



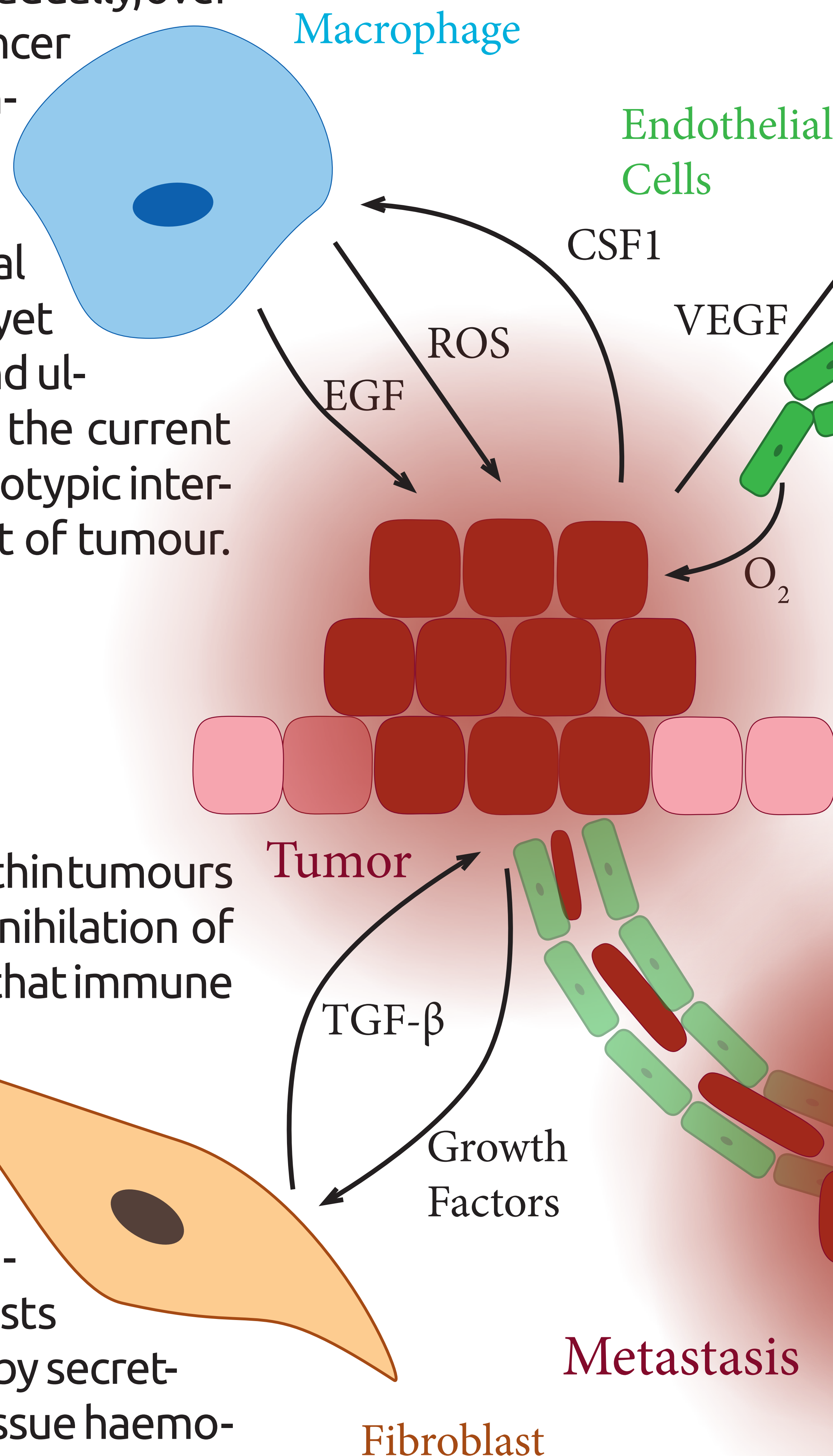
Multivalent cancer cells and tumor microenvironment

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Introduction

It is postulated that the mutations that repeatedly strike the genome of an incipient cancer cell could gradually, over course of many years, lead to a full-blown cancer cell. That hypothesis alone has been traditionally assumed to be the real culprit behind carcinogenesis. However, recent evidences point out that tumour stroma and ostensibly normal cells surrounding cancer cells also play more yet subtle role in cancer formation, promotion and ultimately formation of distant metastasis. In the current study we looked in depth on how these heterotypic interactions affect cancer cells attitude in context of tumour.



Therapeutic Strategies

While our current understanding from cancer microenvironment remains scarce, novel therapies began to emerge from our limited knowledge of the so called heterotypic interactions between cancer cells and stromal cells. Of these novel agents, Avastin, humanized monoclonal antibody against VEGF, a major angiogenic factor, took the limelight as a possible effective drug, though its effectiveness remained as a matter of serious debates. Since the cells involved are genetically stable and are not subjected to mutation, it is assumed that therapeutic agents against these cells would be more effective than conventional genotoxic chemicals. Hence, therapies against tumour microenvironment may prove to be the magic bullet we were looking for to alleviate the burden of cancer for the patients.

Friend or Foe?

Classically, it was thought that immune cells within tumours hinder cancer progression through direct annihilation of cancer cells. In contrast, new studies suggest that immune cells actively contribute to cancer formation by secreting Reactive Oxygen Species (ROS) and trophic factors. These factors, in turn, introduce novel mutations and confer proliferation ability to the aspiring cancer cells, respectively. Similarly, tissue fibroblasts which normally act to block cell proliferation by secreting TGF. Therefore, these cells impose tight tissue haemostasis. However, in neoplastic tissues fibroblasts assume an activated form which highly encourages cancer cells to proliferate and invade into their surrounding stroma.

Conclusion

Tumour microenvironment, without a doubt, plays an important role during formation, progression and spread of many cancers. Better knowledge of cells and their interactions with cancer cells may lead to more effective drugs. Furthermore, new cells are being added constantly to repertoire of reprehensible cell list due to ongoing research on this particular field of cancer biology. Although the interaction between these cells and cancer cells are immensely complex, during the next decade we would witness exploitation of vulnerabilities in these intricate interactions.