**MDK-202: an empirically-designed peptideyl agonist of IL-2/15yc receptor, devoid of RAα interaction, unrelated to IL-2 or IL-15, and fused to an Fc-domain for PK enhancement**

**Introduction**

Efforts to adapt IL-2 for immuno-oncology applications focus on modifying the receptor selectivity of IL-2 by reducing RAα, internalizing (to remove) costimulatory Rbγc subunits. We here demonstrate that IL-2, when fused to an IgG-Fc partner for enhanced PK, elicits a biased agonism profile, with reduced effector cell activation and significantly increased proliferation compared to IL-2 alone, consistent with a shift in the ratio of IL-2/DGE to Rbγc cytokine responses, indicating no significant interference with the biology of "off target" Rbγc cytokine receptor interactions.