My First Medical Writing

SECTION EDITOR

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Editorial

For this release of *My First Medical Writing*, we are delighted to showcase Geoff Churchill (right), a New Zealand-born science professional currently based in France. With an impressive background spanning molecular biology, ecology, biodiversity conservation, and scientific communications, Geoff brings a wealth of diverse expertise to the field. He holds an MSc in Biology, backed by hands-on experience in genetics and physiology research laboratories. Furthermore, Geoff spent more than a decade driving regulatory, scientific, project management, and communications initiatives within the environmental and biodiversity sector. He is now transitioning into regulatory medical writing, actively expanding his professional portfolio and pursuing advanced studies in the discipline. Read on to discover the unique insights and expertise this talented medical writer has to offer.

Evguenia

Nobody wants multiple sclerosis: Why don't we stop it?

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Abstract

A world without multiple sclerosis (MS) might be attainable. Recent findings have linked the very common Epstein-Barr virus (EBV) as being the trigger to the development of MS. A focus of research in this field has turned to trials of an EBV vaccine as a means of preventing MS. However, numerous challenges must be overcome before realising this worthy goal.

A little bit about multiple sclerosis

Multiple sclerosis (MS) causes the body to begin attacking its own nervous system, leading to a progressive decline in the brain's ability to control the body. This autoimmune disease gradually leads to loss of mobility, vision, balance, sensation, and cognition. This is caused by the immune system attacking the myelin sheath proteins that surround and protect nerves, leading to scarring called sclerosis. In cases of MS, there are many of these scleroses, thus the name multiple sclerosis. These result in nerve signals slowing down and eventually being blocked completely.

MS is generally considered a disease of young adults, with onset often occurring between 20 and 40 years of age. The cause of the body suddenly attacking its own nerve cells has long been unclear. Until recently, there was little evidence for why approximately 3 million people worldwide have this disease. Over the last few years, a significant body of literature has emerged linking one of the world's most common human viruses to triggering this condition: the Epstein-Barr virus (EBV).[1]

A little bit about Epstein-Barr virus

You, the reader, are probably infected with EBV, which infects approximately 90% of the human population.[1] This herpes virus is transmitted by saliva and is often asymptomatic. Occasionally, it can cause infectious mononucleosis (glandular fever), resulting in fever, fatigue, and other flu-like symptoms. Generally, it becomes inactive after the initial infection and causes no further problems in the host.

You, the reader, are probably infected with EBV, which infects approximately 90% of the human population

It infects the immune system's B cells for the host's lifetime and, in the vast majority of cases, is effectively inert (latent) after the infection. So how can this extremely common virus cause such a debilitating disease in a tiny percentage of those infected?

What's the link?

Evidence that EBV is involved as the principal trigger of MS has been mounting since 2022. Multiple studies, including more than 10 million participants, have shown that EBV infection is a near-universal precursor to MS. These studies have included people from many different populations, including men, women, different age groups, and different ethnicities.[2,3]

The results suggest an approximate 30-fold increased risk of developing MS following EBV infection. The vast majority of infections occur in childhood or adolescence, whereas MS is usually diagnosed in people 20–40 years of age, suggesting either decades of harmless presence of the virus or a slow accumulation of damage, finally resulting in MS.

While there is no agreed biomarker for pre-MS, markers of neurodegeneration have been shown to increase only after EBV infection in

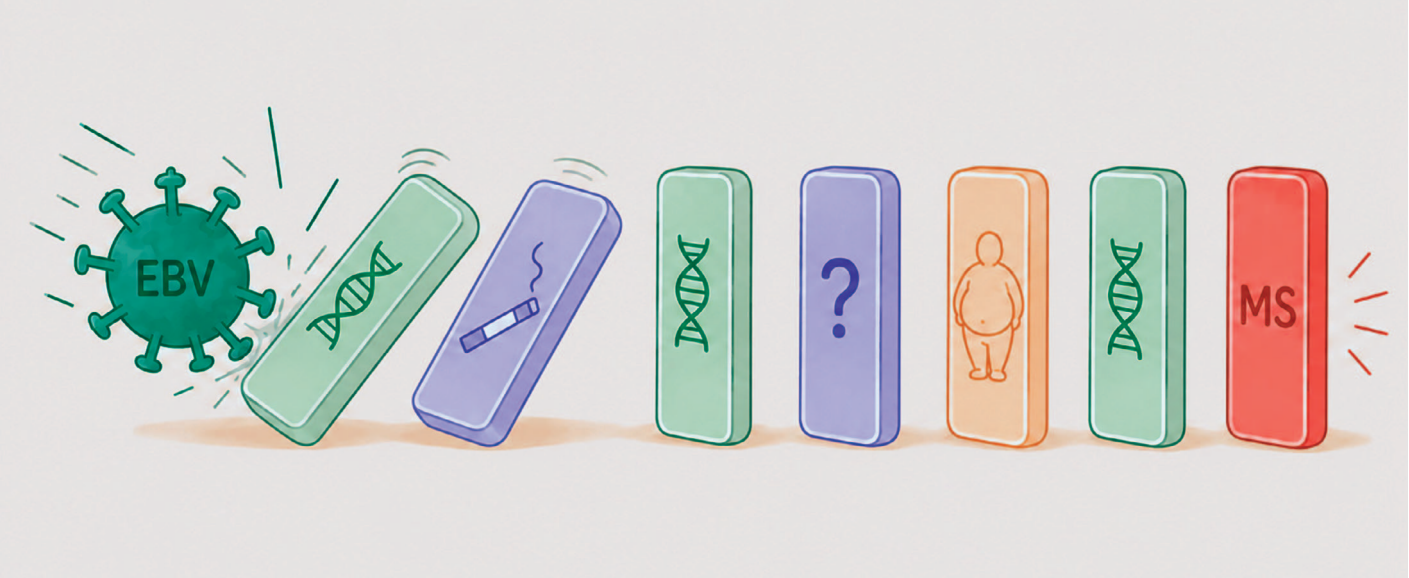

Image: ChatGPT

individuals who later develop MS. This suggests that EBV infection may initiate a gradual neurodegenerative process that eventually results in MS.[2,4]

How could this happen?

How can a viral infection of immune cells lead to the immune system attacking neurons in some people?

EBV establishes lifelong infections in B cells. This critical cell type of the immune system produces antibodies. Two mechanisms of EBV-triggered MS have been suggested. One is molecular mimicry. In some cases, antibodies are produced against a specific EBV protein. To the immune system, this protein resembles certain brain proteins, both of which are attacked. These proteins are involved in the maintenance of myelin, whose deterioration is a hallmark of MS.[5]

Another proposed mechanism is immune dysregulation. EBV-infected B cells may proliferate abnormally, cross the blood–brain barrier, and contribute to chronic inflammation in the central nervous system, a key feature of MS.[5] These cascades from EBV infection to developing MS are plausible mechanisms for the role that EBV plays in this disease.

The search for the genetic factor in MS

EBV infects approximately 90% of the human population, whereas there are fewer than 3 million cases of MS globally. So why do only a very small proportion of infections result in neurodegeneration? While other environmental factors, such as smoking and obesity, also increase the risk of developing MS, there must be a genetic component to this disease.

Genome-wide association studies have identified hundreds of genetic variants associated with MS, primarily in genes involved in immune function.[6] These studies involve searching the genomes of a large cohort of people who have MS and finding their shared genetic mutations. However, all of these identified genetic mutations do not appear in every single case of MS. Instead, certain mutations increase the risk of developing MS more or less than others. The current estimates suggest genetics contributes approximately 20% of the overall risk.[7]

Importantly, genetic risk alone is insufficient for prediction. Many individuals with high genetic risk do not develop MS, while others with lower risk do. Recently, studies have introduced environmental factors into these risk analyses, including EBV antibodies present in the body. These have resulted in greater accuracy for MS prediction, but they remain far from clinical precision.[8] The influence of environmental factors is more significant than the genetic component, above all else – EBV infection.

Is an EBV vaccine even possible?

Developing a vaccine for EBV represents a pathway to preventing MS. By blocking the initial infection, the downstream autoimmune cascade to neuron damage may be avoided.

Early vaccine candidates targeting a single viral protein (gp350) reduced the incidence of infectious mononucleosis but did not prevent infection.[9] Current approaches include mRNA vaccines targeting multiple viral proteins, aiming to induce broader immune protection.[10]

However, significant challenges remain. There are multiple features of the virus that contribute to these challenges. EBV infects both B cells and epithelial cells via different entry mechanisms, meaning an effective vaccine must block multiple infection pathways. In addition, the virus, post-infection, quickly becomes dormant within the cell. This makes it very difficult to eradicate once established. This, combined with the general early age of infection, requires that the vaccine be administered either to children or adolescents prior to infection.[11]

Image: ChatGPT

Image: Freepik

Those who develop MS have generally been infected with EBV for many years or decades. This adds a distinct challenge to clinical trials for a preventative EBV vaccine. Either huge cohorts must be retained in trials for decades to ensure that MS does not develop after the vaccine, or a reliable surrogate endpoint could be substituted. As mentioned above, current genetic markers lack the accuracy to be an effective biomarker. Identifying an early biomarker that reliably predicts future MS risk would significantly accelerate vaccine development and evaluation.[11]

Summary

EBV infection does not equal MS; however, it appears to be a necessary trigger in starting the disease's development in a small proportion of genetically susceptible people. This recent finding has accelerated efforts to develop an EBV vaccine. However, things are never as easy as desired, and many challenges stand in the way of making this a quick reality.

Focusing research on finding an alternative, earlier endpoint as a substitute for MS diagnosis will allow clinical trials of a vaccine to progress much more efficiently. This could lead to a future with MS being categorised as an extremely rare disease.

Disclosures and conflicts of interest

The author declares no conflicts of interest. AI tools were used for editing support and image generation.

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