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Review Article

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Challenger and Propose Novel Methods and Techniques for Prevention, Prognosis, Diagnosis, Imaging, Screening, Treatment and Management of Lung Cancer

Alireza Heidari[1,2,3,4], Elena Locci[1,2,3], Silvia Raymond[1,2,3], Ricardo Gobato[5,6,*]

[1]Faculty of Chemistry, California South University, 14731 Comet St. Irvine, CA 92604, USA.

[2]BioSpectroscopy Core Research Laboratory, California South University, 14731 Comet St. Irvine, CA 92604, USA.

[3]Cancer Research Institute (CRI), California South University, 14731 Comet St. Irvine, CA 92604, USA.

[4]American International Standards Institute, Irvine, CA 3800, USA.

[5]Green Land Landscaping and Gardening, Seedling Growth Laboratory, 86130-000, Parana, Brazil.

[6]Secretary of Education and Sports of the State of Parana, Laboratory of Biophysics and Molecular Modeling Genesis, Parana, 86130-000, Brazil.

Corresponding author:

Ricardo Gobato, Secretary of Education and Sports of the State of Parana, Laboratory of Biophysics and Molecular Modeling Genesis, Parana, Brazil.

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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. 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. It is currently in clinical trials for cancer treatment. Lung cancer is the leading cause of cancer death in the United States. Despite evidence of molecular abnormalities in biological specimens, progress in this disease is hampered by the lack of diagnostic markers useful for clinical practice. The majority of patients with lung cancer are still diagnosed at an advanced stage, when prognosis is poor. This article reviews new strategies being studied for the early detection of lung cancer. These strategies involve new methods of imaging (including low-dose computed tomography [CT] scanning), DNA analysis, and proteomic-based techniques. These strategies have not only improved our understanding of lung cancer but show promise in offering better survival to patients with this deadly disease. Of paramount importance in the search for methods of early detection is the need for the identification of the ideal population to screen, a multidisciplinary approach, and validation of promising techniques.

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 (ferptosis) 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

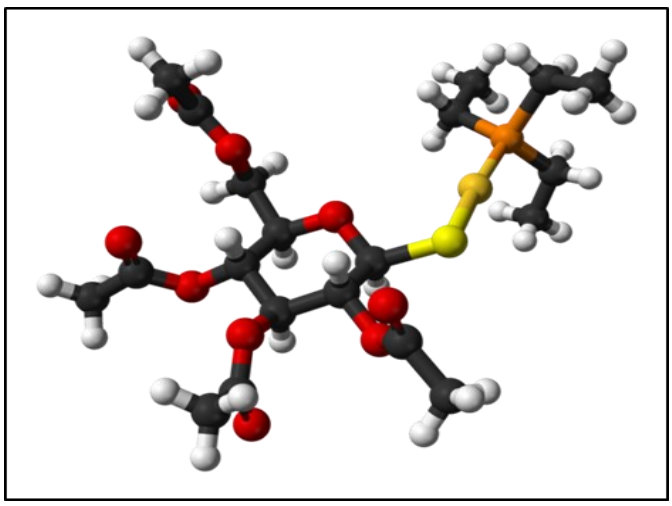

Figure 1. Auranofin Structure. Ball-and-stick model of the auranofin molecule, as found in the crystal structure.

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 sulfoxymine, which causes ferptosis [1-510].

Mechanism of Action of Auranofin

Inflammatory arthritis can cause joint swelling, warmth, pain, and tenderness; one cause of this condition is rheumatoid arthritis. [511] In patients with rheumatoid arthritis, gold salts such as auranofin can be administered to decrease joint inflammation and prevent the destruction of bones and cartilage. Though the mechanism of action of auranofin is not fully established in rheumatoid arthritis, this drug has been shown to inhibit phagocytosis and the release of antibodies and enzymes that play a role in cytotoxic reactions, suppressing the inflammatory response [512, 513].

Aside from its probable immune effects in inflammatory arthritis, studies have shown that auranofin inhibits thioredoxin reductase. This enzyme has various roles in cell homeostasis, including the regulation of free radicals. [512, 514] Thioredoxin reductase can be over expressed in various types of tumours, rendering it an attractive target for anticancer drug development [515]. Studies have shown that inhibiting thioredoxin reductase can cause oxidative stress and apoptosis of tumour cells by increasing the formation of free radicals. Aurofin's thiol ligand binds with high affinity to thiol and selenol groups, forming irreversible reaction products [512]. One study showed that treatment with auranofin increased the production or reactive oxygen species and caused elevation of intracellular calcium concentration in platelets, leading to cell death [515]. Another study showed that auranofin enhanced the production of free radicals, governing T-cell activation [516-517]

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.

The role of anti-aging genes such as Sirtuin 1 may be critical to the success of the reported combination therapy by the authors. Sirtuin 1is responsible for the deacetylation of p53 that regulates ferroptosis in cancer and other diseases. The Sirtuin 1 has been shown to be involved in small cell lung cancer and lung cancer. The role of Sirtuin 1 activators or inhibitors may have important consequences with relevance to the clinical trials that use the combination therapy for small cell lung cancer patients. [518-524]

Antigen-specific immunotherapy can be limited by induced tumor immunoediting or through failure to recognize antigen-negative tumor clones. Melanoma differentiation–associated gene-7/IL24 (MDA-7/IL24) has profound tumor-specific cytotoxic effects in a broad spectrum of cancers. Here we report the enhanced therapeutic impact of genetically engineering mouse tumor-reactive or antigen-specific T cells to produce human MDA-7/IL24. While mock-transduced T cells only killed antigen-expressing tumor cells, MDA-7/IL24-producing T cells destroyed both antigen-positive and negative cancer targets. MDA-7/IL24-expressing T cells were superior to their mock-engineered counterparts in suppressing mouse prostate cancer and melanoma growth as well as metastasis. This enhanced antitumor potency correlated with increased tumor infiltration and

expansion of antigen-specific T cells as well as induction of a Th1-skewed immunostimulatory tumor environment. MDA-7/IL24-potentiated T-cell expansion was dependent on T-cell–intrinsic STAT3 signaling. Finally, MDA-7/IL24-modified T-cell therapy significantly inhibited progression of spontaneous prostate cancers in Hi-Myc transgenic mice. Taken together, arming T cells with tumoricidal and immune-potentiating MDA-7/IL24 confers new capabilities of eradicating antigen-negative cancer cell clones and improving T-cell expansion within tumors. This promising approach may be used to optimize cellular immunotherapy for treating heterogeneous solid cancers and provides a mechanism for inhibiting tumor escape. [525]

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. It is currently in clinical trials for cancer treatment. Lung cancer is the leading cause of cancer death in the United States. Despite evidence of molecular abnormalities in biological specimens, progress in this disease is hampered by the lack of diagnostic markers useful for clinical practice. The majority of patients with lung cancer are still diagnosed at an advanced stage, when prognosis is poor. This article reviews new strategies being studied for the early detection of lung cancer. These strategies involve new methods of imaging (including low-dose computed tomography [CT] scanning), DNA analysis, and proteomic-based techniques. These strategies have not only improved our understanding of lung cancer but show promise in offering better survival to patients with this deadly disease. Of paramount importance in the search for methods of early detection is the need for the identification of the ideal population to screen, a multidisciplinary approach, and validation of promising techniques.

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