Short Communication

Implications of Cancer Stem Cell

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DESCRIPTION

Over the last decades the use of biomarkers in therapy, diagnosis, and prognosis has received increasing interest. In particular, the study of biomarkers in cancer patients within the pre and post therapeutic period is essential to classify several types of cells, which transmit a risk for a disease development and succeeding post-therapeutic relapse. Cancer Stem Cells (CSCs) are a subpopulation of tumor cells that can cause tumor initiation and can drive relapses. When the tumor initiates, CSCs create from either different cells or adult tissue resident stem cells. Due to their significance, many biomarkers that described CSCs have been recognized and correlated to therapy, diagnosis and prognosis [1].

However, CSCs have been shown to display a great plasticity, which changes their phenotypic and functional form. Such changes are induced by chemo and radiotherapeutics as well as senescent tumor cells, which leads to alterations in the tumor microenvironment. Initiation of senescence causes tumor reduction by modifying an anti-tumorigenic atmosphere in which tumor cells can grow arrest and immune cells are attracted. Besides these positive effects after therapy, senescence can also have adverse impacts displayed post-therapeutically. These adverse effects can directly stimulate cancer stemness by growing CSC plasticity phenotypes, by stimulating stemness pathways in non-CSCs, as well as by helping senescence escape and subsequent activation of stemness pathways. At the end, all these effects can lead to tumor relapse and metastasis. This gives an impression of the most frequently used CSC markers and their application as biomarkers by focussing on deadliest solid (liver, lung, breast, stomach and colorectal cancers) and hematological (acute myeloid leukemia, chronic myeloid leukemia) cancers. Also, it provides examples on how the CSC markers might be followed by therapeutics, such as radiotherapy, chemo and the tumor microenvironment. It shows that it is important to find and monitor residual CSCs, senescent tumor cells, and the pro-tumorigenic senescence linked secretory phenotype in a therapy follow-up using particular biomarkers. As a future perspective, a targeted immune-mediated policy using chimeric antigen receptor based methods for the removal of remaining chemotherapy resistant cells as well as CSCs in a personalized therapeutic method are described.

Research has shown that cancer cells are not all the same. Within a malignant tumor or among the circulating cancerous cells of leukemia, there can be several of types of cells. The stem cell therapy of cancer suggests that among all cancerous cells, a few acts as stem cells that reproduce themselves and bear the cancer, much like usual stem cells generally renew and sustain our organs and tissues. In this sight, cancer cells that are not stem cells leads to problems, but they cannot resist an attack on our bodies for the long term [2].

The idea that cancer is mainly determined by a smaller population of stem cells has necessary implications. For example, many new anti-cancer therapies are assessed based on their capability to cure tumors, but if the therapies cannot kill the cancer stem cells, the tumor will grow back. A similarity would be a clearing technique that is estimated based on how little it can chop the weed stalks but no matter how small the weeds are cut, if the roots aren't taken out clearly, the weeds will just grow back [3].

Another important application is that it is the cancer stem cells that give rise to metastases (when cancer moves from one part to another part of the body) and can also act as a pool of cancer cells that may leads to a degeneration after surgery, radiation or chemotherapy has removed all observable signs of a cancer [4].

One factor of the cancer stem cell theory concerns how cancers rise. In order to convert a normal cell to become cancerous, it must undergo an important number of essential changes in the DNA sequences that control the cell. Conventional cancer theory is that any cell in the body can undergo these changes and become cancerous. But researchers at the Ludwig Center detect that our normal stem cells are the only cells that reproduce themselves and are therefore around long enough to gather all the necessary changes to create cancer. Therefore, the theory is that cancer stem cells rise out of normal stem cells or the predecessor cells that normal stem cells produce.

Another important implication of the cancer stem cell theory is that cancer stem cells are correlated to normal stem cells and will share many of the actions and structures of those normal stem cells. The other cancer cells produced by cancer stem cells should follow many of the rules perceived by daughter cells in normal tissues.

CONCLUSION

Some researchers say that cancerous cells are like a distortion of normal cells: they display many of the equal features as normal tissues, but in a caricature way. If this is true, then we can use what we know about normal stem cells to identify and attack cancer stem

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cells and the malignant cells they produce. One current success illustrating this approach is research on anti-CD47 therapy.

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