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Perspective

Cell Function of Macrophages in the Development of Oral Organ's Soft Tissues

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DESCRIPTION

Macrophages have been demonstrated to be malleable cells with the ability to operate in a pro- or anti-inflammatory manner (M1 or M2). Lipopolysaccharide (LPS) or Interferon (IFN-) are the activation for the induction of M1-like macrophages, which produce cytokines such as Tumour Necrosis Factor (TNF)-a, Interleukin (IL)-1, and IL-6. They have large quantities of inducible Nitric Oxide Synthase (iNOS), which produces Nitric Oxide (NO), starting the processes that kill bacteria. In addition to their role in tissue remodeling, angiogenesis, and cell proliferation, M2-like macrophages have an anti-inflammatory effect. *Mannose receptor (CD206)*, *Arginase 1 (Arg1)*, and *Transforming Growth Factor-beta (TGF-β)* are just a few of the genes expressed by M2 macrophages. On their shared substrate, L-arginine, arginase and iNOS expression compete. *Arg1+* macrophages prevent osteoclast development by fighting with iNOS. Notably, new research indicates that the markers for M1 and M2 macrophages can coexist in the same cell, indicating a hybrid state of macrophages. The plasticity of the M1/M2 transition, as revealed by a recent study, further emphasises the need for therapeutic trials incorporating macrophage polarisation in the future.

Due to macrophages' remarkable flexibility and their presence in a variety of illness states, they have been classified into subtypes based on how they perform. According to studies conducted in the last ten years, M2 macrophages can be further divided into M2a, M2b, M2c, and M2d; however, more research is required to fully comprehend these subtypes *in vivo*. An in-depth understanding of the sub-populations and heterogeneity of macrophages, including but not limited to their gene profiles, roles, and the communication between cell types, is imminent with the advent of single-cell RNA sequencing (scRNA-seq).

We will discuss the most recent research on macrophages in the oral organ's soft tissues, specifically the dental pulp, gingiva, and periodontal ligament.

Dental pulp

In a healthy dental pulp, fibroblast cells, resident stem cells, and macrophages are present. The removal of senescent cells, remodeling of the tissue after inflammation, and activators of the immune response by secreting cytokines and chemokines are all responsibilities of these resident macrophages for maintaining tissue homeostasis. However, nothing is known about how they behave when their teeth are developing and getting damaged.

The switch from M1 to M2 macrophage during tissue repair signifies that the tissue is prepared for stem cell proliferation and differentiation in order for repair or regeneration to occur. Our findings showed that M2 *CD206+* macrophages are critical for hastening the healing of post-damage dental pulp inflammation. The association between stem cell and macrophage function in the dental pulp is suggested by the rise in Wnt-receiving stem cells in the dental pulp, *TGF-1* expression, and decrease in apoptosis observed by increased Wnt activity. This was further supported by our finding that macrophages may be killed off chemically, which lowers the number of Wnt-responsive cells in the pulp. The next generation of dentine/dental pulp treatment may involve the modulation of stem cell niche and milieu using small molecules; as a result, practical application of macrophage information may be useful for extending the lifespan of a dental organ.

Gingiva

At the gingival barrier, the preservation of immunological tolerance and tissue homeostasis depends on tightly regulated immune cell networks capable of generating the necessary responses. Macrophages are a core immunological population in health and inflammatory illness due to their phagocytic capacity and functional adaptability. However, it is unknown about the ontogeny, heterogeneity, or function of gingival tissues. In a recent work, we found two macrophage populations that dynamically changed under various settings utilizing single-cell RNA sequencing of human gingival tissues. As both M1 and M2 classes displayed a pro-inflammatory phenotype, we were unable to distinguish between them clearly. In fact, recent single-cell investigations have argued against a binary definition of macrophages because it is based on imprecise *in vitro* data. The histological study of macrophage polarization in gingival tissues, however, revealed no differences in periodontitis, gingivitis, or health. Our investigation revealed that Macrophage 2 (M2) is

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enriched for antigen presentation pathways, while Macrophage 1 (M1) had increased complement transcript expression and a pro-angiogenic character. Both groups exhibit transcriptional regulator expression in bone metabolisms, including NF-B and NFAT signalling, according to transcription factor analyses.

Periodontal ligament

By recognizing bacteria metabolites like LPS via the Toll-Like Receptor (TLR4) and CD14 via LPS-Binding Protein (LBP), macrophages can get polarized into the M1 state. For the metabolism of hard tissues, M2 macrophage activation is advantageous since it can stop bone loss in periodontitis. In periodontitis, an imbalance of M1/M2 cells results in further tissue deterioration. The macrophage depletion experiment in dental pulp demonstrates their capacity to encourage the growth of Wnt-receiving stem cells, which are producing the dentintissue. In a P. gingivalis-induced periodontitis mouse model, macrophage removal also reduces pathogen and bone resorption; this suggests that these macrophages may play an analogous role in Periodontal Ligament (PDL). Since clodronate eliminates all macrophage populations, it is still unknown if a particular macrophage subpopulation plays a crucial role in controlling stem cells in the periodontal tissues.

CONCLUSION

Macrophages are incredibly flexible cells that are controlled by their microenvironment. Understanding the functions of macrophages in development in these tissues, their molecular profiles in homeostasis, the resulting damage, and the numerous actions and interactions with other cells are among the many unanswered problems regarding their role in the dental pulp, gingiva, and PDL.

The behavior of inflammatory macrophages and their interactions with tissue, resident non-immune cells have long been linked to the balance between cell removal after tissue damage and new cell formation to aid in repair. The main aim of the inflammatory response is to modulate the tissue environment by removing unwanted cells and recruiting cells and soluble factors from the bloodstream to help protect the damaged tissue against infective foreign bodies. Such procedures are necessary for tissue remodeling, repair, and regeneration where there has been injury. Macrophages exert their effects in various tissue repair and regeneration effects by exerting their marks in various tissue repair and regeneration effects by exerting their marks in multiple ways during these processes, and as a result, they play a significant role in tissue repair and regeneration. Furthermore, through 'cloaking' microinjuries and controlling neutrophil recruitment, resident macrophages are characterized as regulators of inflammation levels.