that won’t goes away

EBV really is the virus that won’t go away. Once infected with Epstein Barr (EBV) the virus stays in your body forever. The same might be said about EBV’s presence at big MS conferences. Once MS researchers start talking about EBV, the subject never goes away.

With more than 60 abstracts and papers dedicated to EBV in MS, the virus was ever present at the recent ACTRIMS/ECTRIMS MS conference held in Toronto, Canada. And more than just talking about EBV there were several attempts to explain how EBV actually causes MS.

To start at the very beginning, it is well known that 95% of the population is infected with EBV but the vast majority don’t get MS. It’s also known that EBV infection comes before MS in virtually every case, and that the EBV rate in MSers is not 95% but 99.9%. So, clearly EBV is involved with MS, but how? Given the above, there’s a lot of explaining to do.

In 2023 Prof. Alberto Ascherio of Harvard University published a landmark paper in Nature titled ‘Epstein-Barr virus as a leading cause of multiple sclerosis’. This paper looked at blood samples taken from people pre and post EBV infection.

The paper noted that MS risk is minimal in individuals who are not yet infected with EBV but that risk increases over 30-fold following EBV infection. Ascherio’s paper said molecular mimicry and an altered immune response to EBV infection may be involved in converting EBV into MS in a small susceptible minority. I will try and explain what that means later. But first let’s talk about the virus itself.

EBV is a herpes virus. Like other herpes viruses such as cold sores and chickenpox it is quite likely that EBV infection, which manifests as glandular fever, could return years later. However, and this is a big however, cold sores always return as cold sores and chicken pox returns as shingles, a fairly similar illness to chickenpox. MS is a neurological disease, nothing like symptomatic EBV (glandular fever.) So doesn’t this theory fail the plausibility test?

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Continued from page 5 Well, not necessarily, at least not according to several papers presented in Toronto. The current thinking is that EBV, which is always present and under control in the vast majority of the population (95%), is 'poorly controlled' in the tiny minority of us who are later diagnosed with MS. Several studies at the Toronto conference supported this and outlined the biological process involved in this 'poor control' idea.

At the conference several brain-tissue studies were presented which found large numbers of immune cells (CD8 "killer" T cells) in the MS brain. This is significant because CD8 "killer" T cells are the cells that target EBV in glandular fever and this suggests MS brains are in a state of 'hyper-vigilance' for EBV.

The implications of this hyper-vigilance were explained perhaps in another study presented in Toronto. This showed that CD8 T cells can cross-react with EBV-infected cells in those with poorly controlled EBV. This 'cross-reaction' is called molecular mimicry. Sadly, the biology is too complex for a non-scientist like me to explain but the important point is that molecular mimicry starts a cascade of autoimmunity ending in demyelination and MS.

So, if EBV is accepted as the cause of MS, the next question is to ask which treatments can target EBV in MS? Rituximab and Ocrelizumab lower antibodies against the EBV protein called EBNA1. This may help explain why these drugs work so well in MS.

And what about the drugs that have recently been trialled to specifically target EBV in MS One of these drugs, vidofludimus calcium, has recently been tested in a phase 2 progressive MS trial called CALLIPER. Trial data showed a 23.8% overall reduction in 24-week confirmed disability worsening (24wCDW). Immunic, the biopharma behind the drug, plans to start a phase 3 trial in progressive MS later in 2026.

So, we may have to wait a few years for a drug that treats EBV in progressive MS. In the meantime, it is reassuring that the treatments we already have, particularly therapies, like rituximab appear to dampen EBV activity. Rituximab is a treatment for prog-MS in Scandinavia, so shouldn't it be offered to us in the UK? And what about ocrelizumab which also appears to have some anti-EBV activity? Shouldn't that be offered to more of us? Let's hope that Toronto. With its 60 EBV papers on EBV has shown us that EBV really is the virus that won't go away, and strange as it may sound that may be a good thing for us with prog-MS.

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EBV virus pic P5 Wikimedia Commons

US health watchdog gives fast track review to PPMS drug

The U.S. Food and Drug Administration (FDA) is reviewing an application from bio-pharma Roche seeking approval for its BTK inhibitor drug fenebrutinib as a treatment for primary progressive MS (PPMS).

Further, the FDA has granted the application 'priority review', under which the regulatory agency aims to make its decision within six months, compared with the 10 months it usually takes. This means a decision on whether or not to approve the drug would be expected by late March or early April 2027. Roche said the filing makes fenebrutinib— a drug that blocks the abnormal growth of B cells — the first BTK inhibitor to have an application accepted for review in PPMS.

Fenebrutinib is designed to block a protein called Bruton's tyrosine kinase (BTK), hence its BTK inhibitor label. BTK is essential for the activity of certain immune cells involved in MS. Inhibiting BTK is expected to reduce the inflammatory attacks that drive MS relapses, as well as inhibit the smouldering inflammation inside the brain that contributes to gradual disease progression over time across progressive MS types.

The application for fenebrutinib is supported by data from three ongoing Phase 3 clinical studies FENhance 1 (NCT04586010), FENhance 2 (NCT04586023) and FENtrepid (NCT04544449) In the first two studies Fenebrutinib significantly reduced the number of new and enlarging lesions as well as lesions with active inflammation, by more than 70%, and showed a positive trend toward slower disability progression.

In the third study, called FENtrepid (NCT04544449), fenebrutinib was shown to be at least as effective in adults with PPMS as the approved treatment Ocrevus (ocrelizumab). While the study was not trying to compare the two drugs, the data demonstrated that fenebrutinib numerically reduced the risk of confirmed disability progression by 12% compared with Ocrevus, the researchers noted.

The scientists noted that a treatment effect was also observed in patients without signs of active inflammation on MRI scans, indicating that fenebrutinib may help slow disability progression even in individuals with less inflammatory activity. This is particularly relevant because patients without signs of active inflammation have been shown to benefit less from Ocrevus.

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New BTKi drug to be tested in SPMS

A new BTK inhibitor drug, Remibrutinib, is currently being evaluated in a Phase 3 trial NCT07225504 called 'REMASTER' to test the drug's efficacy in patients with secondary progressive MS (SPMS), including non-active (non-relapsing) forms. The trial aims to recruit 1,275 patients and completion date is 2034.

Bruton's tyrosine kinase (BTK) inhibitors (BTKIs) are oral medications (pills) that block the BTK enzyme, a signalling protein critical for activating specific immune cells involved in MS. Being small molecules BTKi drugs can easily get into the brain where they target brain cells called microglia and macrophages, slowing progression and long-term disability worsening.

The trial is worldwide and UK trial locations are Bristol, Coventry, Glasgow, Leeds, London, Nottingham, Stoke-on-Trent, Brighton, Romford in Essex,

The main goal of the trial is to find out if remibrutinib is effective in treating SPMS and is safe to use. Some participants will receive remibrutinib, while others will get a placebo at first. Later, all participants will receive remibrutinib. The study is a crucial step in potentially bringing a new treatment option to people living with SPMS. It is currently recruiting.

To join this study, people must be between 18 and 65 years old and have a confirmed diagnosis of Secondary Progressive MS (SPMS). It's important that you haven't had any new MS attacks or relapses in the last two years, and your disability has shown some progression in the past year.

If you decide to take part in this study, you would first go through a screening process to check that you're suitable. Once you're in, you'll be randomly assigned to either receive remibrutinib or a placebo (a dummy drug) for the first part of the study. Neither you nor the study team will know which one you are getting. After this initial 'blinded' period, everyone will then get the chance to receive remibrutinib openly. The drug comes from biopharma Novartis.

Throughout the study, you'll have regular hospital or clinic visits. During these visits, you'll likely have physical exams, blood tests, and MRI scans to monitor your health and see how the medication is affecting your MS. The doctors will also ask you questions about your symptoms and how you're feeling. The study is designed to continue until a certain number of progression events have occurred in the study group, which means the total duration could vary and will be explained by the study team.

For details of all news stories go to page 14.

Neuro-protective therapy in Phase 2 testing in progressive MS

A Phase 2 trial of 'neuroprotective' drug Lucid-MS is currently underway in people with progressive MS.

The trial is taking place following recent clearance from the U.S. Food and Drug Administration (FDA). It will evaluate the safety, tolerability, and efficacy of Lucid-MS in people with primary progressive MS (PPMS) and secondary progressive MS (SPMS).

Most MS disease-modifying therapies work by suppressing or modulating immune activity, which can help reduce relapses and lesions and slow disease progression. However, these therapies generally cannot repair damaged myelin.

Lucid-MS, also known as Lucid-21-302, takes a different approach and is instead designed to prevent myelin damage. It works by blocking enzymes that become overactive in MS. These overactive enzymes alter myelin proteins in ways believed to make myelin more susceptible to autoimmune attacks. In simple terms Lucid-MS is designed around a novel neuroprotective approach that targets demyelination rather than solely modulating the immune system.

The Phase 2 trial will mark the first time Lucid-MS is tested in people with MS. Earlier studies in mice and healthy volunteers supported moving the therapy into this next stage of testing. Lucid-MS has been developed by Quantum Biopharma working with Thermo Fisher Scientific.

In preclinical studies involving mice with MS-like symptoms, Lucid-MS treatment for about 50 days restored motor function to levels similar to those of healthy mice. The treatment was also reported to be safe and well tolerated in healthy volunteers after both a single dose and consecutive daily doses in two Phase 1 studies (NCT05821387 and NCT06595706).

"Our team brings deep experience in [central nervous system] and MS trials, along with an integrated approach that connects strategy and execution to help deliver these complex studies," said Michael Cioffi, Thermo Fisher's vice president and general manager of neuroscience and clinical research. "By combining scientific expertise with operational excellence, we can help bring promising new therapies closer to the patients who need them," he added.

For further information go to page 14



De-ciphering the CAR T cell puzzle

It has been all over the MS news recently, supposedly a ground breaking new MS treatment. But what actually is 'Chimeric antigen receptor (CAR) T-cell therapy'?

And could this jumble of letters soon become standard treatment for those of us with MS, even progressive MS?

Well, in a nutshell (CAR) T-cell therapy is a therapy designed to remove harmful cells and 'reset' the immune system. It's already used to treat certain blood cancers, and is now being tested in all types of MS including primary and secondary progressive MS. The therapy uses immune cells called T cells, which are reprogrammed in a lab to find and deplete specific cells. In MS these are B cells, (bone marrow cells) a type of immune cell which are thought to mistakenly attack the protective myelin coating around nerves causing de-myelination and essentially cause MS.

CAR T-cell therapy does not use high-dose chemotherapy like stem cell treatment. Instead, just a few days before receiving the treatment, patients typically undergo a short, low-to-moderate intensity course of chemotherapy so it's far less invasive.

Researchers are presently studying two types of CAR T-cell therapy: Autologous CAR T-cell therapy where T cells are collected from the person's own blood, reprogrammed in the lab, and then given back to the patient. The second type of therapy is called Allogenic CAR T-cell therapy: Here T cells are taken from a healthy donor, reprogrammed in the lab and then given to the person with MS.

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Continued from p10: There are several trials going on at the moment. First, there's 'Breakfree-2', a phase 1 clinical trial. It's currently taking place at several sites across the world, including four in the UK. The trial is testing whether an autologous CAR T-cell therapy, developed by biopharma Bristol Myers Squibb, is safe in progressive or relapsing MS. The study is due to finish in 2027, so watch out for results.

The second trial, KYV-101, is also an autologous CAR T-cell therapy. This one is developed by biopharma Kyverna. This is phase 1 clinical trial based in the US. It aims to recruit up to 10 people with primary or secondary progressive MS who haven't responded to therapies to slow progression. Researchers are looking at whether the therapy can enter the brain and spinal cord, and which dose is safest. The team behind this trial presented encouraging early results from the first four trial participants at ECTRIMS 2025. Their results suggest that KYV-101 can cross into the brain and spinal cord and is well-tolerated with no serious side effects. All four participants showed stable or improved disability scores and a substantial improvement in fatigue after 12 months. This trial is due to finish in 2029.

Thirdly, a trial called 'NCT06680037' is a phase 1 study taking place across the US. Researchers are testing if donor T-cells (allogenic) can safely and effectively target the immune cells that drive inflammation and damage in MS. Researchers are recruiting up to 32 people with progressive MS. The study is due to finish in 2029.

Finally AUTO1-MS1 is a phase 1 clinical trial that began in 2025. The team are recruiting 18 people with relapsing or progressive MS The trial will test whether an autologous CAR T-cell therapy is feasible and safe in MS. The trial is due to finish in 2029. There will be a long-term follow-up phase of up to 15 years.

Like HSCT (stem cell transplant), CAR T-cell therapy aims to 'reset' the immune system. But while HSCT rebuilds the immune system using stem cells, CAR T-cell therapy uses reprogrammed T cells to specifically target B cells. This could offer a more precise 're-programming' approach that is less invasive than HSCT. Given that the first trial is due to report in 2027 we may have some idea, as early as next year, whether CAR T-cell therapy is the way forward in treating MS. Watch this space!

'Picture p1 and 5 By Camille Gévaudan - Own work, CC BY-SA 4.0, https://commons.wikimedia.org/w/index.php?curid=43310910

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Positive stem cell therapy response seen in progressive MS

US researchers have identified four cerebrospinal fluid (CSF) biomarkers that may track response to MSC-NP (mesenchymal stem cell-neural progenitor) stem cell therapy in progressive MS. Early results look promising.

Levels of specific inflammatory and repair-related molecules altered significantly following MSC-NP treatment in clinical study participants. This indicates a positive outcome, showing the treatment may work in prog-MS. The levels of these four molecules — biomarkers of inflammation, immune responses, and potential repair processes — were found to be significantly altered after a type of stem cell treatment called MSC-NP (mesenchymal stem cell-neural progenitors) treatment.

A study outlining this work, "Effect of intrathecal injection of mesenchymal stem cell-neural progenitors on cerebrospinal fluid biomarkers in progressive multiple sclerosis," was published in Stem Cells Translational Medicine.

MSC-NP stem cell therapy involves collecting MSCs from a patient and engineering them into neural progenitors, a type of stem cell that can generate new nerve cells (neurons) and other types of brain cells. The engineered MSC-NPs are administered to the central nervous system via intrathecal injection (into the spinal canal), with the aim of reducing inflammation and promoting neurological repair.

MSC-NP treatment has shown promise as a potential MS treatment in early clinical trials, but its exact biological effects on the body are not yet fully defined. Dr. Saud A. Sadiq and his team of scientists at the Tisch Multiple Sclerosis Research Center of New York (Tisch MSRCNY) set out to identify MSC-NP-specific biomarkers — that is, objectively measurable molecules that could provide insight into the effect of MSC-NP treatment. Their analysis involved data from nearly 100 PPMS and SPMS patients who received repeated MSC-NP injections across two clinical studies: a Phase 2 trial (NCT03355365) and its expanded access study (NCT03822858), both of which were sponsored by MSRCNY. The expanded access study included patients who were not eligible for the Phase 2 trial.

Results showed that in both trials, MSC-NP treatment altered levels of four key biomarkers in the cerebrospinal fluid (CSF, the liquid around the brain and spinal cord) but not in the blood. Specifically, CSF levels of CCL2, a proinflammatory molecule that's increased in MS, and SCF, a molecule thought to be involved in immune cell behaviour, dropped after MSC-NP treatment. In contrast, CSF levels of CHIT1 and MMP9, two biomarkers of the early stages of MS thought to promote neurological tissue repair, increased. **For details of all stories go to page 14**

News

€1.1M grant for novel skin patch which promotes remyelination

Researchers in Germany will use more than €1 million in new funding to investigate a novel skin patch for MS that promotes myelin repair.

The experimental patch delivers theophylline, an asthma drug, to help rebuild the protective sheath surrounding nerve fibres. The grant will support the treatment's development toward a planned early trial which will test the drug-patch safety in healthy volunteers.

Theophylline, the drug being administered is a drug long used to treat respiratory conditions such as asthma. In preclinical studies, use of the medication helped repair myelin.

The money will support the development of the patch to meet pharmaceutical manufacturing standards and advance the treatment toward an early clinical trial in healthy volunteers.

Claire Jacob, PhD, a professor at Johannes Gutenberg University Mainz (JGU), leads the team that developed the patch. He said:

“With this funding, we are taking a decisive step toward our goal of translating our research findings into a potential treatment for people with MS,” Jacob said in a university news story.

Jacob’s team has spent more than 20 years investigating the mechanisms by which the nervous system repairs damaged myelin. This research has demonstrated that increasing the activity of histone deacetylase 2 (HDAC2), an enzyme that regulates gene activity, could promote molecular processes involved in remyelination.

The team later identified theophylline — a type of bronchodilator, an oral medicine that relaxes airway muscles in the lungs — as a potential activator of HDAC2. In mouse studies, low-dose theophylline increased HDAC2 activity and promoted myelin rebuilding in the nervous system.

For details of all news stories see page 14

References further details, etc

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https://www.ucl.ac.uk/news/2026/sep/existing-drug-shows-early-promise-treat-progressive-ms

Page 3 HIV drug reverses paralysis and vision loss in 'mouse MS' model

https://medicalxpress.com/news/2026-09-experimental-drug-reverses-paralysis-vision.html

Page 4 More activity link to less nerve damage in prog-MS

https://multiplesclerosisnewstoday.com/news-posts/2026/09/28/nerve-damage-marker-tied-lower-daily-activity-progressive-ms/

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

This feature is based on reports from Prof. Gavin Giovannoni's 'MS selfie' post on Medium. Although I pay for the content (about £70 pa) I can forward content as long as you commit to subscribe. Prof. Giovannoni can be contacted at gavingiovannoni+ms-selfie-research@substack.com

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https://multiplesclerosisnewstoday.com/news-posts/2026/10/01/fda-grants-priority-review-high-efficacy-oral-drug-treat-2-ms-types/?mc_euid=9e6eaae291&utm_source=MS&utm_campaign=f53c66c543-Email_ENL_NON-US_MS&utm_medium=email&utm_term=0_b5fb7a3dae-f53c66c543-71290133

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https://www.novartis.com/clinicaltrials/study/nct07225504

https://clinicaltrials.gov/study/NCT07225504

Page 9: Neuro-protective therapy in phase 2 clinical trial

https://multiplesclerosisnewstoday.com/news-posts/2026/10/08/new-therapy-moves-closer-phase-2-testing-progressive-

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Pages 10-11 CAR T cell therapy for prog MS

The best basic introduction to this is on the MS Society's website https://www.mssociety.org.uk/research/explore-our-research/emerging-research-and-treatments/explore-treatments-in-trials/car-t

Prof. Gavin Giovannoni's 'MS selfie' post on Medium contain some material on CAR T cells and their use in MS. Although I pay for the content (about £70 pa) I can forward content as long as you commit to subscribe. Prof. Giovannoni can be contacted at gavingiovannoni+ms-selfie-research@substack.com

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https://multiplesclerosisnewstoday.com/news-posts/2026/09/22/molecules-point-stem-cell-therapy-response-progressive-ms/?mc_euid=d11b71cbfa&utm_source=MS&utm_campaign=a860529f60-Email_ENL_NON-US_MS&utm_medium=email&utm_term=0_b5fb7a3dae-a860529f60-74170629

Page 13: Skin patch may promote myelin repair

https://multiplesclerosisnewstoday.com/news-posts/2026/09/03/million-plus-grant-moves-ms-skin-patch-closer-first-human-trial/?mc_euid=d11b71cbfa&utm_source=MS&utm_campaign=e78448baad-Email_ENL_NON-US_MS&utm_medium=email&utm_term=0_b5fb7a3dae-e78448baad-74170629

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