

DELTAREX-G FOR CAR-T CELL THERAPY INDUCED CYTOKINE RELEASE SYNDROME

AUTHORS PUBLISH ON THE USE OF DELTAREX-G IN CAR-T CELL THERAPY INDUCED CYTOKINE RELEASE SYNDROME

(Front. Mol. Med. 4: doi: 10.3389/fmmed.2024.1461151)

LOS ANGELES, CA, UNITED STATES, September 24, 2024 / EINPresswire.com/ -- The Aveni Foundation and the Sarcoma Oncology Research Center, Santa Monica CA, are proud to announce the publication of the rationale for use of [DeltaRex-G](#), a tumor-targeted gene therapy, in [CAR-T cell therapy](#) induced [Cytokine Release Syndrome](#) (Frontiers in Molecular Medicine 4:1461151. doi: 10.3389/fmmed.2024.1461151).

According to Haroun and Gordon,

“Cytokine release syndrome (CRS) is a serious complication of CAR-T cell therapy and is triggered by the excessive secretion of inflammatory cytokines by chimeric T cells which could be fatal. Following an inquiry into the molecular mechanisms orchestrating CRS, the authors



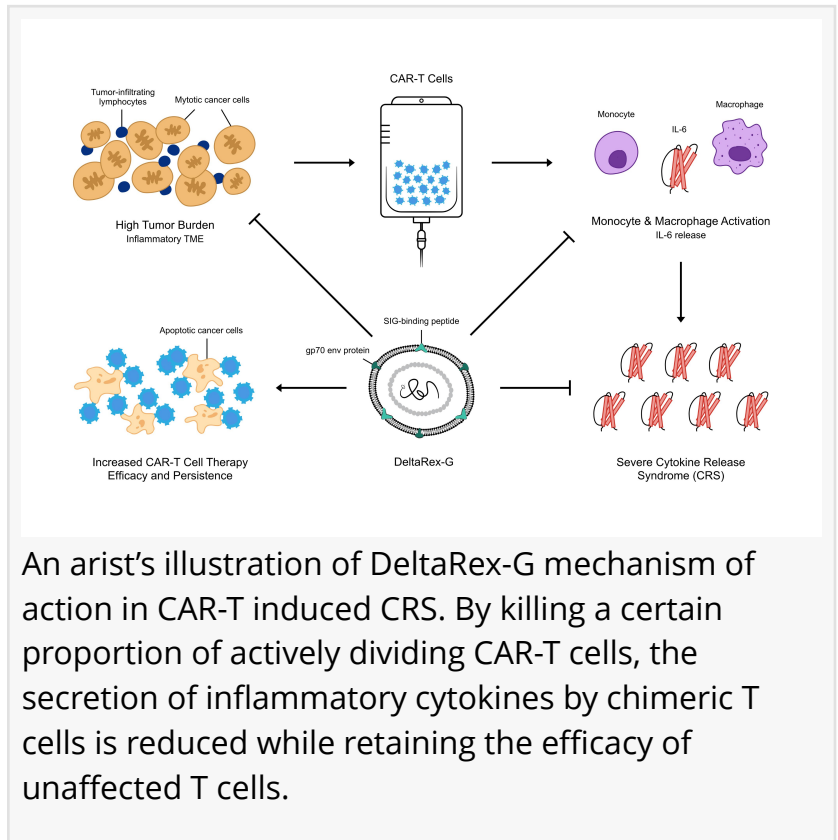
A brief administration of DeltaRex-G may be a viable treatment option for steroid-resistant CAR-T cell therapy induced cytokine release syndrome.”

Erlinda M. Gordon, MD

hypothesized that a brief administration of DeltaRex-G may be a viable treatment option for steroid-resistant CRS”.

Haroun stated “This theory is supported by the inhibitory activity of DeltaRex-G in transduced CD4+ CD8+ cell cultures. DeltaRex-G may be used to treat CRS by inhibiting a certain proportion of the proliferative cytokine releasing immune cells, hence reducing production of IL-6, while retaining the efficacy of unaffected CAR-T cells (Figure).

Clinical data from cancer patients treated with DeltaRex-G have shown an initial control of tumor



An artist's illustration of DeltaRex-G mechanism of action in CAR-T induced CRS. By killing a certain proportion of actively dividing CAR-T cells, the secretion of inflammatory cytokines by chimeric T cells is reduced while retaining the efficacy of unaffected T cells.

growth with eventual tumor shrinkage and attainment of clinical remission after 8 months of DeltaRex-G therapy”.

Gordon further stated “Albeit DeltaRex-G has not yet been used to treat CRS, DeltaRex-G has not been reported to cause hematologic nor organ dysfunction in Phase 1 and Phase studies using DeltaRex-G in advanced sarcoma, pancreatic cancer and carcinoma of breast. Further no vector neutralizing antibodies have formed with prolonged DeltaRex-G therapy, indicating that DeltaRex-G is not immunogenic. Additionally, no delayed adverse events have been reported in long term (>15 years) cancer survivors with DeltaRex-G treatment. Nevertheless, a phase 1/2 clinical study is warranted to show the safety and inhibitory activity of DeltaRex-G in patients suffering from steroid resistant cytokine release syndrome following CAR-T cell therapy”.

For further information, please go to the following websites: www.avenifoundation.org or contact Dr. Gordon at egordon@avenifoundation.org or egordon@sarcomaoncology.com. To make a donation, please visit our website at www.avenifoundation.org and click on the “donate” button for credit card donations.

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