

My prog-MS ezine

For people with progressive MS and other interested people

Issue 61, July August 2025

Hello and welcome to the 61st 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 from Birmingham, UK. My EDSS is still 6) my age is still 66. I'm 67 in August, all gifts gratefully accepted!

The ezine is now entering its 11th year and as ever there is some progress in the world of progressive MS (No pun intended). In issue 60 there are eight pages of prog-MS news stories and two features. One feature, on page 5, looks at hyperbaric oxygen as a therapy for MS. It has been around for decades but some new research suggests it may be effective and worth investigating.

There's another feature on page 10 looking at the weird world of MS drug names. We read about these drugs and often struggle with their long and unpronounceable names but does 'de-coding' them make us more informed as patients and help us better understand what we are swallowing or infusing?

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News

Nose spray Foralumab lessens disability in nonactive SPMS

Foralumab nose spray, a new therapy, being developed by Tiziana Life Sciences lessened disability in non-active Secondary Progressive MS. It also eased fatigue. That's according to the latest results of a Phase 2a study

The study (NCT06292923) is currently testing foralumab against a placebo in adults with nonactive SPMS. The trial is still recruiting at sites in the U.S. Early results found Foralumab was 'well tolerated' as well as being effective.

Foralumab is designed to treat MS by reducing the activity of T-cells, a type of immune cell that plays a central role in the inflammatory attack on the nervous system that drives MS. It also boosts the activity of certain immunosuppressive Regulator T-cells called 'Tregs', which can dampen the inflammatory function of other immune cells. The therapy is administered as a nasal spray three times a week, following a two-week on, one-week off schedule.

In the open-label trial, 10 people with nonactive SPMS whose disease continued to progress despite being on other drugs were treated with foralumab for six months or longer. Results from the study were made available in the preprint, "Nasal foralumab treatment of PIRA (Progression Independent of Relapse Activity) induces regulatory immunity, dampens microglial activation and stabilizes clinical progression in non-active secondary progressive MS." The paper has not yet undergone peer review, the process in which other scientists review the data for technical issues.

After six months on therapy, scores on the Expanded Disability Status Scale (EDSS), a standard measure used to track disability in people with MS, showed three of the ten patients experienced reductions in EDSS scores, suggesting less severe disability. Four participants were continuously treated with foralumab for a full year. Three of these four patients experienced a reduction in EDSS scores after a year on the therapy.

After six months of treatment with foralumab, six of the 10 participants reported clinically significant improvement in vision and significant reductions in fatigue severity.

Biomarker data from the study indicated that foralumab treatment reduced inflammation in the nervous system. It also dampened the activity of nerve cells called microglia in the brain, which is believed to be a main contributor to the silent disease progression experienced by people with nonactive MS.

For details of sources for all news stories go to page 14

EBV alters B-cell movement through body causing MS

The Epstein-Barr virus (EBV) — a well-established risk factor for MS and its progression — is able to alter the movements of infected immune cells setting the stage for MS, a new study claims.

The study sheds new light on how EBV may allow MS to develop several years after infection and it could potentially form the basis for new treatments for MS and other EBV-related diseases, such as Hodgkins Lymphoma and Burkitt's Lymphoma, according to researchers. The study, "Epstein–Barr virus induces aberrant B cell migration and diapedesis via FAK-dependent chemotaxis pathways," was published in the journal Nature Communications.

When EBV enters the body, the virus mainly infects B-cells (bone marrow calls). In this study, researchers discovered EBV-infected B-cells can change their behaviour and movements. B-cells and other immune cells normally reside in the blood or in immune organs called lymph nodes. But when there's trouble, like an infection or cancer, B-cells turn into 'homing cells', so named because they rush, or 'home', to the site of trouble.

Researchers from the German Cancer Research Centre (DKFZ), found EBV-infected cells produce certain inflammatory molecules that cause infected and non-infected cells to turn into homing cells. Through a series of experiments in cell and mouse models, the researchers showed EBV-infected B-cells move to other parts of the body, and they also undergo diapedesis, the process where immune cells squeeze out of blood vessels and move into the body's tissues.

This process may allow EBV-infected B-cells to move to the brain — and because B-cells in the brain are known to 'drive' MS, this could potentially set the stage for the disease. Deciphering the reaction cascade that triggers EBV-infected B-cells to enter homing mode offers the possibility of targeted intervention to prevent B-cell migration.

In their paper the researchers also say they found that defactinib, a cancer drug that has been extensively tested in clinical trials for multiple malignancies with variable efficacy, but with a good safety profile, is able to eliminate infected B cells. Defactinib is a small molecule drug so could in theory cross the blood brain barrier which might make it a potential treatment for MS.

For details of all news stories go to page 14

Fenebrutinib has benefits in active prog-MS says new study

Almost two years of treatment with fenebrutinib — an investigational tablet being developed by biopharma Genentech - helped patients with active secondary progressive MS remain nearly free of disability progression and relapses.

That's according to new data from the open-label extension of FENopta (NCT05119569), a Phase 2 clinical trial that tested fenebrutinib against a placebo, taken twice daily for three months, among a patient group including those with active secondary progressive MS. Following the trial, participants were allowed to enter the open-label extension to receive fenebrutinib for up to 192 weeks (about 3.5 years).

Fenebrutinib works by inhibiting Bruton's tyrosine kinase (BTK), an enzyme that activates immune cells such as B-cells and brain cells called 'microglia'. In MS, these immune cells are overly active, contributing to inflammatory damage in the brain and spinal cord. As a BTK inhibitor, fenebrutinib is expected to reduce inflammation and prevent damage.

In the FENopta trial three months of treatment with fenebrutinib reduced by 90% the number of new brain lesions with active inflammation on MRI scans compared with the placebo. No patient experienced worsening on the Expanded Disability Status Scale (EDSS), a widely used tool to measure disability in MS, in other words there was no progression.

MRI scans revealed no new T1 gadolinium-enhancing lesions, which show active inflammation in the brain. In patients who had initially received the placebo and then switched to fenebrutinib, the number of new or growing T2 lesions, which represent long-term damage, decreased from 6.72 to 0.34 by week 96.

Three fenebrutinib Phase 3 clinical trials are now underway. One of the trials, FENtrepid (NCT04544449) is comparing it with Ocrevus (ocrelizumab), an approved treatment, in primary progressive MS. Data are expected as soon as the end of this year, 2025**.**

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Is it time to look again at hyperbaric oxygen therapy for MS?

When I was diagnosed, some 30 years ago, hyperbaric oxygen (HBO) was the 'go-to' therapy for those open to radical new approaches to treating MS.

HBO had a dedicated group of followers and I once wrote a story about a man with MS who had a personal HBO chamber or "tank" built in his back garden at a cost of many thousands of pounds.

There was also a certain amount of evidence suggesting that HBO could work wonders in MS. And there was a network of more than 50 HBO centres dotted around the country. So what exactly is HBO, and why am I talking about it today? Well HBO is basically the medical use of pure oxygen (usually greater than 99%) administered in a sealed chamber with a higher pressure than atmospheric pressure. And I'm talking about it today because a recent academic paper has shown HBO brings dramatic improvements in a model of MS. There are plans for more research.

HBO was developed decades ago as a treatment for "the bends" or 'decompression sickness'. This is where divers come up from the deep too quickly. A quickly ascending diver has nitrogen bubbles released into their blood (air is 80% nitrogen and just 20% oxygen). An HBO chamber with pure oxygen at an increased pressure helps a diver with 'the bends' to "re-compress" and then "de-compress" safely reducing the volume and number of nitrogen bubbles in the bloodstream. HBO has saved countless divers' lives. About 40 years ago it was proposed as a treatment for MS.

After this, and very quickly, a network of around 50 MS therapy centres was set up around the UK to provide hyperbaric oxygen therapy(HBOT) These centres were independent charitable organisations, each with quite a large local following. Many people with MS still attend these centres for oxygen therapy and report noticeable improvements in their symptoms, particularly fatigue. There is strong anecdotal evidence for HBO as an MS treatment although the scientific/ medical community has been by and large sceptical about HBO**.**

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Continued from page 5 Despite this there have been one or two interesting recent studies about how HBO might, after all, benefit MS patients. There have been some recent pathology studies showing a lack of oxygen in the tissues of MS lesions. Another interesting discovery is that animal models have shown oxygen reduces demyelination and loss of oligodendrocytes - the cells that make myelin. HBO has also been suggested as a stem cell stimulator by increasing the mobilisation of bone marrow-derived stem cells in animal models of MS.

Perhaps the most interesting new research is a recently published paper which showed a reversal of quite profound neurological deficits in animals with experimental allergic encephalomyelitis (EAE) a model of the type of acute inflammation that causes MS relapses. This paper - Oxygen treatment reduces neurological deficits and demyelination in two animal models of multiple sclerosis – was written by researchers at the Department of Neuroinflammation, Queen Square Institute of Neurology, London University.

There is clearly something worth looking at here, a point made by Prof. Gavin Giovannoni in his MS-Selfie post on Medium. He says: "I am a big supporter of the need for more research in this area. . . I was helping write a grant to test HBO in acute optic neuritis when COVID-19 came along, and, like many other projects, it was abandoned. Maybe we should revisit it." Most of the research into HBO is decades old so perhaps Prof. Giovannoni is right – this subject should be re-visited. Could the UK's 50 or so HBO centres that are still operating get together and start the process of getting a new trial into HBO and MS? This suggestion comes not from me but from Prof. Giovannoni.

When I put Prof. Giovannoni's suggestion to the Neuro Therapy Network, the umbrella group for 42 of these HBO centres, a spokesperson said: "Funding a clinical trial is likely to be beyond the reach of most of our members as they vary in size and finances differ greatly."

So, will other funders come forward to fund this research, or will the efficacy of HBO in MS remain one of those many unanswered questions surrounding the condition? If anyone has a spare £2.5M stuffed down the back of the sofa, that's Prof. Giovannoni's estimate of the cost of the study using the existing infrastructure of the MS Centres, please get in touch with him - his email is g.giovannoni@qmul.ac.uk

Photo (p 5) thanks to the Neuro Therapy Network **For further details of sources for feature see page 14**

Gene variant plus glandular fever raises MS risk - large-scale study

The risk of MS is significantly higher in people who carry a variant of a gene called HLA-E*01:01 and who have a history of glandular fever, also called Infectious Mononucleosis, a disease caused by the Epstein-Barr virus (EBV). That's according to a study based on U.K. Biobank data.

The higher MS risk tied to HLA-E*01:01, which was not found among those without records of a glandular fever diagnosis, was independent of other MS-related gene variants.

The study, "Stratifying Multiple Sclerosis Susceptibility Risk: The Role of HLA-E*01 and Infectious Mononucleosis in a Population Cohort," was published in the European Journal of Neurology. EBV infection is "a key environmental trigger" of MS, "with infectious mononucleosis (IM), its symptomatic manifestation, being strongly associated with increased MS risk," the researchers wrote.

It's thought that certain EBV proteins have a similar shape to proteins within the myelin sheath. So, when immune cells make antibodies to fight the virus, they mistakenly target the myelin sheath. Because most people have been infected with EBV but only a few develop MS, unique genetics is likely to be involved in disease susceptibility.

HLA is a family of genes that help the immune system discern infectious agents from healthy tissues. In the context of MS, people carrying HLA-DRB1*15:01, a form, or allele, of an HLA gene, have a higher risk of the autoimmune disorder, while those with the HLA-A*02:01 allele appear to be protected. MS research has recently focused on the HLA-E gene, which include two forms: HLA-E*01:01 and HLA-E*01:03. Cell-based studies suggest that HLA-E*01:03, but not HLA-E*01:01, may protect against MS. At the same time, no connection between HLA-E*01:01 and MS was found in genome-wide association studies (GWAS), which look for statistical links between genetic variants and a given feature or disease.

The team of researchers in Italy and France who performed the study said they "hypothesized that the association between HLA-E*01 and MS risk may be modified by the presence of a significant immune response triggered by EBV, such as the occurrence of IM."

"These findings suggest that HLA-E*01 is associated with increased MS risk in EBV-infected individuals diagnosed with glandular fever, the researchers wrote. "If confirmed, HLA-E*01:01 screening, along with other MS-related alleles, could improve MS risk assessment and become a key tool for targeted monitoring and early detection strategies in EBV-exposed individuals, particularly those with a history of IM diagnosis."

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News

Phase 2 trial for myelin repair drug moves one step closer

Quantum Biopharma has signed an agreement with a global pharmaceutical to continue developing Lucid-21-302, an experimental therapy for promoting myelin repair in MS patients.

The collaboration will help the company put together an investigational new drug (IND) application for the therapy, which is also known as Lucid-MS. Expected by the end of 2025, the submission would seek the U.S. Food and Drug Administration's (FDA) approval to launch a Phase 2 clinical trial of the therapy in people with MS.

While several approved treatments can modify the course of MS and delay disability progression, these mostly work by reducing inflammation. To date, no therapy has been shown to help with myelin repair, which could potentially reverse some of the nerve damage and restore lost functions.

Lucid-MS is an experimental therapy Quantum is developing to prevent and possibly repair myelin damage, without suppressing the immune system, which can increase the risk of infections and other complications.

The patented medication works by blocking certain enzymes that are overactive in MS. These enzymes, called peptidyl arginine deiminases, induce protein modifications that can disrupt myelin structure and function and make it more susceptible to immune attacks.

Lucid-MS restored motor function and eased symptom severity in animal models of MS, which prompted the launch of two Phase 1 safety studies in healthy volunteers. Those now-completed studies tested single and multiple ascending doses of Lucid-MS, and data so far show the therapy is safe and well tolerated.

A 90-day study testing the toxicity and pharmacological effects of Lucid-MS was also conducted to support the IND application.

This agreement takes Quantum a step closer to initiating the phase 2 trial of Lucid-21-302, a 'first-in-class' treatment for MS.

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News

Cancer drug Methotrexate stabilises PPMS and SPMS

Injections of the anti-cancer therapy methotrexate into the spinal canal given every three months helped adults with primary and secondary progressive MS maintain stable disease levels for up to nine years.

That's according to data from a small, open-label Phase 1 clinical study (NCT02644044) that tested how safe and well tolerated intrathecal methotrexate is, and how well it works to slow disability worsening in people with these forms of MS. The study, "Intrathecal methotrexate in progressive multiple sclerosis: a phase 1 open-label study with long-term follow-up," was published in the Journal of Neurology.

Methotrexate is an anti-cancer drug that may help slow disability worsening in MS by reducing inflammation in the brain and spinal cord. It works by interfering with the normal production of immune cells. It has been widely used in other autoimmune diseases, and has been tested in relapsing and progressive forms of MS with some promising benefits.

The researchers set out to investigate the safety and efficacy of methotrexate in progressive MS given as an intrathecal injection instead of via infusions into the bloodstream. They conducted an open-label Phase 1 trial involving 22 patients — 10 with primary progressive MS and 12 with secondary progressive MS — who had experienced disability worsening in the past six months but had no relapses or active inflammatory lesions in that period.

Participants' average age was 57.6, and they had been living with MS for about 13.1 years. They were treated with intrathecal methotrexate every three months for one year. The long-term safety and efficacy of treatment was assessed in a smaller group of 10 patients who continued on methotrexate after completing the Phase 1 trial. These patients received treatment for 2-9 years.

The main goal was to watch for changes in disability levels, as measured with the Expanded Disability Status Scale (EDSS), the 25-Foot Walk (25FW), and the Symbol Digit Modalities Test (SDMT), after one year. 25FW measures walking function, while SDMT is frequently used to assess cognition. Researchers found, more than two-thirds of patients had stable or improved disability (EDSS) scores after six months (72.7%) and after one year (68.2%) of treatment. Of the 20 who completed the first six months of treatment, only 30% had disability progression in one of the three clinical measures used to assess disability , while 30% had disability improvement in one measure over the same period.

For further information go to page 14



What's the difference between an 'alumab' and a 'zumab'? The weird world of MS drugs names

If, like me, you have prog-MS, (I have naSPMS – that's non-active secondary progressive MS) you will be aware there are a few disease modifying drugs we may soon be able to take.

Two new drugs - tolebrutinib and foralumab may soon be available to people with naSPMS. This is, of course, great news for the likes of me, and in the ezine I always try to explain as much as I can about these new drugs, things like what they are and what they do.

One thing I rarely look into is the drugs' rather exotic and unusual names. I often wonder what their names actually mean, or whether you can discover any useful information about the drugs by 'de-coding' these long and unpronounceable drug names. Well, the answer is 'yes you can'. The drug names do actually mean something. So, I thought I would try and de-code the information locked in these names and see what they are telling us.

I thought I would start with tolebrutinib as this seems most likely to be the one I will be taking when it clears all the regulatory hurdles. When I did a little asking around, I was told that it's best if you de-code the meaning of a drug's name by working backwards. Stay with me while I try to explain.

Tolebrutinib is actually tolebru-ti-nib. And the meaning goes as follows. The ending "nib" indicates a small-molecule inhibitor ("nib" is shorthand for "inhibit") Small molecule inhibitors like tolebrutinib can get through the blood brain barrier after which the drug inhibits kinase enzymes. Helpfully the suffix "tinib" indicates tyrosine kinase inhibitors. So, we know that tolebrutinib is a tyrosine kinase inhibitor.

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Continued from p10 Tyrosine kinase inhibitors (TKIs) block chemical messengers (enzymes) called tyrosine kinases, BTK inhibitors (BTKi) cross the blood-brain barrier and work on B cells and microglia, major cellular players in active and chronic active lesions. In stopping this and expressed in very simple terms these drugs put the brakes on what is the runaway train of prog-MS which is fuelled by BTK.(Bruton's Tyrosine Kinase). The rest of the drug's name is a bit of a mystery because the manufacturers – Sanofi – haven't got back to me despite my asking them.

So, while we wait for Sanofi let's move on to Foralumab. Here we come to a whole new and different class of drug. Foralumab belong to a class of drugs called Mono Clonal Antibodies or 'MABs'. A monoclonal antibody is an antibody produced from a cell line made by cloning a type of white blood cell (B cell – "B" stands for 'bone marrow'). All subsequent antibodies derived this way can be traced back to a unique parent cell. This is one advantage of using MABs. Being clones, ie: exact copies, they're more precise than other treatments.

Sometimes you can gain more information about MABs by looking at the two letters that come before the letters "mab" in their name. Take Ocrelizumab (licensed for PPMS) and rituximab, (used off-label in SPMS). The "zu" in ocrelizumab is a two letter code for humanized monoclonal antibody. That means the monoclonal antibody has been made 'human friendly' and makes the human body most likely to treat it as a "friend" rather than a "foe".

Antibodies made from "zumabs" are less likely than "ximabs" to cause a neutralizing antibody response in the patient who receives them. Ximabs, like rituximab, by contrast, are "chimeric" (part human, part nonhuman) so, in theory, at least, they are at greater risk of causing a neutralizing antibody response (in other words your body may not respond to them or it may stop them working – 'neutralizing' them).

Understanding a little about the names of MS drugs is useful because it gives you the basis for a longer and more informed chat with your neurologist. It also opens up the possibilities of exciting scrabble games with them, should you ever move from being a patient to becoming a friend. Don't forget that "z" gives you 10 points in scrabble, "x" gives you 8. Ocrelizumab is 26, rituximab a mere 20. Whatever way you look at it, either better knowledge of the drugs you are taking or higher scrabble scores, I call that a win-win.

Picture p1 and 10 By Camille Gévaudan - Own work, CC BY-SA 4.0, https://commons.wikimedia.org/w/index.php?curid=43310910

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Early success in CAR T-cell therapy trial in prog-MS

US bio-pharma Kyverna Therapeutics' CAR T-cell therapy candidate KYV-101 was safe and well tolerated, and also showed preliminary signs of working in people with primary and secondary progressive MS.

That's according to data from two investigator-initiated Phase 1 trials, which enrolled individuals with secondary progressive MS (SPMS) and primary progressive MS (PPMS) who had not experienced recent relapses. Early findings from both trials were shared in two back-to-back presentations at this year's American Academy of Neurology (AAN) Annual Meeting, held in San Diego, California.

KYV-101 belongs to a class of treatments called CAR T-cell therapies, which involve collecting a patient's immune T-cells and engineering them in the lab to carry a man-made receptor — called a chimeric antigen receptor, or CAR — on their surface that makes them more efficient at targeting a specific protein.

The modified T-cells in KYV-101 are designed to target the CD19 protein at the surface of B-cells, allowing them to attack and destroy the disease-causing immune cells once infused back into the patient. Before cells are infused patients receive a round of chemotherapy to deplete white blood cells and create an environment that is favourable for CAR T-cell expansion and survival.

KYV-101 is being tested in an investigator-initiated open-label Phase 1 trial (NCT06138132) taking place at the Stanford Multiple Sclerosis Centre in California. In an open-label study, both participants and researchers know the exact treatment being given. The trial has so far enrolled 12 patients, ages 18 to 65, with progressive forms of MS. Among these four participants, who ranged in age from 47 to 54, were three patients with SPMS and one with PPMS who failed to respond adequately to other treatments. After a round of chemotherapy three patients received KYV-101 at a dose of 33 million cells, while one was included in a higher dose group and received 100 million cells.

Data showed that CAR T-cells were able to expand in the blood and spinal fluid, peaking between 10-21 days after KYV-101 infusion. Although the Phase 1 trial was not designed to look at clinical benefit, nonetheless, the investigator reported that the first treated patient showed a complete eradication of oligoclonal bands, a marker of brain inflammation, at six months. A clinically meaningful reduction in fatigue levels was also reported in the two patients with three-month data. **For details of all stories go to page 14**

News

Placenta stem cells eased secondary progressive MS symptoms

Stem cells from donated placentas appear safe and may help reduce symptoms of secondary progressive MS according to a small, open-label Phase 1 clinical trial involving five patients.

"Our results suggest possible neuroprotective effects" from these stem cells, researchers wrote in "Cell therapy with placenta-derived mesenchymal stem cells for secondary progressive multiple sclerosis patients in a phase 1 clinical trial," published in the journal Scientific Reports. They cautioned that larger trials are needed to confirm the benefits.

The goal of a stem cell transplant is to reset the immune system using either a patient's own stem cells (autologous) or stem cells from healthy donors (allogeneic). Donor stem cells are typically derived from circulating blood or bone marrow, but can also be collected from other tissues, such as the placenta.

In a Phase 1 clinical trial (NCT06360861) led by Tehran University of Medical Sciences in Iran, researchers tested whether stem cells from donated placentas could be safely used in adult patients diagnosed with secondary progressive MS by monitoring the side effects over six months after a transplant. They also assessed the initial treatment's efficacy.

Five patients receiving off-label treatment with rituximab. All had disease durations of more than two, but less than 16 years. Before the transplant, their Expanded Disability Status Scale (EDSS) scores ranged from 5.5 to 7, indicating a level of disability severe enough to prevent daily activities or require a wheelchair.

The patients' EDSS scores decreased in the first month after the cell injection. By the third month, two patients showed a continued decline in their scores, while the remaining ones maintained stable levels. After the transplant, the patients performed better on cognitive and psychological tests, indicating better mental function. They also tended to report less fatigue, a common symptom of MS. Functional MRI scans showed stronger connections in areas of the brain important for memory, spatial processing, and cognitive functions.

Blood tests showed a reduction in the number of B-cells, these are immune cells responsible for producing antibodies, which may be overactive in MS, and lower levels of inflammatory proteins over the three months after the transplant. Levels of IL-10, a protein that helps reduce inflammation, increased.

For details of all news stories see page 14

References further details, etc

Page 2 Nose spray Foralumab lessens disability in naSPMS

https://multiplesclerosisnewstoday.com/news-posts/2025/05/07/foralumab-shows-promise-early-study-nonactive-

Page 3 EBV role in causing MS better understood

https://multiplesclerosisnewstoday.com/news-posts/2025/05/22/ms-risk-factor-ebv-alters-way-b-cells-move-body-study/?utm_source=MS&utm_campaign=21f5ce56f5-Email_ENL_NON-US_MS&utm_medium=email&utm_term=0_b5fb7a3dae-21f5ce56f5-71290133

Original research at https://pmc.ncbi.nlm.nih.gov/articles/PMC12089463/

For details of defactinib go to https://en.wikipedia.org/wiki/Defactinib

Page 4 Fenebrutinib halts progression in trials

https://multiplesclerosisnewstoday.com/news-posts/2025/06/03/benefits-fenebrutinib-ms-sustained-2-years-study-data/?utm_source=MS&utm_campaign=118ca5a9b3-Email_ENL_NON-US_MS&utm_medium=email&utm_term=0_b5fb7a3dae-118ca5a9b3-71290133

Pages 5-6 Hyperbaric Oxygen

Academic paper at https://pmc.ncbi.nlm.nih.gov/articles/PMC10107096/

For your nearest HBO centre see https://www.neurotherapynetwork.org.uk/

Other organisations offering HBO can be found online.

Page 7 Gene variant plus glandular fever increases MS risk

https://multiplesclerosisnewstoday.com/news-posts/2025/04/23/gene-variant-plus-mono-raises-ms-risk-large-scale-study/

Page 8: Myelin repair drug moves one step closer

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Page 9 Cancer drug methotrexate may stop disability worsening

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Page 10-11 Drug names

It's a complex subject but if you want to find out more start at **https://en.wikipedia.org/wiki/Drug_nomenclature#:~:text=Nonproprietary%20names%20begin%20in%20lowercase,doxorubicin%20(Adriamycin)%22).**

See also https://accesspharmacy.mhmedical.com/content.aspx?bookid=1549&sectionid=93411751

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Page 12 Car-T-cell therapy shows improvement in trial

https://multiplesclerosisnewstoday.com/news-posts/2025/04/15/aan-2025-car-t-cell-therapy-shows-safety-efficacy-ms-trials/

Page 13 Sacral Neuromodulation

https://multiplesclerosisnewstoday.com/news-posts/2025/03/21/sacral-neuromodulation-eases-ms-related-bladder-dysfunction/?utm_source=MS&utm_campaign=1195ee58bd-Email_ENL_NON-US_MS&utm_medium=email&utm_term=0_b5fb7a3dae-1195ee58bd-71290133

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