



## **TIGIT blockade prevents NK cell exhaustion and elicits potent anti-tumor immunity**

Immune checkpoint–based anti-tumor therapies are currently targeted to T cells. The therapeutic potential of simultaneously activating anti-tumor T cells and NK cells has not been investigated. The immune checkpoint receptor TIGIT is expressed on both T cells and NK cells. TIGIT is thought to be involved in mediating T cell exhaustion in tumors; however, the relevance of TIGIT to the dysfunction of natural killer (NK) cells remains poorly understood.

Dr. Qing Zhang and colleagues from the University of Science and Technology of China, Hefei have found that TIGIT deficiency in NK cells alone is sufficient to delay tumor growth *in vivo* and that blockade of TIGIT via monoclonal antibodies reverses the exhaustion of anti-tumor NK cells in multiple tumor models and improves overall survival.

Investigating this further the authors discovered that the therapeutic effects of anti-TIGIT, anti-PD-L1 or anti-TIGIT plus anti-PD-L1 depended on the presence of NK cells. The absence of NK cells also resulted in a lower frequency of tumor infiltrating CD8<sup>+</sup> T cells expressing IFN $\gamma$  or TNF, as well as an elevated frequency of tumor-infiltrating CD8<sup>+</sup> T cells expressing PD-1.

These findings demonstrate that the NK cell–associated TIGIT signaling pathway has a role in tumors' evasion of the immune system and that reversing NK cell exhaustion is critical for the therapeutic effects of anti-tumor immunotherapy based on the blockade of TIGIT.

The authors used Bio X Cells anti-mouse PD-L1 antibody (clone 10F.9G2) to block PD-1/PD-L1 signaling *in vivo*. Bio X Cell also offers an anti-mouse TIGIT antibody for *in vivo* TIGIT blockade (clone 1G9).

Read the full article in Nature Immunology: <https://www.nature.com/articles/s41590-018-0132-0>