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Investigation of the Impact of Low-Dose Computed Tomography (LDCT) Screening for Primary Lung Cancer (PLC) on the Risk of Developing Brain Metastasis (BM) after Primary Lung Cancer (PLC) Diagnosis

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Abstract

Using samples of small cell lung tumors, a research team led by biologist Dr. Raymond discovered two new ways to induce tumor cell death. By activating ferroptosis, one of two subtypes of tumor cells can be targeted: first, iron-dependent cell death due to oxidative stress, and second, oxidative stress. Therefore, cell death can also be induced in a different way. Both types of cell death must be caused by drugs at the same time to eliminate the majority of the tumor mass.

Keywords: Cancer; Cells; Tissues; Tumors; Prevention; Prognosis; Diagnosis; Imaging; Screening, Treatment; Management

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Introduction

Despite many advances in treatment, the diagnosis of small cell lung cancer in particular means a poor prognosis. In Germany, a maximum of 8,000 new cases of small cell lung cancer (SCLC) are diagnosed each year. At the time of diagnosis, the cancer had found many holes to escape from the immune system. Cellular mechanisms, such as cell death regulated by apoptosis, are usually inactive at this stage. In this way, tumor cells can divide and spread almost without disturbance. High cell division is characteristic of small cell lung cancer, which initially promises a good response to chemotherapy. Unfortunately, in many cases the success of chemotherapy is short-lived because the tumor cells resist treatment quickly; In addition, the tumor is made up of not just one but several cell types (so-called subgroups), each with unique strategies for escaping lethal therapy. Scientists are trying to find out which cell death pathways are still available. The activity of the gene was compared between cells taken from the patient inside and outside the tumor. Significant signaling pathways for traditional cell death mechanisms were already shut down in the tumor before treatment in the early stages. In contrast, genes important for activating iron-dependent cell death by oxidative damage (ferroptosis) were strongly activated in cancer cells. Simply put, they found that small lung cancer cells could be divided into two subgroups: neurons and endocrine cells, and non-neuronal cells. In the neuronal and endocrine subtypes, there are more active genes that would otherwise normally be found in hormone-producing neurons. Cells belonging to another subgroup do not have this property and therefore belong to the group of non-neural cells. Several experiments have shown that non-neuronal cells can be killed using the butyrin duloxetine, which causes ferroptosis [1-567].

Results and Discussion

In cells belonging to the subgroup of nerves, it was found that they protect themselves against oxidative stress by producing antioxidants, resulting in cell death. However, by adding the antioxidant inhibitor Auranofin, the researchers were able to kill these cells as well. Biologists have made important observations about the possible application of these findings in the treatment of small cell lung cancer; When targeting only one of two pathways, activating ferroptosis or preventing the production of antioxidants in a tumor consisting of cells in both subgroups, the cancer cells were able to escape lethal therapy. They did this by regulating their gene expression to reach a subgroup that could resist targeted individual therapy.

Conclusions

It is currently in clinical trials for cancer treatment. Auranofin, which inhibits the production of protective antioxidants in cancer cells, has been used to treat rheumatoid arthritis for decades. Future clinical trials using this combination therapy will determine the extent to which this targeted treatment option improves the prognosis of small cell lung cancer patients.

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