FOUNDATIONS OF MS NURSING

PART 1
WRAPPING YOUR HEAD AROUND WHAT MS MEANS

MS AUSTRALIA

MS Nurses Australasia Inc

IOMSN International Organization of Multiple Sclerosis Nurses

Contents

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Fiona is a clinical nurse specialist in MS working in Auckland, New Zealand. She is passionate about service improvement to bring about better outcomes for people with MS and the use of co-design methodology as a method to achieve this. Fiona has published and presented her work at international MS meetings. She loves teaching and learning and is an enthusiastic member of the MSNA education subcommittee and the current MSNA secretary.

Introduction to Part 1 is provided by Dr Therese Burke, Registered Nurse MS Nurse Specialist & Adjunct Senior Research Fellow, School of Nursing, University of Notre Dame, Sydney Campus

Session overview

In this session you will learn about:

- Overview of MS and the prevalence of MS
- Stages of MS including prodromal MS and MS phenotypes
- Risk factors – environmental and genetic
- The topographical model of MS
- Diagnosing MS including MS tests
- MS mimics
- The challenges of an MS diagnosis
- Gender specific considerations: Women and MS/Men and MS

What is MS?

Multiple sclerosis (MS) is the most common acquired chronic neurological disease affecting young adults, often diagnosed between the ages of 20 to 40. As yet, there is no cure.

There is no known single cause of MS, but many genetic and environmental factors have been shown to contribute to its development. In MS, the body's own immune system mistakenly attacks and damages the fatty material – called myelin – around the nerves. Myelin is important for protecting and insulating nerves so that the electrical messages that the brain sends to the rest of the body, travel quickly and efficiently.

Myelin is a white fatty substance that surrounds the axon of some nerve cells. The myelin sheath forms an electrically insulating layer. It is essential for the proper electrical conduction of nerve signals along nerve axons. The production of the myelin sheath is called myelination.

As the myelin breaks down during a MS attack – a process called demyelination – patches of nerves become exposed and then scarred, which render the nerves unable to communicate messages properly and at risk of subsequent degeneration. This means that the brain cannot talk to other parts of the body, resulting in a range of symptoms that can include a loss of motor function (e.g. walking and hand and arm function, loss of sensation, pain, vision changes and changes to thinking and memory).

Learn more about MS in the What is MS? video

MS Symptoms

MS has many symptoms, which can be variable and unpredictable. No two people will experience exactly the same symptoms, and these symptoms can be a one-off occurrence, can come and go or change in severity over time.

MS symptoms can be experienced in different parts of the body, depending on which part of the brain, optic nerve or spinal cord is affected. Some symptoms of MS are invisible, meaning people can't visibly see that someone is experiencing a symptom.

Symptoms can include anxiety and depression, cognitive problems, fatigue, sensory issues and visual disturbances, ataxia, bladder and bowel issues, heat sensitivity and pain.

Learn more about MS symptoms

Learn more about MS symptom assessment in Part 2: The Nuts & Bolts of MS

MS prevalence

3 out of 4 Australians diagnosed with MS are women

The incidence and prevalence of MS in Australia and New Zealand is significantly increasing.

MS affects **three times more women than men**.

In Australia and New Zealand, MS prevalence follows a **strong latitudinal gradient**, where it increases significantly the further south a person lives. The highest prevalence rates in Australia are in Tasmania and in New Zealand in the Southland region

Prevalence in Australia

There has been a significant increase in the number of people living with MS in Australia over the last three years[1].

There were **37,756** people living with MS in Australia in 2024, an increase of 4,421 people since 2021 (from 33,335 people). Since the first MS prevalence report in 2010, the number of Australians living with MS has risen by 77.4%, while prevalence has risen 45.5%, now at **139.2 per 100,000 Australians** (from 95.6 per 100,000 in 2010).

Prevalence in New Zealand

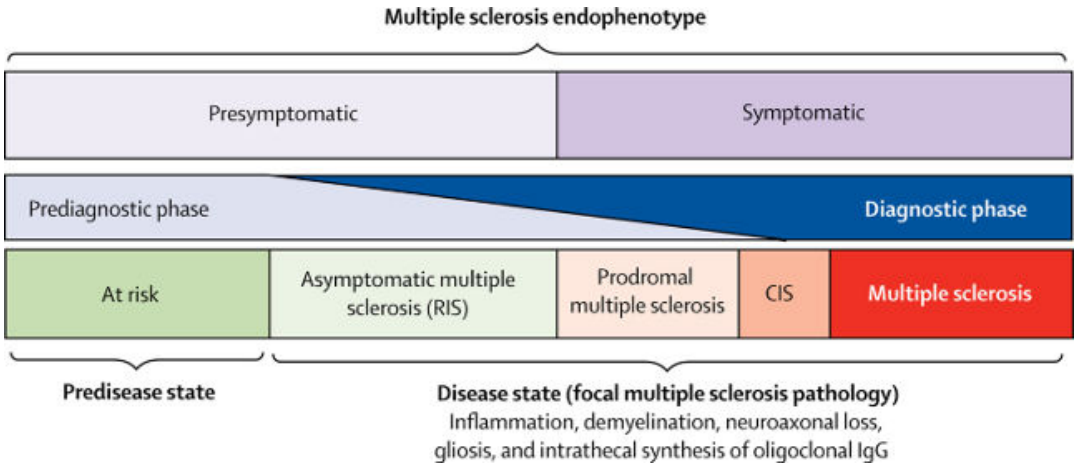
There has also been a significant increase in the number of people living with MS in New Zealand.

In 2022, **4,860** people identified as living with MS in New Zealand (up from 2,917 people in 2022) and a significant increase in prevalence with a rate of **96.6 per 100,000** people (up from 72.4 per 100,000 in 2006)[2]. Overall, about **one in every 1,000** New Zealanders is estimated to have MS. Prevalence is highest among those of European descent (approx. 132.4 per 100,000), while rates among Māori - historically much lower - more than doubled between 2006 and 2022, rising from 15.9 to 33.1 per 100,000.

Prodromal MS

A prodrome is an early set of signs or symptoms predating the onset of classical disease, which in turn predates a definitive diagnosis. The MS prodromal phase is detectable at least 5-10 years before MS symptom onset and possibly up to 20 years in people who develop primary progressive MS. People in the prodromal phase of MS may complain of fatigue and pain, mood changes like depression and anxiety and cognitive issues.

Clinically isolated syndrome (CIS) is diagnosed when a person has experienced a single episode of neurologic symptoms. While not everyone that experiences CIS will actually go on to develop MS, about 70% of people will.



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The **prodromal phase of MS** provides an opportunity for early intervention including reducing modifiable risk factors such as smoking, obesity and low sunlight/inadequate vitamin D. Continued research could also lead to earlier prevention measures such as an EBV vaccine, earlier MS diagnosis and earlier use of disease modifying drugs (DMDs).

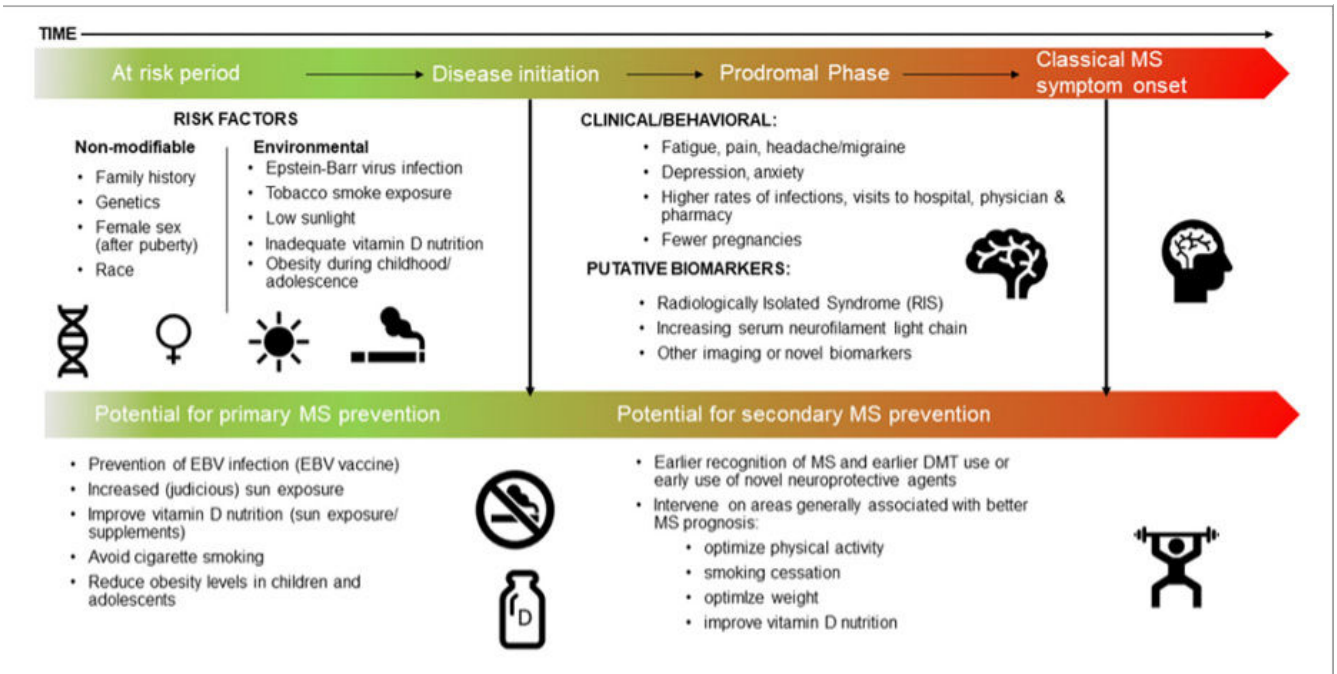

FIGURE 1 | The MS continuum, a proposed timeline: the at risk period, the prodrome, and potential for prevention.

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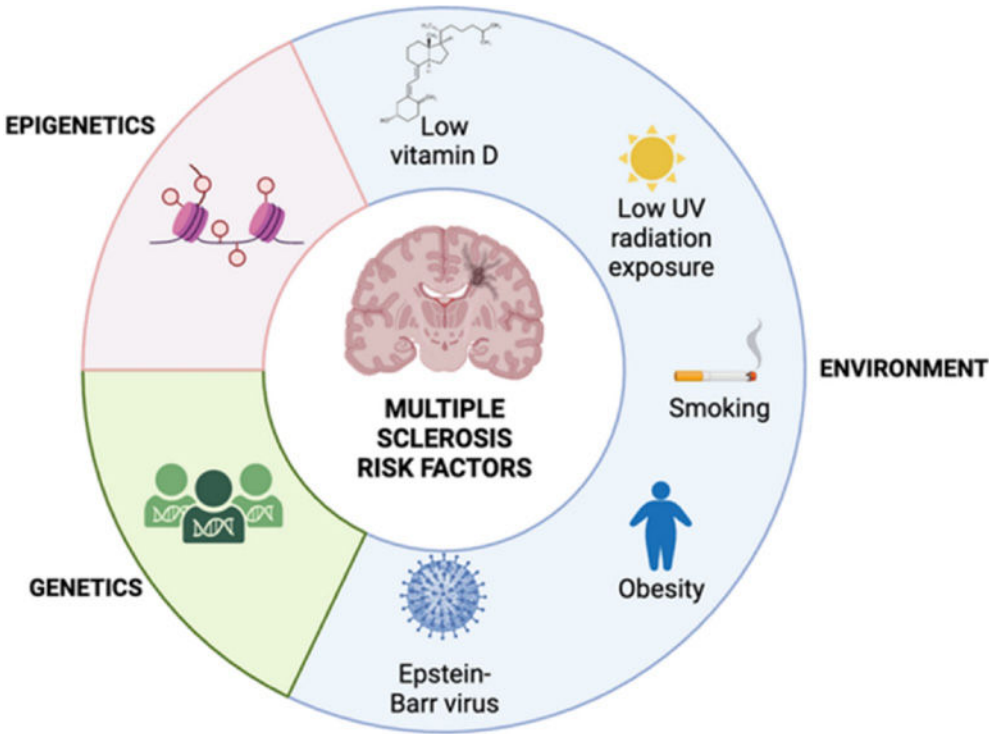
MS risk factors

Epstein Barr virus (EBV) is a very common viral infection that spreads through bodily fluids, especially saliva.

Risk factors for MS include:

- **Environment**: low vitamin D and low UV radiation exposure, smoking (including second hand smoke and childhood obesity.
- **Viral infections**: close to 100% of people diagnosed with MS have been infected with the Epstein-Barr virus (EBV).
- **Genetics**: MS is not caused by a single gene, but by the combined effect of many genes, each contributing a small amount to risk. Large international studies have now identified more than 200 genetic variants associated with MS, together accounting for around 30-50% of a person's overall risk. Your chance of developing MS is slightly higher if a close relative, such as a parent or sibling, has MS, but most relatives will never develop the disease, showing that genes alone are not enough to cause MS. There is a higher chance of getting MS if you are a women - in Australia, three times more women than men have MS. Some genetic risk factors, particularly immune-related genes, interact with environmental factors (such as infections, vitamin D levels and smoking) to influence who develops MS and when.
- **Epigenetics**: These are external modifications to DNA that turn genes 'on' or 'off'. These modifications do not change the DNA sequence, but instead, they affect how cells 'read' genes. Researchers are trying to understand how epigenetics interact with other risk factors.

Read more about risk factors



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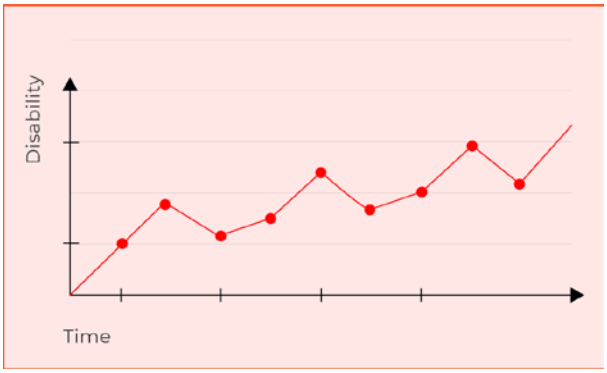
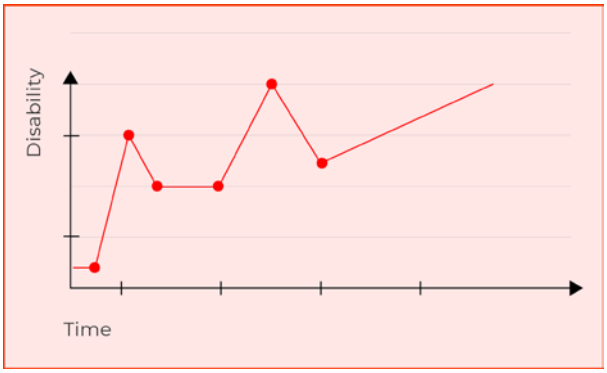
MS phenotypes

There are three main types of MS as outlined below:



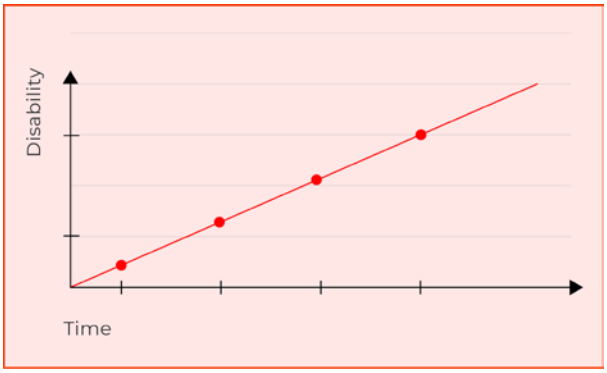
Relapsing remitting MS (RRMS) is the most common form of MS. About 85% of people with MS are diagnosed with RRMS. It is caused by flare ups or exacerbations of the neurological symptoms of MS, also known as relapses, followed by periods of recovery or remission.

Watch the MS in a Minute RRMS video



Secondary progressive MS (SPMS) is a secondary phase of relapsing remitting MS that can develop years to decades following the initial onset of symptoms. SPMS is characterised by a reduction in relapses and a progressive worsening of symptoms (accumulation of disability) over time, with no obvious signs of remission.

Watch the MS in a Minute SPMS video



Primary progressive MS (PPMS) is diagnosed in approximately 10-15% of people with MS. PPMS is characterised by a progressive worsening of symptoms and disability right from the beginning, without periods of recovery or remission. Relapses for most people are possible, but not common. Additionally, there can be periods of "plateau" where progression can stabilise for a period of time.

MS Continuum

Researchers are increasingly seeing MS as existing on a biological continuum rather than as distinct subtypes. Under a continuum model, MS phenotypes are best explained by variations in neurologic reserve - the brain's capacity to compensate for injury.

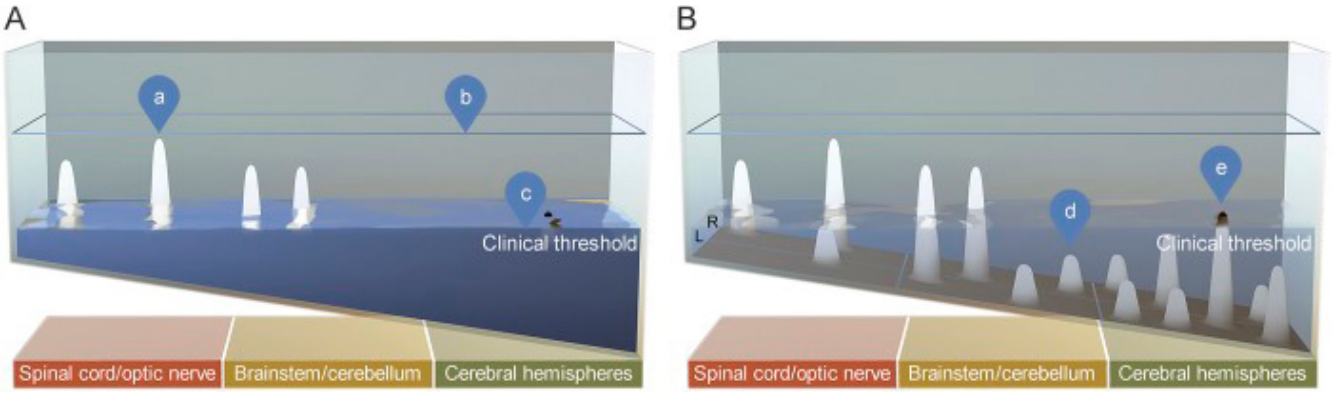
Early in MS, inflammatory damage causes neuronal loss and brain atrophy, but symptoms may remain minimal because neurologic reserve buffers functional impact. As ageing and ongoing MS-related damage continue to deplete this reserve, the brain can no longer compensate, and progressive disability becomes clinically apparent.

New MS Terminology

As we learn more about MS, there is a move towards different terminology. You may hear the following terms used to describe types of MS:

- **Active MS** (lots of inflammation) vs **inactive MS** (not much inflammation)
- **Progressive MS** vs **non–progressive MS**
- **Smouldering MS** – the slow, chronic, underlying disease progression that continues even when a patient is free of relapses and new inflammatory lesions.

Topographical model of MS



(A) Clinical view: water is opaque, only above-threshold peaks are visible.

- Above-threshold topographical peaks depict relapses and quantified Expanded Disability Status Scale/functional system disability measures. Each peak yields localisable clinical findings; the topographical distribution defines the clinical picture for an individual patient.
- Water level at outset reflects baseline functional capacity and may be estimated by baseline brain volume.
- Water level decline reflects loss of functional reserve and may be estimated by metrics of annualised brain atrophy.

(B) Subclinical view: water is translucent, both clinical signs and sub-threshold lesions are visible.

- Sub-threshold topographical peaks depict T2 lesion number and volume.
- The tallest peaks (i.e., the most destructive) in the cerebral hemispheres are shown capped in black as T1 black holes.[6]

The **topographical model of MS** also known as the Krieger model, 'swimming pool' or 'aquarium' was developed by neurologist Dr Stephen C. Krieger.

The model uses a swimming pool analogy to visualise how MS symptoms and disability manifest over time. In this model, MS lesions are peaks rising from the pool's floor. The floor is tilted to show that different parts of the nervous system have different levels of neurological reserve, or the brain's "backup" capacity.

The shallow end represents the spinal cord and optic nerves, where even small lesions easily break the water's surface to cause noticeable symptoms. The **deep end** represents the cerebral hemispheres, where many lesions can remain hidden underwater.

The water's surface acts as the clinical threshold. While a relapse occurs when a peak grows tall enough to acutely break the surface, progression is visualised as the water level slowly dropping over time. This 'recapitulation hypothesis' suggests that sinking water levels unmask previously hidden peaks, causing old symptoms to reappear and disability to worsen.

This framework explains the clinical/MRI paradox, where scans show a high lesion burden despite few physical symptoms. By tracking localisation, frequency, severity, recovery, and progression rate, the model provides a personalised map of MS.

Learn more about the Topographical Model of MS including a simulation app

Genetic risks of MS

Genes do play a role in the risk of developing MS, but they are not the whole story. Both genes and other factors, such as environmental exposures, must combine to lead to the development of MS.

At present, over 200 genetic changes have been linked to the risk of developing MS and this, in turn, has provided evidence on the mechanisms of MS. The gene with the strongest link to the risk of MS is called **HLA-DRB15*01** and this is a major gene of the immune system. This gene is also more commonly found in people of northern European ancestry, which also partially explains why these populations are more likely to develop MS than people with other ethnic backgrounds.

Many of the other more minor risk genes are also known to play a role in regulating the immune system. This makes sense since it is known that MS happens when the immune system mistakenly attacks the brain and spinal cord.

Risk in relatives of people with MS

It has been estimated that genetics accounts for just over half of the risk of developing MS, and those with a family history of MS are at more risk than the general population.

In an Australian study[7], researchers found that the risk of developing MS varies according to how closely related a person is to a relative with MS. Compared to the worldwide risk of 1 in 625 for the general population, people related to a person with MS have the following rates of risk:

- Identical twins - 1 in 6 (or 18.2%)
- Siblings of a person with MS - 1 in 37 (2.7%)
- Parents of people with MS - 1 in 67 (1.5%)
- Children of people with MS - 1 in 48 (2.1%)
- Nieces and nephews - 1 in 100 (1%) and cousins - 1 in 142 (0.7%).
- Learn more about the genetics of MS

Learn more about the genetics of MS

Case study – 'Camille'

'Camille' background

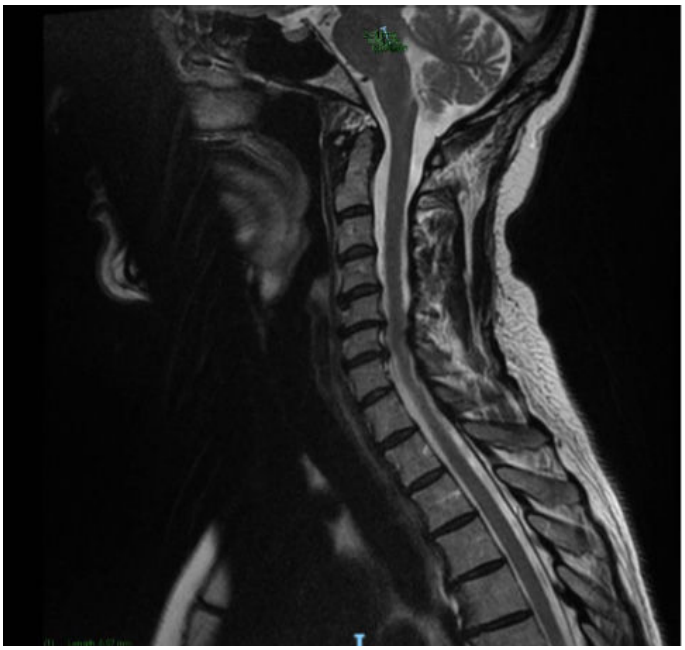
'Camille' is a 52-year-old female who has experienced numbness and tingling in both hands, with symptoms commencing overnight. Her medical history is as follows:

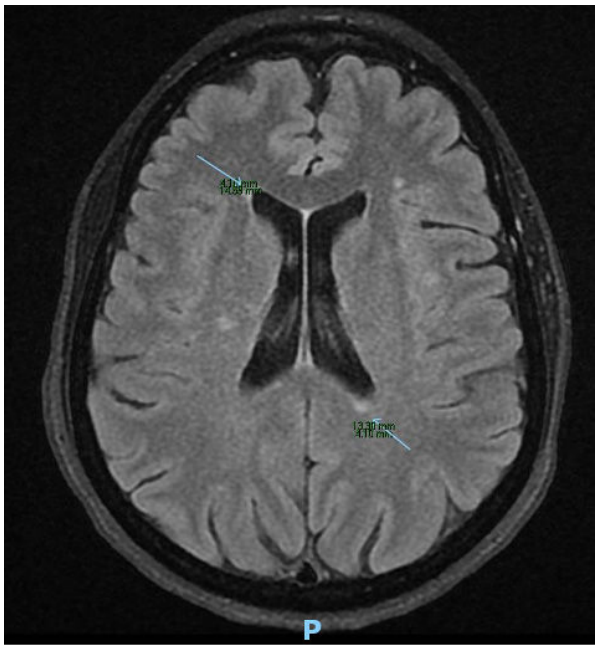
- *Medical history:* none
- *Family history:*
  - » Paternal – father bowel cancer, aunt breast cancer.
  - » Maternal – aunts with breast, bowel and lung cancer.
- *Medications:* none
- *Social:* works full time (account officer), single, no children, lives independently, travels/hikes a lot.

'Camille' visited her GP who undertook some screening:

- Blood tests: B12 levels and thyroid – both normal
- Nerve conduction study: carpal tunnel was confirmed
- MRI: Cervical radiculopathy (pinched nerve) was confirmed, and they also found a **6mm demyelinating lesion in pons, non-enhancing**.

Non-enhancing lesion: Approximately halfway through an MRI the patient will be given gadolinium-a dye which will highlight the areas where inflammation is happening. If a lesion is present but does not light up, it is known as a non-enhancing lesion. These are likely older lesions that indicate the person has had MS for a longer period of time.





'Camille' MS diagnostic testing

The neurology team undertook some further blood tests to rule out MS mimics – these tests were all negative for Camille:

- Antinuclear antibody (**ANA**) and Antineutrophil Cytoplasmic Antibodies (**ANCA**) tests.
- Aquaporin-4-IgG antibodies (**AQP4-IgG**) test (used to diagnose the NMOSD) and a Myelin Oligodendrocyte Glycoprotein (**MOG**) antibody test (used to diagnose MOGAD).
- Testing for **strongyloides parasitic infection** (threadworm)

- Malignancy and virology screening.
- A cerebrospinal fluid (**CSF**) culture was performed via lumbar puncture which was negative and **oligoclonal bands** (OCB) were negative.

The team also undertook some evoked potential tests. The somatosensory evoked potential (**SEP**) was normal and visual evoked potential (**VEP**) indicated her left eye was consistent with optic neuritis.

Pons: a part of the brainstem, a structure that links the brain to the spinal cord. It handles unconscious processes and jobs, such as the sleep-wake cycle and breathing. It also contains several junction points for nerves that control muscles and carry information from senses in the head and face.

See more detail on MS Diagnostic Testing below.

MS diagnostic testing

There are range of tests that people may undergo when obtaining a MS diagnosis including:

- **Blood tests**: these cannot confirm a diagnosis of MS, but they can help rule out other conditions or point to a diagnosis. This may include testing for autoimmune disorders, vitamin deficiencies, infections, and metabolic issues.
- **Magnetic resonance imaging (MRI):** an essential tool for diagnosing MS. It can detect brain or spinal cord lesions that indicate inflammation and demyelination of the central nervous system. Contrast dye (gadolinium) often gets injected intravenously to highlight the problem areas.
- **Lumbar puncture:** also called a spinal tap, is a diagnostic test for MS that involves removing and analysing a sample of cerebrospinal fluid (CSF), the fluid that surrounds the brain and spinal cord within the skull and backbone. The fluid is then examined to see whether there is a higher-than-normal white blood cell count and/or higher levels of antibodies (also known as oligoclonal bands due to the test carried out on the fluid).
- **Evoked potential test:** measures how fast electrical messages travel from your sensory nerves to the brain by measuring the brain's electrical activity in response to specific visual and auditory stimuli. Abnormalities in the test could indicate evidence of nerve damage and demyelination associated with MS.
- **Optical coherence tomography (OCT):** a non-invasive imaging technique measures the thickness of the retinal nerve fibre layers in your eyes. The OCT method applies to diagnosing multiple sclerosis because it can detect damage to the myelin and the optic nerves, both indicators of neurodegeneration in MS.

Oligoclonal bands are bands of antibodies that are seen in a patient's blood serum, or when cerebrospinal fluid (CSF) is analysed. They are an important indicator in the diagnosis of MS. More than 95% of all patients with MS have permanently observable oligoclonal bands.

MS and MRIs

As outlines above, MRI scans are a useful tool in diagnosing MS. They can also be used to assist with prognosis and ongoing monitoring of MS, including to see if a treatment is working.

The MRI scan will look at the brain and may also scan the spinal cord. The gadolinium will highlight the areas where inflammation is happening. If the lesion does not light up (known as a non-enhancing lesion), then it is likely to be an older lesion, and more than three months old.

T1 and **T2** are MRI techniques that use different timing between magnetic pulses to produce images of the brain and spinal cord. These techniques highlight different tissue features and are used to assess different aspects of disease. T1 and T2 lesions refers to whether the lesions were detected using either the T1 or T2 method.

T1 MRI images supplies information about current disease activity by highlighting areas of active inflammation, while **T2 MRI images** reflect the overall disease burden, showing the total number and extent of lesions, both old and new.

Some patients may be anxious about undergoing an MRI and be overwhelmed by the sound of the machine, sensations of confinement and/or the need to stay as still as possible. Talking through the experience of the MRI beforehand can assist patients to be prepared.

A lesion is an area of abnormal tissue or scarring (sclerosis) of the brain tissue or spinal cord due to a previous inflammatory attack.

MS mimics

There are many diseases which manifest symptoms similar to MS. These are called **MS mimics**. Research suggests that up to 1 in 5 with a diagnosis of MS might be incorrectly diagnosed[8], highlighting the challenges faced when diagnosing MS.

The most common alternative diagnoses are:

- **Migraine:** a complex neurological condition that causes moderate to severe throbbing headache episodes, typically on one side of the head. Other symptoms of migraine can include: nausea and vomiting, sensitivity to light, touch and sound, and blurred vision.
- **Radiologically isolated syndrome (RIS):** a neurological condition characterised by changes or lesions in the brain or spinal cord.
- **Cervical spondylosis:** a type of osteoarthritis that affects the vertebrae of the neck.
- **Neuropathy:** the medical term for nerve damage or dysfunction and can include physical damage to the nerves resulting from injury or surgery, infections (including shingles, Lyme disease and HIV), underlying health conditions (including vitamin B12 deficiency, lupus and hypothyroidism) and exposure to certain toxins (including arsenic, mercury and lead).

Learn more about MS Mimics

NMOSD and MOGAD

Neuromyelitis optica spectrum disorders (NMOSD) and **Myelin oligodendrocyte glycoprotein antibody-associated disease (MOGAD)** are demyelinating diseases of the central nervous system (CNS) that share many similar features to MS. Many people with these conditions are often misdiagnosed as having MS.

NMOSD was previously called Devic's Disease. It is an autoimmune condition where the immune system attacks the optic nerves, spinal cord and parts of the brain. NMOSD can look like MS early on, but it is a different disease with different treatments. In NMOSD, antibodies wrongly attack a protein called aquaporin4 in brain cells. Symptoms depend on where attacks occur and commonly include vision loss, mobility problems, sensory changes, bladder or bowel issues, and sometimes severe nausea or hiccups.

MOGAD is an inflammatory autoimmune condition affecting the brain, optic nerves and spinal cord. In MOGAD, the immune system makes antibodies that attack a protein called MOG, which helps protect nerve cells. This causes damage to myelin and disrupts nerve signals. Most people develop optic neuritis, causing blurred vision, colour problems, vision loss and eye pain. Less commonly, the spinal cord or brain are affected, leading to weakness, sensory problems, or encephalitis, especially in children.

Learn more about NMSOD and MOGAD

MS diagnosis

McDonald Diagnostic Criteria

The McDonald Diagnostic Criteria are guidelines to help clinicians provide an accurate diagnosis of MS. The criteria was first developed in 2001 and has been updated several times to account for new information about MS, and were last updated in 2024.

The updated criteria aims to allow people with MS to be diagnosed earlier, with fewer symptoms, and with greater accuracy. Getting diagnosed with MS earlier means people can start treatment sooner, and that can make a big difference in long-term health and quality of life.

The 2024 updates were developed by an international committee of 56 experts and are based on the latest MS research. The updated criteria now incorporates new tools – such as advanced MRI features, simple eye scans, and improved spinal fluid tests – to make diagnosis more precise and accessible.

Key changes include:

- **Earlier recognition of MS:** Radiologically isolated syndrome (MSlike lesions without symptoms) can now be diagnosed as MS when supported by additional tests.
- **Simplified criteria:** Clinicians no longer need to show that MS damage occurred at different times; a single clinical episode may be enough if other evidence is present.
- **Better use of the visual system:** The optic nerve is now a core diagnostic area, and Optical Coherence Tomography (OCT) can identify early eyerelated nerve damage.
- **New laboratory tools:** Kappa free light chains (kFLCs) in spinal fluid can sometimes replace older tests (such as oligoclonal band testing), speeding up diagnosis.
- **More accurate MRI guidance:** Signs such as the central vein sign (CVS) and paramagnetic rim lesions (PRLs) help differentiate MS from other conditions. Additional checks are recommended for children and adults over 50.

CLINICAL PRESENTATION – WHAT EVIDENCE FOR MS IS ALREADY PRESENT?	OBJECTIVE LESIONS	WHAT ADDITIONAL DATA IS REQUIRED FOR AN MS DIAGNOSIS?
2 or more relapses	2 or more	None, clinical evidence will suffice (additional evidence desirable but must be consistent with MS)
2 or more relapses	1	**Dissemination in space** (DIS) shown by one or more MRI detected lesions typical of MS **OR** A further relapse showing damage to another part of the CNS
1 relapse	2 or more	**Dissemination in time** (DIT) shown by oligoclonal bands **OR** MRI evidence of new lesions since previous scan **OR** A further relapse
1 relapse	1 (known as Clinically Isolated Syndrome (CIS))	**Dissemination in space** (DIS) shown by one or more MRI detected lesions typical of MS **OR** a further relapse showing activity in a different part of the CNS **Dissemination in time** (DIT) shown by: oligoclonal bands **OR** MRI showing new lesions since a previous scan **OR** a further relapse
Neurological progression is suggestive of MS. This is typical for a diagnosis of primary progressive MS (PPMS)		**Continued progression for one year (from previous symptoms or by ongoing observations) plus any two of these** One or more MRI detected lesions in the brain typical of MS, two or more MRI detected lesions in the spinal cord, Oligoclonal bands in the spinal fluid.

Challenges of a MS Diagnosis

MS is a complex disease, and diagnosis is not always straightforward. While for some, a diagnosis can be made in a few quick steps, others can wait for a long period of further testing from the time of their first symptoms until they are officially diagnosed.

Factors that can impact a diagnosis include location, socioeconomic status, age, and diversity. If people do not have access to local affordable testing (including MRIs) and health professionals (including neurologists) this can significantly impact their ability to get a timely diagnosis.

In April 2024, The Menzies Institute for Medical Research reviewed the data from the Australian MS Longitudinal Study (AMSLS) to look at trends in MS diagnosis over the last 25 years[9]. The report found that since the introduction of the first DMD for MS treatment in 1996:

- The **average time from symptoms to diagnosis has reduced from five years and almost four months** (median two years) in 1997-2000 to three years and almost 11 months in 2017-21 (median one year).
- There remain people who are not diagnosed as having MS for many years. This reflects the complexity of MS, with some people taking many years to manifest.
- The fall in the median time to diagnosis from two years to one year is very significant.
- Those with a longer time to diagnosis are likely to be older at diagnosis and more likely to be deceased (and no longer part of the study).

Read the full report and lived experience stories about the challenges of getting a MS diagnosis in MS Australia's 2024 World MS Day 2024 'My Diagnosis' report.

Support for people diagnosed with MS

MS Clinics

MS clinics exist across Australia and New Zealand to provide expertise in the diagnosis and management of MS, and many have a range of neurological services available. Some are called MS clinics whilst others are neurology or neuroimmunology clinics. All listed will have specialist experience with MS

Some are large, tertiary referral, multidisciplinary, clinics and others are smaller, private or visiting specialist clinics. Most clinics require a referral from a GP.

Find a list of Australian MS Clinics

Peer Support Groups

People living with MS can benefit from access to a peer support group. This provides a safe place to share their experiences, ask questions, pool resources and find new connections with people on a similar journey.

Support groups may be online or in person and centred on location, age, gender, diversity or special interests. Information on support group scan be found at:

- MSWA – support groups in Western Australia
- MS SA & NT (under the auspices of MSWA) – support groups in South Australia and the Northern Territory
- MS Queensland – support groups in Queensland
- MS Plus - support groups in NSW, ACT, Victoria and Tasmania
- MS New Zealand – support groups operated in each regional MS Society

Brain Health

The **Brain Health - Time Matters report** focuses on the latest advancements in understanding and managing MS, NMOSD and MOGAD. The report provides valuable insights and practical recommendations to improve brain health and patient outcomes. The guide and associated resources can be found here.

Learn more about the Brain Health – Time Matters report in Part 4: Regaining Control

Sex-specific considerations

It is important to consider sex-specific issues when working with people living with MS. Current research suggests that while women are more susceptible to developing MS, men that develop MS will tend to exhibit greater cognitive impairment and accumulated disability.

Learn more about MS and Women's Health

Conclusion

Understanding MS starts with recognising it as a chronic, immune-mediated disease of the CNS that can affect people in highly individual ways. In this module we explored what MS is, why demyelination disrupts communication between the brain and body, and how symptoms can be visible or invisible and fluctuate over time.

We also reviewed current prevalence trends in Australia and New Zealand, key environmental, viral and genetic risk factors, and how MS phenotypes sit within an evolving continuum model. Using the topographical ('swimming pool') framework, we considered how lesions, neurological reserve and progression can explain the clinical–MRI paradox and support personalised discussions with patients.

Finally, we outlined contemporary diagnostic pathways (including the updated McDonald criteria), common MS mimics, and practical supports such as specialist clinics, peer groups and brain health resources – while keeping sex-specific considerations in view. Together, these foundations strengthen clinical reasoning and help clinicians communicate clearly, compassionately and confidently from first suspicion through diagnosis and beyond.

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IOMSN has reviewed this project that was developed by Therese Burke as a resource for MS Nurses. IOMSN has concluded that this project is fair balanced and accurate and is valid for educational purposes.