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Innate immune cell transitions during auto-immunity and inflammation

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Summary

Our immune systems are designed to protect us, but sometimes they malfunction and attack our own bodies. In diseases like multiple sclerosis (MS), this goes wrong in the brain, where immune-like cells called microglia — which normally act as the brain's maintenance crew, keeping neurons healthy — switch into a damaging, inflammatory mode. This ultimately harms the nerve cells that control movement, leading to paralysis.

Scientists have spent years studying what triggers microglia to turn harmful. But far less is known about how microglia carry out this switch at a molecular level — that is, what internal machinery drives the change. This matters enormously, because current MS treatments work by broadly suppressing the immune system (for example, wiping out immune cells called B cells). While this slows the disease, it leaves patients vulnerable to infections and doesn't cure them.

This research focuses on how key transcription factors modulate microglial ability to transition from homeostasis to a proinflammatory state.

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