

My prog-MS ezine

For those with progressive MS and other interested people

Issue 66, May-June 2026

Hello, and welcome to the 66th edition of my ezine about progressive multiple sclerosis and MS progression in general. My name is Ian Cook. I'm a non-active secondary progressive MSer (naSPMS) from Birmingham, UK. My EDSS is 6.5 and rising, and my age is 67 and also going up (68 in August).

In issue 66 there are eight pages of prog-MS news stories. There are also two features. The first, on page 5, looks at ulcerative colitis (UC), a bowel disease linked to MS. I know this because my mum (pictured) had UC. Does this tell us anything about MS and genetics?

On page 9 another feature looks at a recent drugs trial that could be a 'game changer' for those of us with progressive MS because it seems to tackle both inflammation and neuro-degeneration. The drug is called 'vidofludimus calcium' and I think it's one to watch. Remember who told you.

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Tolebrutinib licensed by EMA for non-relapsing SPMS

The European Medicines Agency's (EMA) Committee for Medicinal Products for Human Use (CHMP) has adopted a 'positive opinion', recommending marketing authorisation being granted for Tolebrutinib to treat 'smouldering' MS, (secondary progressive MS without relapses).

Tolebrutinib, a second-generation inhibitor of Bruton's tyrosine kinase (BTKi) will be available in the UK as 60 mg film-coated tablets when it clears the next regulatory hurdle – The UK's Medicines and Healthcare Regulatory Authority (MHRA). To be available on the NHS it will have then to be approved by NICE. Privately it is estimated that the annual cost of the drug will be somewhere between £2,000 and £4,000 for those who can't wait.

The drug will be recommended for the treatment of adult patients with secondary progressive multiple sclerosis (SPMS) without relapses in the last 2 years.

The benefits of Tolebrutinib include a 31% reduction in the risk of 6-month confirmed disability progression and a 38% reduction in the adjusted average number of new and/or enlarging T2-hyperintense lesions per year compared with patients receiving placebo.

The most common side effects of Tolebrutinib (Cenrifki) include infections, haemorrhages, an increased tendency to bruise, heavy menstrual bleeding, abdominal pain, and elevated liver enzyme levels. The most worrisome side effect is the drug-induced liver injury. (DILI) caused by the elevated liver enzyme levels.

Cenrifki should be prescribed by doctors experienced in treating multiple sclerosis. Detailed recommendations for the use of this product will be described in the summary of product characteristics (SmPC), which will be published on the EMA website in all official European Union languages after the marketing authorisation has been granted by the European Commission. (See p14 for link to the EMA website for updates)

Prof Gavin Giovannoni summed up the present situation (EMA approval) on his 'MS-Selfie' post on Medium 'Let's hope the MHRA does likewise, in parallel with NICE. People with smouldering non-relapsing SPMS deserve access to this treatment sooner rather than later.'

The EMA announcement comes as something of a surprise as in December last year The US Food and Drugs Administration (FDA) said it wouldn't approve tolebrutinib for non-active secondary progressive MS (naSPMS) in the US saying that the risks to patients' livers was too great for the drug to be given the 'green light'.

For details of all news stories go to page 14

New trial shows Ocrevus slows disability in advanced PPMS

Ocrevus significantly slows disability progression and hand function decline in PPMS (Primary Progressive MS). Benefits extend to older patients and those with more advanced disease. That's according to results of a Phase III trial.

The trial results were presented at the American Academy of Neurology (AAN) 2026 annual meeting held in Chicago by Ocrevus' developer Genentech, a Roche company. It is based on a Phase 3 ORATORIO-HAND trial (NCT04035005), presented by Jiwon Oh, MD, a neurologist at the University of Toronto.

"ORATORIO-HAND is helpful, because it confirms the significant effect of [Ocrevus] on disability in PPMS, and it confirms that there is an effect in older patients and in those with more advanced disease," Oh said.

Ocrevus, given via infusions into the bloodstream, and its under-the-skin formulation, Ocrevus Zunovo (ocrelizumab and hyaluronidase-ocsq), are the only approved disease-modifying therapies for PPMS. Their approvals were supported by data from the Phase 3 ORATORIO trial (NCT01194570), which showed that Ocrevus significantly reduced — by 24% — the risk of disability progression, as assessed by the Expanded Disability Status Scale (EDSS). The drug showed an even greater effect on hand function, reducing the risk of decline on the 9-Hole Peg Test (9HPT) by 44%.

ORATORIO included PPMS patients up to age 55, and with a maximum disability level of 6.5 on the EDSS scale, where aids like canes or crutches are needed to walk.

Due to these inclusion criteria, "currently, we actually do not know clearly the impact of [Ocrevus] in older people with PPMS and in those with more advanced disease, particularly on the worsening of hand function," Oh said.

Addressing that knowledge gap was the goal of ORATORIO-HAND, which enrolled a broader group of more than 1,000 adults with PPMS, up to age 65. The maximum EDSS score was 8, corresponding to a disability level at which patients usually rely on a wheelchair.

On average, ORATORIO-HAND participants were older, had greater disability, and had been living with MS for longer than people who participated in ORATORIO. The trial "was successful in recruiting an older and more disabled population of people with PPMS," Oh said. Side effects were consistent with the treatment's known safety profile. As expected, infusion-related reactions were more common with the treatment than the placebo, but rates of infections and cancer were similar between the groups.

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Trial of CAR T-cell therapy for progressive MS now recruiting in US

A Phase 1 trial of azer-cel, a CAR T-cell therapy, for progressive MS is now recruiting participants at eight sites across the U.S.

The study (NCT06680037) is expected to enrol up to 32 adults with primary progressive MS or secondary progressive MS to determine whether genetically engineered immune T-cells from healthy donors can safely and effectively improve patient outcomes. The first participant was dosed last year.

Under a licensing agreement, in which TG Therapeutics acquired the development and commercialization rights to azer-cel for autoimmune diseases, the trial's advancement triggered a $7.5 million milestone payment to Precision Biosciences, which developed the therapy.

CAR T-cell therapies work by taking T-cells — a type of immune cell that normally helps fight infections — and genetically modifying them in the lab to produce a special receptor on their surface called a chimeric antigen receptor, or CAR. This receptor allows the T-cells to recognize and attach to a specific target.

In the case of azer-cel, the CAR is designed to recognize CD19, a protein found on the surface of B-cells. Once infused into the bloodstream, the engineered T-cells seek out and destroy B-cells, which is expected to reduce disease activity and slow disability accumulation.

Unlike most approved CAR T-cell therapies, which are made from a patient's own T-cells, azer-cel is produced using T-cells from a healthy donor. This approach allows the therapy to be manufactured in advance as an "off-the-shelf" treatment, potentially making it more readily available to patients.

The ongoing Phase 1 trial is now testing several doses of azer-cel, given as a one-time infusion into the bloodstream. The main goal is to determine a recommended dose for future studies.

Secondary objectives include assessing safety, how the therapy behaves in the body, and potential effects on disability progression, brain lesions, and brain volume loss after up to two years. The trial is set to conclude in 2029, but top-line results are expected later this year.

For details of all news stories go to page 14



My mum's ulcerative colitis and my MS. Is there a link?

Ever since my diagnosis, more than 30 years ago, I have met people with MS who also have ulcerative colitis (UC). This condition, like MS, is an auto-immune disease which affects the colon or large intestine. Inflammation of the colon leads to ulceration, bleeding and worse. It must be hell to have both.

Fortunately, I don't have both, but I come from a family where both conditions are present. My mother had ulcerative colitis all her adult life. Diagnosed in her 30s my mother's life and that of her family was difficult. There were huge restrictions on what we could do, where we could go and what we could eat. Even as a child I realised UC was tough.

When I was diagnosed with MS, also in my 30s, I quickly became aware of the striking similarities between the two conditions. Both are typically diagnosed in young adulthood, both commonly have a relapsing remitting nature, and 'flare-ups' in both are treated with steroids. At least one drug, ozanimod, is now used to treat both UC and MS.

Over the years following diagnosis I did some serious reading and discovered other similarities between UC and MS. Both conditions are most prevalent in Northern Europe, North America and newly industrialized nations suggesting a link to Western lifestyle and environment. Worldwide UC has 1.2 cases per 1,000 people, a figure fairly similar to that often quoted for MS.

Recently an academic paper was published titled: 'Multiple sclerosis and ulcerative colitis: A systematic review and meta-analysis." The paper reveals that UC can increase your risk of MS by more than 50%. The risk of developing UC if you have MS is also greater but less so. Of course, correlation does not mean causation but clearly there is a connection here and for both personal and professional reasons I have been eager to explore it

Continued on page 6

Continued from page 5 .. I am not alone in this task and for years scientists have studied this link between MS and UC as well as a connection between MS and Crohn's Disease (CD) where gastro-intestinal inflammation exists further up the gut. Researchers are just beginning to understand what may be responsible for the association between MS and gastro-intestinal conditions.

MS, UC and CD are all autoimmune diseases, meaning the body's immune system attacks its tissues. It is thought that "leaky gut" may be a contributing factor to MS and UC and possibly CD as well. The term 'leaky gut' means the lining of the intestine becomes more porous, allowing toxins and bacteria to leak from the gut into the bloodstream triggering an autoimmune response. In UC, changes to the lining of the gut lead to inflammation and cause gastrointestinal symptoms. The process in MS is believed to be slightly more complex but is believed to start with similar changes in the gut and its lining or 'epithelium'.

As well as the 'leaky gut' there is also the question of whether a specific gut bacterium or bacteria are responsible for 'gut leakage'. Several studies show MS patients have different gut bacteria or 'gut microbiome' compared to healthy individuals, with certain bacteria like Eisenbergia tayi and Lachnoclostridium being more commonly found. Interestingly, both the above bacteria are also thought to play a role in the development of UC too.

There is also the question of whether a virus has a role to play. The link between Epstein Barr virus (EBV) and MS is well documented but a recent paper suggested that the virus may be a factor in UC too. The paper published in November 2025 in the European Journal of Gastroenterology was titled 'Epstein-Barr virus infection increases the risk for inflammatory bowel disease' and said: 'Six diagnoses, infectious mononucleosis (glandular fever) , Hodgkin lymphoma, multiple sclerosis, SLE (lupus) , Crohn's disease, and ulcerative colitis tended to cluster in the same patient population,". My family is proof of this sad fact.

Over the past few years, I have become increasingly aware of the role of the gastro-intestinal tract in MS. At diagnosis I read about various MS diets in particular the Swank diet which I followed on and off for years. My mother, after her UC diagnosis radically changed her diet too and that affected us, her family. All processed foods were banned, whole grain foods like wholemeal bread became standard fare, the only sugar allowed was honey, a juicer was bought and yoghurt was made on more or less a daily basis.

Interestingly my mother's UC improved as her diet changed, so much so that she returned to the workplace when I was a teenager, got a teaching job and then ran a sub-post office with my father until she retired in her mid-sixties. She died aged 93, not a bad outcome given that she had UC for more than 60 years and after diagnosis told me she had been given just a decade or so to live. My mother's experience has given me much food for thought. **For further details of all news and features go to page 14**

Foralumab reduces microglial activity, inflammation in nonactive SPMS

Treatment with the experimental nasal spray therapy foralumab reduced microglial activity in people with nonactive secondary progressive MS (naSPMS), according to new data from an expanded access programme trial.

Microglia, the brain's resident immune cells, are believed to contribute to the chronic inflammatory processes that drive gradual disease progression in the absence of relapses. These reductions in microglial activity were significantly associated with decreased markers of inflammation in the spinal fluid and increased levels of neuroprotective proteins.

A team led by scientists at Brigham and Women's Hospital in Boston presented the findings in a paper titled, "Nasal foralumab downregulates CSF inflammation and upregulates CSF neuroprotective proteomic pathways which correlate with [F-18]PBR06-PET imaging in na-SPMS with PIRA.(Progression Independent of Relapse Activity) "

Foralumab is an antibody therapy that works by targeting the CD3 protein on immune T-cells. This is believed to activate regulatory T-cells, which help calm excessive immune responses, while suppressing the pro-inflammatory T-cells that cause damage in MS. The therapy has also been shown to reduce the activity of microglia, which are believed to drive disease progression in nonactive SPMS.

An expanded access programme tested foralumab in 10 people with nonactive SPMS, most of whom had been experiencing worsening disability despite treatment. After at least six months of foralumab, disability levels remained stable or were slightly reduced — indicating an easing of symptoms — and most patients experienced reductions in fatigue. Walking ability, hand dexterity, cognition, and visual function also remained largely stable.

Results showed that [18F]PBR06-PET scores decreased significantly with foralumab, indicating that the nasal therapy dampened the activity of microglia. A significant reduction in PET signals was three months after treatment initiation, and levels continued to decline through the last follow-up assessment at a median of 7.5 months. Analyses of spinal fluid done in parallel with the PET scans also suggested a reduction in microglial activity.

Bio-pharma Tiziana is now running a Phase 2a clinical 3 month trial (NCT06292923) to further evaluate the experimental therapy. The trial aims to enrol about 54 adults with nonactive SPMS who have experienced continued accumulation of disability despite at least two years of treatment with available MS therapies. Recruitment is ongoing at seven sites in the U.S. The study's main goals are to assess the safety of the experimental therapy, its effect on nasal symptoms, and changes in microglial activity on [18F]PBR06-PET scans.

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Fenebrutinib set to get FDA approval for PPMS in US

Fenebrutinib, an investigational drug for primary progressive MS (PPMS), met its main goal in a Phase III trial called FENtrepid (NCT04544449) where it was shown to be at least as effective as Ocrevus (ocrelizumab) at slowing confirmed disability progression.

Drug developer Genentech said it plans to use data from this trial to seek regulatory approval of fenebrutinib to treat PPMS in the US and Europe. At present, the main therapy approved for PPMS is Ocrevus — administered by infusion into the bloodstream — and its subcutaneous, or under-the-skin, formula. As an oral therapy, given as tablets, fenebrutinib would ease treatment burden for MS patients.

Trial results of the FENtrepid trial were shared with MSers by researcher Dr David Baker, better known as 'Mouse Doctor' writing in 'The MS-blog'. In the blog Dr Baker revealed 'FENtrepid' showed fenebrutinib reduced the risk of disability progression by 12% compared to Ocrevus as early as 24 weeks and additional analysis showed potential benefit in upper limb function too.

As such, Fenebrutinib now has the potential to become 'first-in-class' oral, brain-penetrant BTK inhibitor for prog-MS, beating the other main BTK inhibitor presently in the prog-MS drug pipeline – tolebrutinib.

Bruton's tyrosine kinase (BTK) is an enzyme essential for the survival and activation of B-cells. B-cells are a type of white blood cell (lymphocyte) whose over-activity may be involved in the inflammation linked to MS. The BTK enzyme also regulates immune cells in the brain called macrophages and microglia which have been linked to MS progression.

Fenebrutinib blocks the actions of the BTK enzyme and it is thought that this will prevent the MS activity which leads to relapses as well as the longer-term damage to nerve cells which causes progression.

d Dr Baker said it looks like Fenebrutinib may make it where other BTK inhibitors (like tolebrutinib) have so far been less successful. In particular, the widely reported liver issues which has so far been a problem seemed to be blocking tolebrutinib in development for progressive MS is less of an issue in fenebrutinib, although Dr Baker says he suspects fenebrutinib will still need to be monitored for 'liver issues' if and when it gets approval. Other BTK inhibitors in the MS drug pipeline include evobrutinib, tolebrutinib, orelabrutinib and remibrutinib.

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Myelin repair clinical trial set to enrol MS patients

Quantum Biopharma soon will be seeking FDA clearance to launch a phase 2 clinical trial testing its oral therapy Lucid-MS in people with MS. The trial is designed to target myelin loss and promote its repair.

If cleared by the regulatory agency, the trial will test the therapy's safety, tolerability, and potential effectiveness in people living with the disease. As part of preparations for the study, Quantum also announced the appointment of Salvatore Napoli, MD, an internationally known neurologist, as the trial's principal investigator, according to a company press release.

Napoli, who Quantum calls "a key opinion leader in MS," is the president and medical director of the Neurology Centre of New England and the MS Centre of New England, where he oversees clinical and research programs focused on advancing innovative treatments for MS and other neurodegenerative diseases.

"Disease progression and demyelination remain areas of significant unmet medical need in MS, and Dr. Napoli's expertise will be invaluable as we prepare for clinical evaluation of Lucid-MS," said Andrzej Chruscinski, MD, PhD, vice president of clinical and scientific affairs at Quantum. "We are pleased to collaborate with Dr. Napoli as we advance our clinical development program."

Most available MS treatments work by suppressing immune activity, which can help slow disease progression. However, such therapies may increase the risk of infections and other side effects, and they cannot reverse existing nerve damage.

Lucid-MS, also known as Lucid-21-302, is designed to take a different approach. The investigational therapy targets a group of enzymes called peptidyl arginine deiminases, which are overactive in MS and make myelin more susceptible to immune damage.

By blocking these enzymes, the therapy aims to prevent demyelination and promote myelin repair. This is expected to potentially reverse some of the nerve damage seen in MS patients and restore lost functions.

The company has been preparing for months to seek FDA permission to begin a Phase 2 trial of Lucid-MS in people with MS. But the regulatory agency had requested two toxicology studies to support the IND application, which have only recently been completed. Now, the FDA submission will come soon, according to Quantum.

For further information go to page 14



Taking a closer look at progression

One of the problems in understanding progressive MS is how much there is to understand. There are several theories about how and why MS progresses, moving from inflammation-driven relapses to chronic, steady neurodegeneration and several theories about what keeps it 'smouldering' in this state. In simple terms there are a lot of dots to join up.

Interestingly a recent drugs trial has joined up at least a couple of these dots. The drug is called Vidofludimus calcium, a tablet, which recently showed success in a Phase 2 trial where it was found to reduce acute and chronic brain lesions in progressive MS and decrease signs of Epstein-Barr virus (EBV) reactivation in patients, EBV being seen as the 'driver' of disease progression.

OK, it may be only two dots joined, but reading the trial results made me think the drug developers may be 'on to something' and the drug may have the potential to join up even more dots and explain MS and its progression.

The Phase 2 trial called CALLIPER (NCT05054140) investigated vidofludimus calcium against a placebo in people with progressive MS. Vidofludimus calcium is designed to control immune cells that cause inflammatory damage in MS. It does this by inhibiting a protein called DHODH. It also has direct neuroprotective effects by activating a protein called Nurr1, and there's evidence that it can interfere with virus-infected cells, potentially preventing EBV reactivation and the whole smouldering process.

In the trial, vidofludimus calcium was tested against a placebo in 467 adults with progressive MS, including 152 people with primary progressive MS and 315 people with secondary progressive MS. Among patients on vidofludimus calcium, the proportion of patients with active inflammatory lesions consistently decreased over time, from 16.4% at the study's start to 7% after about 1.5 years to 0% after about 2.5 years

. **Continued on page 11**

Continued from p10: Drug developer Immunic has previously shared data showing that vidofludimus calcium slowed disability worsening relative to a placebo. This was seen even in patients without signs of active inflammation, supporting the idea that therapy may act on more than inflammation alone, it might work on the neuro-degeneration that characterises prog-MS.

At the recent ACTRIMS meeting, held earlier this year in California, researchers reported the therapy's impact on other key drivers of disease progression. In a first poster, titled "Effect of Vidofludimus Calcium, a Novel Nurr1 Activator and Selective DHODH Inhibitor, on MRI Outcomes in Progressive Multiple Sclerosis: Data from the Phase 2 CALLIPER Trial," scientists showed that vidofludimus calcium was associated with reductions in brain lesions associated with the smouldering inflammation believed to be the driver of progressive MS.

In a second poster, titled "Effect of Vidofludimus Calcium, a Novel Nurr1 Activator and DHODH Inhibitor, on the Anti-EBV T-cell Receptor Repertoire in Progressive Multiple Sclerosis: Data from the Phase 2 CALLIPER Trial," the researchers showed that vidofludimus calcium may be able to prevent EBV reactivation which many consider the disease 'driver'.

To better understand how vidofludimus calcium may affect EBV reactivation, the researchers examined a subset of 87 CALLIPER participants with available data. Specific molecular fingerprints in immune T-cells assessed EBV reactivation. These findings from a subset of the Phase 2 CALLIPER trial participants strengthen the hypothesis that the broad-spectrum antiviral effects of vidofludimus calcium can lead to lower EBV reactivations, potentially addressing MS disease progression related to ongoing EBV reactivations.

Results showed that signs of EBV reactivation seemed to decline over time in people on vidofludimus calcium, but remained stable in people given a placebo and it was a statistically significant difference.

OK, I am not saying the vidofludimus calcium is 'the cure' but I think it may be part of the cure. I am old enough to remember the early days of HIV research. As more became known about HIV it was clear that not one drug but a 'cocktail' of drugs would be needed to keep the viral load at tolerable levels. Given that a virus (EBV) is involved in MS, and is in my opinion is 'the driver' I think it highly likely that something similar to HIV treatment will happen in MS with patients taking a 'cocktail' of drugs including a drug that interferes with the EBV cycle . I would not be surprised if vidofludimus calcium was one of them, perhaps the key one in joining up the dots.

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Pic p10 Thanks to National Cancer Institute/ unsplash

News

B-cell depleting drug shows promise in active SPMS

Obexelimab, a therapy that Zenas Biopharma is developing for secondary progressive MS, almost completely prevented the formation of new inflammatory lesions in the first three months of a clinical trial.

That's according to data from the first part of MoonStone (NCT06564311), a Phase 2 clinical trial testing obexelimab as a weekly, 250 mg subcutaneous (under-the-skin) injection in MSers including those with active secondary progressive MS.

Participants were randomly assigned to receive obexelimab or a placebo for 12 weeks (about three months) in the first part of the trial. In a second part, all are receiving obexelimab for another 12 weeks. After the 24 weeks, patients were then invited to join an open-label extension and continue on obexelimab for a year. The company said it expects to have 24-week data later this year (2026)

Obexelimab targets B-cells where it binds to two proteins CD19 and FcgammaRIIb. This is expected to reduce the production of inflammatory molecules. Unlike other approaches, obexelimab reduces the activity of B-cells rather than destroying them.

The main goal of the trial was to monitor new gadolinium-enhancing lesions on MRI scans, which represent lesions with active inflammation. Results showed that, compared with a placebo, obexelimab almost completely suppressed the formation of new lesions with active inflammation. The cumulative number of new inflammatory lesions was reduced by 95% over eight weeks and maintained throughout 12 weeks.

On average, patients treated with obexelimab had significantly fewer new inflammatory lesions per MRI scan than those on a placebo (0.01 vs. 0.23). Obexelimab also reduced the number of new or enlarging lesions over weeks eight and 12.

The safety profile of obexelimab was consistent with what has been seen in earlier clinical testing for other autoimmune diseases. Some patients experienced infections and exaggerated immune reactions, most commonly mild injection site reactions.

For details of all stories go to page 14

‘Moist Snuff’ use sees less MS progression

The use of ‘moist snuff’ is significantly linked to slower MS disease progression according to Swedish researchers writing in the Journal of Neurology, Neurosurgery and Psychiatry.

The researchers found that while smoking is linked to worse MS progression, the same cannot be said for users of ‘moist snuff’. This is a tobacco where cut tobacco is pouched and placed in the mouth, with users sucking on the pouches. It is commonly used in Scandinavia. In the research a total of 9,089 patients with MS were investigated to examine the impact of smoking, exposure to passive smoking and snuff use. Researchers found there was an ‘inverse association’ between moist snuff use and EDSS (level of disability) among people with MS who were snuff users, compared with those who had never used snuff. Inverse association means high moist snuff use was linked to lower levels of disability.

In the ‘discussion’ section of their paper the researchers try to explain his ‘inverse association’. They say nicotine is the most likely explanation. ‘Nicotine exerts anti-inflammatory and immune-modulating effects through a mechanism dependent on the alpha7 nicotinic receptor31 and has been shown to suppress neuroinflammation and disease severity in experimental autoimmune encephalomyelitis.

‘Experimental data in combination with our finding that users of moist snuff have a more favourable EDSS (disability) progression also suggest a possible strategy to affect MS progression. It should be noted that the use of nicotine as a therapeutic tool has potential disadvantages such as toxicity related side effects, lack of pharmacological specificity and unknown long-term effects on human health. However, selective agonists for the alpha7 nicotinic receptor could represent a pharmacological strategy to reduce neuroinflammation and neurodegeneration and may prove beneficial in patients with MS.’ In their conclusions the researchers say: ‘Snuff use (moist snuff) was associated with slower disease progression, suggesting that nicotine replacement therapy could be an attractive way to increase the chance of quitting smoking among patients with MS.’

Sadly UK readers will be disappointed to know that while snuff (nasal tobacco) is legal to sell and use in the UK, moist snuff – very small packs of tobacco placed in the mouth known as ‘snus’ -are illegal in the UK. Despite ‘snus’ being illegal, chewing tobacco is legal in the UK and can be bought online. Chewing tobacco involves a coarser cut of tobacco that is chewed and often requires spitting, while snus is a finely ground, pasteurized tobacco product (or in a pouch) that is placed under the lip and does not require spitting. Snus delivers nicotine by absorbing through the mouth, unlike chewing tobacco, where it is absorbed through chewing and swallowing the nicotine enriched saliva.**For details of all news stories see page 14**

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Page 13: Moist snuff use as an MA treatment

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https://www.smoke-king.co.uk/collections/chewing-tobaccos

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