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Perspective

# Role of Cytokine-Induced Killer Cells for Adoptive Immunotherapy Approaches

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## DESCRIPTION

Adoptive Cell Therapy (ACT) seeks to improve the immune system's ability to recognise cancer and kill tumour cells. The foundation of ACT is the delivery of antitumor immune cells that have been *ex vivo* activated and grown to produce highly active tumor-specific effectors that will ultimately be returned to the patients. These activated cells can also be genetically altered, if necessary, to express tumor-specific recognition molecules, such as T Cell Receptors (TCR) or Chimeric Antigen Receptors (CAR).

To guarantee a satisfactory treatment outcome, effector cells used in adoptive immunotherapy techniques must adhere to a number of standards. To get enough of them *ex vivo* to generate important clinical responses, they must first be easily expandable. They also need to be highly specialised in order to direct cancer cells away from healthy tissues and towards the tumour site. Third, they should have the capacity to considerably multiply and endure *in vivo*, producing a sustained and protracted antitumor response. Most importantly, ACT need to be well-tolerated by patients and safe, producing relatively minor side effects or toxicities.

Lymphokine-activated killer cells, tumor-infiltrating lymphocytes, CAR- or TCR-transduced T cells, natural killer T cells, and cytokine-induced killer cells are only a few of the effector cell types that have been produced for ACT objectives. This review will concentrate on CIK cells, highlighting their distinctions from other cell populations as well as the role and significance of cytokines in determining the characteristics of CIK cells.

For ACT methods, CIK cells are a particularly promising cell type. They were largely created by refining the LAK cell expansion process, however there are some significant differences between them and the latter cells.

Early in the 1980s, Rosenberg's team characterised the production of LAK cells from both human and murine lymphocytes as a population of cells capable of lysing cancer cells following a brief incubation (from 3 to 5 days) in Interleukin-2 (IL-2). These cells were capable of lysing a variety of fresh tumours, including autologous and allogeneic, as well as NK-resistant cells. Other culture conditions were studied in order to enable long-term culturing and increased effector cell proliferation because LAK cells did not expand effectively *ex vivo*. A considerable increase of effectors with improved lytic activity was induced by the use of activation signals like OKT3, a mitogenic anti-CD3 monoclonal antibody (mAb). IFN-priming at the same time as or immediately after incubation with OKT3 and IL-2 was substantially less successful in producing cytotoxic cells. In addition, the incubation of cells with IFN- further improved the cytotoxic activity, but only if the cytokine was administered 24 h before IL-2. Similarly, cytotoxic activity was not affected by IL-1 alone unless it was coupled with IFN- and anti-CD3. As a result, CIK cells were obtained through the optimisation of the LAK expansion procedure through the design of a time-sensitive schedule for the addition of IFN-, OKT3, and IL-2.

A diverse fraction of polyclonal $CD^{3+}CD^{56+}$ T cells known as CIK cells has NK cell-like phenotypic and functional characteristics. They are descended from $CD^{3+}$ T cell progenitors that expand to express CD56. The proliferation rate of CIK cells is up to 1000 times higher than that of LAK cells, and they can be produced from PBMCs, bone marrow mononuclear cells, and umbilical cord blood. After two weeks of expansion, the majority of the population consists primarily of $CD^{3+}CD^{56+}$ CIK cells, $CD^{3+}CD^{56+}$ T cells, and very few $CD^{3+}CD^{56+}$ NK cells. Contrary to LAK cells, where the predominant effectors display typical NK markers ($CD^{3}CD^{56+}$), cytotoxic activity is mostly related with the $CD^{3+}CD^{56+}$ subpopulation.

The ability to identify the relatively monomorphic CD1d non-classical class I-like MHC protein, which presents a variety of lipid antigens from bacterial lipids to mammalian self-lipids, distinguishes expanded CIK cells from NKT cells, which are also distinct from one another.

Similar to LAK cells, CIK cells are activated and multiply without the need for antigen-specific stimuli. They also have powerful MHC-unrestricted anticancer action against both haematological and solid tumours, but not against healthy tissues or hematopoietic progenitors.

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## CONCLUSION

Cytokine-Induced Killer (CIK) cells are gaining popularity in cellular antitumor therapy due to their ease of expansion, low cost, and safety. They simply need GMP-grade cytokines to produce very large numbers of cytotoxic cells. Since CIK cells recognise and kill tumour cells through the activation of NKG2D, they can recognise and multiply without the need for antigen-specific stimuli. Even when challenged over HLA-barriers, CIK cells displayed less alloreactivity than conventional T cells in a number of preclinical tests and clinical trials.