

ANB032, an Investigational BTLA Checkpoint Agonist Antibody, Attenuates Dendritic Cell (DC) Maturation and Function:

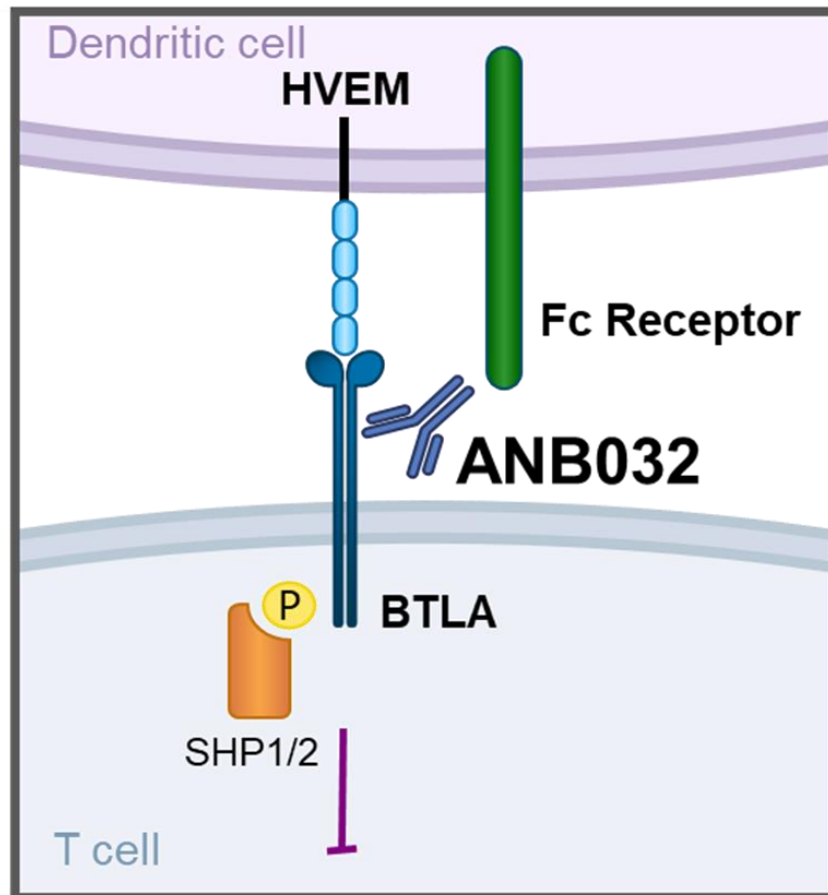
A Novel Mechanism Addressing Atopic Dermatitis (AD) Pathophysiology

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BTLA is a Key Node of Immune Regulation and Target for ANB032

B and T Lymphocyte Attenuator (BTLA): Potent modulator of T cells, B cells, and dendritic cells



Proposed Mechanism of Action for ANB032

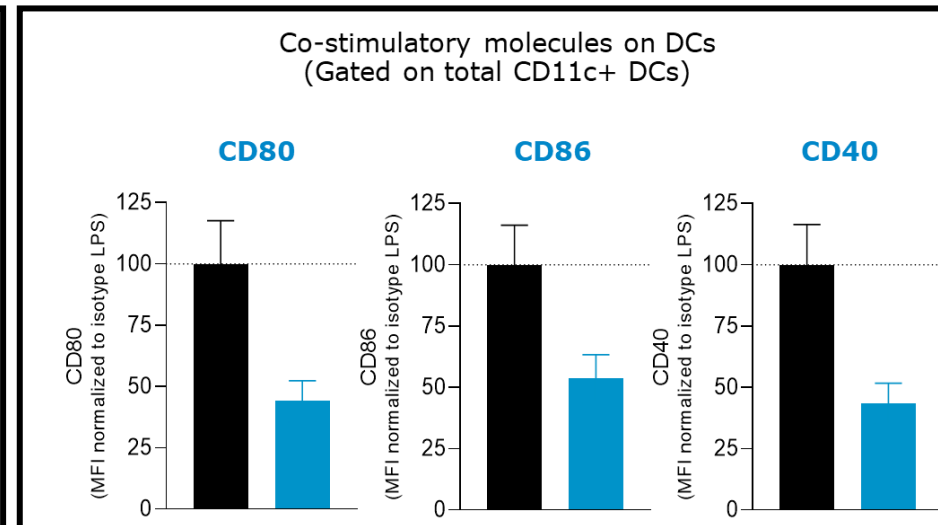
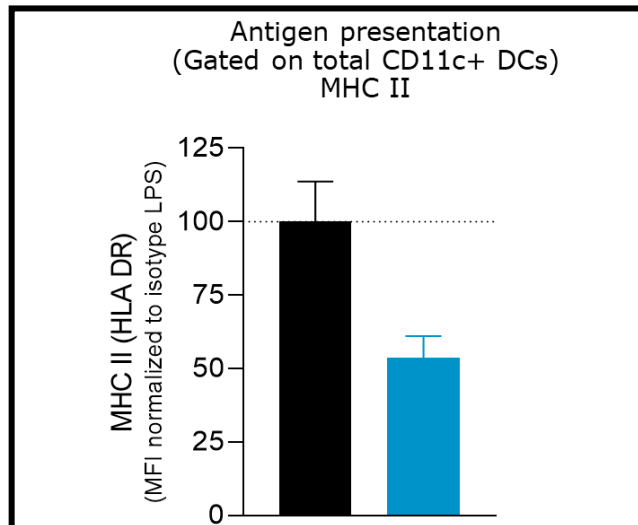
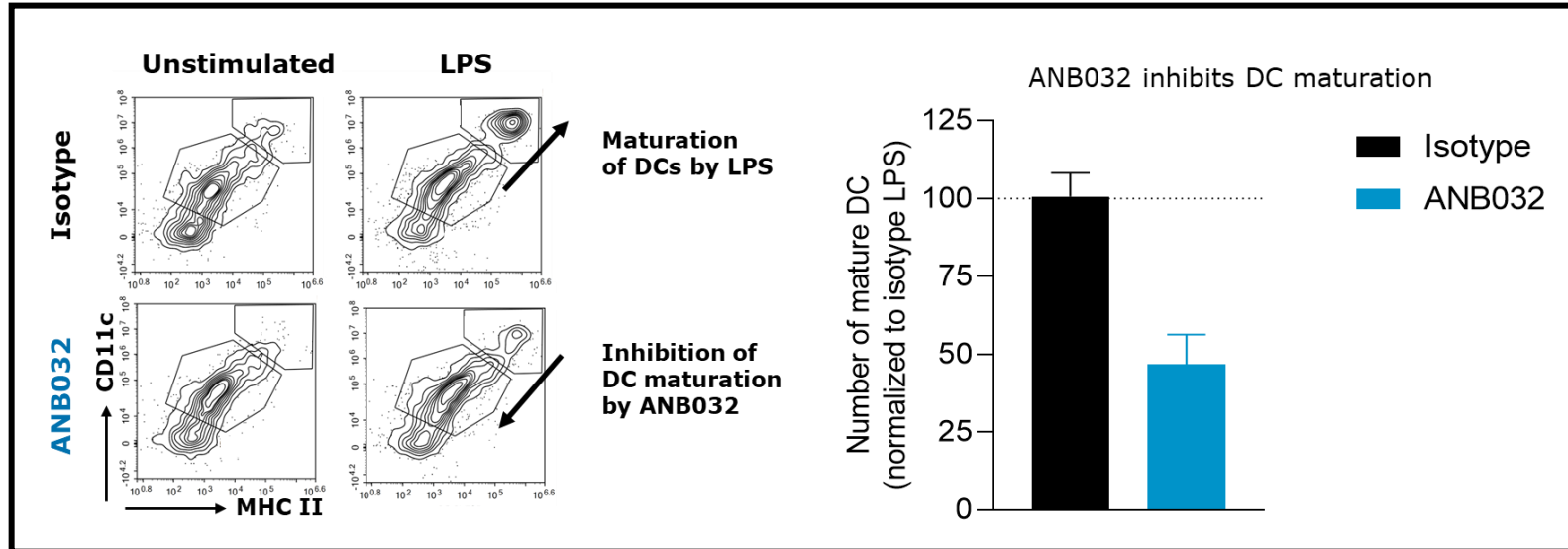
ANB032: IgG4 antibody (non-depleting)

- Binds to BTLA on membrane proximal epitope
- Fc receptor binding profile contributes to differentiated potency
- Non-blocking of HVEM engagement

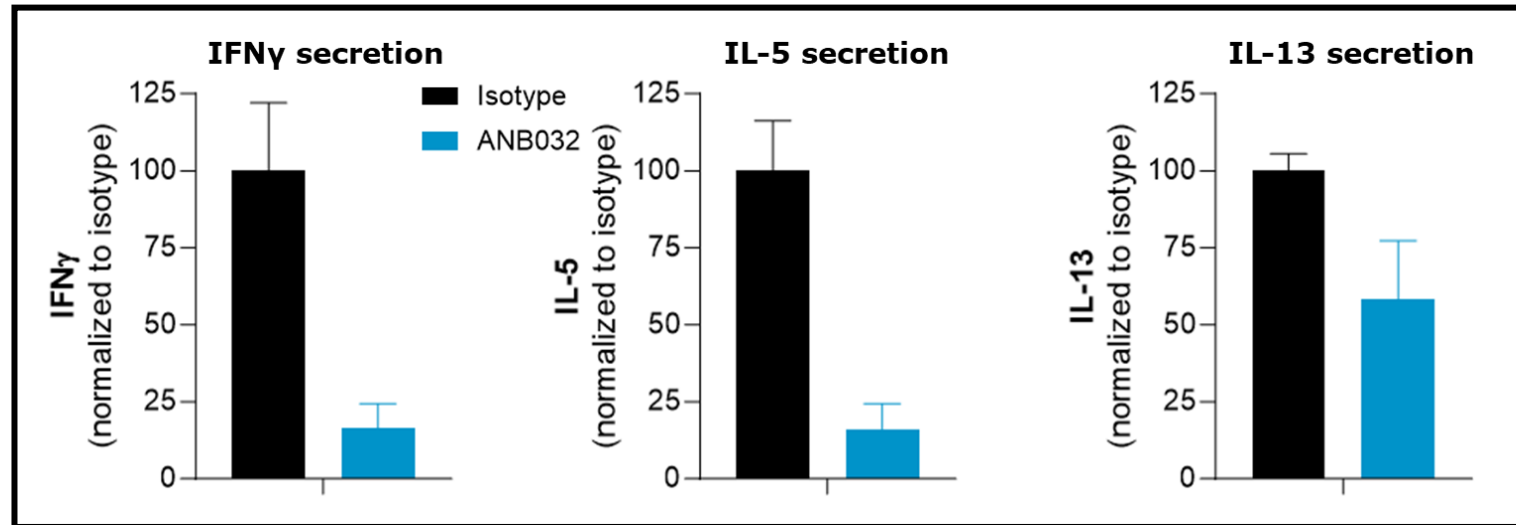
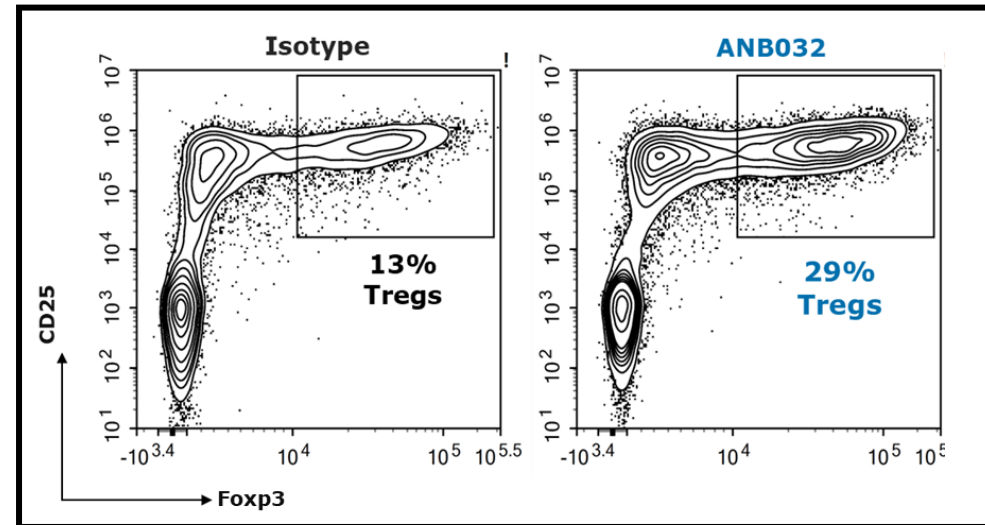
ANB032's agonist signal modulates immune cells

- Inhibits activated T cell proliferation
- Reduces inflammatory cytokine secretion
- Modulates DC function, including inducing T regs

ANB032 Reduced DC Maturation, Antigen Presentation, and Co-stimulatory Molecule Expression In Vitro



ANB032-treated DCs Induced Functional Tregs and Reduced Inflammatory Cytokines in a MLR Assay



Conclusion

- ANB032 reduced the maturation of DCs, reduced MHC II, reduced co-stimulatory molecule expression and contributed to the induction of Tregs in vitro
- ANB032 is being evaluated in an ongoing Phase 2 study in moderate-to-severe AD and data are expected in December 2024 (NCT05935085)

