

## Activation of Microglial Cells: the Bridge between the Immune System and Pain in Central Nervous System

After neural cells and oligodendrocytes, microglial cells are the 3rd well known cell population in brain and spinal cord; though their population "outnumber neurons in the central nervous system" (1).

Recent studies have shown the role of microglial cells in development of pain; especially, the differences between age and gender groups have been claimed to be related to microglial activity in neuropathic pain (1). The role of microglial cells is proposed to challenge with antigens crossing the blood brain barrier or blood nerve barrier and prevent their effects in the central nervous system. Microglial cells function as macrophages; however, as other tissue-resident macrophages, microglial cells are descendants of yolk-sac-derived erythro-myeloid progenitors; this may explain why chronic pain is not a matter of concern in neonates (1, 2).

In this issue of the Journal, Nasser et al have described their findings regarding the role of microglial cells in arthritic pain. They have found that during different stages of Complete Freund's adjuvant (CFA)-induced inflammation, activation of microglial cells plays significant roles in aggravation of pain related behaviors; also, the occurring inflammatory symptoms would alleviate with steady injection of minocycline which is an inhibitor of microglial cells (3).

Though the role of microglial cells and also, the inhibitory role of minocycline on microglial cells has been described earlier in some studies, the study published by Nasser et al demonstrates both the role of spinal microglia activity in induction of pain behavioral symptoms and also, the relief of pain related behavior after minocycline administration. These findings could help us open newer windows towards treatment of arthritic pain. For example, Matsumura et al demonstrated that intrathecal administration of NP-1815-PX (a selective antagonist of P2X4R) could

produce anti-allodynic effects (4, 5); in turn, on the surface of microglial cells, expression of P2X4 receptors is regulated at post-translational level (6). We also know that adenosine triphosphate (ATP)-gated cation channel P2X receptors located on microglial cells are highly involved in CNS pain mechanisms and their antagonism is a potential role in future of clinical pain control (5, 7-9).

The findings of Nasser et al once again demonstrated us that treatment of pain is in evolution. Though this pathway has not reached its final destination, alternate drugs except for opioids are going to emerge with less unwanted drug effects and more targeted pharmacologic effects. The future of pain control seems to emerge from bench-to-bedside studies, demonstrating another feature of translational medicine.

## References

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