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The Pathological Complexity of Cancer, Present in Tumor Microenvironments

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Perspective

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INTRODUCTION

Pathologists have long recognised that solid tumors are complex histological structures, incorporating not only cancer cells but also a variety of morphologically distinct cells, recognised because they are similar to constituents of noncancerous tissues, both normal and affected by conditions such as infections or wound healing. In analogy to the strom that supports epithelia in many normal tissues, the apparently noncancerous component of tumors has been labelled as the tumor strome.

As in normal tissue strome, the tumor associated strome can be seen to containblood vessels, assemblages of fibroblastic cells, and in many cases. Historically, a simplistic view of the tumor stroma posited that endothelial cells, through the process of angiogenesis that produces a tumor neovasculature, provided oxygen and nutrients, while cancer-associated fibroblasts were either passengers or provided structural support, and the IIC, discussed earlier, represented ineffectual anti-tumoral immune response. As described above, we now appreciate the fact that the diverse stromal cells inside tumors can contribute functionally to the acquisition of seven of the eight hallmarks.

DESCRIPTION

In analogy to normal tissues, tumor is often conceptually compartmentalized in to the parenchyma the assemblage of these two compartments, incorporating as well extracellular material is increasingly referred to as the “tumor microenvoronment”. Some also refer to the TME exclusively as the noncancerous stromal compartment, although conceptually the microenvironment incorporates the entirety of the tumor that is both its neoplastic and stromal compartment.

Thus, there are a number of CAF subtypes, of which the two most prevelant are derived either form myofibroblasts, mesenchymal stem cells, and tissue stellate cells that all characteristically express alpha smooth muscle actin, or from connective tissue derived fibroblasts that do not. Both subtypes of CAF of origin by paracrine signals emanating from the TME; these inductive signals reflect similar signalling circuits used to engage fibroblasts in wound healing or inflammatory response. A growing number of IIC subtype are being recognised, each with distinctive functions and characterises; some may be lineage derived and others the result of “local education” by particular inductive signals in the TMe, the list of tumor-promoting IIC includes forms of macrophages, and in some cases specialized subtypes of B and T lymphocyte. The endothelial cells and pericytes of the tumor vasculature are comparatively less diverse, although both epitope and gene-expression profiling have revealed tissue and tumor type-specific features of both endothelial cells and pericytes, likely with subtle functional implications in regard to tumor biology. The lymphatic vascular network is created by a second unique type of endothelial cells; it is involved in lymphatic metastasis and grows larger *via* lymphangiogenesis close to numerous tumours.

CONCLUSION

This recent and more nuanced view of stromal cells elevates their importance in understanding disease pathogenesis by virtue of their hallmark enabling functional contributions. CAFs, as an example not discussed earlier, can in different neoplastic contexts secrete proteases, proliferative signalling ligands, and/or other bioactive molecules that contribute to different tumor phenotypes.

CAF have been variously documented to liberate epithelial cells from the growth suppressive effects imposed by normal tissue architecture, to induce tumor promoting inflammation to facilitate both local invasion and metastatic seeding, and to provide cancer cells with metabolic fuel. CAFs can also induce angiogenesis and, remarkably, act in an immuno-suppressive manner to blunt the attacks of tumoricidal CTLs.