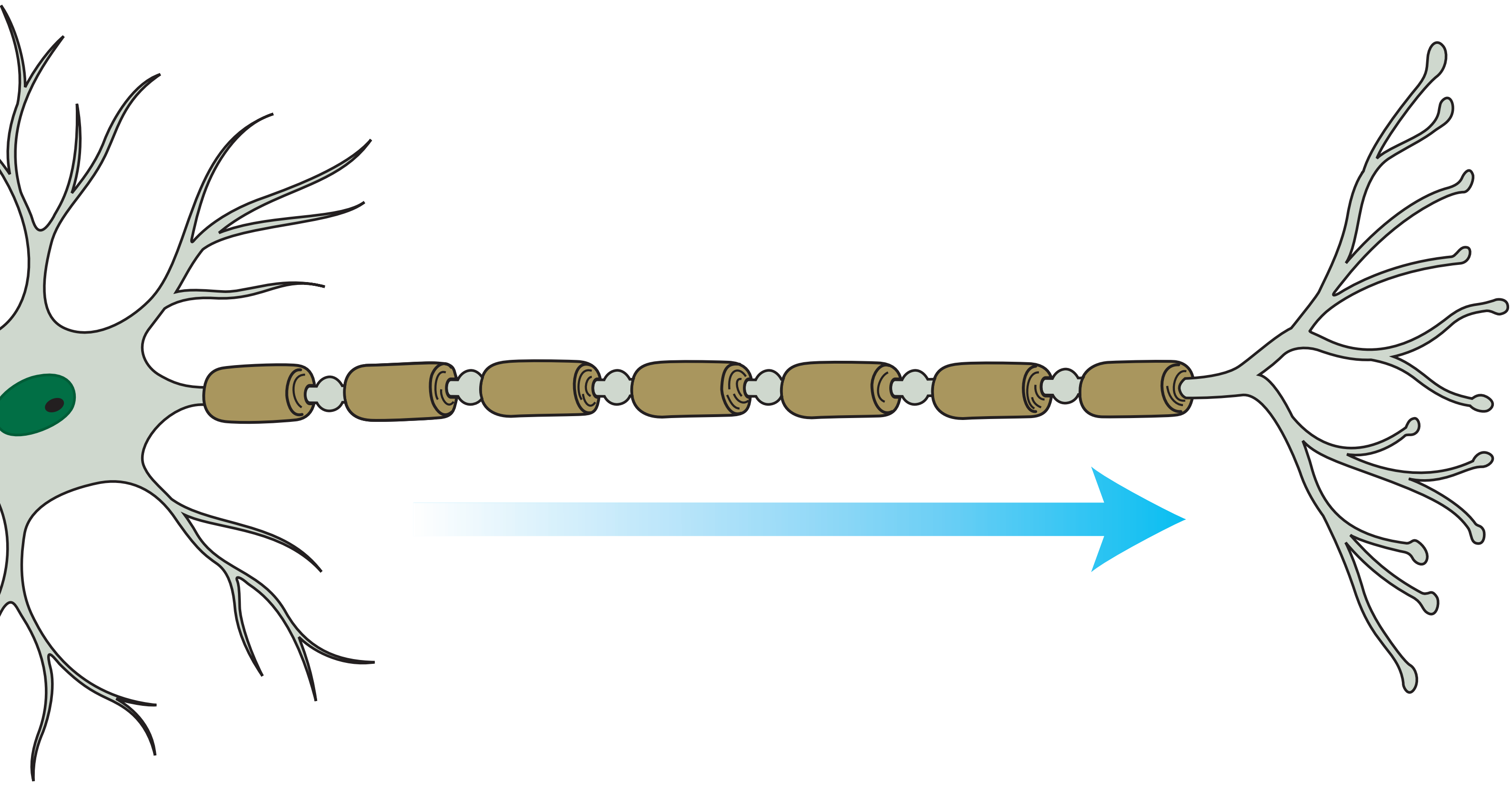
Multiple Sclerosis Society

Helping you explain MS:

a teaching resource for healthcare professionals



UK MSSNA
multiple sclerosis specialist nurse association

THE NATIONAL HOSPITAL FOR NEUROLOGY AND NEUROSURGERY

ABN

contents

This pack contains introductory notes followed by seven sections, as follows:

Helping you explain MS:

a teaching resource for healthcare professionals

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Helping you explain MS is an educational resource for MS specialist nurses, neurologists and other healthcare professionals who want to explain the basic biology of the condition to people with MS.

The need for teaching materials of this kind, for use in clinical practice, is recognised by people with MS and by healthcare professionals. In a one-to-one session with a person with MS who asks for more information about the biology of their condition, professionals may find themselves hastily improvising a diagram of a nerve cell in the absence of clear, high quality, easily accessible illustrations. ***Helping you explain MS*** was designed to meet this need.

The pack is not designed to be used as a sequential set of teaching materials. You may find that for much of the time, you draw upon only two or three illustrations and some will be much more frequently used than others.

Each illustration comes on laminated paper, in both colour and black and white. The colour version will be more useful to you when teaching: the black and white version will be easier for you to photocopy, should the person you are teaching wish to keep a copy.

Each illustration comes with a set of background notes for the professional. At the back of the pack, you will find a CD Rom containing the full pack so that you can reproduce the illustrations as necessary.

The pack was conceived and largely written by Bernie Porter, MS Nurse Consultant and Chairperson of the UK Multiple Sclerosis Specialist Nurse Association (UKMSSNA).

This project has been a collaboration between the MS Society, the UKMSSNA, the Association of British Neurologists and the National Hospital for Neurology and Neurosurgery.

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Your feedback is important to us. Please send your comments to education@mssociety.org.uk

Lynda Finn
Head of Education

SECTION 1

the nervous system

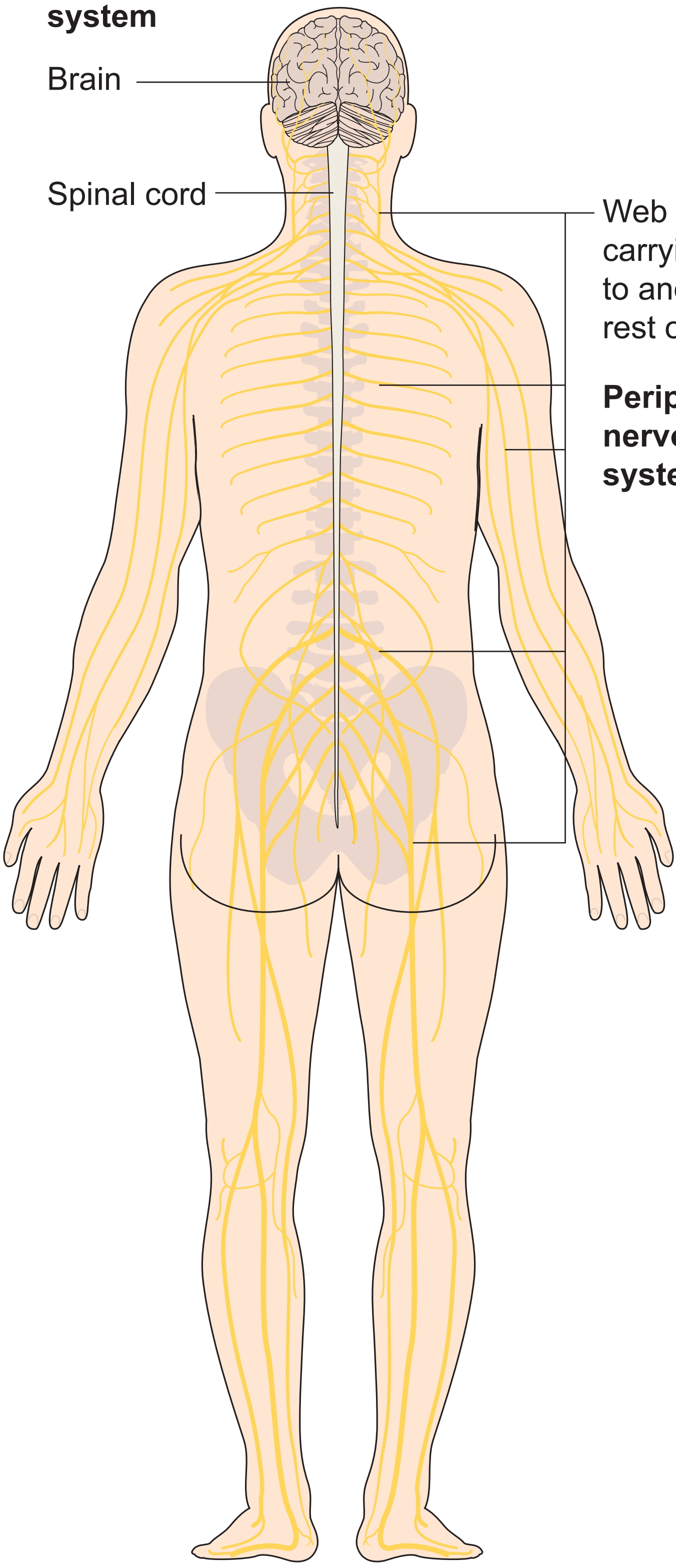
the nervous system

MS affects the central nervous system (CNS). The CNS consists of the brain and spinal cord which are connected to cranial nerves (12 pairs) and spinal nerves (31 pairs). These networks of nerves signal electrical and chemical messages to each other at great speed, controlling body functions.

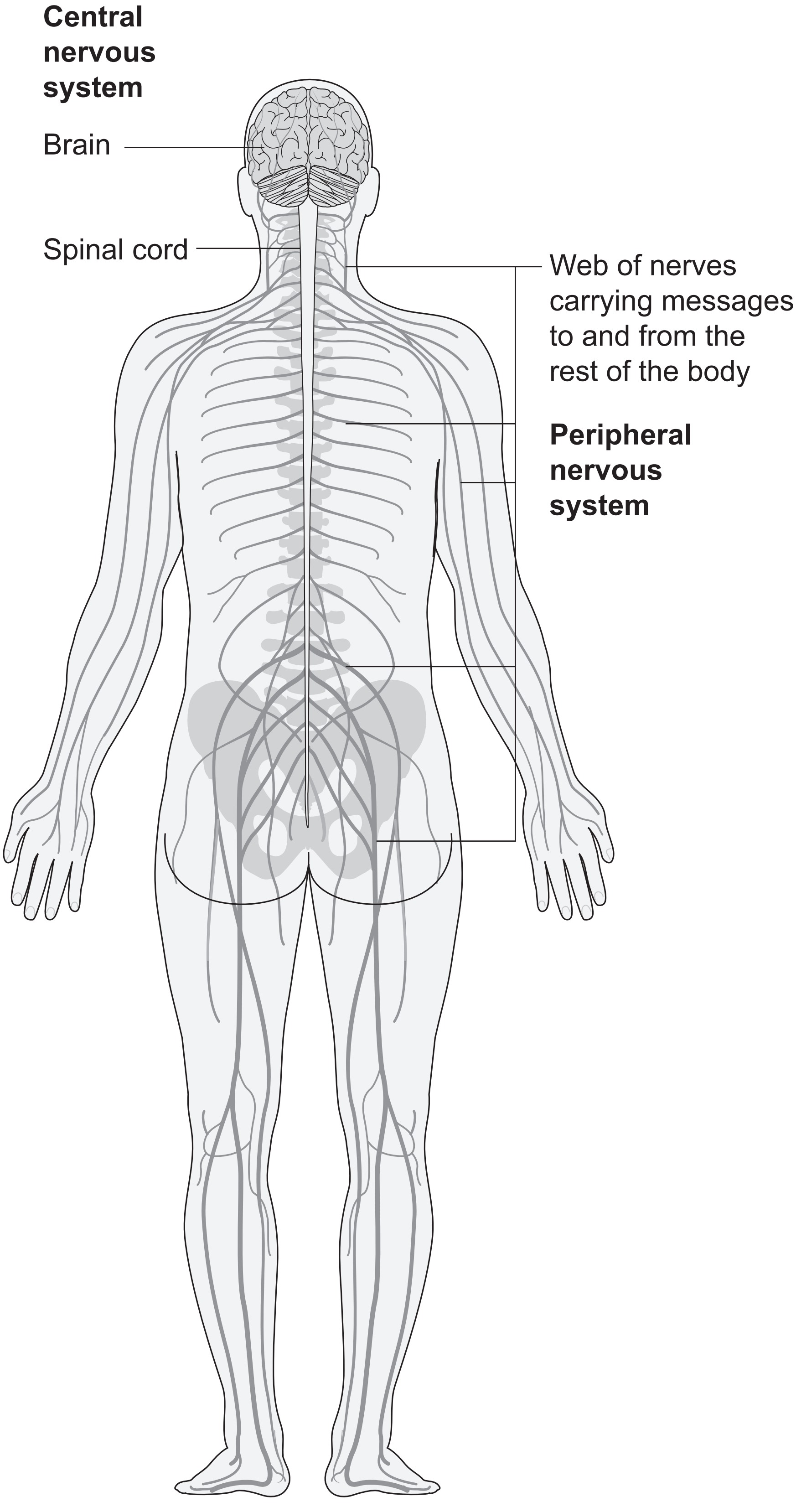
Networks that send messages **to** the CNS in response to stimuli such as heat or pain are known as **sensory** nerves. Networks that transmit messages in the other direction **from** the CNS to peripheral organs and tissues are known as **motor** nerves.

In MS, **myelin**, the fatty protective sheath that surrounds the nerve fibres, becomes damaged. This process causes the messages going to and from the brain and spinal cord to become distorted or slowed down, resulting in MS symptoms.

the nervous system



the nervous system



SECTION 2

nerve cells in the brain

nerve cells in the brain

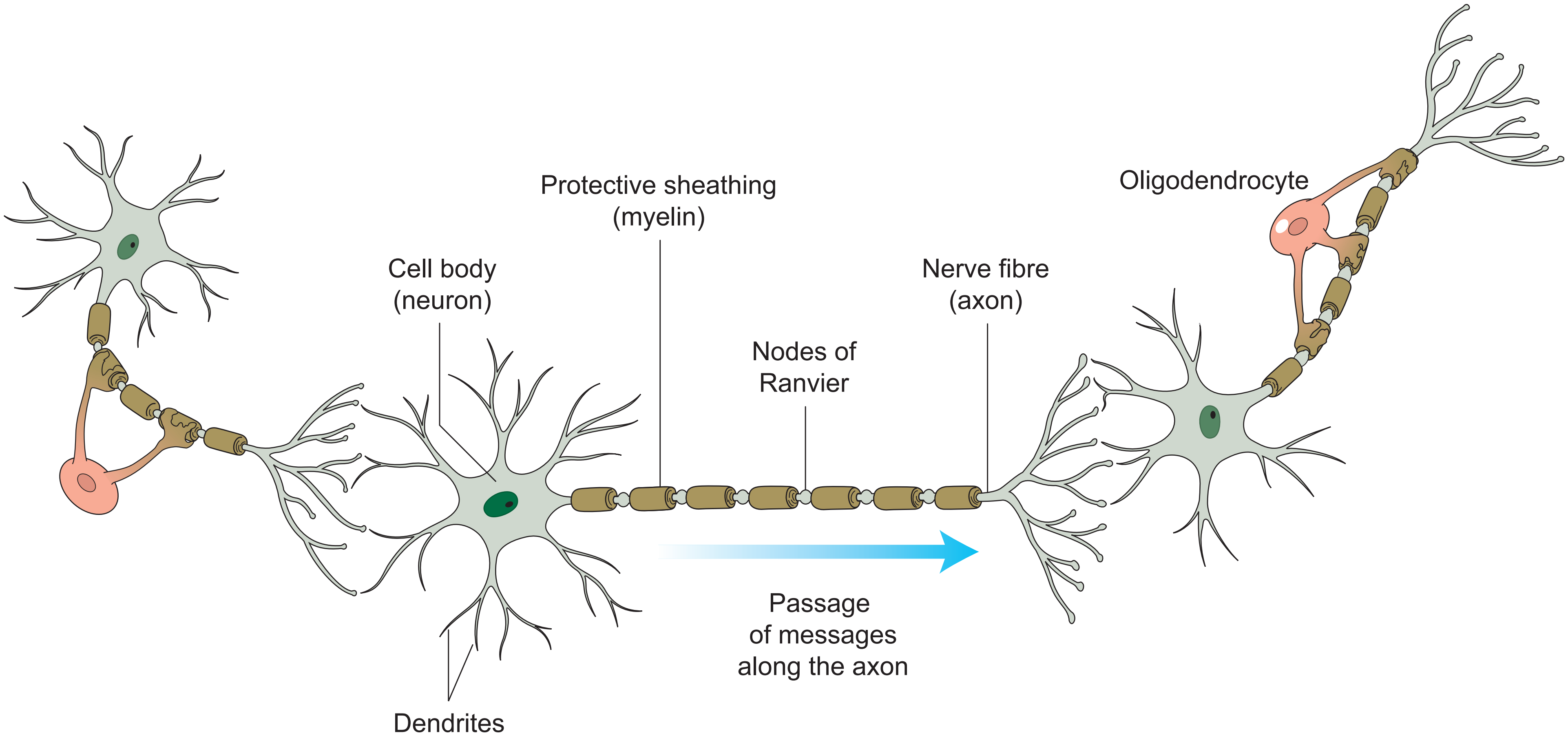
The network of nerve cells in the brain and spinal cord controls all actions and bodily functions.

The central nervous system is made up of both grey and white matter. The grey matter is made up of neuronal cell bodies; the white matter is made up of mainly the **nerve fibres** (axons) covered by a substance called **myelin**. MS is predominantly a disease of the myelin.

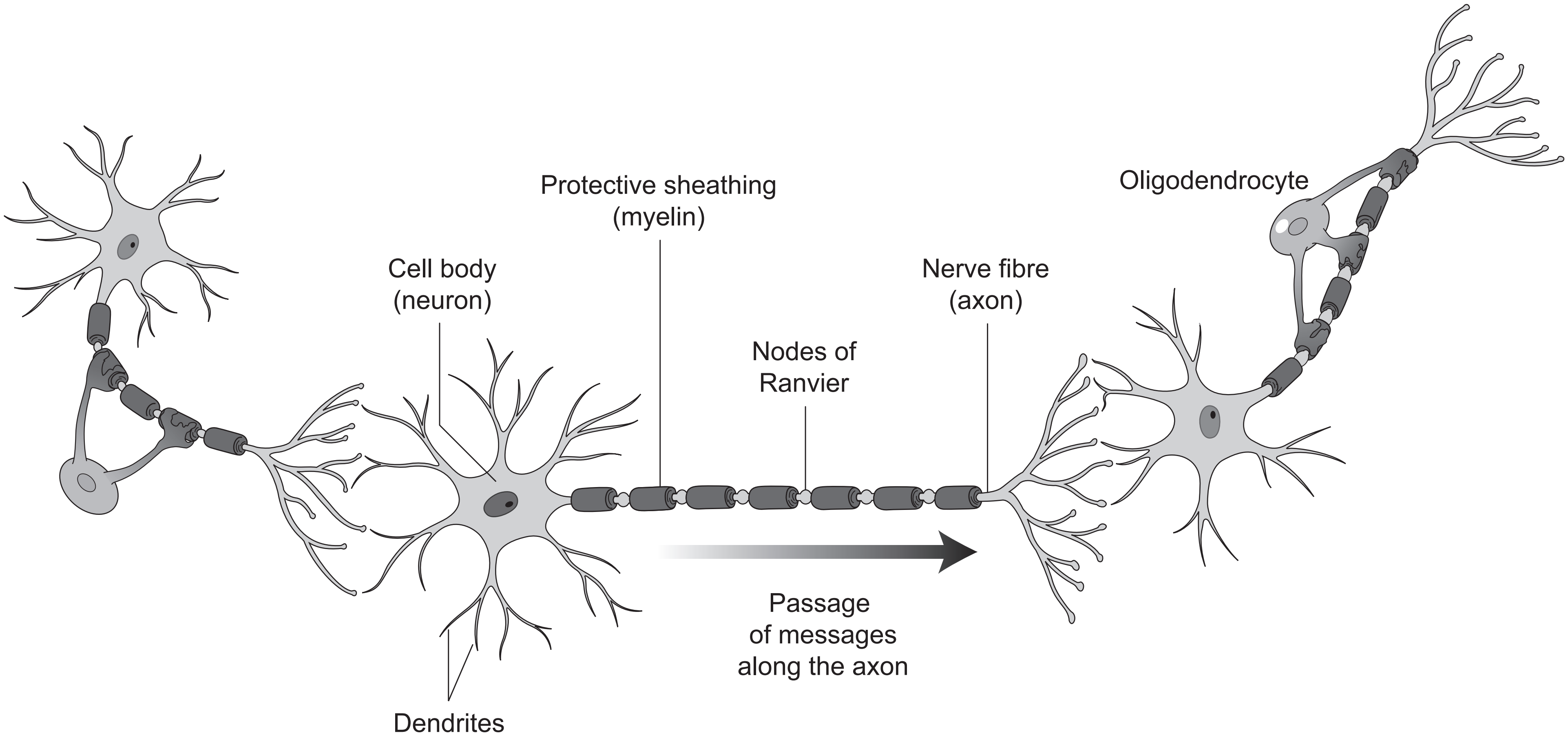
Individual nerve cells, called **neurones**, are made up of a **cell body** – which has multiple extensions called dendrites to receive messages – and an **axon,** a fibre which passes the message along and on to the next nerve cell. Neurones are key components of the central nervous system. They connect to each other to form a complex network of pathways that conduct electrical impulses.

Oligodendrocytes are specialised cells in the brain and spinal cord that form myelin. Myelin wraps around the axon and protects it – much like insulation around an electrical wire – so that messages can pass along the nerve quickly.

nerve cells in the brain



nerve cells in the brain



how MS affects the body

how MS affects the body

In health, the immune system protects us from bacteria, viruses and toxins - known as antigens. These can enter the body, for example, through a cut or through the mouth or nose, activating an immune response. Immune cells – known as white blood cells or lymphocytes – are made in the bone marrow or in the thymus gland. Glands and organs throughout the body then help the immune cells grow and enter the blood stream.

The blood vessels of the central nervous system are structurally different from blood vessels in other parts of the body. The central nervous system is protected from unwanted cells entering by a delicate tissue of cells that are joined together by tight junctions. This structure is known as the blood-brain barrier. The blood-brain barrier allows useful products such as glucose to get into the nervous system. However, it also acts as a defence mechanism and is resistant to the passage of immune cells that may be trying to cross over from the blood into the nervous system.

When MS is active, it is thought that the blood-brain barrier becomes damaged (the cause of this is as yet unknown), resulting in activated immune cells, such as **lymphocytes**, getting into the central nervous system. Once these activated cells get into the central nervous system, they play havoc. Mistakenly, they see myelin as foreign material and start attacking it. This is known as an autoimmune response.

The immune cells attack myelin by secreting toxins that cause inflammation and swelling of the myelin sheath. The inflammation blocks the passage of messages along the nerve fibre (axon) and gives rise to symptoms seen at the time of a relapse. (For example, if the swelling occurs on the optic nerve, the disrupted signal may cause symptoms such as double vision, pain behind the eye or decreased colour vision). As well as causing the inflammatory swelling, these cells may also break down the myelin in a process known as demyelination.

The combination of episodes of inflammation and demyelination are responsible for the acute symptoms that occur during a relapse.

These areas of damage in MS are often referred to as lesions or plaques and show up as white dots on a MRI scan.

IMMUNE SYSTEM CELLS

The immune system is very complex and involves many cells. The cells of particular importance in MS are:

T cells become activated when specific, recognisable antigens (viral/bacteria particles) are presented directly to them. Once activated, T cells multiply and with their many daughter cells, roam the body looking for more target antigens (viral/bacteria particles). T cells produce chemical messengers called cytokines that destroy their captured antigen.

T cells can be divided into two main types – T_1 which are responsible for increasing inflammation and T_2 which suppress and dampen down the inflammatory effect. In MS, the mechanism to keep the balance of T cells in check is not working properly, causing them to trigger an inflammatory cascade of events.

B cells which wait around quietly until they are stimulated by a specific antigen (viral/ bacterial particles). Once stimulated, they multiply and their daughter cells produce antibodies which can destroy viruses, bacteria or toxins. In MS, T cells stimulate B cells to produce antibodies specific to myelin causing demyelination and loss of oligodendrocytes.

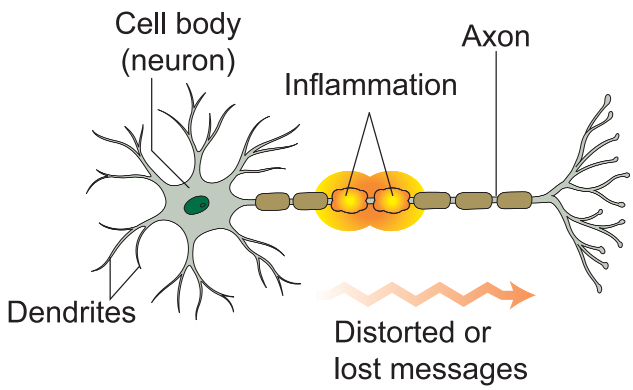
Interferons are anti-viral agents, of which there are three kinds: alpha, beta and gamma. In MS, gamma interferon plays a key role in causing demyelination as it activates and stimulates cells such as T cells to release toxins.

Cytokines are proteins that alter the behaviour of other cells. In MS, cytokines influence other cells to cause inflammation.

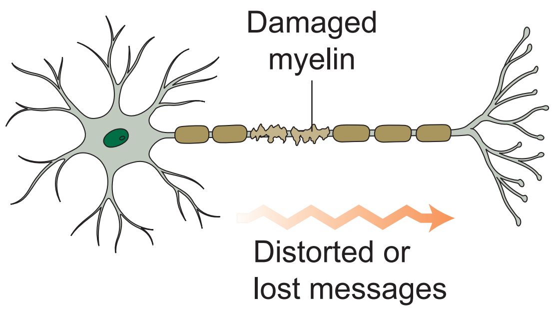
Macrophages are busy immune system cells that engulf foreign material and debris, destroying it. In MS, macrophages directly attack myelin and oligodendrocytes by releasing toxins that cause cell death and erosion of the myelin sheath.

how MS affects the body

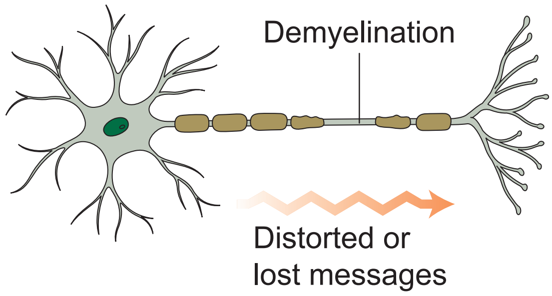
1. Axon with inflammation



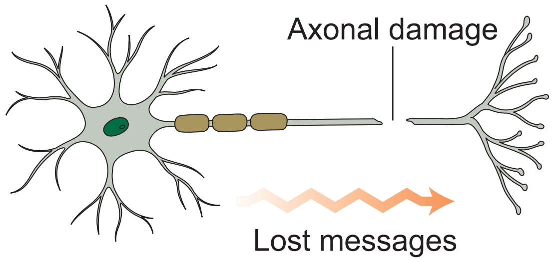
2. Axon with damaged myelin



3. Axon that is largely demyelinated

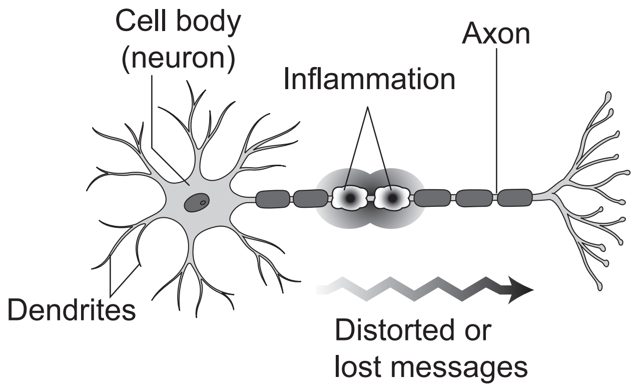


4. Demyelinated axon that is broken



how MS affects the body

1. Axon with inflammation



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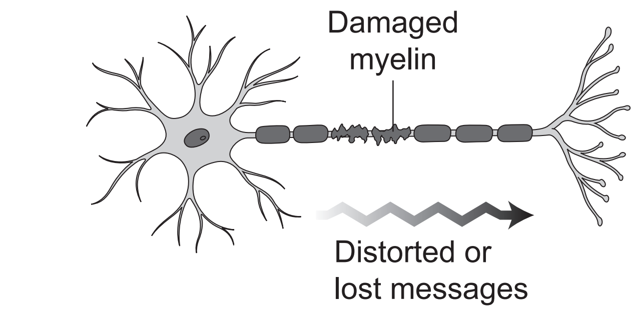

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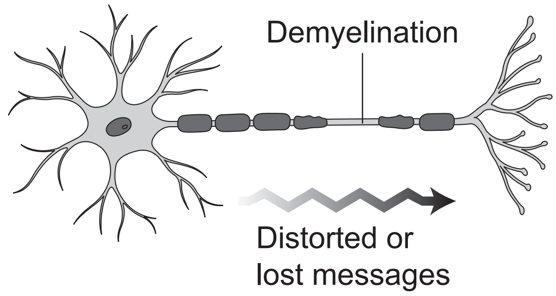

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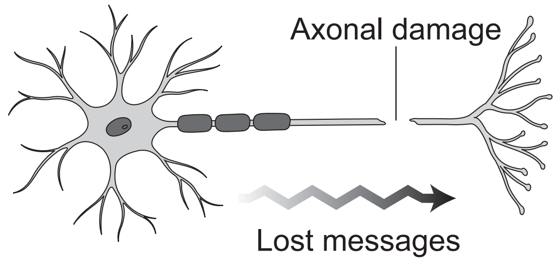
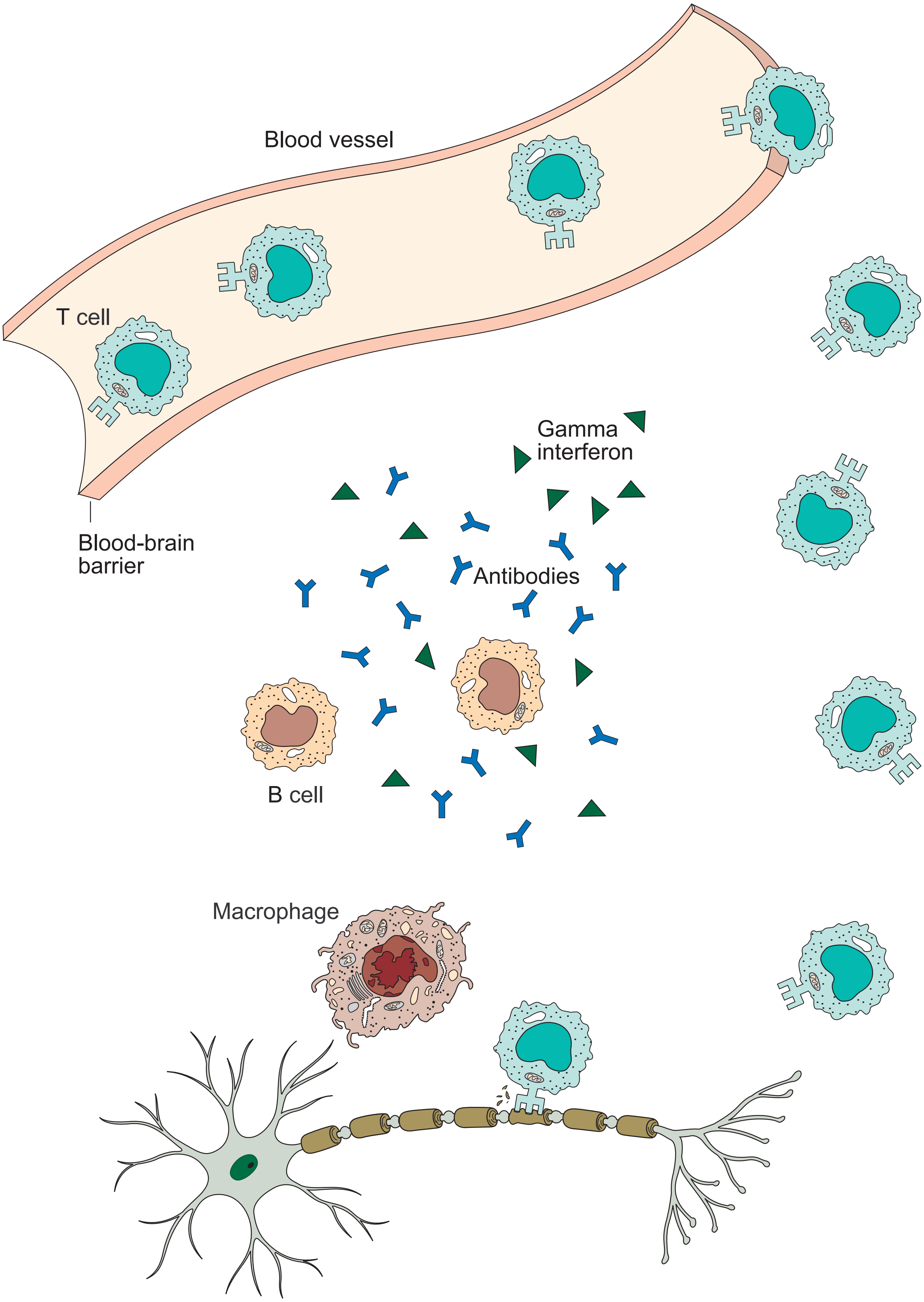
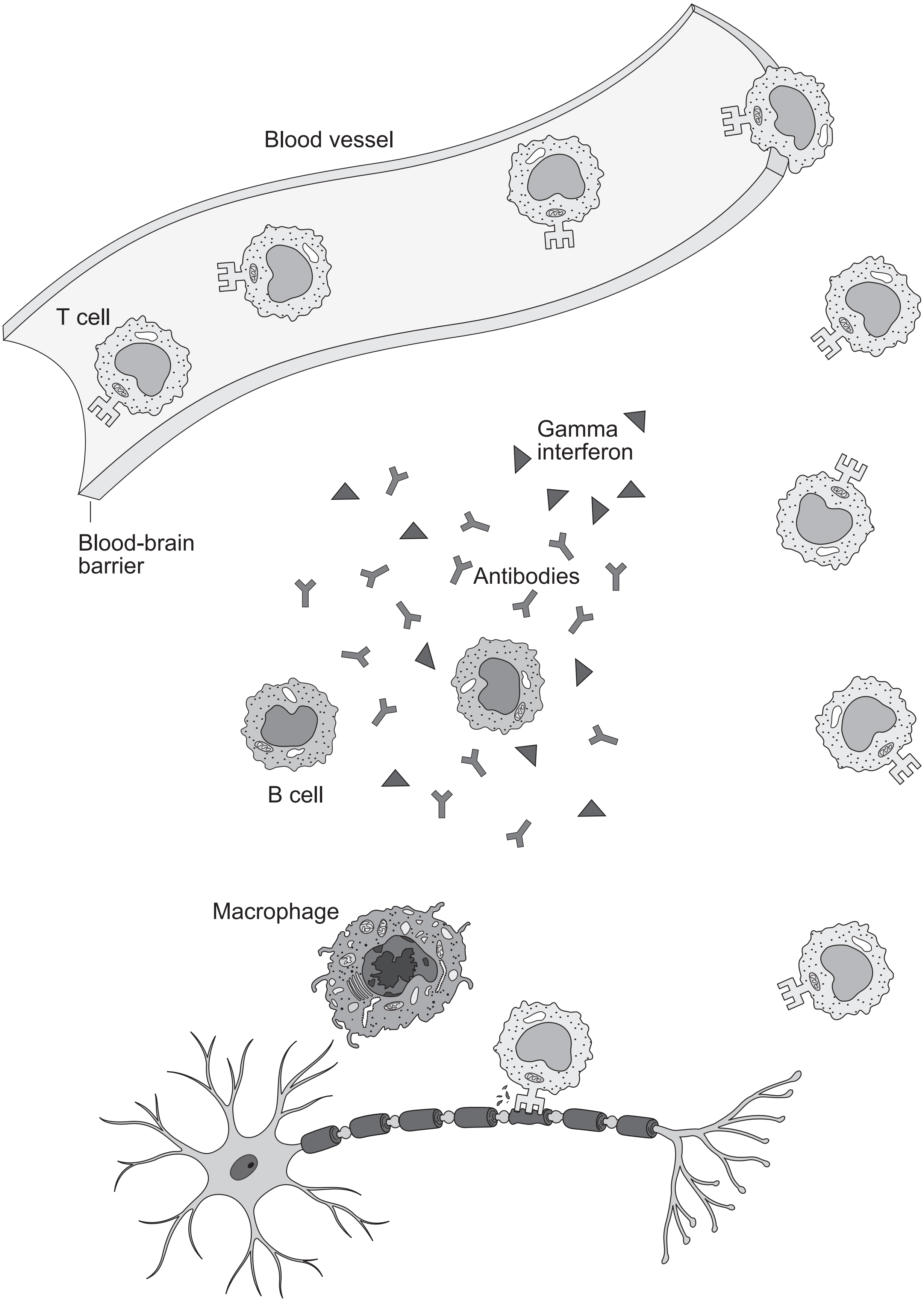


the immune system and MS



the immune system and MS



recovery

recovery

It was once believed that nerve damage was irreversible. However we now know that the body can make new myelin. This process is known as remyelination. Remyelination can take place, especially in the early stages of MS where the oligodendrocytes (myelin-making cells) remain healthy enough to repair damage.

There is no way of predicting how much remyelination may take place after an attack, but it is known that the type and amount of remyelination varies between different types and stages of MS. Although remyelination helps greatly in recovery, it is important to remember that myelin on any remyelinated axon is thinner than normal myelin so the passage of signals may be slower.

Scientists have suggested that in MS, inflammation not only damages myelin directly, but also creates an environment that makes remyelination difficult. This is because:

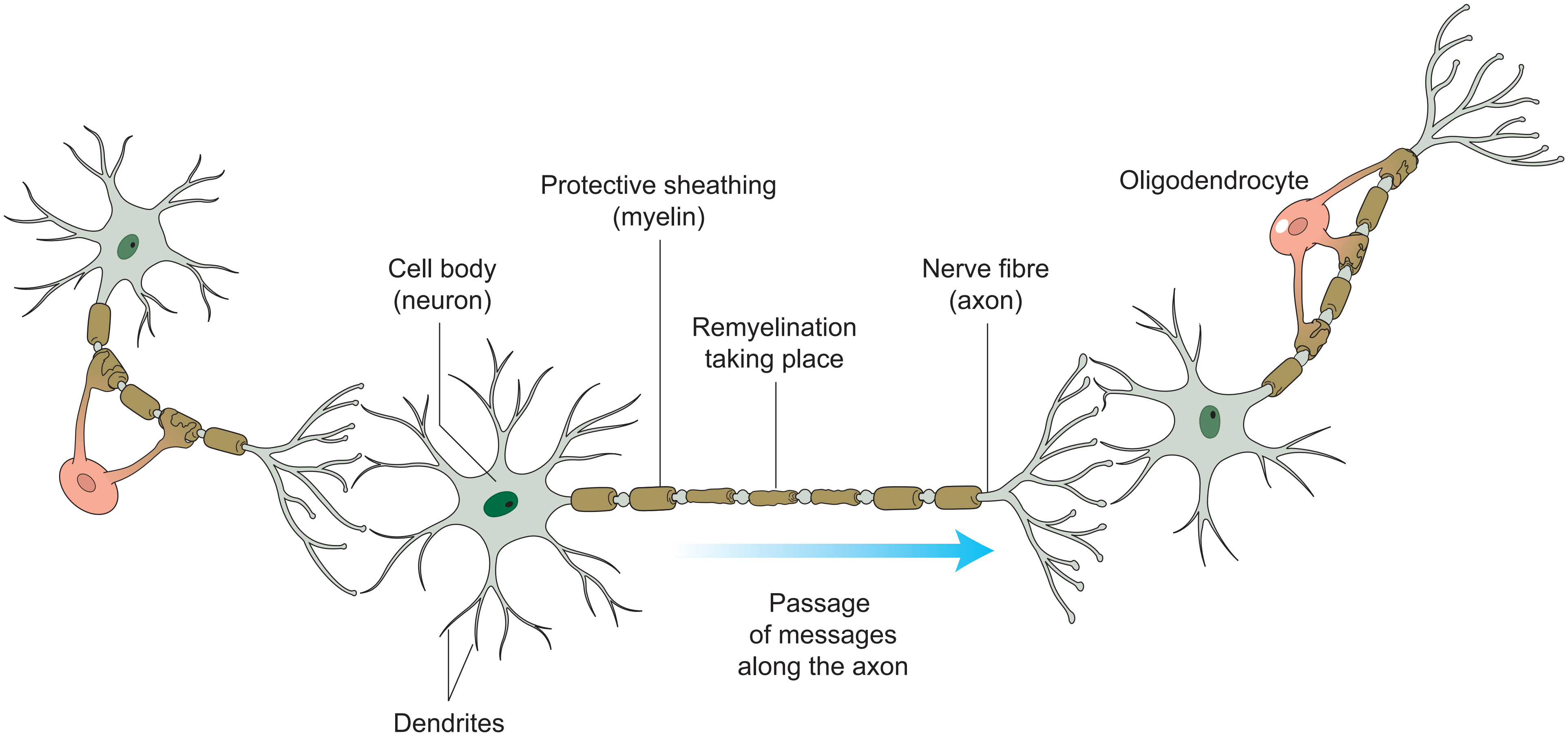
- Oligodendrocytes are lost
- MS inflammation causes signals that inhibit myelin production
- Injured nerve fibres are less receptive to remyelination

But a number of possible ways to improve remyelination are being investigated, including:

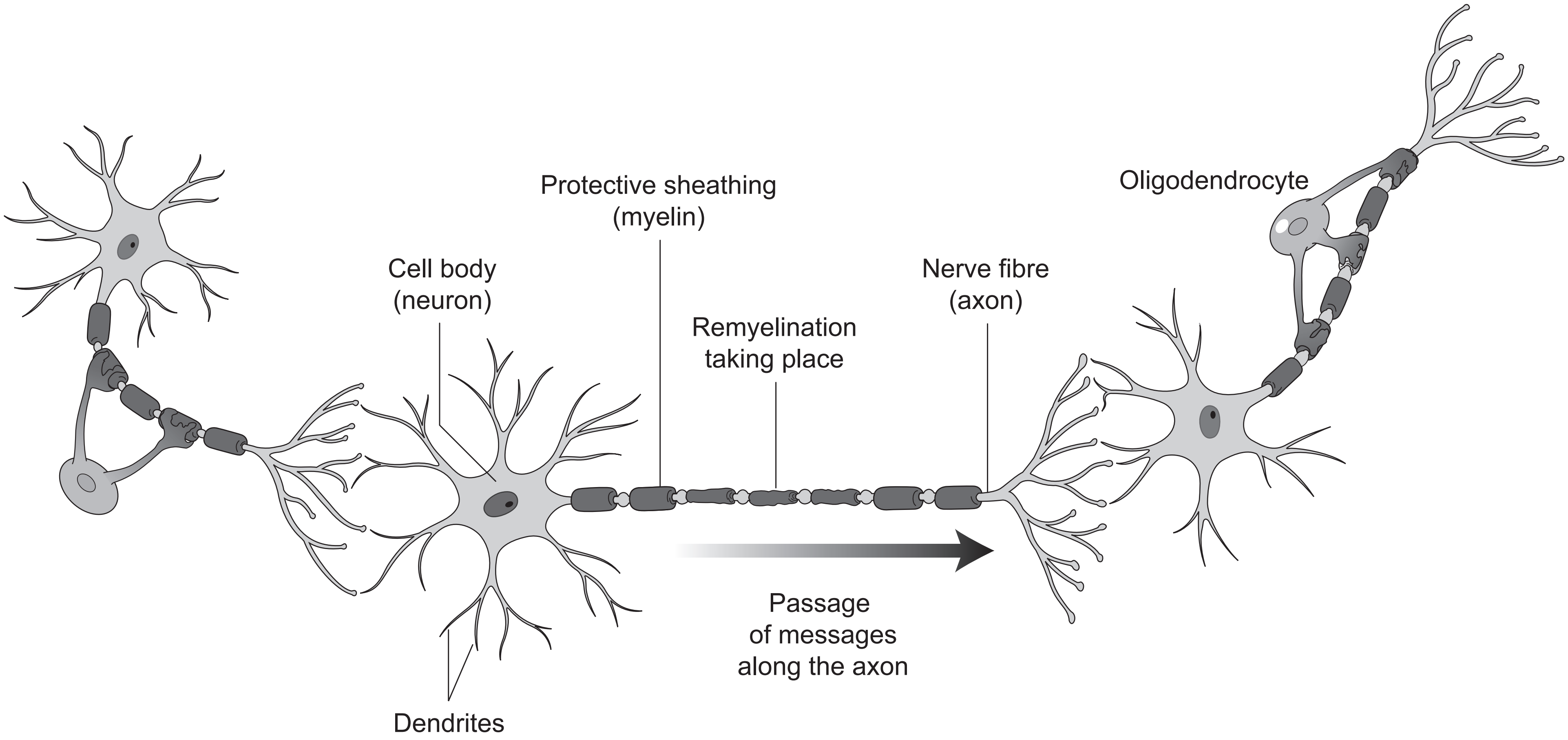
- Protecting oligodendrocytes from attack by the immune system
- Transplanting myelin-producing cells to replace lost oligodendrocytes
- Removing the signals which inhibit myelin production

Another way that the body can compensate against damage caused by MS is through a process called neuroplasticity. This is the lifelong ability of the brain to reorganise neural pathways based on new experiences. In the case of damage to an area of myelin, brain cells surrounding the damaged area can undergo changes that allow them to take on the function of the damaged cells. They can develop a new ‘circuit’ to replace the damaged one so that function can be returned.

recovery


Protective sheathing
(myelin)
Oligodendrocyte
Cell body
(neuron)
Nerve fibre
(axon)
Remyelination
taking place
Passage
of messages
along the axon
Dendrites

recovery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steroids – how they work

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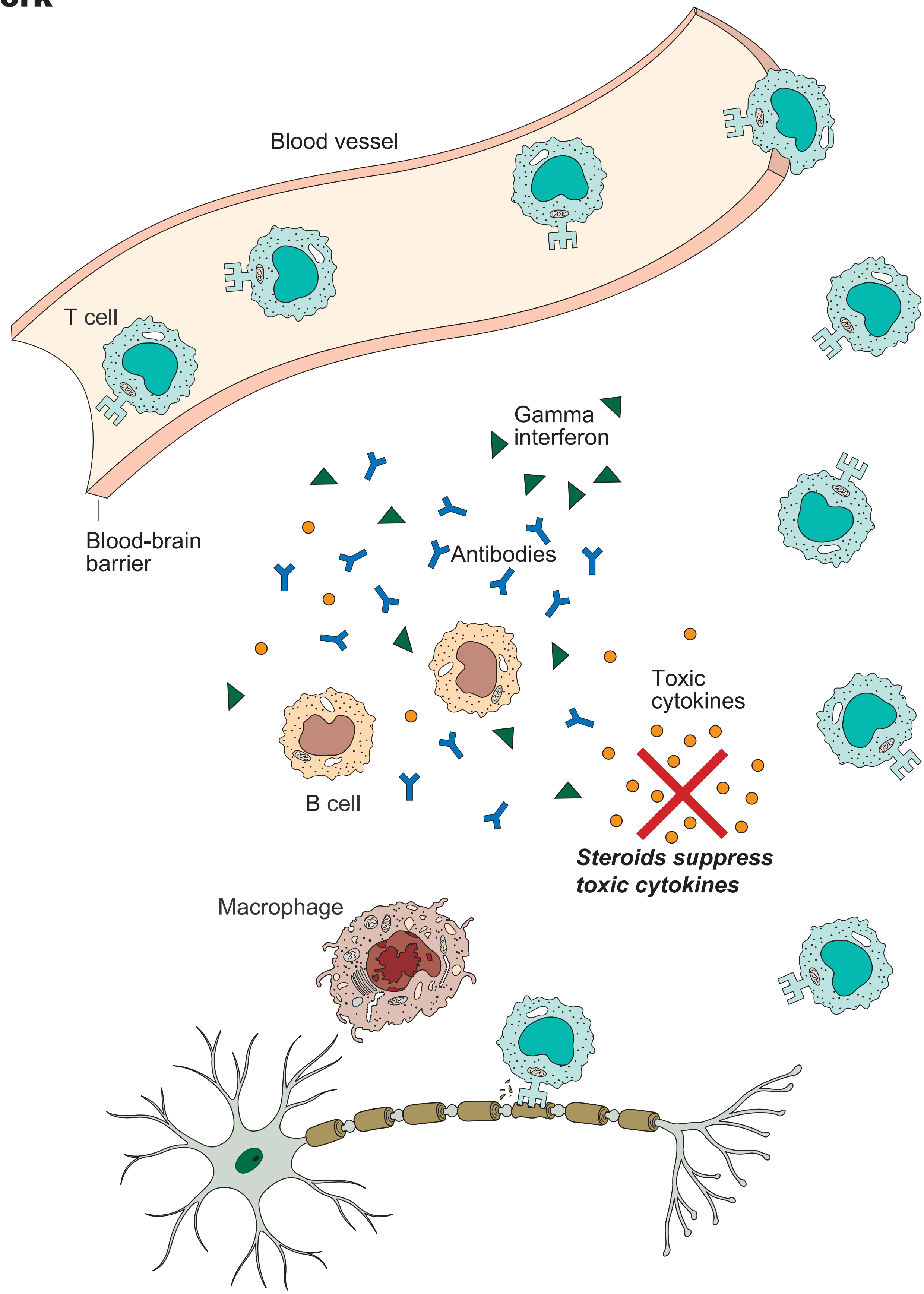
Steroids are naturally occurring hormones secreted by the adrenal glands. Steroids are released by the body in times of stress to help aid recovery and repair. In MS relapse management, a synthetically produced version of steroids is used to help boost the body's natural recovery mechanisms.

The exact way in which steroids work in MS is not known, however it is thought that they may help to:

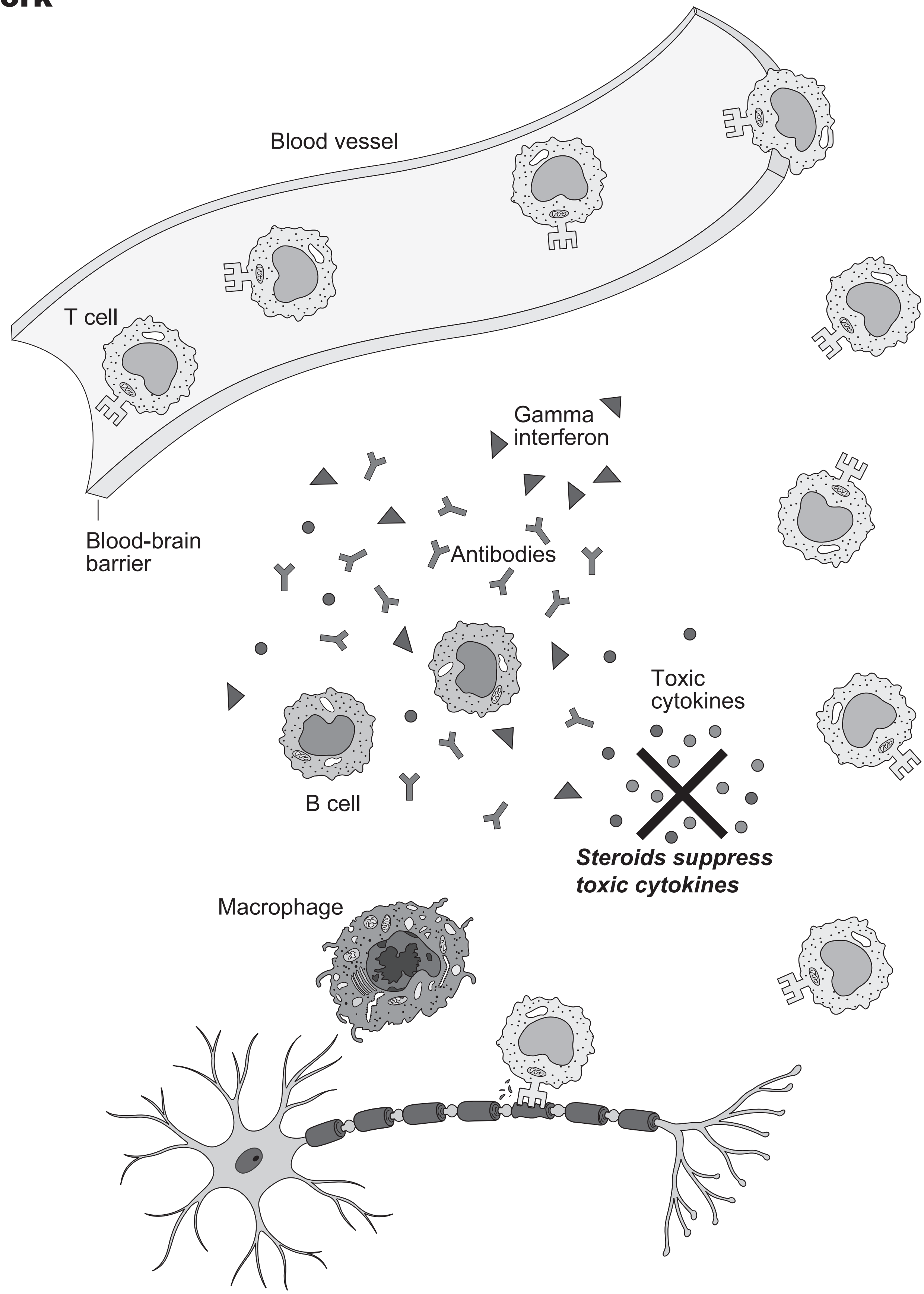
- Restore the integrity of the blood-brain barrier
- Suppress the activity of the toxic cytokines produced by the T cells
- Dampen down inflammation of the myelin

Overall, steroids can help shorten the period of recovery in a relapse but they do not appear to affect the overall course of the condition or how much residual damage will be left by the attack.

steroids – how they work



steroids – how they work



Glatiramer acetate (Copaxone) – how it works

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Glatiramer acetate (Copaxone) is a synthetic compound that resembles one of the proteins involved in making myelin (myelin basic protein). It is licensed for use in relapsing remitting MS and has been shown to reduce relapse rates by an average of 30%.

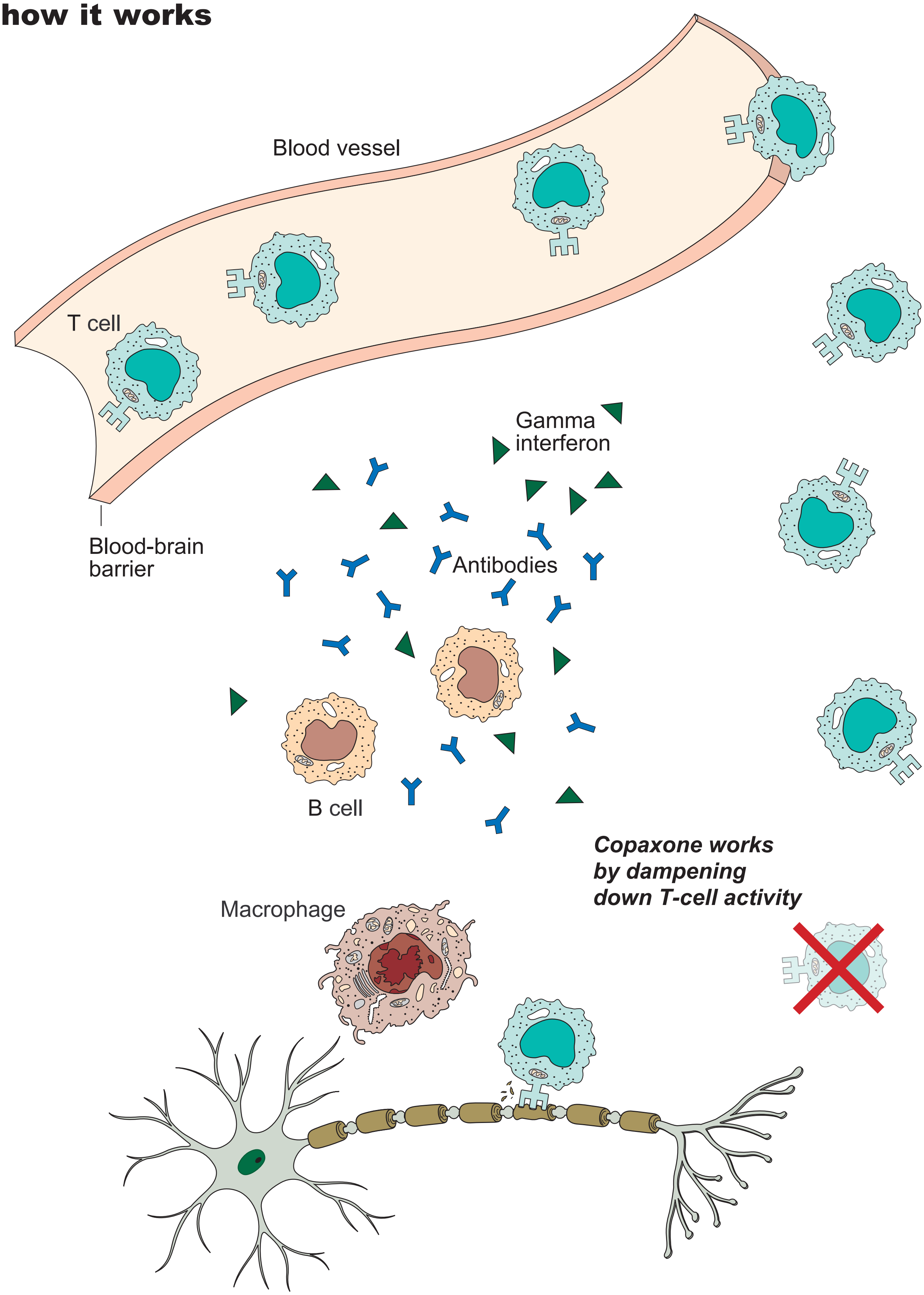
It is given daily by injection under the skin. Once injected, it is thought to work by connecting to cells in the immune system that cross the blood brain barrier to reach the myelin sheath under attack.

Glatiramer acetate (Copaxone) is thought to reduce relapses by:

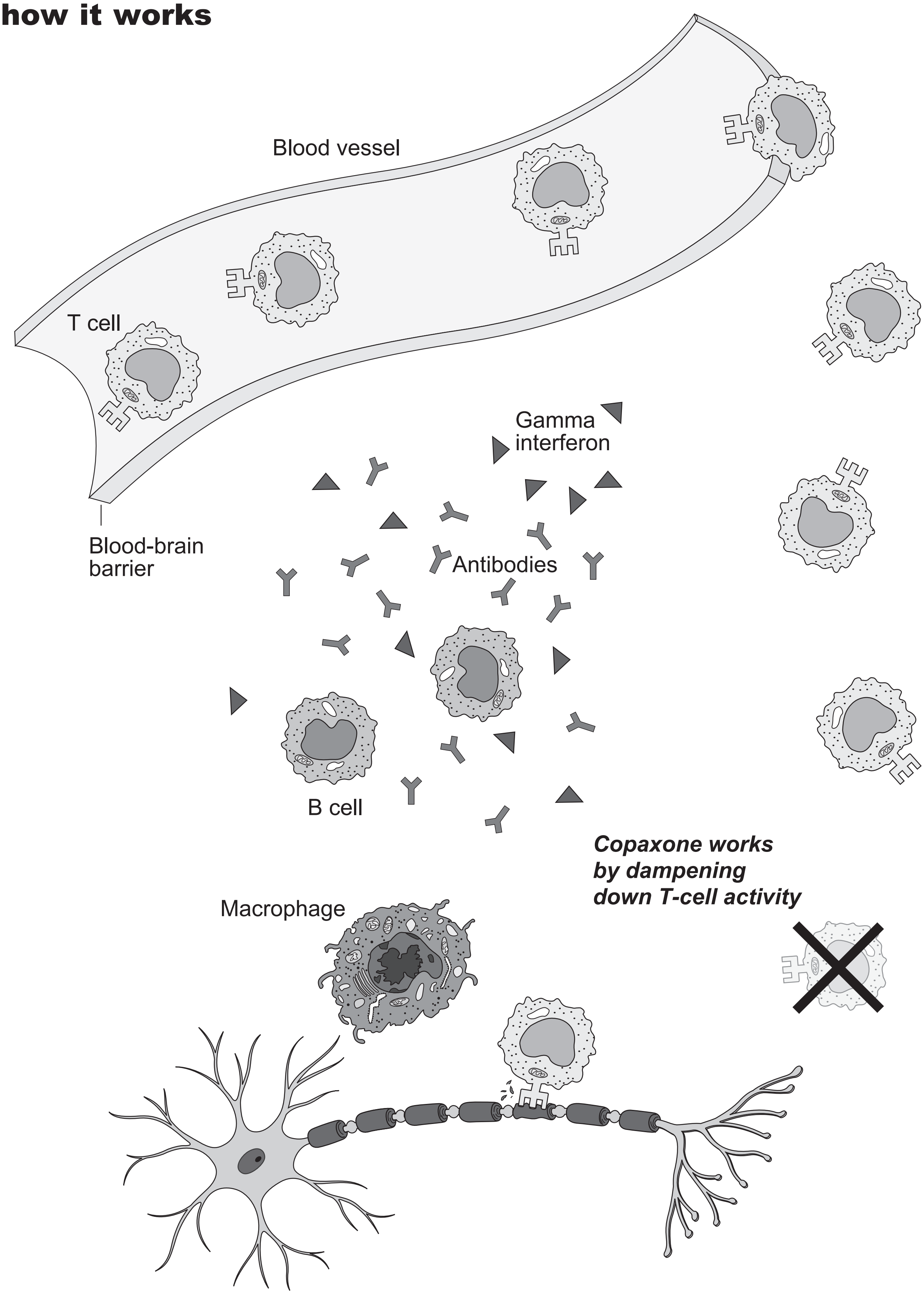
- Blocking T cells from attacking myelin
- Stimulating the body to produce substances which reduce inflammation by switching off the attacking (T_1) cells and increasing the suppressor (T_2) cells. This helps dampen down the inflammatory process.

The criteria for assessment for disease modifying drugs are set out by the Association of British Neurologists.

Glatiramer acetate (Copaxone) – how it works



Glatiramer acetate (Copaxone) – how it works



SECTION 7

Beta interferon – how it works

Beta interferon – how it works

Interferons are proteins that are naturally produced in the body. The body produces three types of interferon: alpha, beta and gamma. All play important individual roles in protecting the body against infections and viruses. Early MS research showed that gamma interferon is involved in causing the damage to the myelin during attacks.

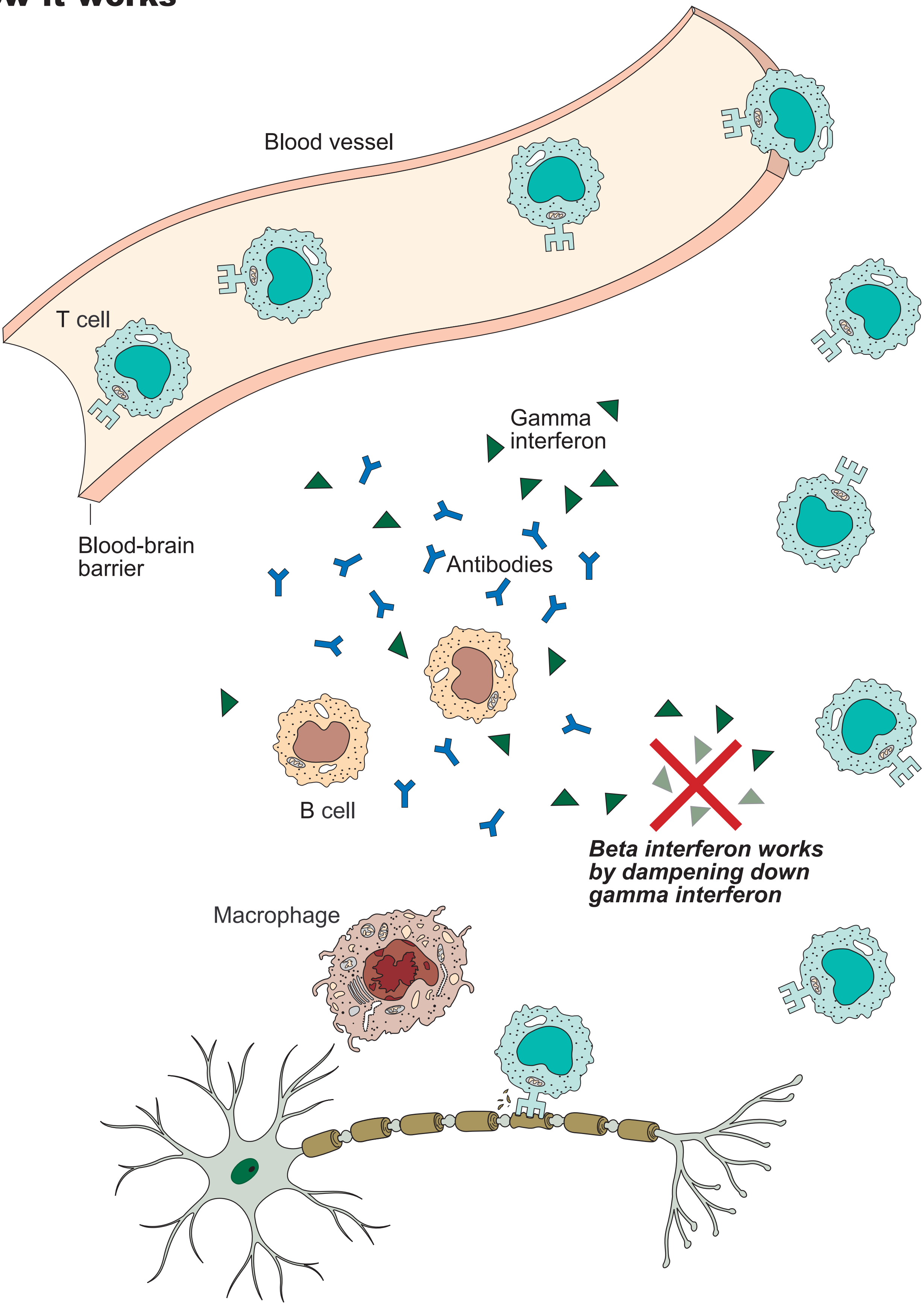
As beta interferon was known to dampen down gamma interferon, trials were carried out to see if it could help reduce attacks. Like Copaxone, the trials showed that beta interferons reduce relapse rates, on average, by 30%. There are three types of beta interferon available and all are given by injection. Each product has its own dosage and frequency instructions.

Overall, it is thought that beta interferon reduces relapses by:

- Reducing the passage of T lymphocytes through the blood-brain barrier
- Inhibiting the production of gamma interferon and toxins
- Suppressing T lymphocyte activity
- Increasing the suppressor (T_2) cells to help dampen down the inflammatory process.

The criteria for assessment for disease modifying drugs are set out by the Association of British Neurologists.

Beta interferon – how it works



Beta interferon – how it works