Editorial

Hackers always attempt to hijack macrophage!

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The world is trying to fight it out with pollution as well Indian government is trying by a solid campaign as "Swachh Bharat mission.[1]" Amusingly, a similar situation has always taken place inside our bodies i.e. by immune system. In an average of 37.2 trillion cells present in human body; some specialized cells constantly executed to clean up the unwanted things in the environment; like cells are dying, bacteria wandering, and the virus is trying to take over on a large scale. Our immune system has always been fighting to destroy these intruders and clear the mess. In particular, the macrophage cell lead the cleanup process, which is consider as a type of white blood cell that execute all the cleanup process by its ability to locate and phagocytosis the particles such as parasites, bacteria, viruses and fungi.[2] Macrophages are monocyte-derived myeloid cells that develop from common myeloid progenitor cells present in the bone marrow of adult mammals. Monocytes move through the bloodstream, and when they leave the blood, they mature into macrophages and live for months, patrolling different cells and organs to keep them clean. There are different cascade, where monocytes derived from the progenitor cells includes the cytokines granulocyte-macrophage colony stimulating factor (GM-CSF), granulocyte colony stimulating factor (G-CSF) and macrophage colony stimulating factor (M-CSF).[3] According to the natural resident, the macrophage populations divided in to lung (alveolar macrophages), bone (osteoclast), nervous system (microglia), connective tissue (tissue cells), intestine, liver, Spleen and peritoneal ones. Each tissue specific macrophage population shows a functional spectrum associated with the different gene expression patterns associated with the tissue specific microenvironment in which it resides. The purpose of the macrophage is very simple to keep all the cells clean in to two processes i.e. by cure or by prevent from the foreign particles. Therefore, macrophages have their own fascinating ability to recognize by examining which cells remain viable and which cells have to destroy. Simply some tags which differentiate between these two proteins where macrophage recognizes and do the rest process according to the need. Broadly the macrophage function is divided into two activity i.e. M1 activity and M2 activity. M1 activity causes tissue damage by inhibiting cell proliferation while M2 activity goes for tissue repair by promoting cell proliferation. The nomenclature of M1 and M2 is chosen because they promote Th1 and Th2 responses and their reaction products (e.g. IL-4, IFN-γ) also down-regulate M1 and M2 activities. In different situation both the activities promote alternate responses (M1 and Th1; M2 and Th2) and work in concert to produce immune responses actually needed for diseased characteristics. It is worth noting that the molecules responsible for these "Fighting" (NO) or "Immobilization" (ornithine) activities are all produced by arginine and by enzymes that undermine each other (iNOS and arginase).[4]

Research has been initiated from the discovery of macrophage by Élie Metchnikoff, a Russian zoologist, in 1884.[5] But the research needs more consistencies in terms of successful outcome as because pathogens (hackers) always attempt to hijack macrophage by means of immune evasion and macrophage polarization. In cancer, macrophages contribute to tumor growth and progression. Macrophages released inflammatory compounds such as tumor necrosis factor (TNF)-alpha which activate the gene switch nuclear factor-kappa B (NF-κB); which in turn enters the nucleus of tumor cells and opens the generation of a protein that stops apoptosis and promotes cell proliferation and inflammation.[6] In Kala-azar disease, *Leishmania* formulates strategies to evade host immune mechanisms to survive in the host. In fact, a sustained battle between the host's powerful immune response and the parasite's anti-evasion strategy will ultimately determine the fate of the disease. The mechanisms of immune evasion by *Leishmania* species included the following mechanisms where it can establish a successful infection: Modification of the complement system and phagocytosis, alteration of toll-like receptor pathways, surviving in the phagosome, defective antigen presentation and co-stimulation, alteration of host cell signaling, modulation of cytokines and chemokines, modification of T cell responses.[7] Likely to the *Leishmania* parasite, toxic *M. tuberculosis* inhibits apoptosis and triggers the necrosis of host macrophages escape innate immunity and delay the onset of adaptive immunity. *M. tuberculosis*-mediated Regulation of eicosanoid formation determines the death pattern of infected macrophages; this in turn has a substantial effect on the outcome of the infection.[8] In the chikungunya virus, the nsP2 protein induces the ubiquitination of Rpb1, the catalytic subunit of the RNAPII complex, leading to its rapid degradation. Six hours after infection, Rpb1 could not be detected in infected cells. This degradation of Rpb1 occurs before other cellular modifications, such as apoptosis, autophagy or inhibition of STAT1 phosphorylation, can be detected. Therefore, nsP2-mediated degradation of Rpb1 appears to be the primary mechanism used by the OW virus to escape cellular antiviral responses.[9]

Like above many more pathogens and causative agents of diseases first target to macrophage to establish their disease, like atherosclerosis, obesity and insulin resistance disease, periodontal disease, HIV infections. However, macrophages play a crucial and potentially decisive role in the outcome of above all cases. More and more regenerative medicine research has begun to timely regulate macrophage phenotype concepts and it has been shown that macrophage phenotypes can be regulated by biological materials to have improved tissue remodeling and long term functional outcome. This suggests that the strategy of providing control of macrophage phenotype may achieve greater success in regenerative medicine applications. A better understanding of the context-specific biological mechanisms that underlie macrophage responses and macrophage polarization transitions is of value in developing constructive and functional tissue remodeling responses that promote site promotion rather than harmful persistent inflammation and scar tissue formation.

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Conflict of interest

The author declares no conflict of interest.

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