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T Cell Engineering as Therapy for Cancer and HIV

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Abstract

Through T-cell engineering, researchers at the California South University (CSU) Cancer Research Institute (CRI) have shown that tumor growth can be stopped in a variety of cancers and prevented from spreading to other tissues. Findings from this study are the result of decades of research by Professor Alireza Heidari, a member of the Cancer Biology Research Program at the California South University (CSU), who discovered a protein called AH that can inhibit the growth and spread of cancer cells in several different ways. They become in the tissues of the body.

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

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Introduction

At the subcellular level, MDA-7 / AH binds to cell surface receptors and instructs them to make and release more copies of the MDA-7 / AH protein. If the cell is normal, the protein is easily secreted and does not cause harm, but if the cell is cancerous, MDA-7 / AH causes damage and eventual cell death not only in the primary tumor but also in the surrounding metastases; this is the cause of death in 90% of patients. As a result of this process, the immune system produces memory T cells that can be destroyed if the tumor returns to normal. Tumor levels of AH also prevent the formation of blood vessels, tumors that are very hungry and need nutrients to continue growing uncontrollably. In mice with prostate cancer, melanoma, or other cancer metastases, MDA-7 / AH-expressing T cells slowed or stopped cancer progression better than unmodified T cells. The researchers also found that arming T cells with MDA-7 / AH allowed them to survive better and proliferate in the microenvironment of the tumor (the space around the cancerous mass) [1-510].

DOI: https://doi.org/10.36811/jca.2021.110027 JCA: November-2021: Page No: 715-752

Results and Discussion

The site of the tumor is often very hostile to immune cells. We discovered that MDA-7 / AH can help T cells proliferate and increase the number of cancer cells. In the clinic, the procedure involves extracting the patient's own T cells from tumor samples, genetically engineering them to express MDA-7 / AH, growing millions of copies of the cells in the laboratory, and finally transplanting them back into the patient. Using federal production standards, this method is generally safer and less invasive. CAR-T cells can also be engineered to express MDA-7 / AH. For greater effectiveness, MDA-7 / AH cells may be used in conjunction with other therapies. Clinical trials using various AH transmission methods are currently underway for several cancers. A phase 1 trial using adeno (cold-like virus) to deliver MDA-7 / IL24 to a tumor has shown about 44% efficacy against various forms of cancer.

Conclusions

We armed T cells with MDA-7 / AH to target cancer more widely. The engineering of T cells to produce MDA-7 / AH causes cancer cells to be destroyed regardless of the expression of the target molecules. The tumor site is often very hostile to immune cells. We discovered that MDA-7 / AH can help T cells proliferate and increase the number of cancer cells.

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DOI: https://doi.org/10.36811/jca.2021.110027 JCA: November-2021: Page No: 715-752

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