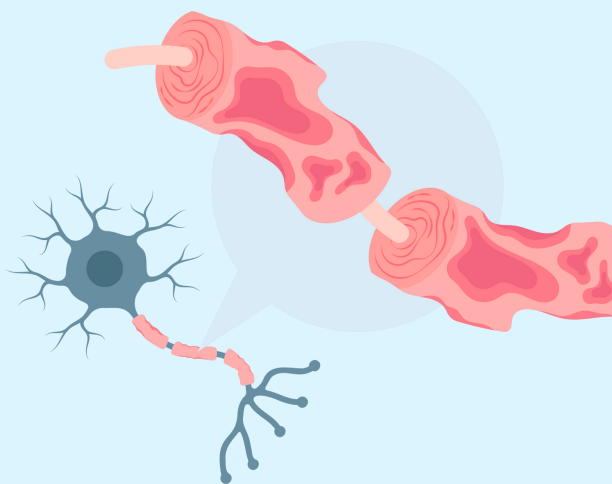


How can normal appearing tissue help us understand MS?

Multiple sclerosis (MS) is the result of the immune system mistakenly attacking the brain and the spinal cord, causing tissue damage (**lesions**) throughout. Scientists at the Netherlands Institute for Neuroscience have come one step closer to understanding it, focusing not on the damaged tissue, but on the so called '**normal-appearing white matter**' tissue instead.

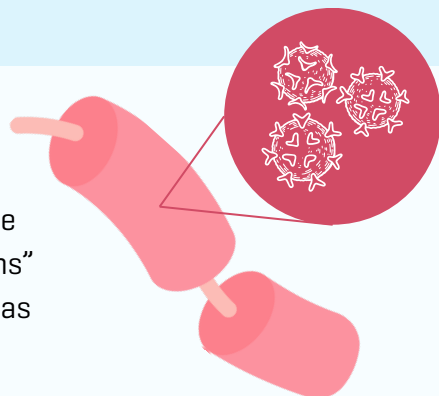


People with MS have clusters, or **nodules**, of a specific type of immune cell known as microglia [the vacuum cleaners of the central nervous system]. Still, their relation to MS was unclear as they appear in almost all brain disorders as well as the aging brain. Whether these nodules are related to the progression of MS was still unclear.

To learn more about nodules in MS patients, researcher Aletta van den Bosch, investigated the pathology of MS in brain donors at the Netherlands Brain Bank. "We found that patients with these nodules have a **worsened pathology**: they have more lesions and the lesions are more active." When van den Bosch looked into their genetic profile, she found more stunning differences.

Mini Lesions

"We recognized many of the genes in the MS nodules quite quickly because they're also expressed in the active lesions" van den Bosch explains. "They could almost be described as mini-lesions".

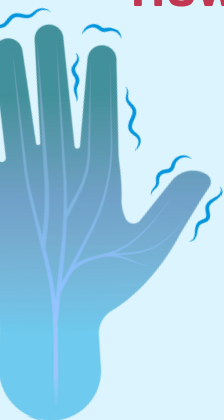


These mini-lesions were absorbing and processing **oxidized lipids**, which is known to result in activation of these microglial nodules. "In fact, when we looked at nerve cells surrounded by the nodules at high resolution, we saw that they were associated with partial damage.

These nodules likely arose to clean up the oxidized myelin. Nodules that are activated by both the lymphocytes and these oxidized lipids may become **very inflammatory**, causing more damage to the tissue surrounding them, leading to a sort of downward spiral."

How will this lead to long-term solutions?

In the end, van den Bosch hopes this will result in new therapeutic targets to stop the progression of MS. "Now we need to explore the relationship between all these inflammatory components in the lab so we can understand exactly what leads to these early signs of breakdown. After that, we can start thinking about which steps we can remove from this process to avoid the development of new lesions altogether."



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