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Will interference with cholinergic effects have implications on the inflammatory process in arthritis?

S Forsgren
Umea University, Sweden

The existence of a non-neuronal cholinergic system is nowadays shown for several organs in the body. In our research group, we have shown this for tendons and the intestine. It is well-known that there is no cholinergic innervation of the synovial tissue of joints. It can therfore be asked as to whether there is a substrate for cholinergic effects in joints. With the above described facts in mind, we have studied knee joint synovial tissue of patients with rheumatoid arthritis (RA) as well as osteoarthritis (OA) and found that there is an expression of the acteylcholine (ACh)-synthesizing enzyme choline acetyltransferase (ChAT) in local cells in the synovial tissue, favouring that there is a local production of ACh in this tissue. In the synovial lining layer the expression is, however, restricted to expression at the mRNA level. We and others have also shown that there is a marked expression of the nicotinic acetylcholine receptor AChRα7 (α7nAChR) in the synovium of human joints. A background fact when interpreting these findings is the newly established concept that there is a cholinergic anti-inflammatory pathway in the body, meaning that ACh released from nerve fibers and local cells, such as inflammatory cells, can have an anti-inflammatory effect. The most important ACh receptor in this respect is the α7nAChR. This raises the question as to whether induction of more ACh effects would be useful in arthritic conditions. The α7nAChR can also promote attempts for repair. Several researchers have actually suggested that interference with the effects of ACh, mainly via effects on the α7nAChR, can be a new strategy in the treatment of arthritis. Experimental studies showing that nicotine can inhibit the TNF-alpha pathway for synoviocytes and that absence of the α7nAChR in knockout mice leads to aggravated arthritis favour this suggestion. There is also evidence which suggests that the cholinergic anti-inflammatory system is suppressed in rheumatoid arthritis, a fact that can be a drawback. A treatment strategy leading to increased ACh effects for humans is nowadays treatment leading to decreased effects of the ACh-degrading enzyme acetylcholinesterase in mb Alzheimer and myastenia gravis. Further studies aimed at evaluating the effectiveness of interference with ACh effects in inflammatory situations, like in arthritis, should be performed.

sture.forsgren@anatomy.umu.se