

Preface

Macrophages are tissue resident phagocytes derived from blood monocytes; they have diverse functions in development and immunity and display enormous phenotypic heterogeneity. Macrophages in different tissues have specialized and specific functions that support organ development and physiology, for example, kupffer cells in the liver filter debris from the blood and aid liver regeneration after injury, Langerhans cells in the skin are important immune sentinel cells and mediate immune surveillance, osteoclasts mediate bone morphogenesis, and microglia in the brain support the development and maintenance of neuronal networks. In response to inflammation or injury, monocytes are recruited into tissue and differentiate locally into macrophages and depending on the nature of the insult or injury these macrophages may acquire distinct phenotypes. Tumours are frequently infiltrated by large number of macrophages and in most cases this is linked with tumour progression and poor prognosis. Macrophage polarization is a poorly defined phenomenon; the mediators and mechanisms that maintain the phenotype of distinct macrophage subsets in both physiology and disease remain to be described. Based primarily on in vitro studies, two particular macrophage phenotypes have been described: “classically” activated or M1 macrophages are characterized by the production of pro-inflammatory cytokines and increased microbicidal or even tumoricidal activity. The second, “alternatively” activated or M2 macrophages, in contrast produce anti-inflammatory cytokines and are linked with angiogenesis, tissue repair, and remodeling. These polarized phenotypes have been described based on in vitro stimulation of macrophages with either interferon (IFN) γ , in the case of M1 macrophages, or interleukin (IL)-4 for M2 macrophages. It still is not clear what correlates these populations have in vivo and their physiological relevance remains ambiguous. While these classifications have been useful in that they allow the functional grouping of different macrophage phenotypes, M1 macrophages being pro-inflammatory cells and M2 macrophages linked with trophic functions and wound healing, there are undoubtedly several intermediates between these polarized phenotypes. However, this classification is too restrictive and it is clear that the functional diversity macrophages in vivo may not be associated with these distinct phenotypic subsets. In fact, the question remains in the context of inflammation and

tumours if “the macrophage” merely displays functional plasticity within tissue responding to environmental cues, or distinct stable subsets of macrophages exist with specialized functions. This issue is particularly pertinent in the case of TAM; these cells often display an M2-like phenotype associated with trophic functions promoting tumour angiogenesis, invasion, and metastasis. However, TAM also often produce pro-inflammatory cytokines and have been associated with the promotion of inflammation-associated cancer. This volume provides an overview of current research on the form and function of TAM, highlighting both the mechanistic roles they play in carcinogenesis and tumour progression as well as the molecular mechanisms that control their phenotype and function.

Marseille, France
London, UK

Toby Lawrence
Thorsten Hagemann

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