

My prog-MS ezine

For people with progressive MS and other interested people

Issue number 48, May-June 2023

Hello and welcome to the latest 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 64.

In this issue are eight pages of prog-MS news stories. There are also two features. One on page 5 is about the seemingly endless search for the reason why MS is less common in sunny places. Is it ultra-violet light, vitamin D or something else? There's also a feature about the search for how to remyelinate nerves. There is details of exciting new research and even an upcoming trial See page 10 for further details. Picture below thanks to - Rajiv Bajaj unsplash

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News

Bio-pharma to launch trial for SPMS nose spray in late 2023

Tiziana Life Sciences plans to launch a Phase 2 clinical trial to investigate its foralumab nose spray in people with non-active secondary progressive MS (SPMS) in late 2023, the company has announced.

Foralumab is an antibody-based treatment being tested in secondary progressive MS and other diseases marked by inflammation in the brain and spinal cord.

Data from earlier research showed foralumab suppressed immune cell activation in the brain and resulted in improvements in cognition and walking that have been sustained for at least six months.

One patient whose disability was worsening despite treatment with Ocrevus (ocrelizumab) saw improvements after starting on formalumab. The patient went from an Expanded Disability Status Scale (EDSS) score of 6 — meaning a cane was needed to walk 100 metres — to a 5 after 11 months, indicating the patient was able to walk 200 metres without a cane. Additional data from all six patients are expected later this year.

Foralumab is designed to block CD3, a protein on the surface of immune T-cells. By targeting CD3, foralumab reduces the activity of certain T-cell subsets that contribute to the inflammatory attacks in MS, while boosting immunosuppressive T-cells that help keep the immune system in check.

Antibody-based medicines often carry a higher risk of side effects than other therapies. Foralumab was designed to have minimal binding to unwanted targets, however, and is expected to have fewer associated side effects. Administering it through the nose also is expected to cause fewer problems than other routes of drug delivery (pills, injections).

In the new Phase II trial Foralumab is being investigated in six patients with nonactive SPMS. All are receiving a 50 microgram (mcg) dose into each nostril in three-week cycles — three doses a week for two weeks, followed by a week of rest.

“Tiziana has reached an important regulatory milestone as it proceeds with the first ever intranasal foralumab clinical trial,” said Gabriele Cerrone, executive chairman and interim CEO of Tiziana. “The FDA’s response to our proposed Phase 2 program allows Tiziana to advance foralumab through the regulatory process as we strive to bring this novel treatment to patients with nonactive SPMS.”

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Progress in EBV vaccine research – potential lead to a cure?

A new collaboration between ModeX Therapeutics and Merck is expected to advance the development of ModeX's MDX-2201, an investigational vaccine targeting the Epstein-Barr virus (EBV).

A history of infection with EBV — known for causing infectious mononucleosis, or mono, also known as glandular fever, and certain types of cancer — is believed to be the strongest risk factor for MS. Indeed, a study last year showed it may increase a person's risk of developing MS by more than 30 times.

So, could an EBV vaccine help those of us with MS? "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," says Alberto Ascherio, MD, DrPH, professor of epidemiology and nutrition at Harvard TH Chan School of Public Health whose landmark paper "Longitudinal analysis reveals high prevalence of Epstein-Barr virus associated with multiple sclerosis," was published in February last year.

Under the new agreement between ModeX Therapeutics and Merck, the two companies will work together to continue the preclinical development of MDX-2201. They also will collaborate to file an investigational new drug (IND) application with the U.S. Food and Drug Administration requesting clearance to test the vaccine candidate for EBV in clinical trials.

After the IND filing, Merck will become solely responsible for the vaccine's clinical development and regulatory activities, as well the therapy's potential commercialization, pending approval by regulatory agencies.

Merck already has developed vaccines with the potential to help protect against certain cancers. Now, the company is setting its sights on a vaccine for EBV which mainly infects and replicates in two cell types: epithelial cells, which line most surfaces in the body, and B-cells, a type of immune cell implicated in MS. After infection, the virus remains in a dormant state inside these cells. Prior research has shown that infected immune cells may be involved in initiating the autoimmune attack that drives neurodegeneration in MS.

This vaccine candidate, in particular, is made of a nanoparticle carrying four types of viral proteins — gp350, gH, gL, and gp42 — that are important for the entry of EBV into cells. A preclinical study confirmed that this multitarget approach elicited a potent immune response in mice, ferrets, and nonhuman primates.

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Another EBV vaccine may enter clinical trials in 2024

German biotech EBViously has announced it has developed a candidate vaccine against the Epstein-Barr virus and plans to bring it into clinical trials starting next year.

The company is a spinoff of Helmholtz Munich, part of a German government research network. It is led by scientists with expertise in EBV research working toward a safe and effective vaccine against the virus and the various diseases associated with EBV infection, including MS. A first clinical trial of the potential vaccine, called EBV-001, will aim to prevent infectious mononucleosis, (glandular fever).

“As a primary indication, we are targeting the prevention of infectious mononucleosis Wolfgang Hammerschmidt, chief scientific officer of EBViously, said in a press release from the German Center for Infection Research, which supported the EBV project.

“As infectious mononucleosis is a known risk factor for multiple sclerosis, there is great hope that our vaccine may also reduce the incidence of this chronic neurodegenerative autoimmune disease,” Hammerschmidt added.

A member of the herpes virus family, EBV is estimated to infect up to 95% of the world’s population. Infections usually go unnoticed and without symptoms, particularly in children. But they can be serious in teenagers and adults who develop infectious mononucleosis, marked by fever, headache and swollen lymph nodes. After infection, EBV remains dormant inside B-cells, part of the immune system. It is believed to make these cells overactive, raising the risk of diseases like MS and certain B-cell cancers, including various lymphomas.

The vaccine candidate is based on virus-like particles that are similar to the virus but do not cause an infection. Specifically, these particles are composed of the virus shell and do not contain any viral genetic material. The shell contains more than 50 viral antigens, molecules that mimic EBV to trigger a highly specific immune response against the virus.

Preclinical testing showed that EBV-001 can elicit an immune response, EBViously reported. In animal models, the vaccine was able to induce a broad immune response reflecting human immunity against the virus.

“Based on our very favourable preclinical proof-of-concept data regarding the immunogenicity of the vaccine, we are confident that EBV-001 can effectively prevent the development of infectious mononucleosis and the often associated chronic fatigue syndrome,” said Axel Polack, MD, the company’s CEO.

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Here comes the sun – at last

With the days getting longer and the sun a little higher in the sky I have found myself reflecting more and more on the complicated and unresolved puzzle of sunshine and MS.

Since my diagnosis, 30 years ago in 1993, I have seen many theories about the causes of MS come and go. The lack of sunshine/ vitamin D theory is one of them. In fact, the theory never goes away or gets disproved or proved. One might say the sun never sets on this theory.

In 2003, ten years after I was diagnosed, I heard a Canadian neurologist Prof. George Ebers talk at a conference about a "latitude gradient" in MS. Basically, he was saying that the further away from the Equator you go and the less sunshine you get, the more cases of MS you find. This is accepted as being as true today as it was in 2003, although no one really knows the reason why.

It was clear to Ebers that something in sunlight either stopped MS developing or perhaps eased its symptoms. The question was: what was this thing? There were two possibilities. The first was vitamin D which is produced when the summer sun hits your skin. The other was sunlight itself, or to be more exact ultraviolet (UV) light, perhaps strong sunlight in the UVB band. It was argued this might be doing something to your body in addition to darkening or tanning your skin.

For the past 20 years there has been an ongoing debate about which of these is responsible for the "latitude gradient" in MS. Those who support lack of vitamin D as a cause of MS point to research such as that carried out in Northern Manitoba, Canada. This showed that native Americans living close to polar regions who had little exposure to strong sunshine but ate a lot of oily fish full of vitamin D had far less MS than you would expect for those living at this latitude. So, did vitamin D, in their fish-rich diet account for their low levels of MS? And does this mean a lack of vitamin D causes MS?

The debate is still going on and you can find academic papers which support and deny the link between vitamin D and MS. Interestingly, the 2022 NICE guidelines on MS say the following: "Do not offer vitamin D solely for the purpose of treating MS". This suggests that vitamin D or its lack isn't generally regarded as the answer to the question of what causes MS or how you can treat it.

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In view of this, attention naturally turns to sunlight itself. Sunlight also has other effects on the body as well as helping create vitamin D. One research study published in the journal Nutrients in 2018 concluded that the protective effect of sun exposure in MS is most likely brought about through "immunomodulatory mechanisms independent of vitamin D". What are these "immunomodulatory mechanisms"? One possibility is that UVB radiation in sunlight hitting the skin may lower levels of bad (LDL) cholesterol which is generally higher in the blood of MSers and may be involved in the disease process itself. One study looking into cholesterol and MS is the current MS-STAT 2 trial, where MSers take high doses of a cholesterol lowering statin (simvastatin) which it is believed may be anti-inflammatory and protect nerves from damage.

It is thought that higher levels of bad (LDL) cholesterol are linked to more severe MS and research into statins in an earlier study (MS-STAT1) found that the rate of brain atrophy (loss of neurons or "brain shrinkage") was reduced in patients taking simvastatin compared to those taking placebo.

So, is it the case that simvastatin lowers levels of bad (LDL) cholesterol and that levels of LDL cholesterol have also been shown to be reduced when you sit out in the sun? In other words, is sunshine more important because its leads to lower levels of bad cholesterol which lessens disease activity and that, although Vitamin D is produced as well, it doesn't really have much of an effect on your MS, hence the NICE guidelines?

One final possibility is that it's not your exposure to sunlight but your mother's exposure to sunlight in pregnancy that counts. Those supporting this theory point to another study carried out in 2016 also by George Ebers involving more than 21,000 people with MS which found that people diagnosed with MS in northern latitudes are more likely to have been born in May, and less likely than average to have been born in November.

What this study suggests is that low sunlight exposure during winter pregnancies, leads to low maternal vitamin D levels which in some way increase the risk of you developing MS later in your life. The role of sunlight in MS is certainly far more complex than originally thought. Sadly, like everything with MS, the answer to the question of what causes it or contributes to its cause remains as elusive as it was when George Ebers looked at it all those years ago. Time, sadly, doesn't appear to answer questions where MS is concerned, only generate new ones.

For further details see page 14

P5 picture thanks to Rajiv Bajaj - unsplash

New prog-MS drug set for Phase 3 trial after positive data

Treatment with the experimental therapy CNM-Au8 led to improvements in vision, cognitive dexterity and brain health for people with prog-MS according to data from the proof-of-concept VISIONARY-MS trial.

Bolstered by these positive results, Clene Nanomedicine is planning to launch a global Phase 3 clinical trial to further evaluate its effectiveness. Assuming the results are positive, Clene expects they could submit CNM-Au8 for regulatory approval.

The upcoming trial will test CNM-Au8's effect on disease progression that occurs independently of relapse activity (smouldering MS). The study will most likely enrol people with nonactive, progressive types of MS — people with primary progressive or secondary progressive disease who haven't had a relapse in at least a year.

CNM-Au8 is an oral liquid that contains a suspension of gold nanoparticles (very small particles) . It's designed to boost energy production in cells. Across a variety of different models, it has been shown that the application of CNM-Au8 to different neurons and different cultures always kept them alive longer, and it's that energetic assist that is believed helps them resist different diseases. Interestingly, the "energy boost" only appears to have an effect on diseased cells.

The upcoming Phase 3 trial will investigate whether CNM-Au8 can slow MS progression independent of relapse activity. After early clinical testing suggested CNM-Au8 could boost energy in nerve cells as designed, Clene launched the Phase 2 VISIONARY-MS trial (NCT03536559) as a proof-of-concept to test the therapy's effectiveness in MS.

Top-line results from the trial, announced last year, showed CNM-Au8 led to improvements over a placebo by an average of about 3 points in the LCLA test. This roughly corresponds to being able to read one additional line of letters in the LCLA test. Low-contrast letter acuity (LCLA) has emerged as a leading outcome measure to assess visual disability in MS research.

CNM-Au8 also outperformed a placebo on measures of visual evoked potential, which assess the strength and speed of the nerve signals that carry data from the eyes to the brain. Although patients usually have one eye more affected by optic neuropathy than the other, improvements were observed in both eyes. What that implies is that neurons that were previously damaged or not contributing to that information processing are now able to contribute. Other data from VISIONARY-MS suggested CNM-Au8 helped stabilize brain structures and preserve the integrity of the myelin sheath, the fatty covering around nerve fibres that's damaged in MS.

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Sativex eases MS spasticity and related symptoms in real-life study

Patients' use of Sativex , a mouth spray cannabinoid treatment, was associated with easing of spasticity and related symptoms for people with MS, according to a recent study carried out in Austria.

Most patients (87.5%) said they were at least partly satisfied with the therapy after three months of use, in contrast to about 30% who were satisfied with previous anti-spasticity treatments. These findings indicate Sativex, also known as nabiximols is an option for patients with MS spasticity who fail first-line oral anti-spasticity treatment, the researchers said. Their study, "non-interventional, prospective, observational study on spasticity-associated symptom control with nabiximols as add-on therapy in patients with multiple sclerosis spasticity in Austria," was published in the journal Brain and Behaviour.

While not approved for use in the U.S., Sativex is available in countries that include Canada and most of Europe. Its mechanisms aren't fully understood, but findings indicate that Sativex as an add-on therapy may ease spasticity for some MS patients in addition to associated symptoms like pain or sleep problems. Still, its potential benefits remain controversial, and it is not found to be effective in all studies.

Scientists in Austria conducted an observational study to evaluate the real-world effects of Sativex on up to 55 patients given the medication at MS centres across that country. They had been experiencing spasticity-related symptoms for an average 7.7 years, most commonly restricted mobility and greater muscle tone/stiffness. These symptoms were largely of moderate or severe intensity.

A majority had tried other spasticity medications and/or non-pharmaceutical interventions like physical therapy. At the time of starting with Sativex, 61.8% still were using non-pharmaceutical approaches, mainly physiotherapy, and 63.6% were using anti-spasticity medications, mainly baclofen or tizanidine.

It was found that easing in spasm frequency and severity with Sativex as an add-on treatment lowering spasm frequency by 68.2% — from a mean of 10.7 spasms a day to 3.4 spasms a day. Spasticity severity also was reported to ease by 39% at three months. Sativex's use eased several other spasticity-associated symptoms after one month of treatment, and these benefits were either stable or greater after three months. Specifically, the medication resulted in a 47.2% reduction in overall sleep impairment scores after three months, and fatigue and pain due to spasticity declined by 26.4% and 40.4%, respectively.

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Ublituximab suggested as treatment for active SPMS

An arm of the European Medicines Agency (EMA) has said it favours the approval of Briumvi (ublituximab) to treat adults with active, secondary progressive MS across the European Union (EU).

The Committee for Medicinal Products for Human Use (CHMP) view is based on data from twin Phase 3 trials, ULTIMATE I (NCT03277261) and ULTIMATE II (NCT03277248), that found the therapy significantly prevented the formation of new lesions. Briumvi is given in an hourlong infusion every six months, after starting doses.

“The positive recommendation … takes us one step closer to delivering Briumvi to patients and healthcare providers in Europe,” Michael Weiss, chairman and CEO of TG Therapeutics, Briumvi’s developer, said in a company news release. “We look forward to hearing a decision on the marketing authorization application by the European Commission in the coming months.”

Briumvi is an antibody-based medication that works by blocking the CD20 protein found on the surface of B-cells. These immune cells become overactive in MS and target the myelin sheath in the brain and spinal cord, causing inflammation and damage.

By blocking CD20, Briumvi lowers the number of B-cells in the bloodstream, which is expected to help reduce relapses and slow disease progression.

It works much like the other approved anti-CD20 antibody treatments, Ocrevus (ocrelizumab) and Kesimpta (ofatumumab). It is administered as a one-hour intravenous (into-the-vein) infusion every six months, after the two first doses.

The U.S. Food and Drug Administration approved Briumvi in December to treat adults with relapsing forms of MS: clinically isolated syndrome, relapsing-remitting MS, and active secondary progressive MS.

Briumvi’s most common side effects are infusion-related reactions and respiratory tract infections. Other reported side effects include herpes virus-associated infections, pain, insomnia, and fatigue.

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Is the 'holy grail' of MS research about to be found?

Remyelination has often been described as "the holy grail" of MS research. The holy grail is an apt metaphor because remyelination is the ultimate prize for any MS researcher. It is ardently sought after but seemingly aways out of reach, like the holy grail itself.

That said, the past two decades have seen massive steps taken in understanding and reducing the immune attacks that cause inflammation and damage to the myelin coating around nerve cells in the brain and spinal cord, and this research has led to a better understanding of myelin and drugs that might protect it.

But, and this is a big but, disease modifying drugs can only reduce damage to myelin, they can't repair MS damage that has already happened. The human body can replace damaged myelin in healthy individuals but in MS demyelination is far bigger and the process is impaired in MS so that remyelination isn't really possible, although things may be changing with a couple of new treatments on the horizon.(See below)

These new treatments, if successful, might offer the possibility of remyelination in MS and would offer the potential to delay, prevent or reverse disability The treatments are based on research at the University of Cambridge and nearby Addenbrookes Hospital . If successful this research and ensuing treatments could be the beginning of a whole new chapter in MS research and, dare I say, a possible cure.

Before we go any further, it is perhaps a good idea to understand just how and why the opposite process - demyelination - happens and what goes wrong in MS. Basically, in the normal (non-MS) brain and spinal cord, cells called oligodendrocytes produce myelin which they wrap around nerve fibres or axons. When it comes to MS the immune system attacks the protective sheath (myelin) that covers these axons or nerve fibres and causes communication problems between the brain and the rest of the body. Eventually, the disease can cause permanent damage or deterioration of the nerve fibres. As all people with secondary progressive MS (like me) know only too well, **Continued on page 11**

Continued from page 10 the early relapsing remitting phase of the illness quickly gives way to a progressive phase where the damage just goes on and gets worse over time.

However, in terms of looking forward and looking at solutions to problems rather than the problem itself, the focus of research so far has traditionally been to try and get the cells that wrap the myelin round exons called oligodendrocytes, in the shape of immature oligodendrocytes (oligodendrocyte progenitor cells, OPCs) to mature and produce new myelin. But these efforts have a huge hill to climb – metaphorically speaking .

To begin with, the debris left over from damage to myelin during numerous MS attacks inhibits remyelination so the clearance of debris by cells called macrophages and the other cell types found in brain tissue, such as astrocytes and microglia, fails probably because the workload is just too great for these young immature oligodendrocyte progenitor cells. It is also known that remyelination is less effective with age and disease duration.

However, all is not lost for older patients or those of us with secondary progressive MS who have had MS for decades not years. Yes, it may be possible to counteract some of the consequences of these problems inherent in remyelinating those like me who are older and have had the disease longer. A recent study reported that in a lab model of demyelination, alternate day fasting or treatment with metformin, a type 2 diabetes drug which can mimic the effects of calorie restriction, can recalibrate ageing oligodendrocytes rather than immature oligodendrocytes (oligodendrocyte progenitor cells, OPCs) to remyelinate axons.

Another drug called clemastine – an anti-histamine often prescribed for hay fever – has also been shown to have promise as a remyelinating agent. Now researchers at Cambridge University are launching a new phase 2a trial testing whether metformin and clemastine can together help the brain regenerate myelin in people with MS. Participants in the trial will be given either a combination of metformin and clemastine or a placebo (dummy drug) alongside their current disease modifying treatment. Researchers will be testing to see whether this combination is able to promote myelin repair in people with MS.

This trial team are recruiting 50 people with MS who are taking a disease modifying therapy (DMT). This research has been held up because of COVID but now in 2023 participants are being recruited and the trial is being organised by doctors at Addenbrooke's Hospital in Cambridge. But other people living within two hours of Cambridge may also be able to take part. If, and this is a big if, assuming the trial is successful then a treatment that puts right the things that have gone wrong may be just a few years away, not too many we all hope. We will all watch the outcome of the trial with great interest, and great hope too.

For details of all news and features go to page 14 **Photo p10 James Coleman-unsplash**

News

Bacterial strain in SPMS patients supports link with progression

A certain bacterial species is enriched in the gut of people with secondary progressive multiple sclerosis (SPMS) and may promote neuroinflammation and drive disability progression, a new study has found.

It's believed this bacterial species promotes inflammation through the activity of its flagella, the hairlike projections that help bacteria to move around, adhere to, and invade target cells. Due to ongoing patent protection, researchers would not disclose the name of the bacteria, calling it "bacteria X."

"The selective elimination of pathogenic bacteria, including this bacteria X, might lead to innovate therapies for SPMS patients," Daiki Takewaki, MD, said in a presentation "Identification of Pathogenic Gut Bacteria Underlying Multiple Sclerosis Progression."

A growing body of evidence has linked the gut microbiome to immune dysregulation in MS. Abnormalities in the balance of these microbes is commonly observed in MS patients, and changes in gut bacteria are linked to earlier and more severe disease in animal models of MS. Takewaki and his team sought to identify the specific gut bacteria linked with MS progression, and the likely mechanisms that drive neuroinflammation.

Using bacterial DNA obtained in faecal samples from 15 SPMS patients, the researchers found 12 bacterial species that were particularly enriched in the SPMS samples. Of them, bacteria X levels showed the strongest correlation with patients' disability levels. This observation "confirms that bacteria X is highly enriched in the progressive stage of MS," Takewaki said.

On further examination, two different strains, or subtypes, of bacteria X were identified. While strain A1 levels were similar among healthy people and RRMS and SPMS patients, strain B1 was significantly more abundant in SPMS samples compared with the other two groups. Using a mouse MS model it was found that SPMS-derived bacteria also led to a relative increase of Th17 cells, a type of pro-inflammatory immune cell considered a main driver of inflammation in MS, in the large intestine and brain.

Notably, certain types of flagella in bacteria are thought to activate receptors called toll-like receptor 5 (TLR5). This prompts the release of the pro-inflammatory molecule interleukin-6 (IL-6) from certain immune cells, which in turn activate Th17 cells. "We confirmed that bacteria X has the capacity of inducing Th17 cells in the gut and subsequently exacerbate neuronal inflammation," Takewaki said, adding that this is "possibly associated with disease progression from RRMS to SPMS."

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News

Gut bacteria may explain disease activity as MS progresses

A specific toxin-producing gut bacteria may be responsible for ongoing disease activity as MS progresses, according to a new study led by a team of researchers at New York-Presbyterian/ Weill Cornell Medical Centre.

The work, published in The Journal of Clinical Investigation, identifies epsilon toxin-producing Clostridium perfringens in unusually high abundance within the gut microbiome of people with prog-MS. The study goes on to show that in a preclinical model of MS, epsilon toxin opens the blood vessels of the brain allowing inflammatory cells to gain access to the central nervous system and cause demyelination characteristic of MS.

"There are many mysteries to MS," said co-senior author Dr. Timothy Vartanian, a professor of neuroscience in the Feil Family Brain and Mind Research Institute at Weill Cornell Medicine. Clostridium perfringens and epsilon toxin may explain many of these mysteries." Perhaps most remarkably, epsilon toxin specifically targets brain blood vessels and myelin, providing a clear mechanism of its action.

In the current study, Dr. Yinghua Ma, an assistant professor of research in neuroscience in the institute's Vartanian lab, led work, showing that more sensitive techniques readily detected these strains in the MS gut microbiome. Using highly sensitive DNA detection techniques, Dr. Ma found that people with MS are more likely to carry epsilon toxin-producing C. perfringens in their small intestines than healthy controls. Having established this, the investigators tested whether the toxin alone could cause MS. For that, they turned to a standard mouse model of MS in which animals are predisposed to autoimmunity, but MS-like disease only occurs if mice are also given pertussis toxin. Dr. Ma substituted epsilon toxin for pertussis toxin and animals developed a disease that more closely resembled MS compared to previous models.

"The finding that epsilon toxin can replace pertussis toxin in a mouse model of MS is very exciting," said co-author Dr. Gregory F. Sonnenberg, the Henry R. Erle, M.D.-Roberts Family Associate Professor of Medicine and a member of the Jill Roberts Institute for Research in Inflammatory Bowel Disease at Weill Cornell Medicine. "It not only advances a more relevant model to study MS, but critically defines a new microbial-derived determinant that provokes a breakdown of immune privilege in the central nervous system to initiate demyelinating disease."

"A treatment that neutralizes epsilon toxin may halt our patients' new disease activity, far more effectively than current treatment modalities that suppress or modulate the immune system." said Dr. Vartanian, who is also chief of the Multiple Sclerosis and Neuro-Immunology Division in the Department of Neurology at Weill Cornell Medicine and a neurologist at NewYork-Presbyterian/Weill Cornell Medical Center. **For details of all news stories see page 14**

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