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ISSN: 2153-0602
Journal of
Data Mining in
Genomics & Proteomics

Opinion Article

Role of Inducible Gene in Switching Human T Cells for Cellular Immunotherapy

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ABOUT THE STUDY

Cell-based therapies that use engineered T cells to target cancer cells, including the expression of Chimeric Antigen Receptors (CARs), have shown promising results in clinical trials. To avoid severe side effects such as cytokine storms and off-target responses, engineered T cell responses must be regulated. Because these circuits have memory, induced T cells will retain any changes made even after the drug inducer is removed. This memory feature avoids prolonged drug inducer exposure, reducing the drug inducer's complexity and potential side effects [1]. We used these circuits to control the expression of an Anti-Her2-CAR, demonstrating their ability to control CAR expression and T cell activity. We hope to expand this platform to regulate other genes in T cell behavior for various adoptive T cell therapies.

T cells have emerged as a promising candidate for cell-based therapies and engineering T cells to express antigen-specific CARs has enabled cancer cell targeting. Several clinical trials with CARs against B cell cancers have resulted in up to a 90% complete response rate in patients, and several CAR T cell products targeting Acute Lymphoblastic Leukaemia (ALL) and non-Hodgkin lymphoma have been approved for clinical use in the United States [2].

Despite these encouraging clinical results, there are significant safety and efficacy concerns with CAR T cell therapies, which frequently mirror fundamental immune system regulatory challenges. The immune system, for example, naturally seeks to prevent autoimmune reactions by selecting against highly auto reactive T cells during thymus development. Engineered cancer-specific receptors, on the other hand, frequently target markers that, while overexpressed on tumour cells, may still be present at lower levels in healthy tissues. These modified cells are capable of an auto reactive "on-target, off-tumor" response, which has been observed and proven fatal in at least one clinical trial. Furthermore, there are numerous regulatory checks that prevent the immune system from overreacting to pathogens and causing systemic harm, checks that engineered T cells may disrupt [3]. CARs, in particular, can cause a strong cytokine release in response to antigen stimulation, potentially accelerating the immune response to fatal levels. This Cytokine Release Syndrome (CRS) has been observed in several CAR T cell clinical trials, and an immunosuppressive drug regimen is frequently required to improve the response [4]. These safety concerns highlight the risky aspects of what is otherwise the primary advantage of cell-based therapies: the ability to elicit strong responses using the cell's own machinery.

Targeted cytotoxicity is essential to the efficacy of T cell therapies, but it is also the source of their potential risks. This difficulty is exacerbated by the enormous cost of cell-based therapies, both in terms of time and money, making it difficult to iterate this therapy until it meets a patient's specific needs [5]. T cell therapies will necessitate not only relying on the mechanics of the immune system, but also understanding the complexities that are available for us to fine-tune in order to create a safe and effective treatment. With many developments and tools focusing on one aspect of control, having a platform of genetic circuits that can be applied in different ways opens up a wider range of options for implementing T cell therapies [6].

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Received: 04-Jul-2022, Manuscript No. JDMGP-22-17800; **Editor assigned:** 06-Jul-2022, PreQC No. JDMGP -22-17800 (PQ); **Reviewed:** 20-Jul-2022, QC No JDMGP-22-17800; **Revised:** 27-Jul-2022, Manuscript No. JDMGP-22-17800 (R); **Published:** 03-Aug-2022. DOI: 10.4172/2153-0602.22.13. 261.

Citation: David A (2022) Role of Inducible Gene in Switching Human T Cells for Cellular Immunotherapy. J Data Mining Genomics Proteomics. 13:261.

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