immune function is unclear, with both costimulatory and proapoptotic roles described. CD30 is transiently upregulated following activation of memory T cells and has been linked to highly activated/suppressive IRF4+ effector Tregs.

**Methods** Here we evaluated the activity of BV on CD30-expressing T cell subsets in vitro and in vivo.

**Results** Treatment of enriched T cell subsets with clinically relevant concentrations of BV drove selective depletion of CD30-expressing Tregs > CD30-expressing CD4+ T memory cells, with minimal effects on CD30-expressing CD8+ T memory cells. In a humanized xenograft model, treatment with BV selectively depleted Tregs resulting in accelerated wasting and robust T cell expansion. The observed differential activity on Tregs is likely attributable to significant increases in CD30 expression and reduced efflux pump activity relative to other T cell subsets. Interestingly, blockade of CD25 signaling prevents CD30 expression on T cell subsets without impacting proliferation, suggesting a link between CD25, the high affinity IL-2 receptor, and CD30 expression.

**Conclusions** Together, these data suggest that BV may have an immunomodulatory effect through selective depletion of highly suppressive CD30-expressing Tregs.

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**Ethics Approval** Animals studies were approved by and conducted in accordance with Seattle Genetics Institutional Care and Use Committee protocol #SGE-024.

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