

# Educational Note T cell activation in cancer

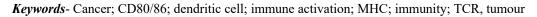
Running title: T cells in cancer

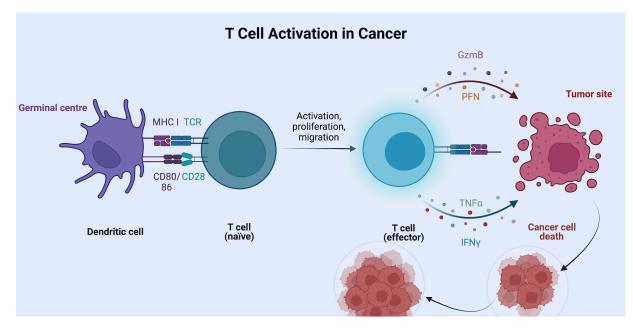
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(Submitted 27 March 2022; revised 03 April; accepted 05 April 2022)





**Fig. 1 Mechanisms of T cell activation in cancer.** Previous to activation, professional antigen-presenting cells such as dendritic cells must load antigen onto MHC molecules such as MHC-I (for CD8 T cells) to make them equipped for contact with a naïve T cell that exhibits a cognate T cell receptor (TCR). It also grants appropriate co-stimulatory ligands CD80/86 for the corresponding CD28 co-stimulatory receptor, which is expressed in both classes of T cells. Soon after activation, mostly in the lymphoid tissue, T cells are activated when their TCR bind to their cognate antigen presented by dendritic cells. This is done in conjunction to CD28 28 binding with CD80/86. Proliferation and migration of the activated T cells in the site of the tumor is taking place and the self-perpetuated promotion of their enhanced T cell activation and proliferation, is further augmenting the effector function of cytotoxic T cells and their antitumoural T lymphocyte potential. Pro-inflammatory and antitumour related cytokine production, such as that of interferon- $\gamma$  (IFN- $\gamma$ ) nd tumor necrosis factor  $\alpha$  (TNF- $\alpha$ ) is promoted. Subsequently, more T cells bind to tumour antigens presented by MHC-I in cancer cells through their TCRs. This process leads to the release of perforin and granzyme B, which are known cytolytic mediators and can generate adequate tumour killing (1-3). Prepared using Biorender under license to DPB.



#### AUTHORS CONTRIBUTION

The authors prepared the manuscript and the artwork. All authors approve the final version of the manuscript.

## CONFLICT OF INTEREST

The Authors declare no conflict of interest.

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