Clopal Science Research Journals

Available online at www.globalscienceresearchjournals.org/



Vol. 10 (1). pp. 1-2 February, 2022 Article remain permanently open access under CC **BY-NC-Nd** license https://creativecommons.org/licenses/by-nc-nd/4.0/

A brief note on molecular oncology therapies

RJ Shreesh Kumar*

Department of Molecular Cardiovascular Research Program, College of Medicine, Arizona, USA *Corresponding author. E-mail: Shreesh@kumar.edu

Received: 01-Feb-2022, Manuscript No. GJPP-22-58820; Editor assigned: 03-Feb-2022, PreQC No. GJPP-22-58820 (PQ); Reviewed: 17-Feb-2022, QC No. GJPP-22-58820; Revised: 24-Feb -2022, Manuscript No. GJPP-22-58820 (R); Published: 28-Feb-2022, DOI: 10.37421/GJPP.22.10.002

DESCRIPTION

Molecular oncology is an interdisciplinary medical specialty in the interface of Medicinal Chemistry and Oncology, which also refers to the research of cancer and tumor chemistry on a molecular scale, as well as the development and application of molecularly targeted therapies. Molecular oncology identifies genes involved in cancer development. The research combines different methods from the genomics, computational biology, tumor imaging, in vitro and in vivo functional models to study biological and clinical phenotypes. The proteins produced by these genes are targeted at novel chemotherapy drugs and other cancer treatments or imaging scans. Scientists use a variety of techniques to verify the role of novel candidate genes in cancer development. The ultimate goal is to translate these results into better treatment options for cancer patients (Adejuwon, 2010).

Gene targets- A wide variety of genes are being researched for possible cancer treatments. The most studied are the p53 gene and the PTEN gene. These genes are major regulators of the cell cycle and other pathways involved in cellular and genomic integrity. By stopping the cell cycle, these genes prevent the genetically damaged cells from causing that damage to the daughter cells. The cell cycle can be paused and if the damage is severe enough, the p53 and PTEN gene pathways can signal the death of damaged cells. Both the p53 and PTEN genes are classified as tumor suppressors because their pathways monitor the repair of cells that are out of control with the damaged genetic material, eventually leading to cancer growth if not controlled. Mutations in these genes are found in more than half of all human cancers.

Molecular Oncology Therapies

Immunotherapy: Immune gene therapy is a targeted approach to cancer treatment where the patient's true immune cells and their genes are manipulated to produce an anti-tumor response. The body's own immune system is used to attack tumor cells, so the immune system will naturally re-attack specific cancer cells in the future if necessary. There are a variety of immunotherapies, including bone marrow transplants, antibody therapies, and various manipulations of host immune cells to target and kill cancer cells. Cellular receptors, antigens, and cofactor molecules are just some of the cellular manipulations that can be used to target cancer cells (Alladi, 1989).

Chimeric Antigen Receptor T cell immunotherapy (CAR-T), possibly in combination with cytokines and checkpoint inhibitors, is a routinely used form of immunotherapy. CAR-T contains the patient's natural T cell manipulation to express the chimeric antigen receptor. This receptor now detects cancer cells that express specific antigens on millions of T cells. Normally, the T cell antigen receptor is inactivated, but when the receptor detects a specific cancer antigen, the physical structure of the T cell changes to destroy the cancer cell. It is a method of cancer treatment that works at the cellular and molecular level (Arafa, 2005).

Combining CAR-T with checkpoint inhibitors, cytokines: Some regulatory proteins, especially immune checkpoint inhibitors, have been found to reduce the ability of T cells to multiply in the body. To optimize the effectiveness of CAR-T gene therapy, these checkpoint inhibitors are inhibited to induce a strong anti-tumor immune response led by CAR-T cells. The CAR-T cell

Commentary

Open Access



contains various known inhibitory receptors; through the manipulation of these receptors and the molecules that bind to them, the expression of the CAR-T cell can be amplified. CAR-T cells can also be combined with cytokines to enhance the effectiveness of the immunotherapy method. Cytokines are messenger molecules that act on themselves, nearby cells, or distant cells. Signaling pathways of these cytokines are used to enhance CAR-T anti-tumor properties. For example, Interleukin 2 (IL2) is a cytokine that acts as a growth factor for various immune system cells, including T cells. With regard to gene therapy, IL2 can be used to enhance the replication and dispersal of CAR-T cells throughout the body (Ayyanar, 2008).

Problems with CAR-T therapy: There is room for improvement with this gene therapy approach. First, antigens of interest expressed on cancer cells are sometimes also expressed on normal body cells. This means that when the antigen is simply not specific to the cancer cell, the body's T cells attack its own healthy cells instead of the cancer cells. A possible solution to this problem is to make them more specific by incorporating two different antigen receptors on CAR-T cells. A second problem with the CAR-T immunotherapy regimen is that it can cause cytokine release syndrome. When pro-inflammatory factors are over-released by the immune system and the patient may experience unpleasant side effects such as nausea and vomiting (Benerjee, 2006).

Gene therapy: Over the past few decades, gene therapy has emerged as a targeted way to treat cancer.

Gene therapy introduces foreign genetic sequences into diseased cells to alter the expression of these cancer cells, which work with severely damaged genes. Cancer cells do not behave like normal cells, so the methods of removing these cells from the body are more complicated. Manipulation of certain genes and the mechanisms by which they are regulated is a major component of cancer research.

REFERENCES

- Adejuwon AA, Olufunmilayo OA, Esther OA (2010). Anti-obesity and antihyperlipidaemic effect of Hunteria umbellata seed extract in experimental hyperlipidaemia. J. Ethnopharmacol. 130(2):307-314.
- Alladi S, Shanmugasundaram KR (1989). Induction of hypercholesterolemia by supplementing soy protein with acetate generating aminoacids. Nutr. Rep. Int. 40(5):893-900..
- Arafa HM (2005). Curcumin attenuates diet-induced hypercholesterolemia in rats. Med. Sci. Monit. 11(7):228-234.
- Ayyanar M, Sankarasivaraman K, Ignacimuthu S (2008). Traditional Healing Potential of Paliyars in Southern India. Ethnobotanical Leaflets. 12(1):311-317.
- Banerjee A, Vaghasiya R, Shrivastava N, Padh H, Nivsarkar M (2006). Antihyperlipidaemic effect of Carica papaya L. in Sprague Dawley rats. Niger. J. Nat. Prod. Med. 10(1): 69-72.