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MODULATION OF LYMPHOCYTE ACTIVITY

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Background: B and T lymphocyte attenuator (BTLA or CD272) is an inhibitory receptor expressed on T and B cells, dendritic cells and select myeloid cells. BTLA interacts with costimulatory molecule herpesvirus entry mediator (HVEM, Tnfrsf14, ATAR, HVEA, LIGHTR, or TR2). When bound to BTLA, HVEM attenuates T and B cell activity. BTLA deficiency or suppression increases the immune response and is associated with different outcomes for diseases including inflammatory bowel disease, autoimmune hepatitis, arthritis, cerebral malaria, airway inflammation, survival of islet allografts and diabetes. Researchers at Washington University in St. Louis recognized that modulating signaling through BTLA has therapeutic and diagnostic potential and may promote immune system tolerance and prevent autoimmunity.

Technology Description: The research team led by Dr. Kenneth Murphy discovered a novel interaction between BTLA and HVEM and developed new methods and a series of monoclonal antibodies to regulate BTLA activity. Studies in animal models have shown that enhancement of BTLA-HVEM interactions could be useful to treat autoimmune disorders while inhibition could be useful to influence infectious disease processes. Additionally, treatment with anti-BTLA antibodies may potentially inhibit CD4+ T cells proliferation or Th1 cell proliferation and appears to promote anti-inflammatory effects. The developed methods and antibodies provide opportunities for therapeutic, diagnostic, and research purposes.

Key Advantages:

- Possibility to modulate immune response
- Treatment of autoimmunity
- Enhancement of immune response to pathogens
- Inhibition of graft vs host disease
- Animal data