



New Trends  
in Psychology

## Inflammatory-Demyelinating Diseases Multiple Sclerosis

Victoria Onofrei <sup>1</sup>

**Abstract:** At birth, only some areas of the brain are fully myelinated, such as the brain stem regions, which direct reflexes. Once their axons are myelinated, neurons achieve optimal function and conduct faster and more efficiently. Although the myelination process begins in an early postnatal period, the axons of the neurons of the cerebral hemispheres carry out this process a little later. The myelination process is slow, starting about 3 months after fertilization. It develops at different times, depending on the area of the nervous system that is formed. Although the myelination process begins in an early postnatal period, the axons of the neurons of the cerebral hemispheres carry out this process a little later. Multiple sclerosis is the most representative condition for CNS demyelinating diseases. In the specialized literature, multiple sclerosis (MS) is a condition characterized by inflammation and focal demyelination with multiple locations, accompanied by a process of axonal degeneration. Multiple sclerosis can lead to pain and muscle spasms, weakness and fatigue, mental problems, disorders of vision, dizziness, digestive problems and dysfunction of the urinary bladder and large intestine.

**Keywords:** multiple sclerosis; demyelination; cerebral cortex; neurons; injury

### 1. Introduction

In order to understand the term ‘demyelination’, it is necessary to first consider where it comes from and what it refers to. Myelin or myelin sheath is a fatty substance that surrounds nerve fibres. Its function is to increase the speed of nerve impulses, which improves communication between neurons<sup>2</sup>. Information is

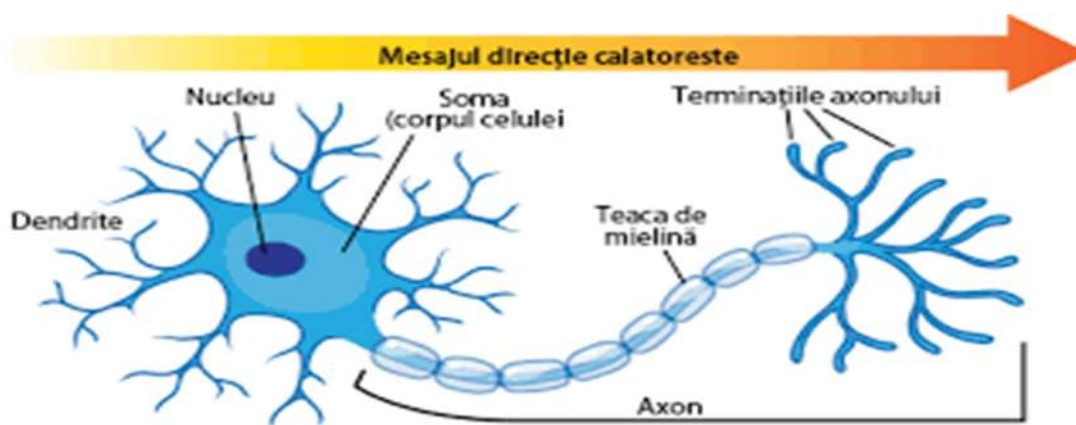
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<sup>1</sup> Student, 2nd year, Faculty of Behavioural and Applied Sciences, specialization Psychology, Danubius University of Galați, Romania, Address: 3 Galați Blvd., Galați 800654, Romania, Tel: +40372361102, Fax: +40372361290, Corresponding author: victoriaonofrei@univ-danubius.ro.

<sup>2</sup> The neuron is a nerve cell specialised for generating and transmitting nerve impulses.

transmitted in the form of an electrical signal. It also allows greater energy savings for the nervous system.<sup>1</sup> It is a spiral sheath made up of 70% protein and 30% lipid (proteolipid) that coats the axons of neurons creating an insulating layer.

The myelin sheath is interrupted from place to place - at the level of the Ranvier's nodes - this configuration ensuring a faster, jumpy transmission of the action potential by reducing the electrical capacity of the axonal membrane. The insulation created by myelin is different between the two nervous systems, the central and the peripheral. Thus, in the CNS the myelin sheaths for several neurons are formed by a single oligodendrocyte<sup>2</sup>, whereas in the PNS each internodal segment is insulated by myelin formed by a single Schwann cell (Dănăilă & Golu, 2015, p. 42).



As shown in the picture, the neuron consists of the cell body and extensions - dendritic and axonal - that provide unidirectional signalling. It is through these that they communicate rapidly and accurately with each other at the synapse. The morphology of neurons is well suited to receive, conduct and transmit signals. Dendrites provide a very extensive branching surface to receive signals. Axons conduct electrical impulses rapidly over long distances to synaptic terminals that release neurotransmitters (Sîrbu, 2020, p. 22).

The myelination process is slow, starting about 3 months after fertilisation. It develops at different times, depending on which area of the nervous system is

<sup>1</sup> <https://ro.warbletoncouncil.org/mielina-13449>.

<sup>2</sup> Small supporting cell of nerve tissue, located around nerve cells, between nerve fibres and along blood vessels. These types of brain cells mainly perform supporting and binding activities. They also have the important function of generating the myelin sheath in the CNS. <https://ro.warbletoncouncil.org/oligodendrocitos-4837>.

forming. For example, the prefrontal region is the last area to be myelinated and is responsible for complex functions such as planning, inhibition, motivation, self-regulation, etc.

At birth, only some areas of the brain are fully myelinated, such as the brain stem regions, which direct reflexes. Once their axons are myelinated, neurons achieve optimal function and faster, more efficient conduction.

Although the myelination process begins in an early postnatal period, the axons of brain hemisphere neurons carry out this process a little later. From the fourth month of life, neurons are myelinated until second childhood (between 6 and 12 years). It then continues through adolescence (12-18 years) until early adulthood, which is linked to the development of complex cognitive functions.

The primary sensory and motor areas of the cerebral cortex begin myelination before the frontal and parietal association areas. The latter are fully developed within 15 years. The commissural, projection and association fibres myelinate later than the primary sites. In fact, the structure that connects both cerebral hemispheres (called the corpus callosum) develops after birth and completes myelination at 5 years of age. Greater myelination of the corpus callosum is associated with better cognitive functioning.

Defective myelination is the main reason for neurological diseases. When axons lose their myelin, known as demyelination, electrical nerve signals are disrupted.



Demyelination can occur due to inflammation, metabolic or genetic problems. Whatever the cause, loss of myelin causes significant nerve fibre dysfunction.

Specifically, it reduces or blocks nerve impulses between the brain and the rest of the body<sup>1</sup>.

Structurally, we distinguish two types of myelin degradation. If we refer to the changes that a myelin layer that was originally normal undergoes, we are talking about the process of *demyelination*. However, when there are primary biochemical abnormalities, the phenomenon is defined as *dysmyelination*. Degradation and loss of myelin is considered the cause of various nervous system disorders such as stroke, spinal cord injury and multiple sclerosis.

Multiple sclerosis is the most representative condition for CNS demyelinating diseases

In the literature, multiple sclerosis (MS) is a condition characterized by inflammation and focal demyelination in multiple locations, accompanied by a process of axonal degeneration, in a person with a genetic susceptibility to the disease and influenced by environmental factors (Sîrbu, 2020, p. 22). In a less scientific sense, Multiple Sclerosis is an inflammatory disease of the central nervous system (brain, cerebellum, brain stem and spinal cord). When myelin is destroyed, nerve impulses are misdirected through axons. The lesions are called 'plaques'. Nerves transmit electrical signals that control all parts of the body. When a portion of the myelin sheath is damaged, the underneath nerve messages are affected and become blurred, creating chaos in the body (depending on the inflamed areas of the nervous system) (William, 2016).

Some patients are born genetically predisposed to "autoimmune" reactions. In childhood, they come into contact with an external factor (possibly a virus) and the autoimmune response is triggered, which years later causes inflammation and demyelination of areas of the brain or spinal cord (Maldonado, 1998).

The Romanian scientist, Gheorghe Marinescu, has argued that the viral origin of this disease is due to the destruction of the myelin sheath of neurons by a myelinolytic lipase contained in the viral pathogen. In demyelinating lesions, genomic material of the agent's *Chlamydia pneumoniae*, *Borellia*, Herpes virus type 6, Epstein-Barr virus (EBV) has been found, but their participation in the disease has not been proven (Sîrbu, 2020, p. 209). Some authors accept a mixed autoimmuno-infectious theory due to the resemblance of a myelin basic protein (MBP) to the structures of some viruses (Mihancea, 2005, p. 40). Although genetic predisposition is recognised in the

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pathogenesis of the disease, a gene responsible for the occurrence of this disease has not yet been discovered. The incidence of MS varies between different ethnicities living under comparable environmental influences, suggesting a genetic link. The relative risk of the disease is 0.3% in the general population and 2.77% in those with affected first-degree relatives. So, as already stated, a specific cause of the disease is not known but is thought to involve three factors:

- genetic vulnerability (inheritance of too many susceptible genes and too few protective genes);
- forms of exposure to one or more environmental pathogens;
- development of pathogenic immune response directed against the central nervous system (CNS) (Mihancea, 2005, p. 55). Autoimmune disease that causes the immune system to mistake the myelin sheath of nerves for foreign invaders and attack it.

Multiple sclerosis can lead to muscle pain and spasms, weakness and fatigue, mental problems, impaired vision, dizziness, digestive problems and bladder and large bowel dysfunction. It can partially or completely paralyse the legs, forcing the person to use a cane, crutches or even a wheelchair. Emotional symptoms such as severe susceptibility to stress, anxiety and over-emotionality, depression, unmotivated mood swings (crying and laughing) and personality changes have also been attributed to the disease since its inception.

However, some researchers, by the evidence provided, declare that MS patients were “emotionally destroyed” long before the signs of organic disease appeared: the premorbid state of multiple sclerosis is characterized by a marked immaturity of personality associated with an excessive need for love and affection. Not satisfied in childhood, this important need has led to the formation of typical traits - in order to maintain a meaningful relationship, the patient is forced to suppress anger and wants to please in an attempt to gain approval points.

Behind the outward calm, the smile or, conversely, the mask without a smile, there is inner tension and significant emotional dependence on key figures. These can include parents, siblings and people from a more distant environment with whom projective identification has been manifested.

The symptoms of multiple sclerosis, observed by a number of researchers, physiologically resemble the regression to infantile states until childhood impotent: “Enuresis, poor coordination, slurred speech, dissociative eye movements, and

ataxic spastic movements are examples of extended physiological regression to falling, more infantile levels, physical activity.”

A number of researchers (Groen and colleagues, 1969) have studied the psychosomatic aspects of multiple sclerosis based on ongoing psychiatric observation of patients' life events, behaviour and attitudes during remission and relapse, and partly in clinical psychological research. It has been found that the disease predominantly occurs in patients who come from families with a tyrannical, subordinate father or mother. In addition, in the parental family, on the one hand, the subject of sex was taboo, on the other hand, situations arose which could be seen as a kind of violence at an early age, which entails a strong sense of guilt and shame, and fear of sexuality. It is not uncommon for MS patients to express aggression, and most of their behaviour is about maintaining a “bad world” in relationships and deep compulsive experiences alone with themselves.<sup>1</sup>

**Concluding** the above, we could complete the 3 causes that lead to the onset of Multiple Sclerosis in humans. In addition to immune sensitivity which leaves the body at the mercy of viruses supposed to affect the nervous system (Chlamydia pneumoniae, Borellia, Herpes virus type 6, Epstein-Barr virus (EBV), genetic vulnerability and autoimmune diseases, we also have the courage to mention the inner conflicts which the subject did not know how to manage at an early age and which led to their somatization, thus causing nerve damage. Of course, all these “supposed” causes are not established to be the concrete and irrevocable reason that causes the destruction of the myelin of the nerve cell axons, sabotaging their activity of communication and transmission of information through impulses. Some sceptical researchers believe that the process of detecting the factors responsible for this *demyelination* damage is still in its infancy and will not be revealed soon enough to prevent its effects. Bibliography:

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