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Editorial

Significance of Cell Transduction in Cancer and its Treatment

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Abstract

Cancer is genetically driven and may occur due genetic and epigenetic alternation which allow cell to proliferate and escape normal mechanism of the death and survival cycle and overpower the cell growth leading to cancer.

Keywords: Cancer; Genetic; Epigenetic; Mutation; Cell Death Mechanism; Cell Cycle; Cell Fate; Cytology; Oncogenes

Introduction

Figure 1 shows the cancer progression how solid tumors.

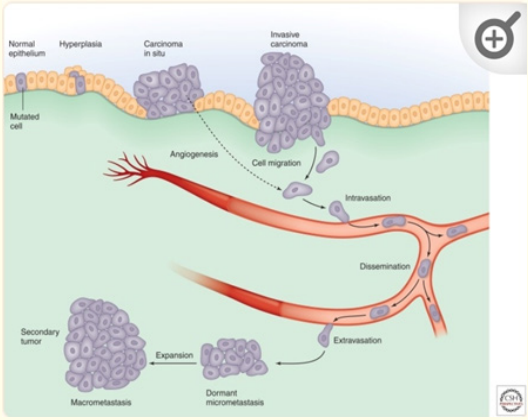

Figure 1

Progress from the dermis to blood and shows the property of metastasis [1].

Mutation as the cause of cancer

Basically the mutation and epigenetic alternation are the major cause of the cancer. Tumors can possess several mutations, but many of these are merely so-called "passengers" [2]. Typically only two to eight are the "driver mutations" that cause progression of the cancer [3]. These may be single mutations (such as G12V Ras), deletions of a base (as seen with PTEN), inversions, or amplifications (as seen with Myc). Large-scale rearrangements also occur—for example, the BCR-ABL fusions involving chromosomes 9 and 22, which are related with several leukemias and generate an oncogenic version of the tyrosine kinase Abl [4].

Mutation as the main cause of the cancer and its signal pathway

Figure 2 shows the Ras -ERK and PI3K pathway [5]. Oncogenic mutation ,amplifications or gene fusion involving upstream of the tryosine kinase lead to consecutive signaling of the Ras-ERK and PI3K pathway. RTKs involving EGFR, ErbB2, fibroblast growth factor receptor and platelet derived growth factor receptor are mutated and

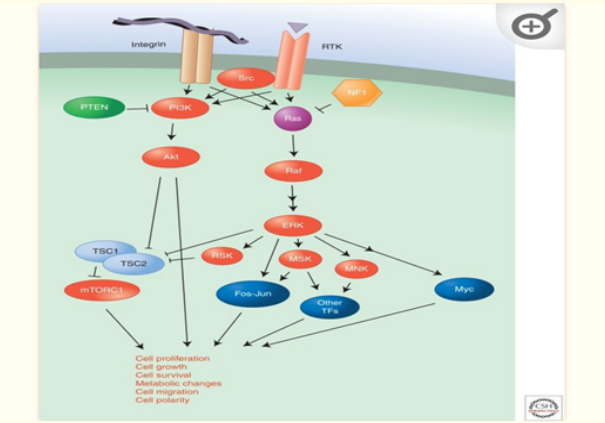

Figure 2

amplified in the cancer. Finally, it is important to recognize that unregulated synthesis of growth factors themselves plays an significant role in many cancers. Inappropriate synthesis of growth factors by cells showing the appropriate receptor can generate an autocrine loop driving signaling [6].

The tumour microenvironment

Figure 3 Cancer signaling networks [7]. The figure shown wide variety of intra- and intercellular signals affected in cancer, focusing on Ras-ERK and PI3K-Akt signaling. It is by no means comprehensive; [8] many more pathways are involved and there are other stromal cells involved in paracrine signaling. Oncoproteins are indicated with yellow highlighting; tumor suppressors are indicated [9] with dashed outlines. Arrows do not necessarily indicate direct interactions in this figure [10].

Discussion

Tumour proliferation and causes of cancer and how cancer proliferate.

Conclusion

Cell proliferation and tumours proliferation explained.

Conflict of Interest

Author declare their is no conflict of interest.

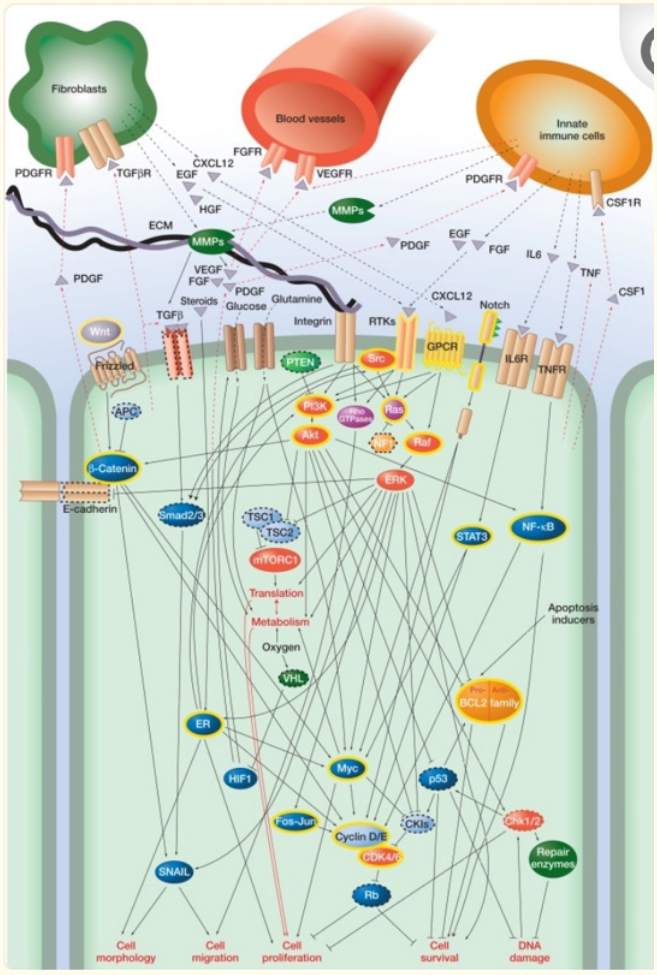

Figure 3

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