

# My prog-MS ezine

## For people with progressive MS and other interested people

**Issue number 42, March/April 2022**

**Hello and welcome to the slightly later than usual 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 63.**

In this issue are eight pages of prog-MS news stories. There are also two features. One on page 5 is about a surprisingly common and under-reported eye problem in MS, and I'm not talking here about optic neuritis.

There's also a feature on "smouldering MS" – that MS is a smouldering progressive disease right from day one. See page 10 for details.

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## *News*

# ‘Compelling evidence’ Epstein-Barr virus is a cause of MS

MS is likely caused by infection with the Epstein-Barr virus, (glandular fever) according to a study led by Harvard T.H. Chan School of Public Health researchers. Their findings were published in the journal “Science”.

“The hypothesis that EBV causes MS has been investigated by our group and others for several years, but this is the first study providing compelling evidence of causality,” said Alberto Ascherio, professor of epidemiology and nutrition at Harvard Chan School and senior author of the study. “This is a big step because it suggests that most MS cases could be prevented by stopping EBV infection, and that targeting EBV could lead to the discovery of a cure for MS.”

The cause of MS is not known, yet one of the top suspects is EBV, a herpes virus that can cause infectious mononucleosis (glandular fever) and establishes a latent, lifelong infection in the patient. Establishing a causal relationship between the virus and the disease has been difficult because EBV infects approximately 95 percent of adults, MS is a relatively rare disease, and the onset of MS symptoms begins about 10 years after EBV infection. To determine the connection between EBV and MS, the researchers conducted a study among more than 10 million young adults on active duty in the U.S. military and identified 955 who were diagnosed with MS during their period of service.

The team analysed serum samples taken every two years by the military and determined the soldiers’ EBV status at time of first sample and the relationship between EBV infection and MS onset during the period of active duty. In this group, the risk of MS increased 32-fold after infection with EBV but was unchanged after infection with other viruses. Serum levels of neurofilament light chain, a biomarker of the nerve degeneration typical in MS, increased only after EBV infection. The findings cannot be explained by any known risk factor for MS and suggest EBV as the leading cause of MS.

Ascherio says that the delay between EBV infection and the onset of MS may be partially due to the disease’s symptoms being undetected during the earliest stages and partially due to the evolving relationship between EBV and the host’s immune system, which is repeatedly stimulated whenever latent virus reactivates. “Currently there is no way to effectively prevent or treat EBV infection, but an EBV vaccine or targeting the virus with EBV-specific antiviral drugs could ultimately prevent or cure MS,” said Ascherio.

***For details of sources for all news stories go to page 13***

## Phase III trial of Tolebrutinib in SPMS and PPMS – results in 2024

Patients with non-relapsing secondary progressive multiple sclerosis ages 18 to 60 years are now being recruited in the UK to take part in the "The Hercules Trial" a Phase III clinical trial designed to test how well the trial drug Bruton's Tyrosine Kinase (BTK) Inhibitor Tolebrutinib works in SPMS and whether it's safe.

Tolebrutinib will be compared to placebo (a tablet that looks the same as the trial drug but is essentially a "sugar pill"). The main effect being tested is whether the trial drug delays disability progression in patients diagnosed with non-relapsing secondary progressive MS (nrSPMS). Primary Progressive MS (PPMS) is also being looked at in a separate trial.

SPMS patients will be randomly selected to receive the trial drug or placebo. Patients are twice as likely to receive the trial drug vs placebo. The trial drug is being compared to placebo because approved treatments specifically for non-relapsing SPMS (nrSPMS) are not available in all countries or covered by insurance in all patients.

Additional goals of the trial will be to test the effect of the trial drug on disability progression, magnetic resonance imaging (MRI) lesions, the ability to learn and process information, and quality of life, as well as the safety and tolerability of the trial medication.

Those who qualify to participate in clinical trials may receive medical care and medication at no cost to the patient. People who participate in clinical trials aid others with the same condition by helping the scientific community find new treatment options for diseases such as multiple sclerosis. The following UK cities are hosting trials: Exeter, Plymouth, Nottingham, Oxford, Cardiff, Newcastle Upon Tyne, Salford , Tooting, London.

Tolebrutinib inhibits an enzyme called Bruton's tyrosine kinase (BTK), reducing the activation of B cells and inhibiting immune cells called macrophages which have been linked to MS progression. By inhibiting this enzyme, it is thought tolebrutinib will reduce the damage caused to nerve cells by MS.

In animal studies tolebrutinib appeared to prevent the loss of myelin by preventing microglia from engulfing and destroying the myelin sheath produced by oligodendrocytes in the animals' brain, while ensuring the survival of myelin-producing cells. It is a tablet taken once daily.

A trial (Perseus) is also taking place into the use of the drug in primary progressive MS. Both Hercules and Perseus trials are due to end in 2024.

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# Malaria drug Hydroxychloroquine Delays Disability in PPMS

A study from the University of Calgary in Canada has found that malaria drug hydroxychloroquine shows promise in treating the evolution of disability of primary progressive MS (PPMS).

Researchers Marcus Koch, MD, PhD, and Wee Yong, PhD, both found that the drug helped to slow worsening of disability during the 18-month study involving participants in the MS clinic in Calgary.

“With primary progressive MS, there is no good treatment to stop or reverse the progression of disease. The disability progressively worsens through time,” Koch said in a press release.

“Dr. Yong’s research team, with whom we closely collaborate, has been screening a large number of generic drugs over several years and the results with hydroxychloroquine show some promise. Our trial is a preliminary success that needs further research. We hope sharing these results will help inspire that work, specifically larger scale clinical trials into the future.”

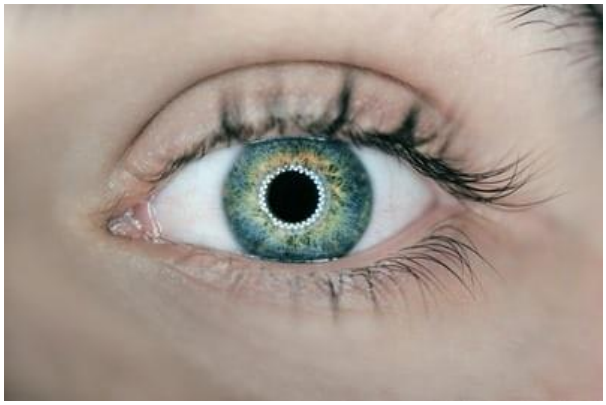
The single-arm, phase 2 trial followed 35 patients with MS between November 2016 and June 2021. The researchers projected that approximately 14 participants would experience a significant worsening of their walking function, however, only 8 participants had their walking function decline.

Hydroxychloroquine was chosen because it is widely used in rheumatological diseases, such as lupus and rheumatoid arthritis, and is mostly well tolerated by these patients, according to the study authors.

“Based on research in our lab on models of MS, we predicted that hydroxychloroquine would reduce disability in people living with MS. Calgary has a vibrant bench-to-bedside MS program and the work from Dr. Koch’s trial offers further evidence which we were pleased to see,” Yong said in the press release.

Koch and the other researchers will continue to study hydroxychloroquine for its potential to achieve greater results as a therapy in combination with other generic drugs, according to the release.

Hydroxychloroquine, sold under the brand name Plaquenil among others, is best known as a medication used to prevent and treat malaria in areas where malaria remains sensitive to chloroquine. **For details of all stories go to page 13**



# MS: the eyes have it!

**I recently had an appointment with my optician to see if I need stronger reading glasses. In case you were wondering the answer was "yes." During the eye test the optician asked me if I ever noticed my eyes were dry.**

I was a little surprised at this question but in truth the answer is yes, my eyes are sometimes dry. The optician then went on to tell me something very interesting. Dry eyes, he said, can be a symptom of MS. I told him that in my 30 years since diagnosis I had never heard this but I would buy some eye drops the next time I visit the chemists and make sure I use them. He said that I could get a prescription for eye drops if I preferred.

A few days later I was reading the popular MS Blog https://multiple-sclerosis-research.org/ and spotted a post by Neuro Doc Gnanapavan (that's Dr Dr Sharmilee Gnanapavan, a neurologist at Barts Hospital in London) The post was titled: "It's in the Eyes" and went as follows:

"I know what you're thinking. You're thinking that I'm going to write about optic neuritis since this is what everyone writes about. But you will be wrong. This post is about the lesser well-known portion of the eye – the conjunctiva. (The conjunctiva is the clear tissue that covers the white part of the eye and the inside of the eyelids, it provides protection and lubrication of the eye by the production of mucus and tears.)

"In a small cross-sectional study of MS patients and controls, a group of investigators from Turkey found that dry eyes are far commoner in MS than you'd expect by chance. The Schirmer test (a test of eye moisture) was below 10mm in 66.7% of the participants, whilst the TBUT (tear break up time) was below 10s in 81.8% of MS patients. On average the Schirmer test result was 8.45mm in MS vs 17.36mm in controls and the TBUT was 8.12s vs 13.06s in controls. (In other words, an MSer's eyes are half as moist and tears break up nearly twice as quickly as those without the condition).

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***Continued from page 5*** Dr Gnapavan went on: "As a result, corneal biopsies showed that the degradation of the cornea (your eye's clear, protective outer layer) was greater in MS than in controls. It is thought that the dry eyes occur as a result of reduced corneal nerve innervation (lack of nerve support) to the eye. In addition to providing sensation to the cornea, nerves provide growth support as well. With a reduction in the corneal nerve fibre density there is an obvious reduction in the health of the cornea." For their part the Turkish authors of the paper which Dr Dr Gnanapavan was quoting concluded by saying that "Patients with MS should be monitored for ocular surface alterations and dry eye disease."

I know that corneas matter a lot in sight, more than is generally recognised, I have a friend who has corneal problems and she is partially sighted as a result. My friend explained to me that in lay terms the cornea is like the eye's equivalent of a car windscreen. It is literally your window on the world and blinking acts like the cornea's windscreen wipers. Blinking keeps your eyes clean of debris with tears acting as a lubricant to ensure that your blinking takes place in a well lubricated environment and cleans the eye properly.

My friend does not have MS and when we spoke I had no idea that MS could affect your corneas although given the above research it's clear corneal damage is a potential problem and one that's hardly surprising given that in MS nerve damage occurs in all parts of your body, including your eyes. Just think of optic neuritis, double vision and nystagmus. They are all well recognised as MS symptoms. Now it seems dry eyes are a symptom too.

The journal "Review of Optometry" put the MS related dry eye problem in the following terms. It said: "Multiple sclerosis is a demyelinating disease that can lead to severe dry eye. In MS, poor corneal sensory impulse conduction can result in insufficient tear production, and lagophthalmos-associated DED (Dry Eye Disease) can occur due to poor motor control. These patients may have limited ability to instil drops and perform lid hygiene properly; thus, punctal plugs, humidifiers and in-office blepharitis therapies may be crucial."

A punctal plug, in case you are wondering, is a tiny medical device that sits in the tear ducts in the eyes to help prevent tears draining away, keeping the eyes moist. Punctal plugs are usually inserted by doctors or ophthalmologists. I have no idea what is meant by in-office blepharitis therapies and little desire to find out, although I do know that blepharitis is where the edges of your eyelids become red and swollen. The good news is I don't have blepharitis.

Fortunately, the optician told me that my dry eye problem is not that serious. And although I have noticed my eyes do get a bit dry occasionally, that's all. However, based on his advice I have started to use eye drops on a regular basis. They are cheap, easy to obtain and I have found them highly effective. I will try to get them on prescription next time I see my GP.

I suppose my "take away message" based on the optician's visit is to keep my eyes moist and to get eye drops on prescription. My message to other MSers would be to ask your optician at your next visit: "Do I have dry eyes?"

**More information on page 13. Picture page 1 and page 5 arteum.ro at unsplash**

# Bio-pharma Abata Plans for prog-MS T-cell Therapies

**US bio-pharma Abata Therapeutics has announced plans to use regulatory T-cells to treat progressive MS. Three clinical trials are planned to start in 2025, including one in primary progressive (PPMS) and nonactive secondary progressive (SPMS) patients.**

“Our initial and urgent focus is on patients living with non-relapsing progressive MS, which remains untreatable, despite exciting advances for relapsing forms of the disease,” Richard Ransohoff, MD, co-founder and chief medical officer at Abata, and a venture partner at Third Rock Ventures, said in a press release.

“The time for T-regs is now, and we are emboldened in our approach to halt the progression of this debilitating disease and others like it,” he added.

Regulatory T-cells, also referred to as Tregs, are a type of immune cell that prevents other immune cells from attacking the body’s own tissues. As such, they are considered ideal candidates to treat autoimmune disease, restore a state of steady equilibrium in the body and promote repair in affected tissues.

T-cells have receptors on their surface that allow them to sense and respond to specific antigens, or molecules that trigger an immune response. In MS, disease-causing T-cells target antigens of myelin, damaging this nerve-protecting sheath.

Researchers can tinker with Treg receptors so that they travel to sites where an autoimmune response has been triggered, preventing other immune cells from doing damage. By selectively targeting the site of disease, Abata hopes to halt its autoimmune reactions without suppressing the entire immune system.

The Tregs used by the company are autologous, meaning they are obtained directly from a patient, minimizing the risk of rejection once engineered cells are returned to the patient. According to Abata, the engineered cells are expected to remain in tissues for long periods of time, which may allow the effects of a single dose to last for years, and potentially lifelong.

The proposed MS trial will enrol PPMS and nonactive SPMS patients with evidence of inflammatory lesions on MRI scans. Pending positive results, the company expects to expand testing to all with progressive MS, including patients who experience relapses.

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## Atara, Fujifilm Enter Deal to Produce Cell T-cell Therapies

Atara Biotherapeutics has reached an agreement with Fujifilm Diosynth Biotechnologies for the production of its cell therapy pipeline, including ATA188, an experimental therapy being investigated for the treatment of progressive MS.

Under the agreement, Fujifilm will acquire the Atara T-Cell Operations and Manufacturing (ATOM) facility in California, for an upfront payment of $100 million. The companies also are entering into a supply agreement that could extend up to 10 years, whereby Fujifilm will provide Atara with access to the capacity and capability needed to manufacture its cell therapies.

Atara is sponsoring a Phase 2 clinical trial called EMBOLD (NCT03283826) that is testing ATA188 in people with progressive forms of MS. The study is recruiting at locations in the U.S. and Australia.

ATA188 is a T-cell-based therapy that is designed to destroy cells infected with Epstein-Barr virus (EBV), a type of herpes virus that some research has shown may be a cause of MS. The therapy uses allogenic, or "off-the-shelf" T-cells — as opposed to autologous cell therapies that use cells derived from the patient — meaning it may be more readily accessible to those who need it.

With the new deal, Atara expects to have sufficient funds to run operations beyond the completion of the above trial, the company announced. As part of the agreement, approximately 140 manufacturing and quality staff at the ATOM site now will be working for Fujifilm.

"We are thrilled that through this acquisition we will add approximately 140 talented staff from Atara's cell therapy manufacturing facility to the Fujifilm Diosynth Biotechnologies family. The collective expertise of the team will further support our efforts as a world-class CDMO," said Martin Meeson, CEO of Fujifilm Diosynth Biotechnologies, which is a subsidiary of Fujifilm Corporation.

A CDMO, or contract development and manufacturing company, is a company within the pharmaceutical industry that provides drug development and manufacturing services, usually in the form of outsourced services for pharmaceutical companies.

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# Masitinib to enter phase III trials for SPMS and PPMS

The Swedish Medical Products Agency has approved AB Science's request to launch a confirmatory Phase 3 clinical trial of masitinib in adults with primary progressive multiple sclerosis (PPMS) and nonactive secondary progressive MS (SPMS).

The decision comes on the heels of a similar authorization by the French Health Authority. AB Science expects the upcoming trial to involve 75 sites in Europe, the U.S., Canada, Israel, and South Africa.

"This latest approval … represents a key step not only for the clinical programme of masitinib in progressive forms of MS, but also for the overall masitinib pipeline in neurology," Olivier Hermine, MD, PhD, AB Science's co-founder and president of the scientific committee, said in a press release.

"To date, masitinib has demonstrated positive Phase 2B/3 results in three neurodegenerative disorders, namely, Alzheimer's disease, amyotrophic lateral sclerosis (ALS), also known as motor neurone disease, and progressive forms of MS. The therapy's success in these three conditions "clearly demonstrates that masitinib's innovative targeting of the innate immune system is a valid strategy," Dr Hermine said.

"We are committed to continue masitinib's clinical development programme in each of these highly challenging neurological disorders, with an ultimate objective to improve patients' lives," Hermine added.

Masitinib, formerly known as AB1010, is an oral therapy designed to suppress the activity of innate immune cells — such as mast cells, microglia, and macrophages — which are thought to drive neuroinflammation and neurodegeneration in progressive MS. It works by blocking tyrosine kinase, a key enzyme for these cells' activity.

Called AB20009 (2021-000639-30), the upcoming international trial will test the therapy against a placebo in up to 800 adults, 18–65 years, with PPMS and nonactive SPMS. Participants will be randomly assigned to receive either masitinib — starting at a daily dose of 3.0 mg/kg and increasing to 4.5 mg/kg after four weeks — or a placebo for up to two years.

The study's main goal is to assess whether masitinib extends the time to disability progression lasting at least three months. Secondary goals include time to six-month confirmed disability progression, changes in EDSS scores over two years, and time to becoming wheelchair-bound. **For further information on news stories go to page 13**



# When your MS is just smouldering away

One of the contradictions of MS, personally speaking, is that my MS doesn't neatly fit the generally accepted view of the illness.

The official view is one in which the MSer moves neatly from episodes of inflammatory disease activity (relapsing remitting MS or RRMS) to one in which the disease gradually gets continually worse (secondary progressive -SPMS- or neurodegenerative MS)

For me things aren't quite so neat or simple. In my own 30 years of MS there have been several episodes of disease activity which started in the first few years after diagnosis (when I was in the RRMS stage) and where the symptoms didn't suddenly appear and go away, in fact they are still with me. I am thinking here of leg spasms and urinary problems which started about five years after diagnosis have come and gone but never really gone away at any point subsequently. Now I have these symptoms daily.

This sense of not fitting in with the accepted view of MS means my view of progression being independent of relapses rather than an accumulation of relapse damage made me interested in an academic paper written by 18 MS experts including Professor Gavin Giovannoni of Barts Hospital In London.

This paper outlines the argument for "smouldering MS being the real MS ". This "smouldering MS" view of MS is that the real MS is not about focal inflammation or acute relapses and remissions but a more diffuse gradually worsening process. In other words, MS is a smouldering progressive disease from day one. This is at odds with the dominant worldview.

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**Continued from page 10** Giovannoni and his co-authors, writing in the journal Therapeutic Advances in Neurological Disorders, provide evidence to back up their argument that the 'real MS' is in fact driven primarily by a smouldering pathological disease process. Their paper points out that in clinical trials, relapses and focussed disease activity revealed by magnetic resonance imaging (MRI) in MS patients were poor predictors of long-term disease evolution and were dissociated from disability outcomes.

In addition, the academics argue that the progressive accumulation of disability in MS can occur independently of relapse activity from early in the disease course (as is my experience of leg twitching and bladder issues). It is argued therefore that the progressive accumulation of disability as MS advances is underpinned by a smouldering pathological process that may affect the entire central nervous system. I am in total agreement with this.

Based on this argument the authors hypothesise that therapeutically targeting a state of 'no evident inflammatory disease activity' (NEIDA) cannot sufficiently prevent disability accumulation in MS. They reject the concept that MS is a two-staged disease, initially dominated by an inflammatory relapsing phase that transitions into a non-inflammatory neurodegenerative phase pointing out that this has been largely based on clinical and MRI observations and is at odds with pathological studies which show that active ongoing inflammation and demyelination in the CNS can be observed even in the terminal or end stage of MS. Overall, these observations agree with pathology studies showing, both in SPMS and PPMS cases, similar degrees of inflammatory infiltrates, axonal loss, and cortical demyelination, thus supporting the notion of MS being a single-stage disorder.

It is further argued that we therefore need to go beyond no evident inflammatory disease activity (NEIDA) and focus on other pathological processes in the end-organ (brain and spinal cord) in order to delay or prevent the slow loss of neurological function in people with MS. In addition, a holistic approach to the management of MS is needed by targeting other processes that have been shown to impact on brain health.

**Page 10 Image by Yaoqi from Unsplash**. **For details of all news and features go to page 13**

# Tevogen to Develop T-cell Remedy That Might Halt EBV Infection

Tevogen Bio is planning to make use of its virus-fighting immune cell technology and turn it against Epstein-Barr virus (EBV), a common virus thought to greatly increase the risk of multiple sclerosis (MS).

The technology employs off-the-shelf cytotoxic T-cells — a type of white blood cell that can kill other cells, such as those infected with a certain virus. This potentially allows the body to replace the infected cells with healthy, uninfected cells.

For those already diagnosed with the neurodegenerative disease, the treatment might dampen the immune attacks that ultimately damage the myelin sheath in the brain and spinal cord, potentially slowing disease progression.

“Anyone that knows someone who has been impacted by this debilitating disease understands the importance of advancing science to develop pathways to alleviate the suffering from MS,” Ryan Saadi, MD, CEO of Tevogen, said in a press release.

In MS, the immune system erroneously launches an attack against myelin, a fatty substance that wraps around nerve fibres and is necessary for an efficient nerve cell communication. The exact causes of MS remain unknown, but there are several theories as to what triggers the immune system to attack the body’s own tissues. One likely explanation is an infection at some point in life.

“Recent studies on multiple sclerosis have suggested a probable link between latent infection with EBV and later onset of the inflammation that degrades the myelin sheath and causes multiple sclerosis,” said Neal Flomenberg, MD, chairman of the scientific advisory board at Tevogen.

The company is currently testing its technology in a Phase 1 clinical trial (NCT04765449) evaluating the safety and efficacy of T-cells in treating COVID-19, a disease caused by the severe acute respiratory syndrome coronavirus 2 (SARS-CoV-2).

The therapy, called TVGN-489, essentially involves collecting T-cells from healthy donors who have recovered from COVID-19 and selecting the cells that specifically recognize particles from the SARS-CoV-2 virus. Tevogen now plans to use its technology and develop a donor-derived T-cell therapy against EBV for the treatment of MS.

“We look to apply our significant expertise in off-the-shelf [SARS-CoV-2 specific T-cells] to explore EBV specific [T-cell] therapy as a possible means of addressing the unmet needs of MS patients,” Flomenberg added.

**For further details of all features go to page 13**

*News*

# Top researcher asks for anti-viral MS drug trial volunteers

Gavin Giovannoni, Chair of Neurology at the Blizard Institute, Barts and The London School of Medicine, is asking prog-MSers: “Would you volunteer to participate in a clinical trial of an antiviral drug cocktail to suppress MS disease activity, in particular smouldering MS?”

He says: “There is now a reasonable body of evidence in the literature that human endogenous retroviruses (HERVs) may play a role in autoimmunity, in particular MS.”

HERVs are viruses (genetic code) that have been incorporated into the human genome over deep time. Some genetic elements from HERVs have taken on important functional roles, for example, they are involved in the development of the placenta and hence are part of our human biology. When HERVs elements are transcribed they may be capable of forming so-called replication-competent viruses, which can reinfect cells and then integrate them back into the genome. These reintegration sites may be important in themselves and may enhance the function of individual cells and hence drive the selection of cells within the body. These processes may underlie the development of autoimmunity and even some cancers. Other HERV elements are replication-incompetent and although they can produce functional transcripts (RNA) and proteins they can’t form an infectious virus.

Some HERV proteins act as danger signals activating so-called toll-like receptors and other danger-signalling pathways. These pathways then upregulate innate immunity and provide the immunological nudge that drives autoimmunity. This is why there have been some attempts to try and suppress HERV activation with antiviral drugs or to neutralise some of the HERV proteins that may activate the immune system, or are directly toxic to myelin-producing cells and/or neurones, as a treatment for MS.

Prof Giovannoni goes on: “It would be a travesty if in 20 years' time the next generation of MS researchers discover that HAART is a very effective treatment for MS when we have the tools to answer this question now, i.e. in the next 4 to 5 years?

“Sadly outside-the-box ideas or paradigm shifts often take generations to occur. So don’t be surprised if the wider MS community continues to reject these hypotheses and nothing happens for decades. So if you know anyone who works for one of the big pharma companies who make antiretrovirals are a wealthy philanthropist please forward this Newsletter (see url on p13) to them.

# References further details, etc

**Page 2 EBV and MS link**

https://news.harvard.edu/gazette/story/2022/01/epstein-barr-virus-may-be-leading-cause-of-multiple-sclerosis/

**Page 3 Hercules/ Perseus trials**

https://clinicaltrials.gov/ct2/show/NCT04411641

**Page 4 Chloroquine and MS**

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**Page 5-6 Dry eyes and MS**

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**Page 8 Atara Therapeutics**

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**Page 13 Anti-viral MS drug trial volunteers sought**

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