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Behind the Sheath: Shining a Spotlight on Multiple Sclerosis

TEAM #32
Health and Wellness

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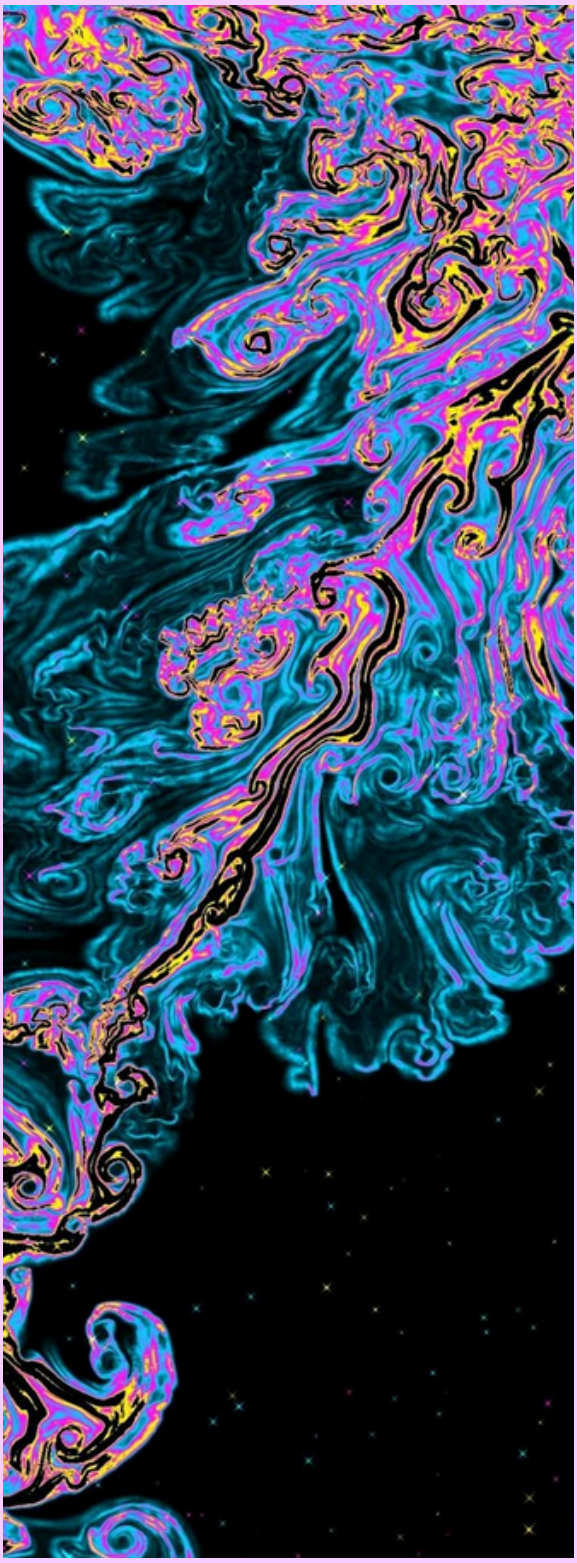
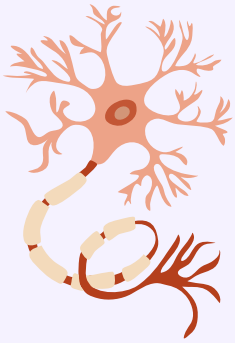


LETTER OF INTRODUCTION

Dearest Reader,

What if one morning you woke up and your legs didn't feel like your own? What if the messages you tried sending your brain got lost along the way, causing your vision to blur or words to slip just out of reach[1]? For millions of people living with **multiple sclerosis (MS)**, this is more than a hypothetical: it is reality.



In this special edition of *The Neural Network*, we delve into MS: a disease as complex as the nervous system it affects. We explore the science behind it, its unpredictable nature, and the courageous individuals who live with it every day.

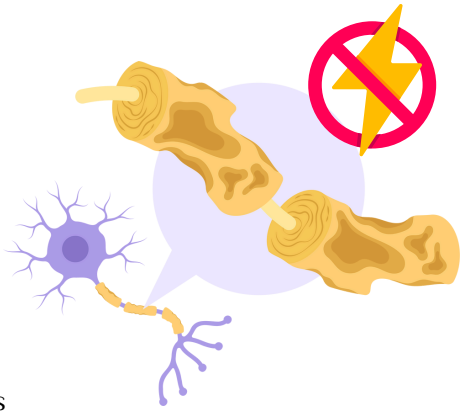
Through our Celebrity Spotlight, you will meet **Christina Applegate**, who has brought visibility to MS with her vulnerability and strength. Throughout this issue, you will also uncover the secrets scientists are unlocking, from viral triggers to immune misfires. Join us as we uncover the silent battles, celebrate resilience, and try bringing science out of the textbook and into lived experience.

With curiosity and compassion,

The Editorial Team

ETIOLOGY
UNDERSTANDING THE DISEASE

Multiple sclerosis (MS) is an **autoimmune disease of the central nervous system** in which the body's immune system mistakenly attacks the **protective myelin sheath** around nerve fibers in the brain, spinal cord, and optic nerves[1-3].



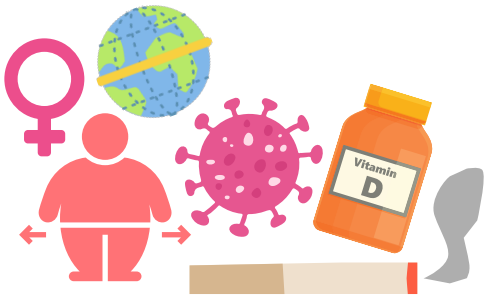
This damage **disrupts nerve signal transmission** and leads to the characteristic neurological symptoms and lesions (scars or "scleroses") of MS[2-4].

The exact cause of MS remains unknown, but current science points to a **multifactorial** etiology; a combination of genetic predisposition and environmental triggers that provoke an abnormal immune response.

Genetic Factors

- The allele *HLA-DRB1*15:01* is the strongest genetic risk factor, increasing risk 3x.
- Over 200 genetic loci have been linked to MS, mostly related to immune function[3,5].

CONCLUSION: Genetics prime the immune system for overactivation but are not sufficient to cause MS on their own.

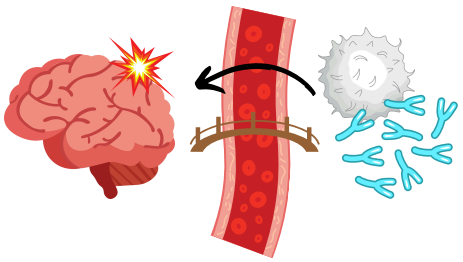


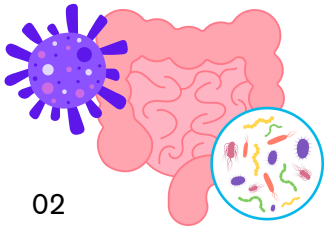
Environmental and Lifestyle Triggers

- Epstein-Barr Virus (EBV) is strongly associated with MS, and nearly all MS patients have had EBV[3-5].
- Vitamin D deficiency due to low sun exposure is linked to increased risk[2].
- Smoking and adolescent obesity are modifiable risk factors[2].
- Women are 3x more likely to develop MS than men[7].
- Higher MS rates can be found further from the equator[2].

Immune System Dysfunction

- In MS, T-cells and B-cells cross the blood-brain barrier and attack the CNS.
- T-cells release cytokines and recruit macrophages, contributing to myelin damage.
- B-cells produce antibodies against myelin and activate T cells[5-6].



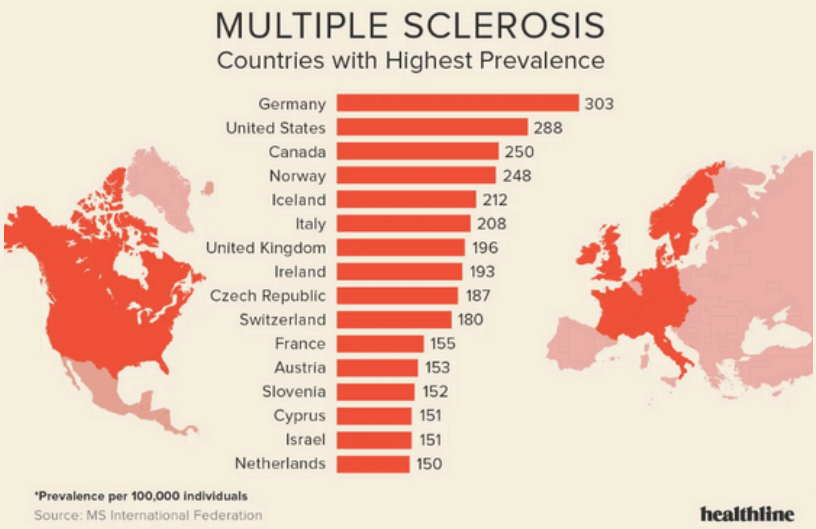


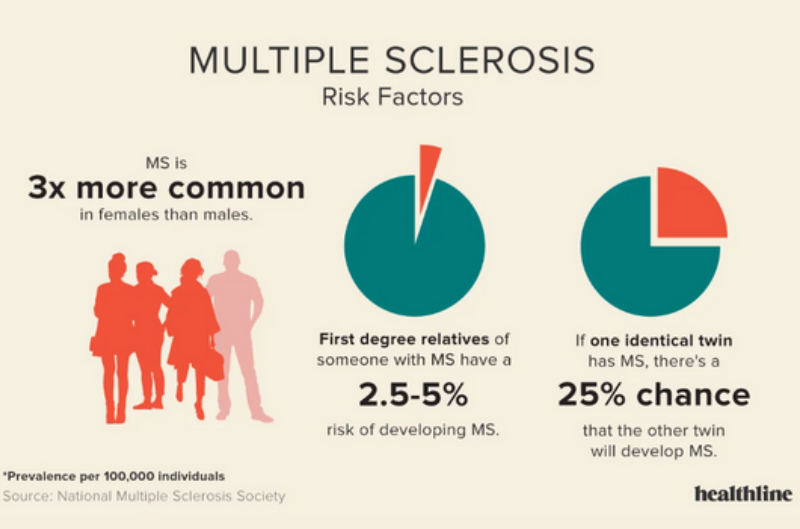
Emerging Hypotheses

Is EBV a primary and necessary trigger for MS?
Ongoing research to determine if EBV is the trigger[4,6].
Can Your Gut Bacteria Influence Multiple Sclerosis?
Certain bacteria may influence immune response or disrupt the blood-brain barrier[8,9].

BY THE NUMBERS

MS affects around 2.9 million people worldwide





- People of Northern European descent have highest risk of developing MS[10].
- Lowest risk appears to be among people of Native American, African, and Asian descent[10].
- Children can get MS, with around 30,000 children diagnosed worldwide[10].
- Sex is a risk factor, thought to be due to hormonal influence[10,11].
- MS is not an inherited disorder, however, first degree relatives of those with MS have slightly increased risk of developing MS[10,11].
- Children of parents with MS may have a 10 to 20 times greater chance of developing the condition than the general population[10,11].
- In the case of identical twins, if one twin has MS, there's a 20% to 30% chance that the other twin will also have the disease[10,11].

Types of MS

Clinically Isolated Syndrome (CIS)	• Considered to be **one course** of MS, and **may or may not** progress to MS. • High risk of developing MS if brain lesions are seen on MRI scan (60-80% chance of progression)[10].
Relapsing-Remitting MS (RRMS)	• Characterized by **clearly defined relapses** of increased disease activity and worsening symptoms followed by **remissions**. • Symptoms may improve or disappear during remission. • 85% of people are initially diagnosed with RRMS[10].
Secondary Progressive MS (SPMS)	• Follows an initial RRMS diagnosis 50% of the time within a decade of diagnosis. • Disability can gradually increase with disease progression, and there may or may not be relapses or MRI scan changes[10].
Primary Progressive MS (PPMS)	• Diagnosed in 15-20% of people with MS • Patients experience steady progression of MS with no clear relapses/remissions[10].

CELEBRITY SPOTLIGHT

Christina Applegate

Fighting MS in the Spotlight - with Strength, Humour, and Honesty

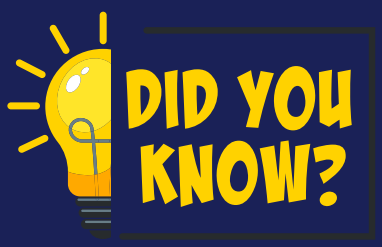
Best known as an actress, producer and advocate, Christina Applegate is best known for her unforgettable roles in *Married... with Children*, *Anchorman*, and Netflix's *Dead to Me*. But in 2021, the Emmy-winning performer took on a new and uninvited role: someone living with MS. Due to advice from her friend and a subtle symptom, she chose to get tested[13].

"This is the first time anyone's going to see me the way I am[12]."

Her first symptom: a tingling sensation in her hands and feet

"I don't say that I have a disease - I say I'm living with a disease[12]."

Since going public with her diagnosis, Applegate has used her platform to raise awareness about the challenges of MS. From openly discussing her fatigue and balance struggles while filming to shamelessly using a cane at public appearances, she has approached this challenge with vulnerability, resilience, and grace[13].



- **Applegate was diagnosed with MS at age 49.**
- **MS affects nearly 3 million people globally.**
- **Fatigue, numbness, & difficulty walking are some of the most common early symptoms.**
- **Applegate previously survived breast cancer before MS[13].**

Christina's journey is a powerful reminder that **MS does not discriminate.** It can affect anyone, even those in the spotlight. She has become a symbol of strength for others with MS, proving that awareness and advocacy can exist with adversity. Her symptoms, like numbness and muscle weakness, reflect a pathology that will later be discussed, so please continue reading to learn all about multiple sclerosis.

PATHOGENESIS

CELLULAR EVENTS LEADING TO DEMYELINATION

Multiple Sclerosis (MS) is a multifactorial autoimmune disease. Factors such as genetics, environment, vitamin deficiencies, and smoking are common contributors to the pathogenesis and progression of MS[14]. These factors facilitate demyelination of axons and destruction of oligodendrocytes found in the Central Nervous System (CNS)[14]. The immune response to the body's own cells is initiated by a a storm of cytokines[14,15].

1 INITIAL DAMAGE

ENVIRONMENTAL FACTORS

Viruses and bacteria can weaken immune regulation in host. Foreign agents may over-activate immune responses[14].

GENETICS

Variability in protein expression related to immune modulation may up or down regulate responses[14].

SMOKING

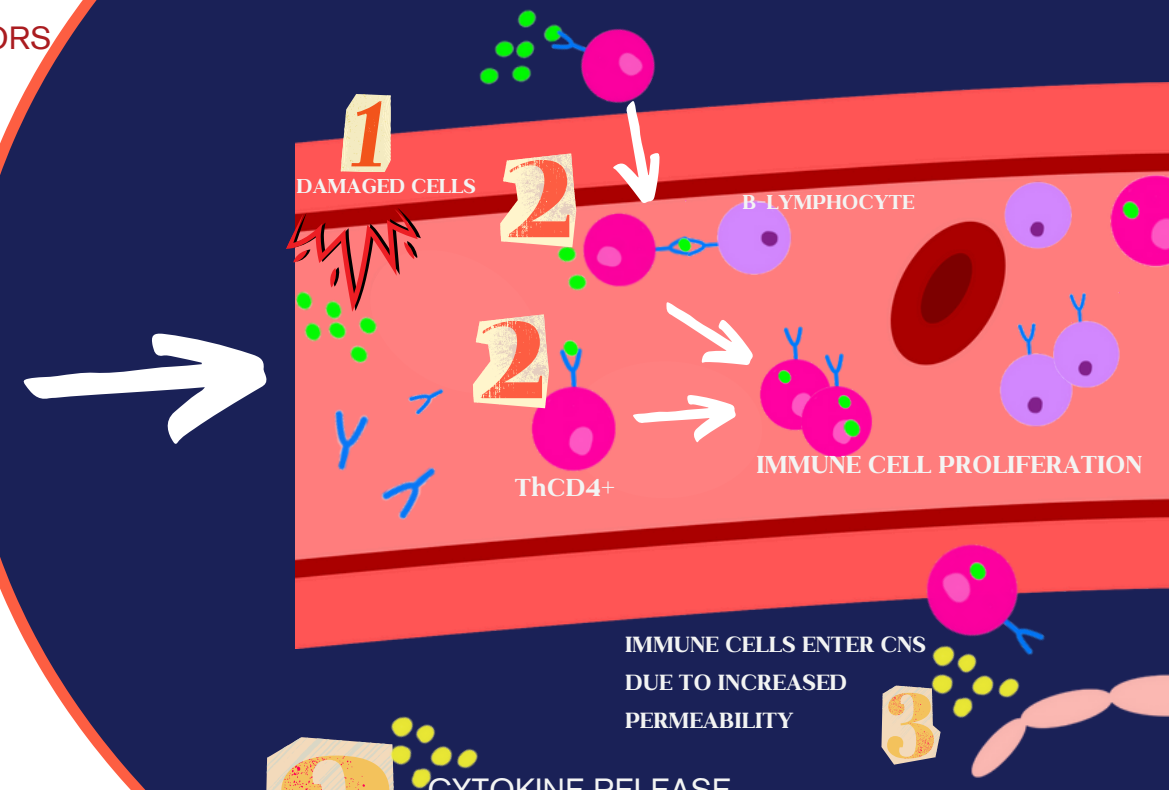
Oxidative stress and accumulation of damaging by-products increase risk of damaging cells and tissues including oligodendrocytes. Changes expression of self-antigens or antigen receptors[14].

VITAMIN DEFICIENCIES

Vitamin D is involved in immune regulation and expression of immunity. Vitamin B12 is a factor required for myelin sheath production. Decreased myelin production may expose neurons to damaging agents[14].

2 IMMUNE CELL ACTIVATION

Immune cells respond to endogenous and exogenous antigens as they bind to receptors expressed on the cell's surface[14]. Antigens activate innate immunity mediating T-Cells. T-Cell secretions and antigen presenting capabilities recruit various other immune cells including adaptive immunity mediating cells such as B-cells[14,16]. Activated cells proliferate to magnify the response.

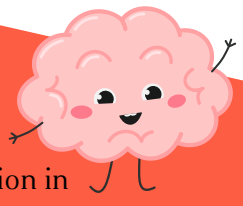


3 CYTOKINE RELEASE

The damaging cytokines TNF-α and Lymphotoxin are released from activated cells in the CNS[14,15].

Cytokines initiate inflammatory responses such as increasing vessel permeability to inflammatory and immune cells so they can reach their target tissues in the CNS[14,15].

TRUE OR FALSE:



Immune cell activation in Multiple Sclerosis must occur in the CNS.

Flip to the next page to find out!

WHAT ARE CYTOKINES?

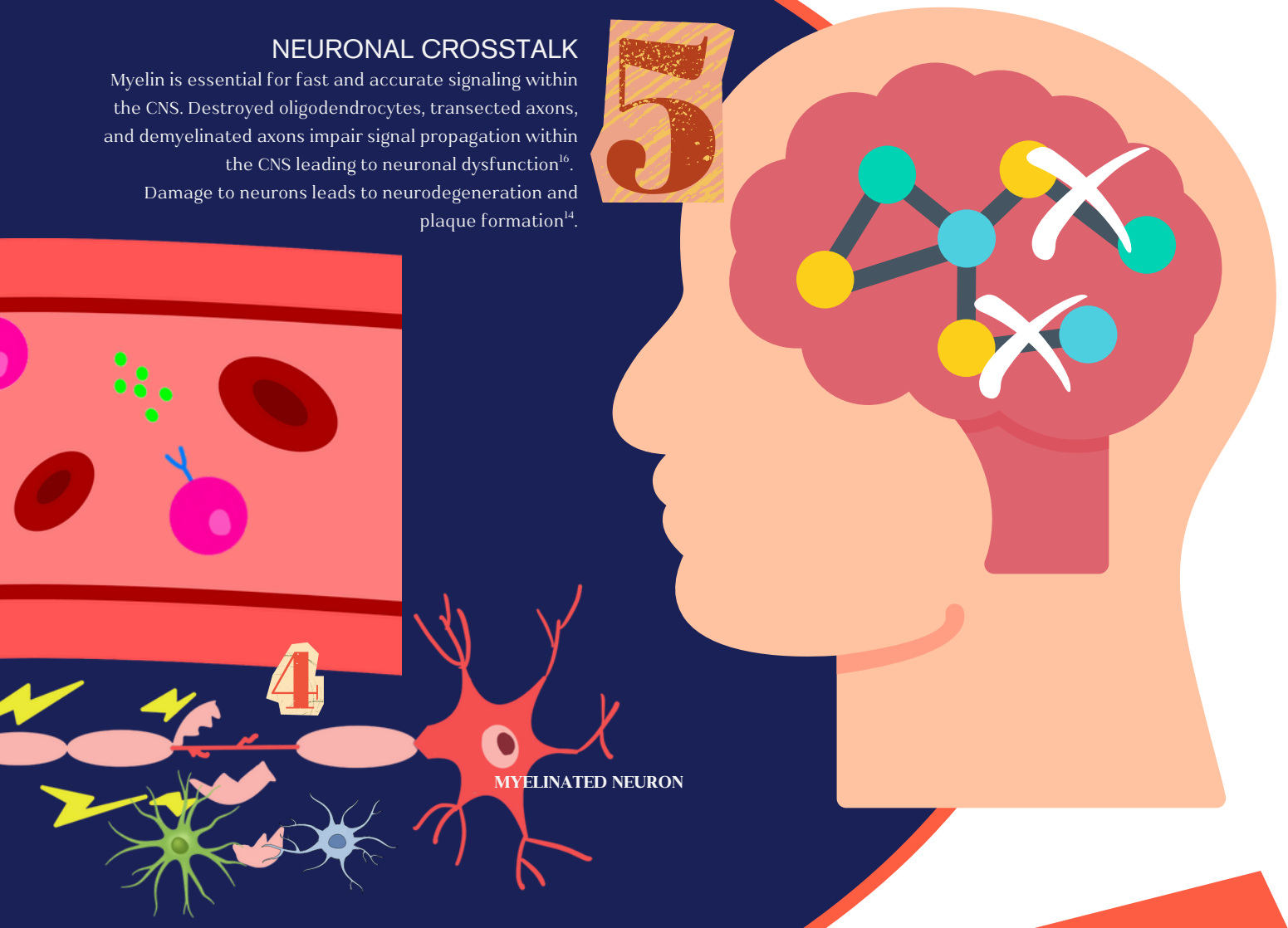
Cytokines are proteins, there are numerous steps to producing proteins. The secretion of proteins from inflammation-mediating cells may take time. Cytokines control inflammation throughout the body by acting as signalling molecules. Various cell types release cytokines of produce local or systemic effects[14,15].

6 NEURONAL DYSFUNCTION

Autonomic and sensorimotor defects
Visual Disturbances
Ataxia
Fatigue
Difficulties Thinking
Emotional Problems[14]

5 NEURONAL CROSSTALK

Myelin is essential for fast and accurate signaling within the CNS. Destroyed oligodendrocytes, transected axons, and demyelinated axons impair signal propagation within the CNS leading to neuronal dysfunction[16].
Damage to neurons leads to neurodegeneration and plaque formation[14].



4 DESTRUCTION OF MYELIN

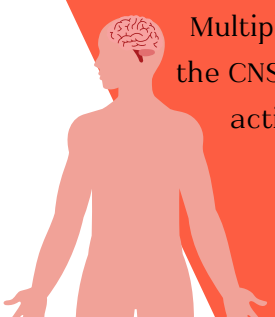
MYELINATED NEURON

Cytokines can modulate inflammation by activating microglia and astrocytes[15]. Microglia can phagocytose myelin and release more antigens that stimulate local immune cell activation [14].
Demyelination by these cells leads to interruptions in signaling[14,16].

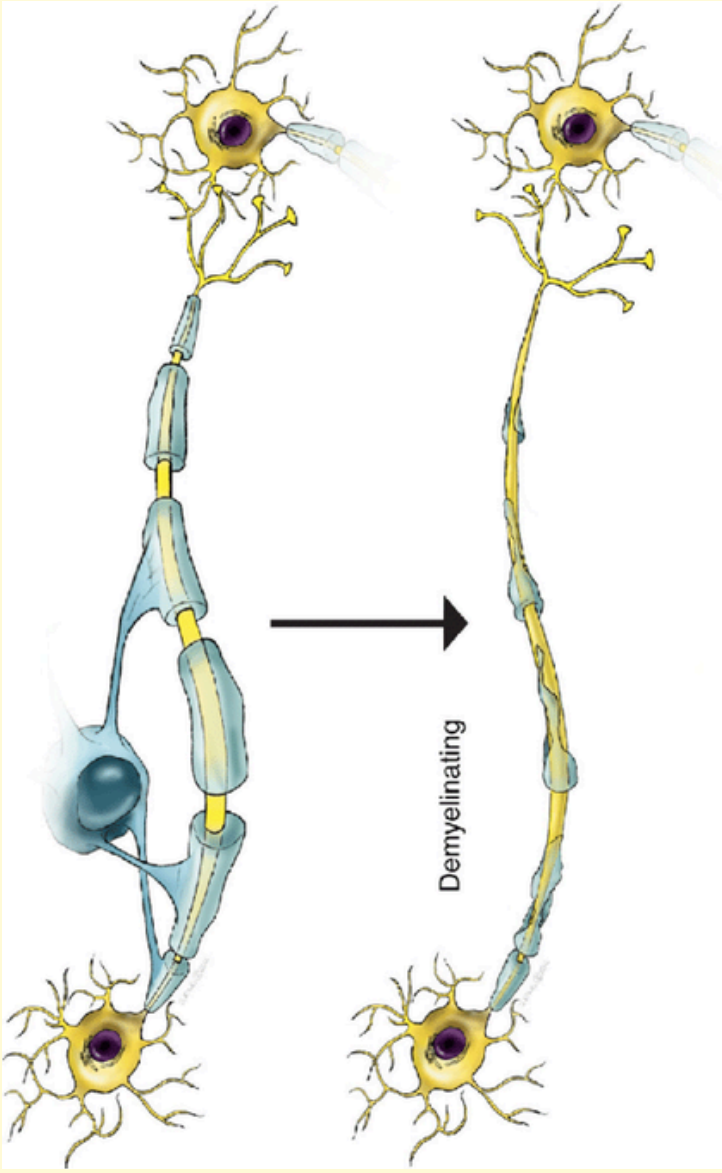
TRUE OR FALSE FROM PAGE 5:

FALSE

Immune cell activation in Multiple Sclerosis can occur in the CNS or in the periphery and activated immune cells can spread to the CNS.[14]



DEMYELINATION STEP-BY-STEP



1) Damage to CNS or peripheral tissues generates antigens. Antigens are picked up by antigen presenting cells (APCs) called dendritic cells[14].

2) APCs present antigens picked up at the site of damage to tissue-resident or circulating CD4+ T-Helper cells[14]. The binding of antigens stimulates innate and adaptive immune responses.

3) The binding of antigens onto APC's toll-like receptors initiates the release of cytokines called interleukins (IL)[14]. Specifically, IL-12, IL-23, and IL-4 are released.

4) Cytokines IL-12, IL-23, and IL-4 signal to T-Helped cells to differentiate into different classes of T-cells that have that ability to release unique cytokines[14,15].
This includes: Th1, Th2, or Th17 cells.

5) Th1 cells produce pro-inflammatory interferon gamma (IFNγ) and tumor necrosis factor alpha (TNF-α) which suppress Th2 differentiation[14].
Th2 cells produce anti-inflammatory cytokines IL-4 and IL-13 which promote macrophage activity.
Th17 cells promote inflammation through various cytokines.

6) Pro-inflammatory mediators increase blood-brain-barrier permeability allowing more cells to permeate it. Activated immune cells such as T-cells, B-cells, and macrophages enter the CNS[14].

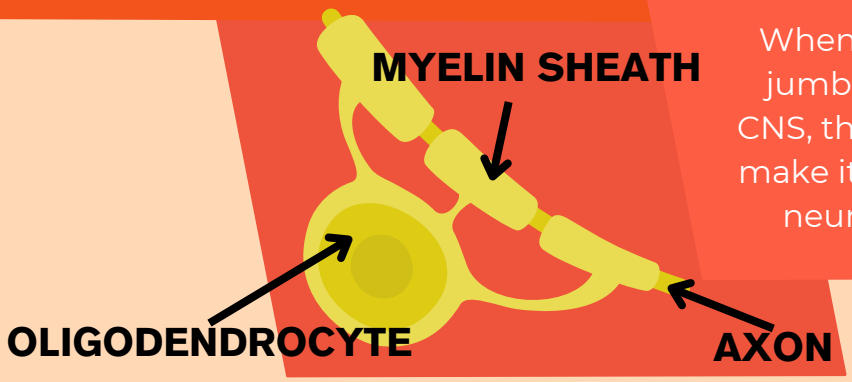
7) Immune cells recognize self-antigens on oligodendrocytes and myelin sheaths as foreign[14,16]. This is known as an auto-immune attack. Macrophages phagocytose myelin, cytotoxic mediators damage axons and myelin, nerve conduction is impaired[14].

DEMYELINATION: WHAT'S THE BIG DEAL?

Myelin is made up of lipids and proteins, it is crucial for signal transmission. A myelin sheath acts like insulation on a wire: it prevents unwanted signals from transmitting to surroundings and it allows signals to travel faster down the axon[16].

In the CNS, oligodendrocytes are responsible for myelinating neurons. Myelinated neurons make up the white mater of the brain. White mater is found within deep cortical structures. Neurons of various regions of the brain must be able to interpret incoming information from the peripheral receptors, communicate with other neurons to coordinate responses and information processing, and perform higher-order functions[14].

When myelin is lost, signals tend to get jumbled as they travel throughout the CNS, they travel slower, or they may never make it to their destination[14]. This leads to neurological deficits and symptoms.



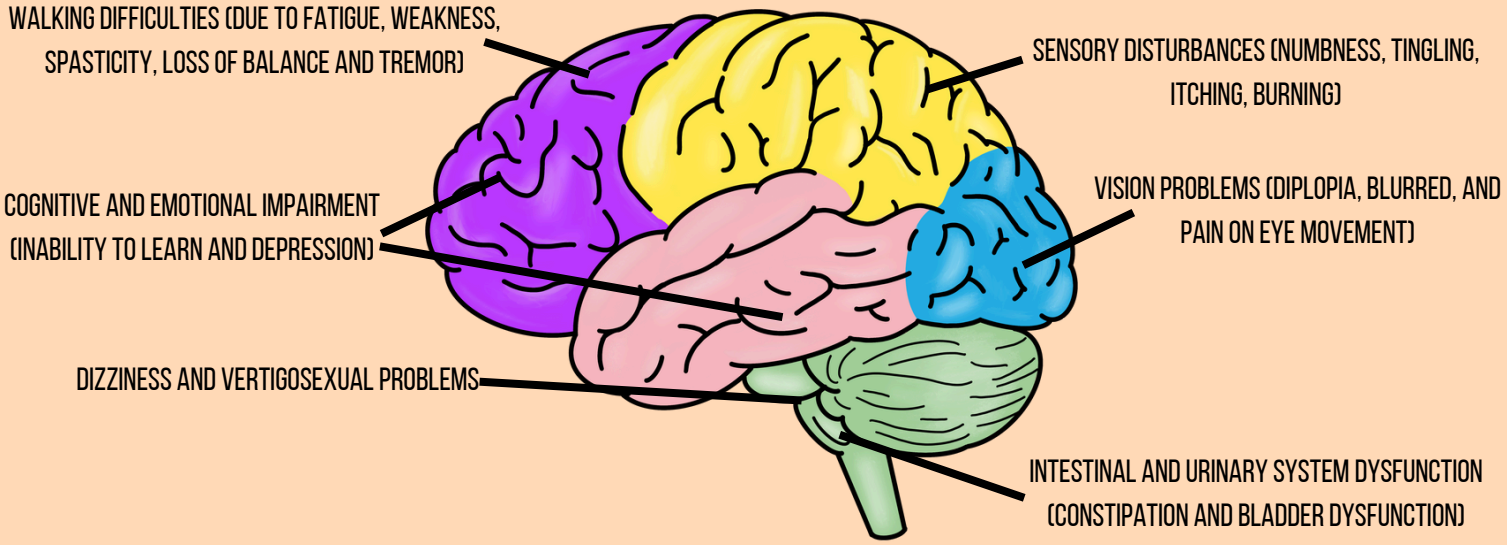
SYMPTOMS

UNPREDICTABLE

UNCERTAIN

MS patients can present with a variety of symptoms. The brain can be organized into functional regions, symptoms vary depending on the extent to which a certain part of the brain faces the most demyelination or plaque accumulation. Below are come common MS symptoms[14].

UNIQUE



TAKING CONTROL OF YOUR HEALTH IN THE KITCHEN

Whether its a proactive measure, or a recommendation made by your healthcare provider to control your symptoms, diet is an important factor of overall wellbeing.

Here are some simple changes from the National MS Society that you can implement to improve the long-term health of your nervous system.

A balanced diet and a steady, healthy body weight is associated with decreased risk of relapse and MS associated activity[17]. Diet has a tremendous effect on what bacteria reside in the gut and make up the gut microbiome. The gut microbiome participates in immune responses by building up a tolerance for pathogens, strengthening the intestinal epithelial barrier and modulating inflammatory responses[18].

MS National Multiple Sclerosis Society

RECOMMENDS:

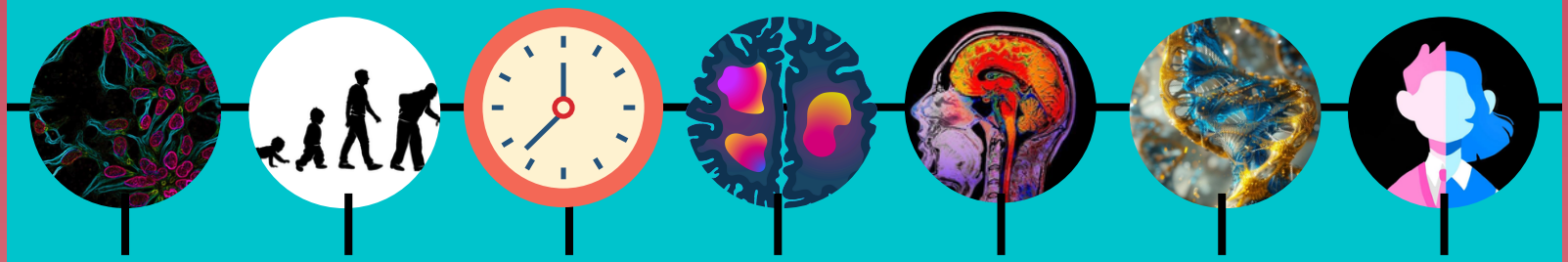
"Prepare meals at home as much as possible.
Incorporate colorful fresh fruits and vegetables daily.
Choose lean proteins and healthy fats.
If you eat grains, choose whole grains over refined grains.
Consider adding herbs and spices to add flavor to meals.
Avoid or limit processed foods and added sugars as much as possible[17]."

Check out this website for MS-friendly recipes and nutritional advice[19]:
https://overcomingms.org/resources/recipes

PROGNOSIS

Prognosis of multiple sclerosis can vary significantly from individual to individual as factors such disease subtype, frequency, severity of responses and responses to treatment influence the degree of this disability.
Understanding the prognosis of MS is crucial for patients, caregivers, and healthcare providers to manage expectations and develop appropriate treatment strategies for individuals.

RISK FACTORS AFFECTING PROGNOSIS OF MS



DISEASE SUBTYPE | AGE OF DISEASE ONSET | TIME INTERVAL BETWEEN ATACKS | LESION BURDEN & LOCATION | BIOMARKERS | GENETICS | SEX

DISEASE SUBTYPE [20]

- Relapsing remitting MS
  - Most common
  - Periods of remission and relapse
  - Treatment can delay progression
- Secondary progressive MS
  - Steady decline in neurological function
- Primary progressive MS
  - Poor prognosis + gradual worsening from onset

TIME INTERVAL BETWEEN ATACKS

- Regain full function → Slower disease progression
  - Indicates better repair of demyelinated areas
  - Compensates for damage, delaying disability
- Lingering symptoms → More aggressive disease course
  - Suggests poorer recovery and ongoing damage
  - Faster accumulation of disability over time

AGE & SEX [20]

- Young individuals - slower disease progression compared
- Symptoms:
  - Sensory symptoms (numbness, tingling, altered sensations) → Favorable outlook
    - Less harmful = no immediately damage tp motor pathways
    - Slower disease progression
  - Motor/cerebellar symptoms (muscle weakness, walking difficulties, balance issues, tremors, cognitive impairment) → Poorer prognosis
    - More extensive nervous system damage
    - Higher risk of disability and faster progression
- MS - common in females (3:1 female-to-male ratio), males tend to have a more aggressive disease course
  - Men - develop Primary Progressive MS (PPMS) = worse prognosis

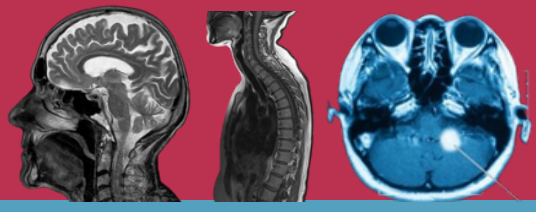
LESION BURDEN & LOCATION [20]

LESION BURDEN & MRI

- **Leison burden**→ More demyelinating plaques = Higher risk of disability
- Critical lesion locations (brainstem, spinal cord) → Affect vital functions (movement, sensation, balance, autonomic control)
- **Dignostic tool : MRI**
  - QSM & DTI detect tissue damage & neurodegeneration
- **Brainstem lesions** → Swallowing (dysphagia), speech (dysarthria), and eye movement issues
- **Spinal cord lesions** → Mobility impairment, spasticity, bladder/bowel dysfunction
- **Gadolinium-enhancing MRI** → Detects active inflammation (higher risk of relapses)
  - No GD+ lesions → Indicates stabilized disease with treatment [21]

CEREBELLAR INVOLVEMENT

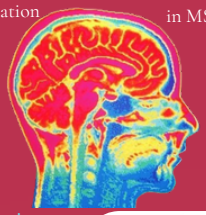
- **Early cerebellar symptoms (ataxia, tremors, dysarthria, vertigo) = Worse prognosis**
- **Leads to poor coordination, balance issues, increased fall risk**
- **Brainstem & cerebellar lesions linked to faster disability progression**
- Fewer lesions at onset → Slower disease progression
- Widespread lesions (brain + spinal cord) → More severe disease course



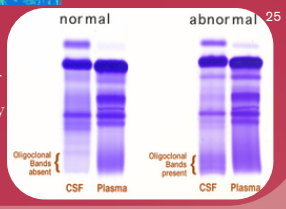
PROGNOSIS

BIOMARKERS

- **Neurofilament Light Chain (NfL)**[22]
  - Structural protein in neurons, found in cytoplasm, axons, dendrites
  - Released into CSF & blood when axons are damaged
  - Elevated NfL = Active inflammation, lesion formation, neurodegeneration
  - High NfL early in disease → More aggressive MS course
  - NfL levels decrease with effective disease-modifying therapy (DMT) = good biomarker
- **Glial Fibrillary Acidic Protein (GFAP)** [23]
  - Located in astrocytes in the CNS, non-myelinating Schwann cells in the PNS and enteric glial cells
  - Modulates astrocyte motility & shape, provides structural stability
  - Reactive astrocytes accumulate around demyelinated areas, contributing to scar formation (gliosis)
  - GFAP levels are detected in MS plaques
  - INJURY
  - Astrogliosis: Astrocyte reaction to CNS injury



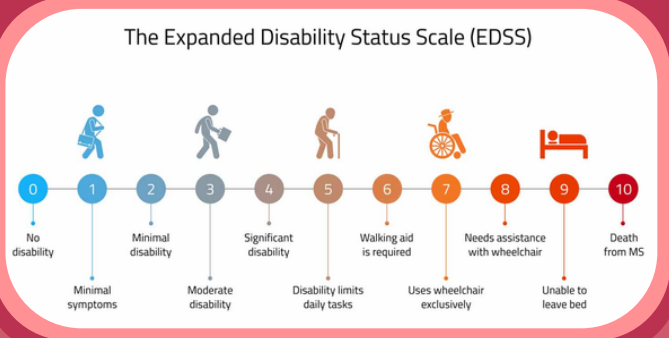
- **Oligoclonal Bands (OCBs)** [24]
  - IgG oligoclonal bands (OCBs) are present in the cerebrospinal fluid (CSF)
  - Presence of IgG in CSF is a hallmark of MS and may correlate with disease activity
  - OCBs → increased levels of disease activity and disability



[25]

EXPANDED DISABILITY STATUS SCALE [26]

- EDSS
  - Used measure to track MS progression through a standardized method
  - Assesses neurological impairment based on a 0 to 10 scale



GENETICS

- Gene:
  - HLA-DRB1*15:01 is the strongest genetic risk factor for MS, particularly in individuals of European ancestry[27].
  - This gene is part of the human leukocyte antigen (HLA) system, which helps regulate immune responses[28].
- Effects:
  - Carriers of HLA-DRB1 = higher risk of developing MS + experience a more inflammatory disease course [27].

PATIENT PERSPECTIVE

MEET MAYA

- Age 27
- Diagnosed with Relapsing-Remitting MS

"It started with a tingling in my left hand. I thought it was from too much typing at work. But when my vision blurred and I stumbled walking, I knew something was wrong."

Symptpms | MONTH 1 | MONTH 3 | MONTH 5

- Numbness in left arm and leg
- Blurred vision (optic neuritis)
- Muscle spams and loss of balance

Diagnosis

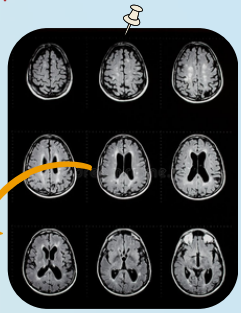
- MRI Scans - showed ultiple demyelinating lesions in the spinal cord and white matter
- CSF analysis - elevated levels of NfL, marks axonal injury

"It was challenging as I feared what my diagnosis would be, all these invisible and common symptoms left me confused and misunderstood"

WHAT HELPED

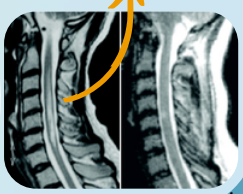
- Joining a support group
- Keeping a symptpm journal
- Open talk
- Adding to my diet

Patient MRI Scans



Relapse shows greater leisons

Demyelination of the SC



If you or a loved one are experiencing symptoms of multiple sclerosis, please consult your primary care doctor for evaluation

TREATMENT

The treatment of multiple sclerosis (MS) focuses on slowing disease progression, managing symptoms, and improving quality of life.

Current therapies target immune modulation, neuroprotection, and symptom management.

A multidisciplinary approach is used involving neurologists, physical therapists, and mental health professionals, for comprehensive care.

Treatment Goals [29]

- Decreasing relapses of MS
- Magnetic resonance imaging to measure axonal damage and activity
- Minimizing permanent disability
- Addressing various patient concerns such as bladder and bowel dysfunction, depression, cognitive impairment, fatigue, sexual dysfunction, sleep disturbances, and vertigo
- Treating beyond symptoms

Treatment Types

FIRST LINE TREATMENTS: [29]

- Used when one is newly diagnosed or milder disease activity
- Reduces relapse rate
- Delays progression
- Decreases formation of new leisons in the CNS
- Fewer side effects

SECOND LINE TREATMENTS: [29]

- Used when disease is highly active
- Stronger suppression of MS disease activity
- Inhibiting attack by blocking immune cells crossing the blood-brain barrier
- Reduces inflammation
- Traps lymphocytes
- Greater side effects

Treatment - Mangement [29]

Diet & Nutrition

- Helps with inflammation + hydration

Exercise

- Helps with spasticity + strength training

Stress Reduction

- CBT can help with mindfullness and reducing flare-ups

Sleep & Support

- Helps with reducing nigttime muscle spams and pain mangement

COMPLICATIONS

MEDICATIONS USED FOR MS THERAPY

Nearly all therapies that combat the disease progrssion of MS require modulating the immune system through the use of immunosuppressive medications. While these treatment methods have positive outcomes on patients, they also introduce the risk of opportunisitic infections[30].

IMMUNOSUPPRESSIVE MEDICATIONS:

Natalizumab

Mechanism of action:

Natalizumab blocks α-4 integrin on lymphocyte surface → Prevents adherence to vascular adhesion molecule-1 → Inhibits migration of T-cells across the blood brain barrier → Induces apoptosis by blocking transmural signaling[30]

Alemtuzumab

Mechanism of action:

Binds to CD52 on T-cells, B-cells, thymocytes, and Natural Killer cells → Causes cell lysis and lymphocyte depletion → Long-term inflammatory suppression may result from the repopulation of regulatory T cell subsets[30].

Ocrelizumab

Mechanism of action:

Ocrelizumab is a humanized antibody that binds to CD20 on B-cells → B-cells drive MS by activating proinflammatory T-cells, secreting cytokines, and producing autoantibodies that attack myelin → Reduces B-cells via antibody-dependent cytotoxicity and complement-mediated lysis → It allows B-cell reconstitution while depleting B-cells[30]

Fingolimod

Mechanism of action:

Inhibits lymphocytes from exiting lymph nodes and from recirculating within the bloodstream and CNS → Redistribution of lymphocytes within lymphatic tissue induces considerable, yet reversible lymphopenia[30]

Teriflunomide

Mechanism of action:

Teriflunomide selectively and reversibly inhibits dihydroorotate dehydrogenase, a critical mitochondrial enzyme in the de novo pyrimidine production pathway[31] → Results in reduced proliferation of activated T and B lymphocytes without cell death[31].

RISKS OF OPPORTUNISITIC INFECTIONS

TUBERCULOSIS

ALEMTUZUMAB:

- Results in prolonged profound lymphocytopenia
- Affects both humoral and cell-mediated immunity
- Associated with high rates of TB among patients with hematologic malignancies
- B occurred in only 2 of the >900 individuals randomized to alemtuzumab in 2 pivotal large-scale phase III clinical trials[32]

TERIFLUNOMIDE

- 3 cases of TB were reported among the >2000 teriflunomide-treated patients, though LTBI (latent tuberculosis infection) screening was not mandated in most of these studies
- Due to these cases, LTBI screening is recommended in teriflunomide's FDA-approved product label[32]

HERPES VIRUS

ALEMTUZUMAB

- Alemtuzumab is associated with very high rates of Herpes simplex virus infections and can require hospitalization
- As a result, clinical trial protocols were altered to include acyclovir 200 mg twice daily during alemtuzumab therapy and for 28 days afterward

FINGOLIMOD

- Two fatal herpesvirus infections occurred in individuals receiving fingolimod in a clinical trial
- Analysis showed a slightly higher risk of total varicella zoster virus in patients treated with fingolimod[32]

OCRELIZUMAB

- Ocrelizumab appears to raise the risk of herpes virus infections, albeit almost all infections were mild to moderate[32]

PROGRESSIVE MULTIFOCAL LEUKOENCEPHALOPATHY

NATALIZUMAB

- More than 650 cases of PML have been reported in association with natalizumab therapy, as of 2017
- Risk factors include prior immunosuppression, prolonged treatment duration with natalizumab, and presence of anti-JCV antibodies
- Due to lack of effective treatment of natalizumab-associated PML, it is very important to discontinue the use of natalizumab when benefits outweigh risks
- Radiographic evidence of PML precedes PML-associated neurologic deficits, thus, serial brain MRI scans are recommended[32]

FINGOLIMOD

- Risks of PML with fingolimod is much lower compared to natalizumab
- Fewer than 10 cases of PML associated with fingolimod[32]

HEPATITIS B VIRUS

OCRELIZUMAB

- Anti-CD20 monoclonal antibodies, such as ocrelizumab, pose a high risk of HBV-associated hepatitis and liver failure, and can sometimes be fatal
- Any patient receiving ocrelizumab with any indication of HBV infection, should undergo antiviral prophylaxis during and for 12 months after discontinuation of immunosuppressive medication[32]

ALEMTUZUMAB

- Profound lyphocytopenia can risk severe disease due to HBV infection

FINGOLIMOD & TERIFLUNOMIDE

- The risk of HBV infection associated with fingolimod or teriflunomide is very low[32]

Myth	Fact
MS is a death sentence.	MS is **not fatal for the vast majority** of people. Most individuals with MS have a **near-normal life expectancy** with proper care and treatment[1-3].
Only older adults get MS.	MS most often begins between the ages of **20 and 40**, but it can occur at **any age**[1-3].
MS is contagious.	MS is **not infectious** and cannot be passed from person to person. It is an **autoimmune disease**[1-3].
Everyone with MS ends up needing a wheelchair.	Many people with MS **never** need a wheelchair. Treatments can help **manage symptoms and prevent disability**[1-3]**.**
MS symptoms are always visible.	MS is often considered an **invisible illness**. Symptoms like fatigue and brain fog aren't always seen but can be impactful[1-3].
If no one in my family has MS, I can't get it.	Most people with MS have **no family history**. **Environmental** factors also play a key role[1-3].
There is nothing you can do to treat MS.	There are **many effective treatments** today that can reduce relapses, slow progression, and manage symptoms[1-3].

TEST YOURSELF

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Thank You Readers

Recap

To Learn More Visit :

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- https://www.mayoclinic.org/diseases-conditions/multiple-sclerosis/symptoms-causes/syc-20350298
- https://mscanada.ca/

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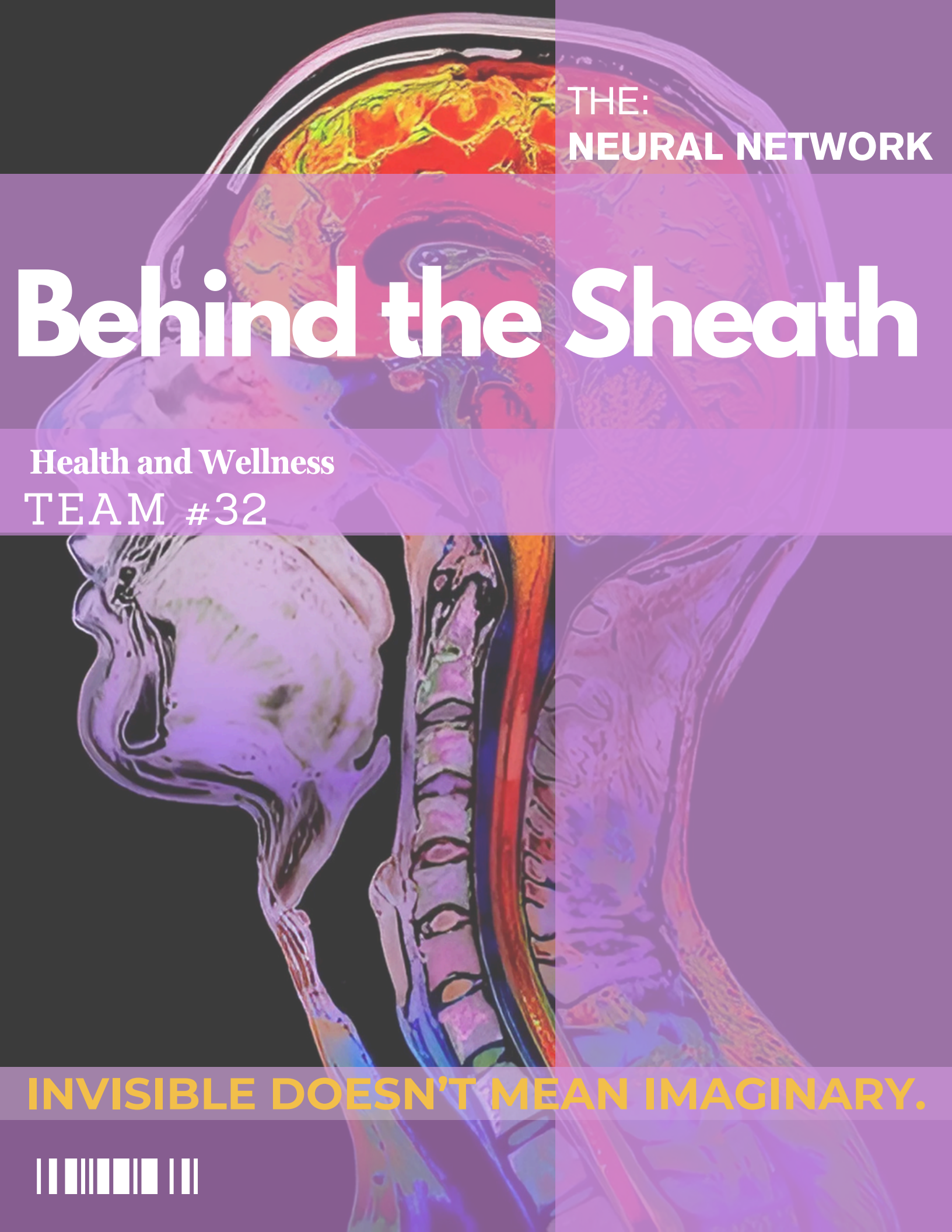

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