

Investigation of Immune System Cells in the Hepatic Microenvironment

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Description

Hepatocellular Carcinoma (HCC) is the most prevalent type of primary liver cancer in the United States and around the globe. HCC has the highest fatality rate of any cancer in the United States, and it is also the top cause of cancer fatalities worldwide, accounting for more than 700,000 deaths each year. So far, the adoption of novel diagnostic methods and therapeutic regimes has not resulted in a substantial decrease in mortality, as HCC is the second most deadly tumour in the United States, trailing only pancreatic cancer. In HCC, the hepatic microenvironment is made up of a diverse community of cells with unique genetic and phenotypic characteristics and impacts on tumour development.

TAMs are a key subpopulation that can support tumour development in the hepatic microenvironment, and distinct phenotypes with varied functional characteristics have been characterized. Because most studies to date depend on conventional *in vitro* and *in vivo* animal models (e.g., rodent models) that inadequately reproduce the Tumour Micro Environment (TME) in humans, the precise function of Transient Abnormal Myelopoiesis (TAMs) in the promotion of HCC is contentious and poorly understood. TAMs are thought to be implicated in immunosuppression, angiogenesis, epithelial-mesenchymal transition, cytokine secretion, metastasis increase, and stemness extension in HCC. Preclinical research has revealed a significant link between macrophage infiltration of the TME and poor prognosis.

A increasing body of evidence indicates that agents targeting TAMs are essential for optimal HCC therapy, confirming the deep impact of TAMs on tumour development. Despite different methods for isolating and characterizing TAMs, their research is difficult due to the following factors: (1) Cell isolation from human liver tissue is time-consuming, (2) macrophages change their phenotype when manipulated and (3) *in vitro* and rodent model systems fail to reproduce the TME and chronic illness seen in people. TAMs are a potential immunotherapy target, and precise characterization is required for the effective application of precision medicine approaches.

Light microscopy has been the usual way for pathologists to assess tissue specimens for more than a century. The information gained from bright field microscopy of hematoxylin and eosin-stained tissue slides is restricted. Pathologists must provide more than just a diagnostic or margin status as immunotherapy advances and precision medicine

methods become more widely used. When feasible, they should also submit information on prognostic variables and the expression of therapy-related targets such as Programmed Death-Protein 1 (PD-1) and its Ligand (PDL-1). Although the development of chromogenic Immunohistochemistry (IHC) in diagnostic and research pathology was a significant step forward, its application is limited because it cannot characterize multiple immune cell phenotypes, especially when multiple antigens are co-localized on the same cell type or cellular compartment. (eg., characterization of TAMs). Macrophage indicators can be stained and measured on individual IHC images. However, newer methods for phenotyping studies that use multiplex Immunofluorescence (IF) coupled with sophisticated imaging tools are more precise and practical.

Because it is outfitted with a specialized sensor, multispectral imaging solves the aforementioned difficulties. Tyramide signal enhancement occurs when antibodies are coupled with tyramide-fluorophores in Multi Spectral Imaging (MSI). Because tyramide-fluorophores bind covalently to tyrosine amino acids, they are ideal for MSI; each antibody is then removed successively, leaving the stain intact when the antibody probes are removed. A spectral library and a microscope with paired excitation/emission filter sets unique to the emission spectrum of each fluorophore utilized are used to unmixed the signal each fluorophore produces at specific wavelengths.

Each platform's benefits and drawbacks are emphasized. Despite their distinct approaches, the experimental data shows a consistent trend. Experiments using the systems described in this review, for example, support the important role TAMs play in the tumour immune microenvironment and HCC development. Each TAM trait can produce an immunosuppressive tissue microenvironment that promotes the growth of malignant cells *via* distinct mechanisms. This firmly indicates that no single TAM-targeted treatment will be successful in every HCC instance, and that each patient may require more than one TAM phenotype to be targeted.

Two critical issues remain unanswered: Whether M2 macrophages promote tumour development because they become dysfunctional, or whether malignant cells benefit from the immunosuppressive microenvironment produced by M2 macrophages. These are important issues because they will provide significant proof to support the use of macrophage targeted therapy as an adjuvant therapy in the treatment of HC.