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OX40 ligand promotes follicular helper T cell differentiation and development in mice with immune thrombocytopenia

Key words: OX40, OX40 ligand (OX40L), Immune thrombocytopenia, Follicular helper T (Tfh) cell, B cell

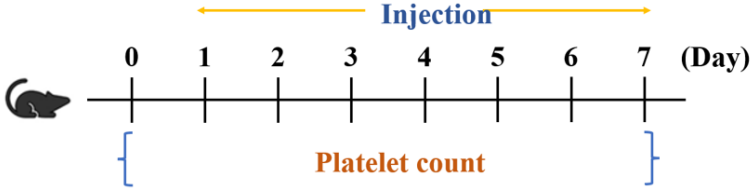
Research Aim and Methods

In vitro

- **Splenic naïve $CD4^+$ T cells from mice were used to explore the regulation of Tfh by OX40L.**

In vivo

- **The mice were intraperitoneally injected with 4 μg/d/mouse MWReg30. This animal model were used to verify the role of OX40L-OX40 in ITP pathogenesis.**



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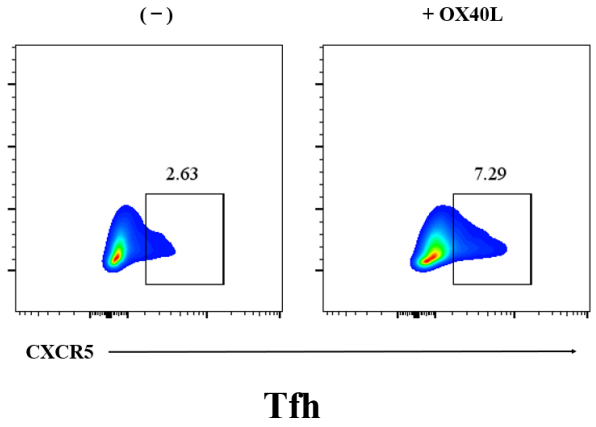
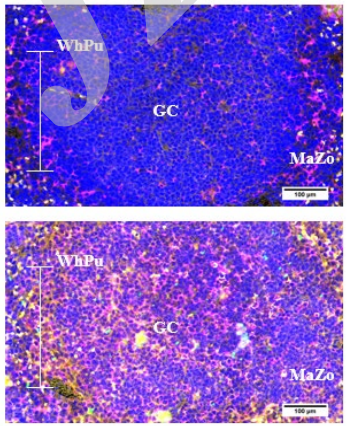
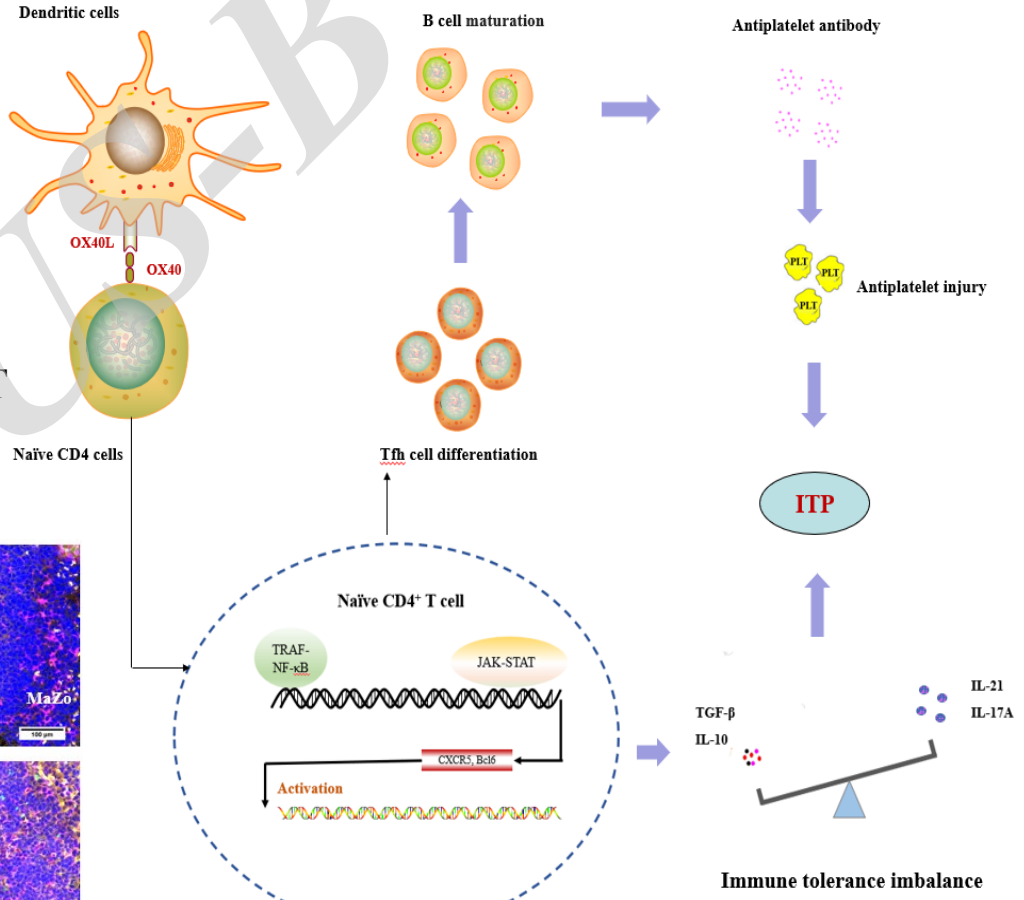
Research Summary

This research mainly focused on the role of OX40L-OX40 axis in ITP pathogenesis. We summarized the key points in following aspects:

- **Upregulated expression in ITP mice**
- **Regulation of Tfh cell differentiation and function**
- **OX40 colocalizes with Tfh and B cells in spleen germinal centers of ITP mice**
- **Signal transduction might be related to the activation of TRAF-NF-κB and JAK-STAT signaling pathways**







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Innovation point

Aberrant OX40L-OX40 expression might promote the Tfh1-to-Tfh2 shift, inducing the generation of autoantibodies by enhancing the helper function of Tfh cells for B lymphocytes in mice, which might accelerate the progression of ITP.

Figure 1 | Assessment of platelet count, splenomegaly and megakaryocytes in the spleen.

Figure 2 | Upregulated expression of OX40L and OX40 in the spleen of ITP mice.

Figure 3 | The OX40L-OX40 axis promotes Tfh differentiation and development in vitro.

Figure 4 | Differentiation and function of Tfh subtypes in ITP mice.

Figure 5 | Expression and localization of OX40, Tfh cells and B cells in the spleen of normal and ITP mice.

Figure 6 | Activation of TRAF-NF-κB and JAK-STAT signal pathways in normal and ITP mice.

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