

My prog-MS ezine

For those with progressive MS and other interested people

Issue 64, January-February 2026

Hello, happy 2026 and welcome to the 64th 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 my age is 67.

In issue 64 there are eight pages of prog-MS news stories. There are also two features. The first, on page 5, looks at a long running debate about sunshine and MS. What is it in sunshine that leads to lower rates of MS in sunny places? Ultra violet (UV) radiation or vitamin D production? Read p5 to see some new research. You may be surprised.

On page 10 there's another feature looking at how we test walking when MS progression is being measured Walking is routinely used in trials for new MS drugs, but arguably the timed 25-foot walking test is overly simple and 'cheatable'. So should it be scrapped in favour of a more reliable test? I my opinion, yes, it should be.

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Antioxidant lipoic acid slows brain shrink in prog-MS

Alpha lipoic acid (ALA) slows brain shrinkage in people with progressive MS. That's according to the results of a two-year study. However, ALA, an over-the-counter anti-oxidant pill, does come with side effects when taken over long periods.

A phase 2 clinical trial (NCT03161028) tested alpha lipoic acid against a placebo over two years in more than 100 adults with progressive MS. The researchers noted that alpha lipoic acid did not easily cross the blood-brain barrier, however once in the brain it seemed to stop brain shrink or brain atrophy, a common sign of MS progression.

"It didn't work clinically in progressive [MS] the way we hoped," Rebecca Spain, MD, an associate professor of neurology in the Oregon Health & Science University (OHSU) School of Medicine in Portland, said in a university news story. "However, the slowing of brain atrophy that we saw in MRI images suggests that we may yet be on the right track, especially if we can find a better way to deliver the beneficial effects of alpha lipoic acid," she added. Her study, "Lipoic Acid for Treatment of Progressive Multiple Sclerosis - A Phase 2 Randomized Clinical Trial," was published in the journal 'Neurology'.

Alpha lipoic acid, also called Lipoic acid, helps turn blood sugar, or glucose, into energy in the body's cells and is found in certain foods and over the counter supplements. It is metabolised by the liver. It reduces oxidative stress, inflammation and limits the entry of immune cells into the brain and spinal cord. In the research trial 115 adults with primary or secondary progressive MS received a daily dose of either 1,200 mg of LA/ALA or a placebo.

The study's primary outcome was walking speed, assessed by the Timed 25-Foot Walk, an increasingly controversial outcome measure (See feature on page 10). The study's results showed lipoic acid did not enhance walking speed compared with the placebo. However, more reliable measures like MRI scans showed that whole-brain volume appeared relatively stable in patients given ALA, while it tended to decrease in those in the placebo group. The effect was significant in the deep grey matter, which is typically most severely affected in MS. These findings suggest possible neuroprotection, even without a clear clinical benefit, according to the research. Researchers hypothesize lipoic acid may have both anti-inflammatory and antioxidant effects that may protect damaged myelin and underlying nerves, assuming enough of it circulates in the blood to reach the brain

In terms of side effects more patients stopped taking lipoic acid than the placebo, due to one particular side effect. Lipoic acid was linked to proteinuria, indicating an excess of protein in the urine — a sign that the kidneys may be damaged — which necessitated closer lab monitoring, a fact that may mean long term lipoic acid is not suitable for everyone**.**

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

New naSPMS drug Tolebrutinib hits twin setbacks

FDA approval for tolebrutinib as a treatment for “non-active secondary progressive” MS will likely come later than was expected. That’s according to a news release from French biopharma Sanofi.

The US FDA (Food and Drug Administration) had previously aimed to make a decision on approving tolebrutinib by Sept 28, 2025 but pushed that deadline back to December 28, 2025 after Sanofi made a “major amendment” to its application. Now, the company suspects recent discussions with the FDA indicate a ruling won’t come until sometime towards the end of March 2026.

The company said it “strongly believes” in tolebrutinib’s risk-benefit profile. In response to an FDA request, an “expanded access protocol” has been drawn up so eligible patients with non-relapsing secondary progressive MS can get hold of the drug, which is already available to patients in the United Arab Emirates (UAE).

In a separate announcement Sanofi said tolebrutinib had failed a Phase 3 study in primary progressive MS and that the company is scrapping plans to get the drug approved for this type of MS.

Sanofi didn’t release any specific data, saying only that tolebrutinib failed to meet the main goal of a late-stage trial titled PERSEUS. The trial enrolled 767 participants with primary progressive MS, and looked at whether tolebrutinib was any better at delaying the disabilities that typically worsen over the course of the disease.

Tolebrutinib comes as a tablet, and is part of a class of drugs designed to inhibit “Bruton’s tyrosine kinase,” (BTK) an immune system-regulating enzyme that plays a key role in the development of B cells (bone marrow cells). These cells make antibodies that alert the body to potential threats. Research suggests a root cause of MS is antibodies mistakenly attacking the protective myelin sheaths that surround axons or nerve fibres.

Sanofi already has one BTK-blocking drug, Wayrilz, on the market for a different autoimmune condition. The class of drugs to which tolebrutinib belongs, known as BTK inhibitors, carry safety concerns, namely the potential for liver injury, which in recent years led the FDA to issue partial clinical holds on similar drugs from bio-pharmas Roche, Merck KGaA and InnoCare Pharma**.**

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First UK prog-MS patient dosed in CAR-T cell therapy trial

The first UK prog-MS patient has been dosed in a Phase 1 clinical trial of Autolus Therapeutics' experimental CAR T-cell therapy, (obe-cel), for people with progressive MS.

Dosing took place at University College London Hospitals NHS Foundation Trust, in the U.K in the Autumn. The trial called BOBCAT (NCT07139743) is evaluating the potential of the therapy to slow disability progression as well as its safety and tolerability.

Obe-cel (obecabtagene autoleucel) is approved as Aucatzyl in the U.S. and Europe for a type of blood cancer called B-cell acute lymphoblastic leukaemia. It is also being studied for other diseases caused by abnormal or overactive B-cells, immune cells with a key role in driving MS and other autoimmune diseases.

Obe-cel belongs to a class of treatments known as CAR T-cell therapies. These involve collecting a person's immune T-cells, modifying them in the lab to produce a man-made receptor that recognizes a specific target, and then infusing them back into the patient's bloodstream.

Obe-cel is specifically made with a person's own T-cells, which are engineered to produce a chimeric antigen receptor (CAR) that binds to the CD19 protein on the surface of B-cells. When infused back into the patient, the modified CAR T-cells can attach to B-cells and destroy them, thereby easing the autoimmune attacks which characterise MS.

CD19 is a protein on B cells, crucial for their function, and its role in MS involves both contributing to the autoimmune attack (through overactive B cells) and being a target for new therapies, like CAR T-cell therapy,

The ongoing BOBCAT study plans to include up to 18 adults with progressive MS who have not responded to at least two highly effective disease-modifying therapies. Its main goal is to see if obe-cel is safe and well tolerated, and whether patients experience any dose-limiting toxicities within 28 days after the infusion.

Researchers will also watch for changes in Expanded Disability Status Scale (EDSS) scores over two years. In addition, changes in walking function, hand dexterity, and cognition will be assessed.

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Is it UV light and not Vitamin D that treats MS?

Over the years I have been well aware of the long running debate about a link between vitamin D and MS. The alleged vitamin D/ MS link is based on the observation that MS is more common in parts of the world that don't get much sunshine. It is argued that vitamin D in sunlight is what stops MS and taking vitamin D tablets will treat it.

Following this very simple argument I have been taking vitamin D tablets daily (2-5,000 IU) for the past 20 years and have had my blood/vitamin D levels tested annually. I presently have levels as high as someone who spends most of their time outdoors exposing lots of their skin to the sun, someone like a lifeguard.

Despite this, my MS rolls relentlessly on, with my EDSS disability levels rising remorselessly upwards. So, is the vitamin D/ MS theory flawed in part or in its totality? Well, what does the science tell us?

Looking at scientific studies, the first thing that strikes you is that they are at best ambiguous. Sunlight affects the body in many ways. Large studies have shown lack of exposure to sunlight is a key risk factor for MS, with the disease being more common the further from the equator you live. This is where people get less sunlight, so there is clearly some sort of link here. But what exactly is this link?

Since the days of rickets it has been known that ultraviolet light helps the skin make Vitamin D, and therefore the relationship between MS and sunlight exposure has long been thought to be driven mainly by vitamin D. However, and this is a big 'however', studies testing whether vitamin D supplements might help ease MS have produced mixed results, often finding little or no benefit. Reading one study after another has made me question why I continue taking vitamin D. Is it having any effect on my MS? Research would suggest not necessarily.

Researchers are now exploring another idea — that it's ultraviolet light itself and ultraviolet radiation (UVB – the sort that tans you) that might protect against MS, rather than vitamin D. To study this idea, researchers recently analysed blood samples collected during a clinical trial called 'Proteomics confirms immune stabilizing effects of narrowband UVB treatment in patients with clinically isolated syndrome and multiple sclerosis'. **Continued on page 6**

Continued from page 5 . This trial enrolled a small group of people with clinically isolated syndrome (CIS), who had experienced a first episode of MS-like symptoms but didn't yet meet the criteria needed to establish an MS diagnosis.

In the trial, half of participants sat in a dark room (the placebo group), while the others sunbathed three times a week for eight weeks. Researchers used the MS Disease Activity (MSDA) scale — a clinically validated test for monitoring MS disease activity — and found the majority of people initially categorized as high-severity disease activity no longer fell into that group after about three months of sunbathing and being exposed to narrowband ultraviolet B (NB-UVB) radiation.

The trial concluded 'NB-UVB caused broad and coordinated reductions in untargeted and targeted inflammatory proteins. These effects support NB-UVB as a promising immunomodulatory strategy in MS in both general inflammation and MS-specific activity, as well as consideration of inflammatory markers and the MS Disease Activity Score as surrogate endpoints in future trials.'

These positive findings support plans for a larger, multicentre Phase 2 trial that will evaluate UVB's impact on fatigue and other MS outcomes.

Looking back at my 20 years interest in the complex area of MS, vitamin D and sunshine, I wonder whether a better investment than buying my bottles of 2,000 IU vitamin D tablets might be to go out and buy a small deckchair and get out in the sun when the sun's strong enough to tan my skin.

This means sunbathing for short periods (just sufficient to tan the skin) in the months surrounding the longest day. That's every day from April 1 to October 1 at mid-day.

The small 'folding beach' deck chair can be bought on Amazon for less than £25. Over the long run his investment will be cheaper than my 'HeathAid' 2,000 IU vitamin D tablets (90 tablets) and sunbathing in my deck chair may actually be better in the long run. It's something I will think about when Spring arrives over the next few months and the sun shines strongly enough to give me a tan.

Pic (p5) – Happy smiling cartoon sun illustration brgfx

For details of sources for feature see page 14

Fenebrutinib posts positive results for PPMS

Bio-pharma Genentech's fenebrutinib has shown positive Phase 3 results in a trial for primary progressive MS (PPMS), the company has announced.

The trial FENtrepid (NCT04544449) found that the therapy was at least as effective as Ocrevus (ocrelizumab) at slowing disability progression in people with PPMS. Genentech plans to soon submit regulatory applications for the drug.

"Fenebrutinib slowed disability progression in PPMS," Levi Garraway, MD, PhD, chief medical officer and head of global product development at Roche, the parent company of Genentech, said in a company press release. "These unprecedented results suggest that fenebrutinib could potentially become a best-in-disease medicine as the first high-efficacy, oral treatment for people with PPMS."

Fenebrutinib is designed to block BTK, Bruton's tyrosine kinase, an enzyme with a key role in the activation of B-cells and microglia — immune cells involved in MS. By inhibiting BTK, the therapy aims to reduce inflammatory immune cell activity, thus slowing disease progression.

The PPMS trial FENtrepid was designed to show that fenebrutinib was non-inferior to (or at least as effective as) Ocrevus in delaying the onset of confirmed disability progression after 120 weeks, or about 2.5 years. That goal was met, making fenebrutinib the first BTK inhibitor to achieve its main goal in Phase 3 clinical testing for PPMS.

Genentech reported that a numerical benefit favouring fenebrutinib was observed as early as six months and lasted for the duration of the trial.

Liver safety data from the studies were consistent with earlier studies of fenebrutinib, and additional safety data are still being analysed. According to Genentech, full data from the two studies will be presented at upcoming medical meetings.

Fenebrutinib is one of several BTK inhibitors being developed for progressive MS. Tolebrutinib has shown positive results in trials for non-active secondary progressive MS, although not primary progressive MS and is awaiting regulatory approval by regulatory authorities in the US for naSPMS.. BTK inhibitors were originally developed for treating certain types of cancer and some have been 're-purposed' as MS treatments.

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

CAR-M cell therapy shows promise in preclinical studies

US biopharma Liberate Bio's CAR-M cell therapy almost completely depleted B-cells in nonhuman primates, suggesting the therapy could be used to treat MS where treatments often involve changing these cells.

CAR-M cell therapy is designed to genetically engineer two types of immune cells called monocytes and macrophages, targeting and eliminating B-cells (bone marrow cells). That modification is done inside the patient's body, eliminating the need to wait several weeks as seen with traditional cell therapies. It also doesn't require patients to undergo chemotherapy. It is hoped to launch a trial in the second half of 2026.

Preclinical data into the CAR-M therapy were presented at the American Society of Gene and Cell Therapies' Advancing Cell and Gene Therapies for Cancer conference, held recently in Philadelphia, USA.

MS is, of course, caused by the immune system mistakenly attacking healthy parts of the brain and spinal cord. B-cells are key immune cells involved in these attacks, and are also involved in certain blood cancers. Researchers have so far been exploring T-cells, another immune cell type, as a potential approach to eliminating B-cells. T-cells can be engineered to produce a man-made receptor, called a chimeric antigen receptor (CAR), that recognizes a specific protein at the surface of disease-causing cells, making them extremely effective at eliminating these cells. This approach is called CAR T-cell therapy, and it usually involves collecting the T-cells from patients, modifying them in the lab, and then introducing them back into the patient's bloodstream to target B-cells.

Liberate Bio is developing a different kind of cell therapy that harnesses monocytes and macrophages instead of T-cells, and modifies the cells inside the patient's body. The company's CAR-M therapy uses a messenger RNA (mRNA) molecule containing instructions to produce a CAR receptor targeting the CD20 protein in B-cells. mRNA is a temporary molecule used as a template to make proteins. This mRNA molecule is inserted into lipid nanoparticles that target monocytes and macrophages, ensuring the therapy is directly delivered to these immune cells.

Researchers screened multiple nanoparticles in non-human primates using the company's proprietary RAPTOR platform. After two doses, which were well tolerated, the lead nanoparticle, dubbed LNP LB01, led to a more than 99% depletion of B-cells. After each dose, there was a transient increase in the levels of inflammatory molecules, including interleukin-6 and TNF-alpha, but these resolved within two days.

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EBV vaccine could eventually become MS treatment

A clinical trial is now recruiting participants to test whether an experimental vaccine for the Epstein-Barr virus (EBV) is safe and could help reduce disease activity in people diagnosed with MS. Those with active secondary progressive MS are invited to take part .

The Phase 2 study (NCT06735248) aims to enrol 180 adults, ages 18 to 55, who are neurologically stable (as assessed by an investigator), and who are EBV-seropositive at screening. Enrolment is underway at six sites in the U.K (see below) .

As it's an early-stage trial safety is the study's main focus. Researchers will also look at whether the vaccine affects different measures of MS disease activity.

Over the past few decades research has shown EBV, which causes glandular fever, is a key risk factor for MS, some research even saying it is the 'leading cause' of MS. After infection, usually in childhood or adolescence, EBV can remain dormant in the body's cells for decades and may reactivate later in life. The relevance of the vaccine to those of us with MS is that it is theoretically possible that the vaccine may stop the process by which the virus continually re-activates through a process known as 'latent lytic' cycling. The vaccine will be given to MSers who are EBV positive and could theoretically stop MS disease activity, putting 'the brakes' on MS by preventing this re-activation process.

The experimental vaccine, called mRNA-1195, is designed to prevent this reactivation, which researchers hope could help reduce MS disease activity. The vaccine is being developed by Moderna, which is sponsoring the trial.

The vaccine uses mRNA technology — the same platform used for Moderna's COVID-19 vaccine. Instead of delivering a piece of the virus, mRNA vaccines provide instructions that the body's cells use to make a small viral protein, training the immune system to recognize the virus and triggering a response similar to traditional vaccines.

The trial, which is expected to conclude in 2029, is open to people with active secondary progressive MS as well as those with relapsing forms of MS — including clinically isolated syndrome. It is also open to people with radiologically isolated syndrome, where MRI scans show MS-like changes before any symptoms appear.

UK trial locations include Bristol, Cardiff, Edinburgh, Glasow, Sheffield and Newcastle.

For further information go to page 14



Is key MS progression test fundamentally flawed?

When I was diagnosed, some 30 years ago, I had a lumbar puncture at a local hospital. In the 'small talk' that followed the test I asked the doctor why he had wanted to work in neurology. He looked shocked, telling me that he was a junior doctor and when he finished training, he was definitely not going to be a neurologist.

The junior doctor went on to say that neurology was a 'backwater' of medicine which attracted candidates of low ambition and ability. Ouch. To support his rather controversial claim, he pointed out that the most high-tech equipment a neurologist used was a hammer to tap patient's knees. Okay, this was in the days before MRIs but you kind of know where the guy was coming from.

It's true that an air of low tech, cheap and arguably poor-quality tests surround much in the MS world. Take the 25 foot timed walk test (T25FW). This 'gold standard' test, used to measure disease progression and used in many drug trials, is praised for its simplicity, its low cost, and speed of testing. It may have these three positives but I would argue these are outweighed by its many negatives which are that it fails to capture the complexity of walking and particularly MS 'walking' or wobbling particularly in terms of endurance, and fatigue. Also, walking seven paces (25 feet) along a hospital corridor is hardly a test of walking in a 'real world' sense.

To make the T25FW test even less reliable, some people taking the test, myself included, "sprint" to achieve a normal or near-normal time, masking underlying issues. I dash through the seven paces required to get me across a distance of 25 feet. The reason is that I have lost the ability to walk for much longer than 10 seconds so I lurch forwards, using momentum to keep me on my feet for the short distance. In that sense I 'cheat' the test, making it an unreliable measure of my real 'walking ability'. The same is probably the case for many other prog-MSers being tested.

As well as failing to reliably measure my 'real walking' ability the other thing the T25FW test fails to measure is fatigue. Given the test typically takes less than 10 seconds to complete, it does not allow enough time to induce fatigue (progressive slowing down) or foot drop that characterise 'real life' MS walking. **Continued on page 11**

Continued from p10: In real life I walk the first 10 feet normally but I begin dragging my foot (foot drop) or stumbling afterwards. The T25FW misses this entirely because these things happen after 25 feet. Perhaps the most important area where the T25FW test lets us down is in clinical trials where it is often used to measure the success of a new MS drug or treatment.

One example of this shortcoming is the recent phase 2 trial of alpha-lipoic acid, which used T25FW as a primary outcome measure (see p14 for trial details). The trial was negative for the primary outcome (walking ability) but positive on MRI measures that capture progressive MS very well, i.e. deep grey matter volume loss (brain shrink). Deep grey matter volume remained stable in ALA participants and decreased in placebo participants, so I would argue the test was more positive than has generally been reported. That said, the chances of alpha lipoic acid being recommended or even dispensed as a simple and cheap treatment for prog MS have been damaged irreparably due to a poor and 'cheatable' trial test measure (T25FW) being used as its 'primary outcome' measure.

So, if the T25FW test is so bad, one might ask: are any other 'cheap and easy' tests which are any better? Well, yes there are. One of these is the 9HPT (9 hole peg test). This hand (manual dexterity) test is a very different sort of test, as it captures MS worsening or improvement with much greater sensitivity. It integrates vision, coordination, sensory function, and fine motor coordination into a single outcome measure and it's virtually impossible to cheat, trust me I've tried.

I suspect that if the trial into alpha lipoic acid had focused on measures such as the 9 hole peg test, the ALA study would have been positive. In simple terms I think the T25FW test skewed the results. So, in my opinion, the T25FW should be discarded as an outcome measure in clinical trials before we throw out another baby (alpha lipoic acid) with the bathwater.

For my part, I will continue taking alpha lipoic acid. This is a personal choice not a recommendation. In the meantime, I urge readers to read the research paper carefully. It says: 'Whole-brain volume seemed stable in ALA participants, whereas it trended toward a decrease in placebo, even after accounting for an increased total T2-weighted lesion volume that was greater in the ALA group. Deep grey matter volume remained stable in ALA participants and decreased in placebo participants.' In simple terms the researchers are saying that ALA will stop brain shrink, a major marker of MS progression. I think that's a very good reason for taking ALA.

Thanks to Prof Gavin Giovannoni whose Medium posting is the source of some of this material **For details of all features go to page 14**

News

B-cell depleting drug shows promise in active SPMS

Obexelimab, a therapy Zenas Biopharma is developing for secondary progressive and relapsing remitting 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 may join an open-label extension and continue on obexelimab for a year. The company said it expects to have 24-week data early in 2026.

Obexelimab targets B-cells — the immune cells that produce antibodies — including those involved in autoimmune disease. It binds to two proteins on B-cells, CD19 and FcgammaRIIb, which is expected to reduce the production of antibodies and other 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 show 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**.**

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News

Researchers $800,000 plan to test new type of MS ‘nerve repair’

An international research team has secured nearly $800,000 to advance a potential ‘regenerative’ therapy for MS that would repair damaged nerves. The work is now moving toward clinical trials.

While existing treatments can slow disease progression, none can repair the nerve damage that has already occurred. This new project aims to help to repair damaged nerves and restore lost function for those living with MS.

The project is led by Fang Liu, MD, PhD, a senior scientist at the Centre for Addiction and Mental Health in Toronto, in collaboration with Iain Greig, PhD, researcher at the University of Aberdeen. Funding comes from Brain Canada, the U.S.-based National MS Society through its Fast Forward program, and Health Canada via the Canada Brain Research Fund.

The therapeutic approach is designed to counter ‘excitotoxicity’ — that’s when nerve cells fire more than they should, leading to injury and cell death. If successful, the new approach would help restore normal nerve signalling.

While currently approved MS therapies can reduce disease activity and slow disability progression, none can repair the damage that’s already occurred. Liu’s team has been working on small molecules that protect nerve cells and promote remyelination, the natural process of repairing and replacing the damaged myelin sheath.

Although a press release did not specify the compound now being advanced, the researchers have previously reported the development of ZCAN262, a treatment candidate designed to modulate a receptor involved in excitotoxicity.

ZCAN262 is able to modulate the activity of AMPA glutamate receptors specifically. In preclinical studies, the compound prevented myelin loss and led to milder disease in animal models, without interfering with important brain functions. The experimental therapy being developed is now entering the final stages of preclinical testing to prepare for clinical studies, according to a press release from Brain Canada**.**

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