

Exploring novel macrophage-associated mechanisms in autoantibodies-induced pain

by Zerina Kurtovic (ESR5)

You likely know somebody suffering from rheumatoid arthritis (RA). It might be obvious that joint deformations which these individuals have also come with a lot of pain. However, did you know that individuals affected by RA experience pain even prior to inflammation and deformation of joints? Furthermore, despite being treated with very effective treatments and achieving disease control many patients still live with joint pain. Our lab is interested in the reasons for these phenomena. We explore the link between pain and autoantibodies (antibodies against the patients' own body) which are also present in patients prior to disease onset and also despite disease control. We do this by injecting RA-related autoantibodies into mice. The mice then develop RA-like joint inflammation and changes in their behavior, e.g. they dig and move less and have an increased responsiveness to pressure applied to the paw, which represents measures of pain-related behaviors.

My particular project explores the involvement of an immune cell type called macrophages and autoantibody-induced joint pain within the scope of the TOBeATPAIN network. More precisely, on how immune cells effect nociceptors (special neurons-cells transmitting pain stimuli) in specialized structures called dorsal root ganglions which are part of the peripheral nervous system. We think that targeting receptors on macrophages can help attenuating pain for the many patients whose life quality is diminished. Our initially findings also show that the involvement of these immune cells in signaling toward pain sensing neurons could be what causes differential pain sensation between sexes. If proven to be the case, this could guide clinicians on how to adapt the treatment of pain conditions to better fit the individual patients.



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