

My prog-MS ezine

For people with progressive MS and other interested people

Issue 53, March/ April 2024

Hello and welcome to the 53rd edition of my ezine about progressive Multiple Sclerosis and MS progression in general. My name is Ian Cook. I'm a secondary progressive MSer from Birmingham, UK. I'm 65.

In this 53rd issue, there are as usual eight pages of prog-MS news stories. There are also two features. One on page 5 looks at beef and MS. For a long time cow's milk has been suspected as the environmental factor that causes MS to be more prevalent in places where cows are reared, As well as being places where cows' milk is drunk these are also big beef eating places. So should MSers be excluding beef as well as milk from our diets.? One academic thinks so. Picture below : Annie Spratt- Unsplash

There's another feature on page 10 looking at a question that has vexed me for ages: why do MS researchers carry out experiments on mice. Mice don't get MS, so how much use is the "mouse model" in developing treatments for a very human illness?

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News

FDA agrees to cancer treatment trial for prog-MS – possible 'cure'?

The U.S. Food and Drug Administration (FDA) has agreed to a phase 2 clinical trial to test an existing cancer treatment in people with treatment-resistant progressive MS. The "c" word (cure) been used by one eminent UK neurologist impressed by the treatment. A UK trial has been talked about.

The early stage US trial (NCT06138132), will enrol 12 patients with either primary progressive or secondary progressive MS who will take a single dose of the treatment, called KYV-101, which aims to equip the immune system with the ability to eliminate antibody-producing B-cells which are implicated in MS. The treatment is called a CAR T-cell therapy (Chimeric Antigen Receptor) T cell therapy and is a new and promising treatment currently used for blood cancer. It uses the body's own immune system to help fight cancer.

In using the therapy in MS, a person's own immune T-cells are isolated and engineered in the lab to contain a man-made receptor, designed to recognize the CD19 protein found on B-cells' surfaces, which is believed to be the "cause of MS auto-immunity.

When the engineered T-cells are returned to the body, they're equipped to specifically target and attack problematic B-cells, thereby resetting the immune system. It is believed that this will offer sustained disease remission for progressive MS patients with just a single treatment, in theory a treatment quicker and easier than stem cell transplants.

As well as attacking problematic B cells KYV-101 is also designed to reduce certain side effects and improve tolerability relative to previous CAR T-cell therapies. The new study will answer whether CAR T-cell therapy offers a new treatment for those with progressive MS.

Interestingly in his latest MS Selfie post titled "Will CD19-targeted CAR T-cell therapy cure MS?" Prof: Gavin Giovannoni of London's Barts Hospital said: "I have spent the better two years trying to get CAR T-cell companies interested in MS. The good news is that three phase-1 CAR T-cell safety studies will be starting in MS, with several other companies planning to explore this technology in MS as well. . . An accompanying editorial discussed the possibility that these subjects have been cured of their autoimmune diseases."

Prof. Giovannoni ended his post by asking: "Knowing what you know about MS and its possible cause, would you be willing to volunteer for a CD-19-targeted CAR T-cell trial?" His post is really worth reading. See link on page 14

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Ocrevus may also help secondary progressive MSers

Ocrevus (ocrelizumab), a disease modifying drug approved in the UK for relapsing remitting and early primary progressive MS may also help people with secondary progressive MS. That's according to new US research.

The research, "Ocrelizumab in Highly Disabled Progressive Multiple Sclerosis Patients," was published in the journal Multiple Sclerosis and Related Disorders. It found Ocrevus may stabilize disability progression in people with MS who have severe walking problems — a group who were excluded from clinical trials supporting the therapy's 2017 approval.

The 2017 trials that supported Ocrevus' approval excluded people with MS with higher levels of walking disability, such as those with secondary progressive MS, so its impact on this group was not established and hence not approved, at least not in the UK. This is unlike in the US where Ocrevus has been approved for active secondary progressive disease (SPMS) with relapses) as well as primary progressive MS (PPMS)

To learn more about the drug's efficacy on secondary progressive MSers, researchers at the Brigham and Women's Multiple Sclerosis Centre in Boston reviewed medical records of MS patients with substantial disability — specifically, those with an Expanded Disability Status Scale (EDSS) score of 6.5 or higher at the time they began Ocrevus treatment.

Among those studied, 49 (79%) had secondary progressive MS, and 13 (21%) had primary progressive MS. The results showed that 66.1% of patients experienced no disability progression after about three years on Ocrevus, as assessed by a reduction in EDSS scores. A total of 8.1% had some easing of disability — reported as improvements — as seen in their EDSS scores. About one-quarter of the patients (25.8%) experienced disability worsening.

According to researchers, "progressive patients with higher levels of disability may experience comparable benefits from [Ocrevus] treatment to progressive patients that are more ambulatory [able to walk]." Importantly, there was no difference in outcomes between those with PPMS for whom it is NICE approved or SPMS.

"Our study reports on the safety and effectiveness of [Ocrevus] in MS patients with significant levels of disability, and suggests that it may be a useful therapeutic strategy in up to a third of these patients in appropriate clinical setting," the researchers concluded." More research is needed to help identify those highly disabled [people with MS] who would most benefit from [Ocrevus] use," they added. It should be noted that many of those taking Ocrevus discontinued treatment before the end of the study period. Their reasons for stopping the therapy primarily were side effects (58.6%) or MS progression or lack of perceived benefit (41.4%). **For details of all news stories go to page 14**

Ibudilast slows growth of brain lesions tied to worsening MS

An asthma drug ibudilast, being explored as a treatment for progressive MS, has been shown to significantly slow the growth of 'chronic active lesions' in the brains of MS patients, according to a study called SPRINT-MS.

Chronic active lesions are areas of damage marked by inflammatory cells at their edge, with an inactive core without inflammatory activity. They're thought to contain compartmentalized regions of inflammation and to grow slowly over time — contributing to a gradual worsening of disability in MS patients, something called "smouldering MS".

Treatment with the asthma drug ibudilast decreased slowly enlarging lesion volume, by as much as 23%," researchers found. A study, titled "Ibudilast reduces slowly enlarging lesions in progressive multiple sclerosis," was published in the Multiple Sclerosis Journal. The lesions, are thought be linked to gradual disability worsening.

Ibudilast is a pill that lowers the levels of pro-inflammatory signalling molecules that drive inflammation and nerve cell damage in MS. Unlike many approved MS therapies, ibudilast can cross the blood-brain barrier, a tight membrane that regulates the passage of substances from the bloodstream into the brain. In Japan, ibudilast capsules have been used to treat asthma for 20 years so the drug should be easier to licence than new medications.

In recent research called SPRINT-MS Phase 2b clinical trial (NCT01982942) it was shown that almost two years of daily ibudilast treatment significantly slowed brain volume loss by 48% compared with a placebo in patients with primary progressive MS (PPMS) or secondary progressive MS (SPMS), with or without relapses. MRI data from 195 trial participants showed a significantly smaller median SEL (slowly enlarging lesion) volume among ibudilast-treated patients than those given the placebo. Across all trial participants, ibudilast was associated with a 23% reduction in SEL volume compared with the placebo.

For patients, a larger SEL volume significantly correlated with reduced walking abilities — as indicated by a longer time to walk 25 feet. Worse manual dexterity, measured by the nine-hole peg test, and impaired cognitive function, assessed using the symbol digit modalities test, also were significantly associated with larger SEL volumes.

These data suggest that "ibudilast's mechanism of action is likely to be different from conventional anti-inflammatory therapies in MS," which normally exert an effect of total new or enlarging lesions and inflammatory lesions, the researchers noted .Before re-purposing a drug a larger phase III trial will be needed, but encouragingly the US Food and Drug Administration (FDA), responsible for licensing drugs in the US, have granted ibudilast fast track status. This is designed to speed up the development and review of new treatments. **For further details see page 14.**



Beef and MS – food for thought

Dairy has long been considered the environmental cause of MS. The reason is simple - MS is more common in countries where cow's milk is drunk.

Recently, Nobel Prize winning researcher and virologist Harald Zur Hausen widened this theory to look at beef and MS too. Zur Hausen made several interesting observations about the connection between cow products and MS in academic papers published in 2014 and 2021. In the 2014 paper he said: "Epidemiological data point to the involvement of a cow milk factor in the aetiology of multiple sclerosis (MS)." In the more recent paper he went further, suggesting that MS is associated with the consumption not just of milk but beef too.

He noticed isolated ring-shaped DNA molecules closely related to BMMF (bovine meat and milk factors) from dairy and cattle blood present in the brain lesions of patients with MS. Zur Hausen was amazed at his findings. As he put it: "The result was electrifying."

Zur Hausen didn't go so far as to say that beef and milk cause MS. Rather he thought MS is caused by a double infection of brain cells by the Epstein Barr virus (EBV) and the presence of BMMF in those EBV infected brain cells.

Zur Hausen is the latest in a long list of researchers to make a connection between MS and cow products although traditionally most researchers have limited their attention to milk rather than beef. In 2018 a German academic Stefanie Kürten from University Hospital Bonn looked into a link between cows' milk and MS. "We injected mice with different proteins from cows' milk. We wanted to find out if there was a constituent that they were responding to with symptoms of disease." **Continued on page 6**

Continued from page 5 Kürten and her group did indeed find exactly what they were looking for. When they administered a cows' milk constituent called casein together with an effect enhancer to the mice they went on to develop neurological disorders. Electron microscopy showed damage to the insulating layer around the nerve fibres, the myelin sheath.

The insulating sheath was massively perforated in the mice, apparently triggered by casein administration. "We suspected that the reason was a misdirected immune response, similar to that seen in MS patients," explains Rittika Chunder, a postdoctoral fellow in Prof. Kürten's research group. "The body's defences actually attack the casein, but in the process, they also destroyed proteins involved in the formation of myelin."

Casein is present in beef as well as cows' milk. Clearly it is possible that casein is part of the process that causes BMMF to be in the brain lesions associated with MS. But the question remains: how does all this tie in with the exposure to the Epstein Barr virus? In his 2021 paper Zur Hausen hypothesized that EBV is reactivated in MS and then the BMMF multiply and are eventually converted into proteins which cause auto-immunity leading oligodendrocytes to malfunction, which leads in turn to the destruction of myelin.

According to Zur Hausen and Kürten the case against cow products is a strong one but if further justification were needed, a paper published in Multiple Sclerosis Journal in 2016 found that one of the biggest cattle rearing areas in South America, Córdoba, Argentina, also has the highest prevalence of MS in Latin America with 88 cases per 100,000 inhabitants.

This prevalence dwarfs that of the Chilean city of Punta Arenas at the very Southernmost and least sunny tip of the continent, which is known for its sheep farming, and has a prevalence of MS of just 14 per 100,000, suggesting that cow products may be a far more powerful predicator of MS even than latitude and lack of sunlight and vitamin D.

Interestingly when I carried out a 93 food IgG (Immunoglobulin) food intolerance screening test I found that cow's milk was the animal product that I had the highest level of sensitivity to, and beef was the meat I was most sensitive to.

If further justification for this radical change to my diet were needed it would come not from my own anecdotal evidence or classical medical research but from a study of occupational risks and MS. The study was carried out by a Danish pension provider PensionDanmark into critical illness insurance cover and those occupational groups most likely to claim it.

This research showed that the frequency of MS among "dairy operators," was twice as high as the rest of the study's population. It's hard to think of what more proof you need when it comes to reasons for giving up cow's milk. The evidence against beef is growing but when it comes to giving up cow's milk, I would say the evidence is already there. Given the fact that beef has only been linked to MS relatively recently, it may be just a matter of time before beef is next on the banned list for enlightened MSers. I am sure that time will come and come soon.

Pic p5 Thanks to Annie Spratt- Unsplash

For further details of sources for feature see page 14

Sanofi launches Phase 3 frexalimab trials after positive Phase 2 data

Two phase 3 clinical trials have been launched in the US to test a new injectable drug frexalimab as a treatment for both non-active and active progressive MS.

One of the newly launched Phase 3 trials (NCT06141473) will recruit adults, aged 18-55, with active secondary progressive MS (SPMS). The other trial (NCT06141486) will involve adults with non-active SPMS. Both trials are actively recruiting at sites in the U.S. The first trial has a primary and study completion date of May/ June 2027. The second trial has a primary completion date of December 2026 and a study completion date of March 2028.

Frexalimab is designed to block a protein that forms part of the pathway that activates many parts of the immune system. This pathway has been implicated in inflammation that characterizes MS. Sanofi, the bio-tech company behind Frexalimab believes it has potential to address both acute and chronic inflammation in MS without causing widespread immune cell depletion.

The two studies build on promising data from a proof-of-concept Phase 2 trial (NCT04879628) that initially was presented at an annual meeting last summer. A Sanofi-sponsored study that details those findings, titled “Inhibition of CD40L with Frexalimab in Multiple Sclerosis,” has been published in The New England Journal of Medicine.

In earlier trials, frexalimab was associated with significant reductions in new inflammatory brain lesions between weeks eight and 12 compared with the placebo group. Other positive results included reductions in the number of new or enlarging lesions compared with the placebo group and the total number of inflammatory lesions. Reductions were also seen in neurofilament light chain, a biomarker of nerve damage, and CXCL13, a marker of inflammation.

“Progression is where the true clinical value of frexalimab, and its place in the therapeutic armamentarium against multiple sclerosis, will need to be defined,” a company spokesperson said. That’s the goal of the one of the Phase 3 trials, in which investigators will assess whether frexalimab can delay confirmed disability progression among non-active SPMS patients compared with a placebo.

Participants will be followed for nearly four years. Disability progression in this trial will be determined as an increase in Expanded Disability Status Scale (EDSS) scores, or a 20% worsening in dexterity or walking ability scores, that’s sustained for at least six months**.**

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EU agency OKs new drug study to boost mitochondrial function in prog-MS

Bio-pharma Mitochon Pharmaceuticals is launching a pilot clinical trial to evaluate MP101, its treatment candidate for improving mitochondrial function, in people with progressive MS.

The announcement follows the trial's clearance by the European Medicines Agency (EMA), which enables Mitochon to begin enrolling patients with MS and other neurodegenerative conditions in the trial.

The Phase 1-2 trial will enrol people with MS, amyotrophic lateral sclerosis (ALS), Alzheimer's disease, and Huntington's disease, each of whom will receive the experimental therapy for 14 days. The goal is to demonstrate whether the therapy is safe in these groups and induces relevant changes in disease-related biomarkers.

MP101 designed to work as stimulator for mitochondrial function. Mitochondria are small cellular structures that produce most of the energy cells need to function — including the energy required for nerve cells to transmit electrical signals to other cells. They're also involved in other essential cell functions.

Research has shown that mitochondria are dysfunctional, or don't work properly, in most neurodegenerative conditions, including MS. Thus, impaired mitochondrial function is thought to play an important role in neuronal damage and disease progression. Some readers may recall Dr Terry Wahls 2010 book "Minding My Mitochondria. How I overcame secondary progressive multiple sclerosis (MS) and got out of my wheelchair." Dr Wahls was a pioneer in suggesting mitochondria were involved in MS and claimed great improvement in her condition by following what came to be known as "The Wahls Protocol".

MP101 is an investigational, brain-penetrant drug that works as a mitochondrial stimulator, improving the function of mitochondria in the brain and spinal cord cells. This increases the amount of energy produced by cells and also induces the production of neuroprotective factors, which help protect cells from neurodegenerative disease processes.

In preclinical studies, researchers showed that MP101 was able to protect nerve cells from the demyelination that occurs in people with MS. It also reduced disease severity and improved mobility in animal models of the disease. Similar results were obtained with MP201, a similar candidate drug. The upcoming trial is expected to confirm the benefits of MP101 in neurodegenerative conditions. If positive, these findings are expected to support Phase 2b studies in people with primary progressive and secondary progressive MS, ALS, Huntington's, and Alzheimer's. No timeline for the trial has yet been provided by Mitochon.

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Gender, age at PPMS onset has no impact on disability progression

Gender and age at onset of primary progressive MS (PPMS) don't seem to have an impact on disability progression, according to a recent study in Argentina.

Disease progression, based on the Expanded Disability Status Scale (EDSS), was analysed in patients between the age of 20 and 50 at disease onset over 10 years. The study, "Clinical impact of gender and age at onset on disease trajectory in primary progressive multiple sclerosis patients," was published in the Multiple Sclerosis Journal.

Several factors have been claimed to be possible indicators for disease course and progression including age and gender. In relapsing-remitting MS (RRMS), men have been shown to have a worse disease course and more severe disability than women.

However, "the current understanding remains inconclusive as to whether gender and age at onset play a significant role in influencing the progression of disability in patients with PPMS," wrote the researchers, who studied 125 people with PPMS and looked at the progression of their disease and disability.

Men were slightly older at disease onset than women (average age, 45.1 vs. 42.6) and had a longer disease duration Significantly more women were treated with Ocrevus (ocrelizumab) than men (38% vs. 23%), but no differences were observed on the time to treatment since disease onset or treatment duration.

At the first, or baseline, evaluation, men and women had similar mean EDSS scores, but men had a higher score at the last evaluation (6.4 vs. 6). There were no significant differences between genders in the annual increase of disability, between one and 10 years after PPMS onset, however.

There was a trend for a faster decline in disability rate in older patients at progressive onset compared to younger ones, a phenomenon the researchers attributed to the progressive decline in immune function as people age, called immunosenescence.

Finally, for a difference in 20 years in age at onset, there were no clinically relevant differences in disease disability between men and women.

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Of mice and MS

There is a saying among medical researchers that "mice lie and monkeys exaggerate". In other words, experiments using mice give results that are wrong and those involving monkeys give results that are plain misleading.

Despite this, testing on mice continues to be a typical early step in researching MS medications, and academic journals are filled with reports about mouse or "murine" studies. The practice of experimenting on mice to find new MS treatments is so widespread and open that a Professor of Neuro-immunology at Queen Mary University of London goes by the title "mouse doctor" and tweets @prof_mouse.

While some MS researchers may be enthusiastic about mouse studies, many MSers are more sceptical. Ed Tobias, an American who has lived with MS since 1980 writes a column in "MS News Today" and recently took up this issue in his column "The MS Wire". The column was titled "Let's not get overexcited about any mouse study used in research". Ed asked: what exactly are the odds of an experimental treatment making it from mouse or monkey to human?

He said that according to a 2014 study published in the American Journal of Translational Research less than 8% of cancer treatments make it from animal studies into tests on people. And, according to a study published in the July 2022 Acta Pharmaceutica, only 10% of these medications make it through to government approval. That means less than 1% of the treatments that work on animals ever make it as cancer treatments for humans.

In spite of this, a lot of us read reports of a promising mouse MS study and think it will lead to a blockbuster MS treatment that will be available and quickly. Not so, says Ed. "On average, it takes a drug about 12 years to get from discovery to market." That's according to an article in the Muscular Dystrophy Association's Quest magazine. A clinical trial alone can take five to seven years. Then, if all goes well, government approval is sought.

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Continued from p10 The small percentage of mouse trial drugs that work in humans are not likely to make it as human treatments very quickly.

I have no doubt the MS researchers who carry out mouse experiments sincerely believe their work will help find new treatments and ultimately uncover the "holy grail" of MS research – the cure. And it is true that a few disease modifying drugs have come to market after starting out by being tested on mice. However, there have been criticisms of how similar the mouse model is to human MS, not to mention how many new treatments start by showing promise in mouse MS models and then fail to translate to treatments for MS in humans, what is known as "failure in translation".

Many things do indeed get lost in translation, so to speak. At least one researcher acknowledged this, but more of that later. For me, the real problem with the mouse MS model called EAE (Experimental Autoimmune Encephalitis) which involves demyelinating mice is that it may tell us about the process of demyelination, what I call the "how" question. However, does it tell us much about why we humans demyelinate (ie: get MS) in the first place, what I call the "why" question? All of which begs the question- is there a better way of finding out why we get MS?

I think there is, and this approach is based on observation rather than experimentation. It has been known for years that there are other demyelinating diseases in animals and in humans too. There was a well reported outbreak of an MS-like illness among Macaque monkeys in a zoo in Oregan USA in 2011. "Monkey MS" was found to be caused by a herpes virus in the same group as Epstein Barr virus or glandular fever.

In humans there is also a demyelinating condition known as Tropical Spastic Paraparesis (TSP) which can lead to demyelination. This condition is caused by a virus too. In this case it's a retrovirus (HTLV1) and it shares some similarities with HIV. Unsurprisingy perhaps there are occasional stories of HIV positive MSers who are "cured" of their MS after taking a combination of HIV anti-viral medications.

I suppose my message to all mouse doctors would be: "yes carry on with your work. Studying demyelination may be useful in studies that focus on the process of how to get the body to re-myelinate." Beyond that, I would argue there is more value in looking at why viruses, not just EBV, might lead to demyelination in the first place. And from where I am sitting this doesn't seem to me to require much, if any, work on mice.

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News

Nitric oxide metabolites in blood could aid diagnosis of PPMS - study finds

Measuring levels of nitric oxide metabolites in the blood could help diagnosis of primary progressive MS, (PPMS) recent research suggests.

Nitric oxide molecules, collectively called NOx, were elevated in the blood of people with primary-progressive MS compared to people with a noninflammatory neuropsychiatric condition.

The research study titled "The Use of Nitrosative Stress Molecules as Potential Diagnostic Biomarkers in Multiple Sclerosis," was published in the International Journal of Molecular Science.

Nitric oxide, or NO, is a molecule that plays a number of roles in the body, including in blood vessel widening, or vasodilation. High concentrations are associated with nitrosative stress, a type of cell damage that's implicated in MS and other neurodegenerative diseases.

In MS, it's thought that NO contributes to the dysfunction of the blood-brain barrier (BBB), a tight-knit layer of cells lining the blood vessels of the brain that helps keep potentially harmful substances in the bloodstream out of the nervous system. Vasodilation caused by NO disrupts this barrier, allowing immune cells to cross the blood brain barrier contributing to the self-reactive inflammatory attacks that mark MS.

The current study included 16 people with PPMS, all untreated. PPMS patients had higher levels of NOx in the blood.

The study didn't include people with secondary progressive MS (SPMS). The researchers said they wanted to evaluate NOx levels in untreated patients, and SPMS patients have typically already received some type of MS therapy.

"NOx in SPMS would be particularly relevant as a progression and therapy response marker, which was not the focus of our study," they wrote.

"A combination of NOx with other biomarkers might increase sensitivity in the future," they added. **For details of all stories go to page 14**

Study suggests EBV-specific T cells play key role in MS development

The body's immune response to Epstein-Barr virus (EBV) may play a key role in neurological damage to people with MS, according to a new study led by University of Texas Health, Houston, USA.

EBV infection has long been associated with MS, but how the infection might contribute has not been clear. More than 95% of people have been infected with EBV - a very common virus; however, it typically remains in its latent stage and doesn't cause any issues. In some cases, though, the T cells specific for the EBV infection are believed to cause problems.

Now, research led by first author Assaf Gottlieb, Ph.D., assistant professor with the Centre for Precision Health at McWilliams School of Biomedical Informatics at UT Health Houston, and senior author J. William Lindsey, MD, professor in the Department of Neurology with McGovern Medical School at UT Health Houston, demonstrates that T cells specific for EBV-infected cells are present in high numbers in the cerebrospinal fluid of people with MS at its earliest stages. The study is published in a publication “Proceedings of the National Academy of Sciences.”

On average, 13% of the T-cells in the cerebrospinal fluid of people with the first symptoms of MS were specific for autologous B lymphocytes infected with EBV, demonstrating a clear link between EBV and MS.

T cells for the three other common infections tested -varicella zoster virus (chicken pox); influenza virus, and candida - did not show a similar abundance in the cerebrospinal fluid.

For details of all news stories see page 14

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Page 13 Study suggests EBV-specific T cells play key role in MS development

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