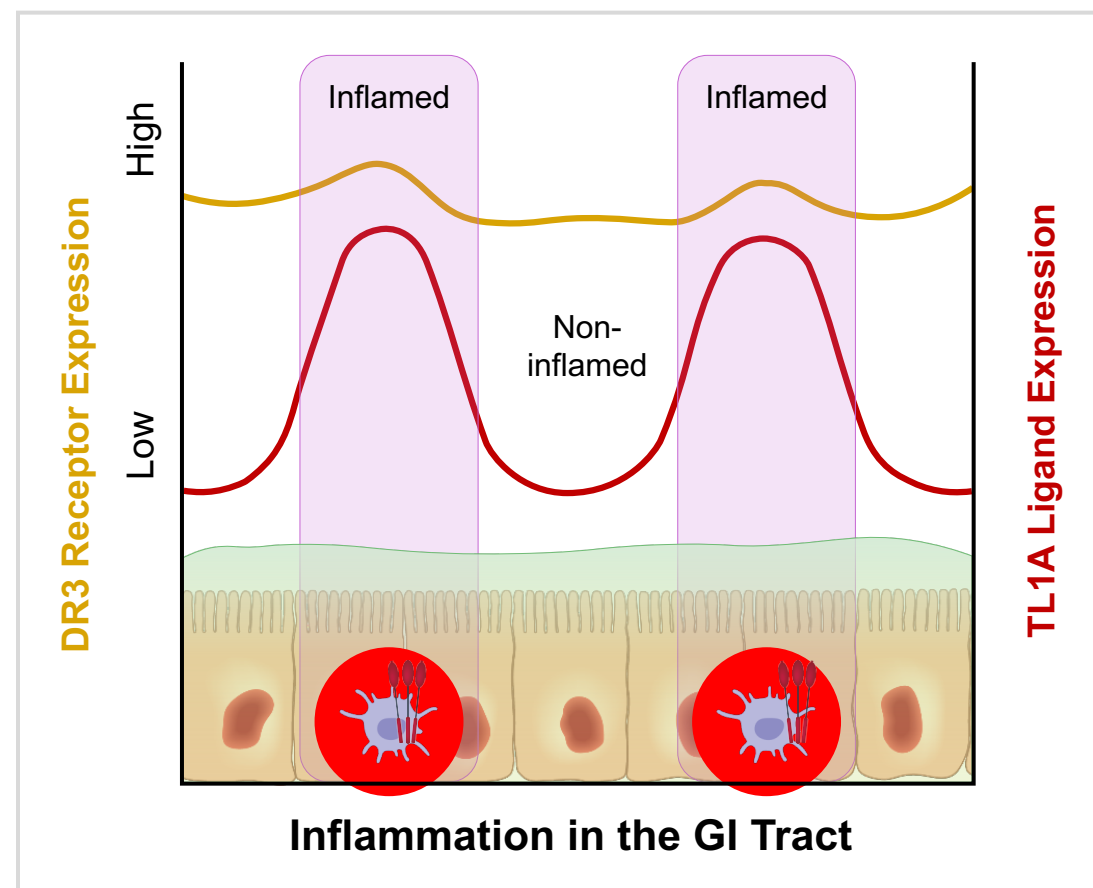
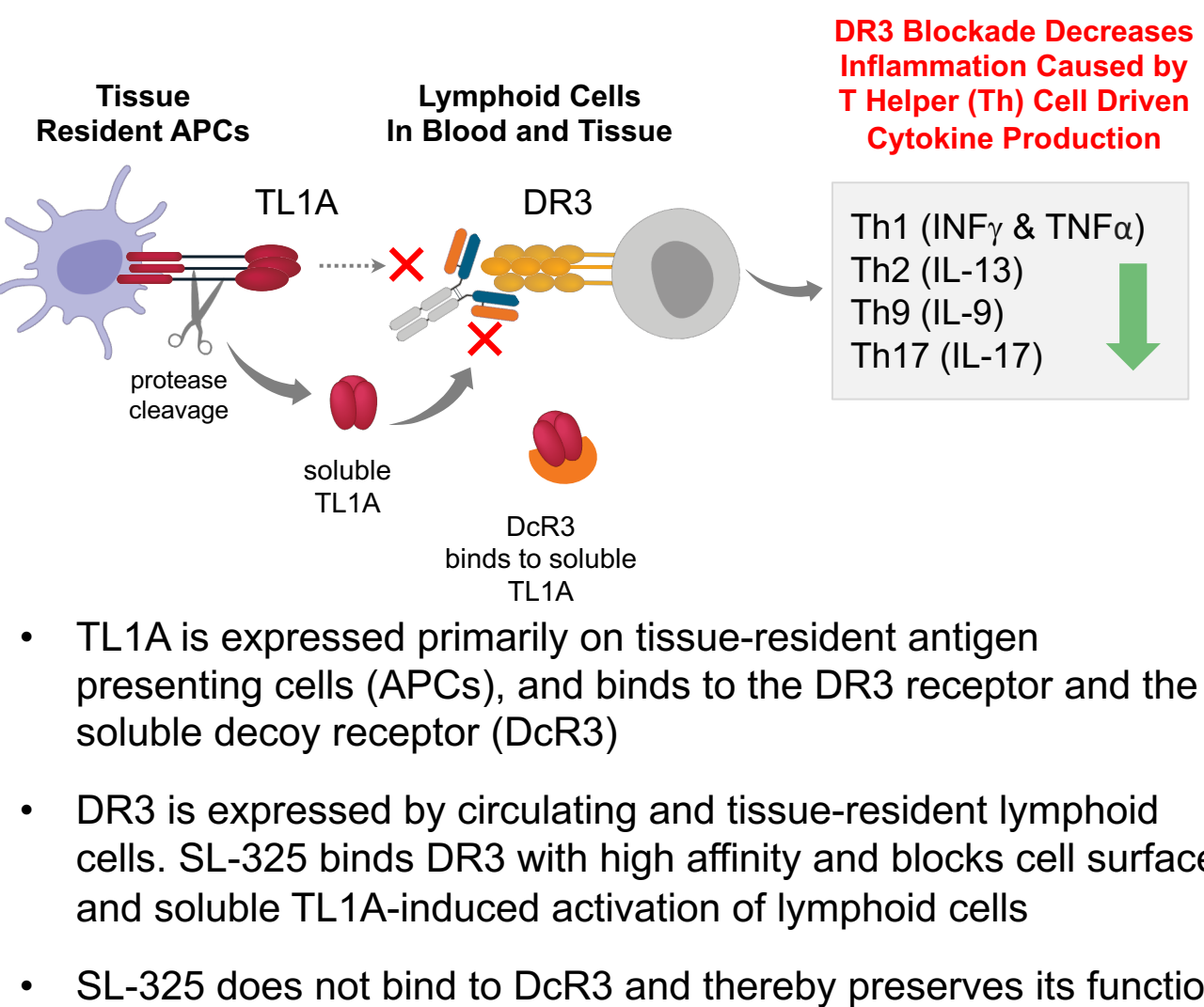


1. Rationale for Targeting DR3 in IBD

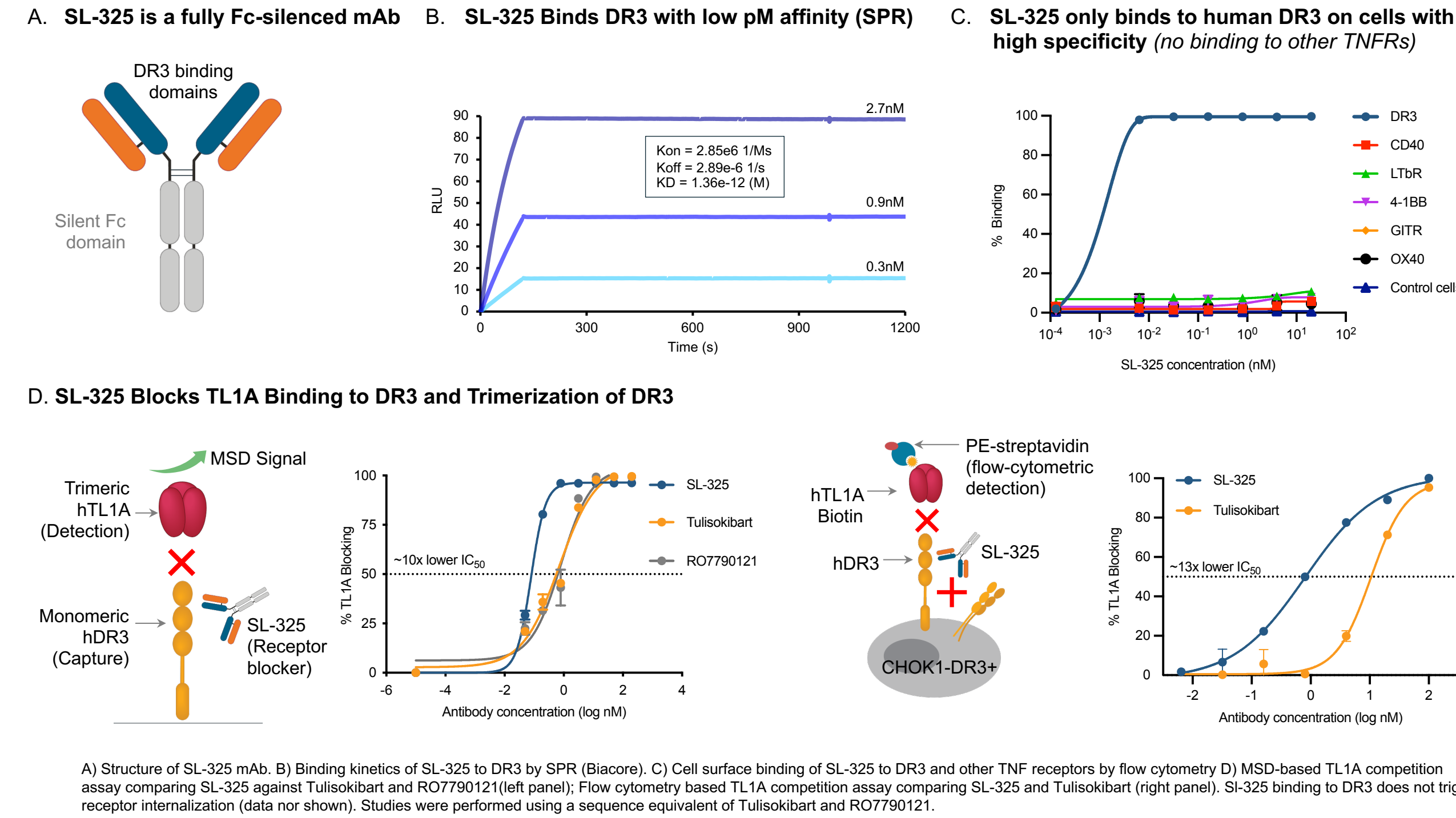
- Death Receptor 3 (DR3, TNFRSF25) is the sole signaling receptor for TL1A.
- Dysregulation of the TL1A/DR3 axis has been implicated in multiple inflammatory diseases, including inflammatory bowel disease (IBD).
- TL1A is found selectively in actively inflamed tissue, where it is primarily expressed by antigen presenting cells in an inducible and transient manner.
- Neutralization and blockade of soluble and membrane forms of TL1A, respectively, by monoclonal antibodies (mAb) has shown significant clinical responses in IBD patients.
- In contrast, DR3 is constitutively expressed by lymphocytes, and is found in greater abundance than TL1A in both actively inflamed and adjacent uninfamed tissue.



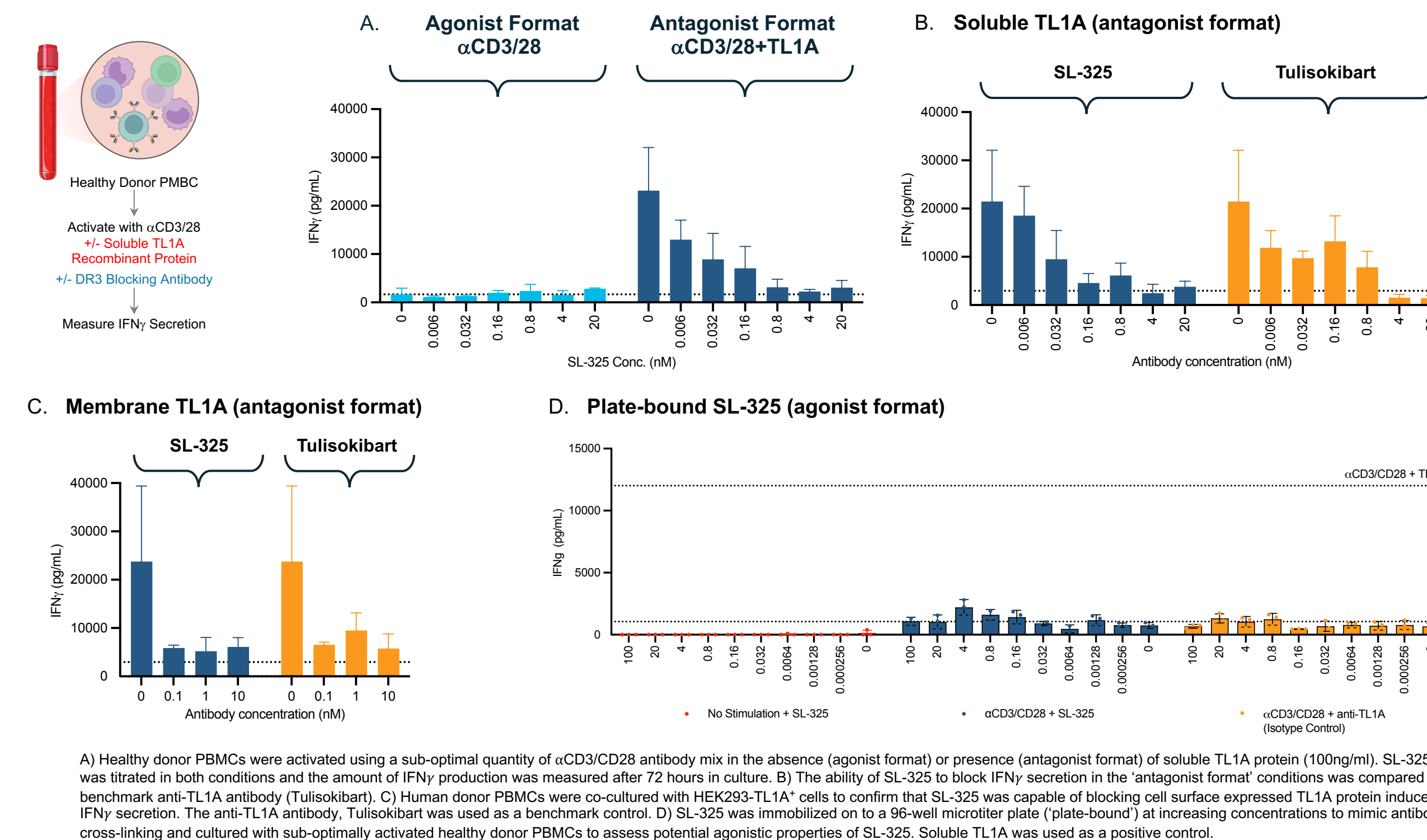
2. SL-325 Mechanism of Action



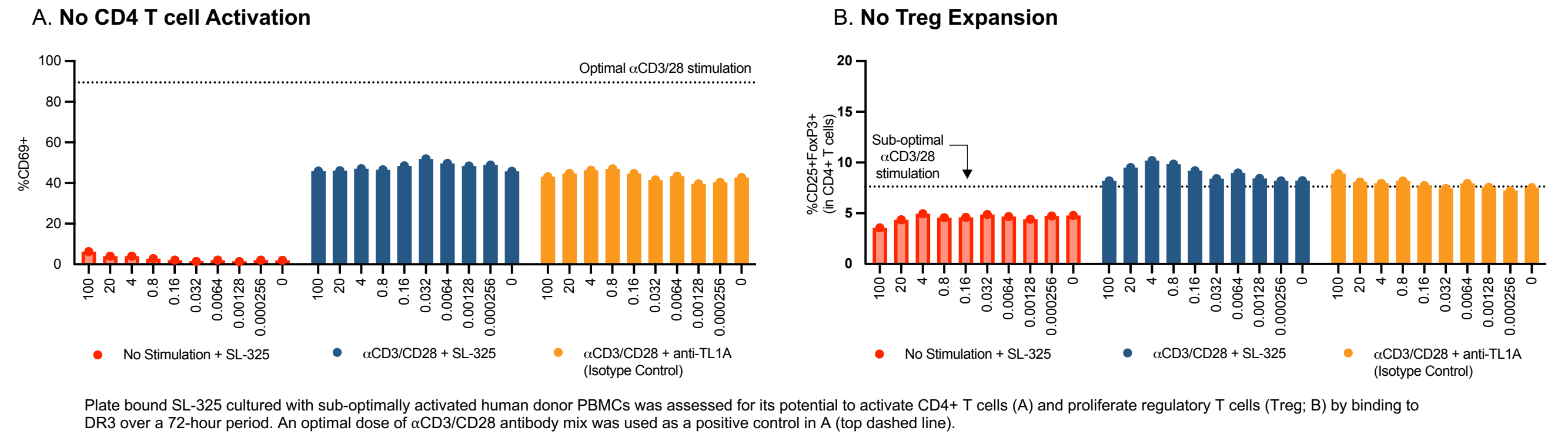
3. High Affinity DR3 Binding, Specificity and TL1A Blocking Activity



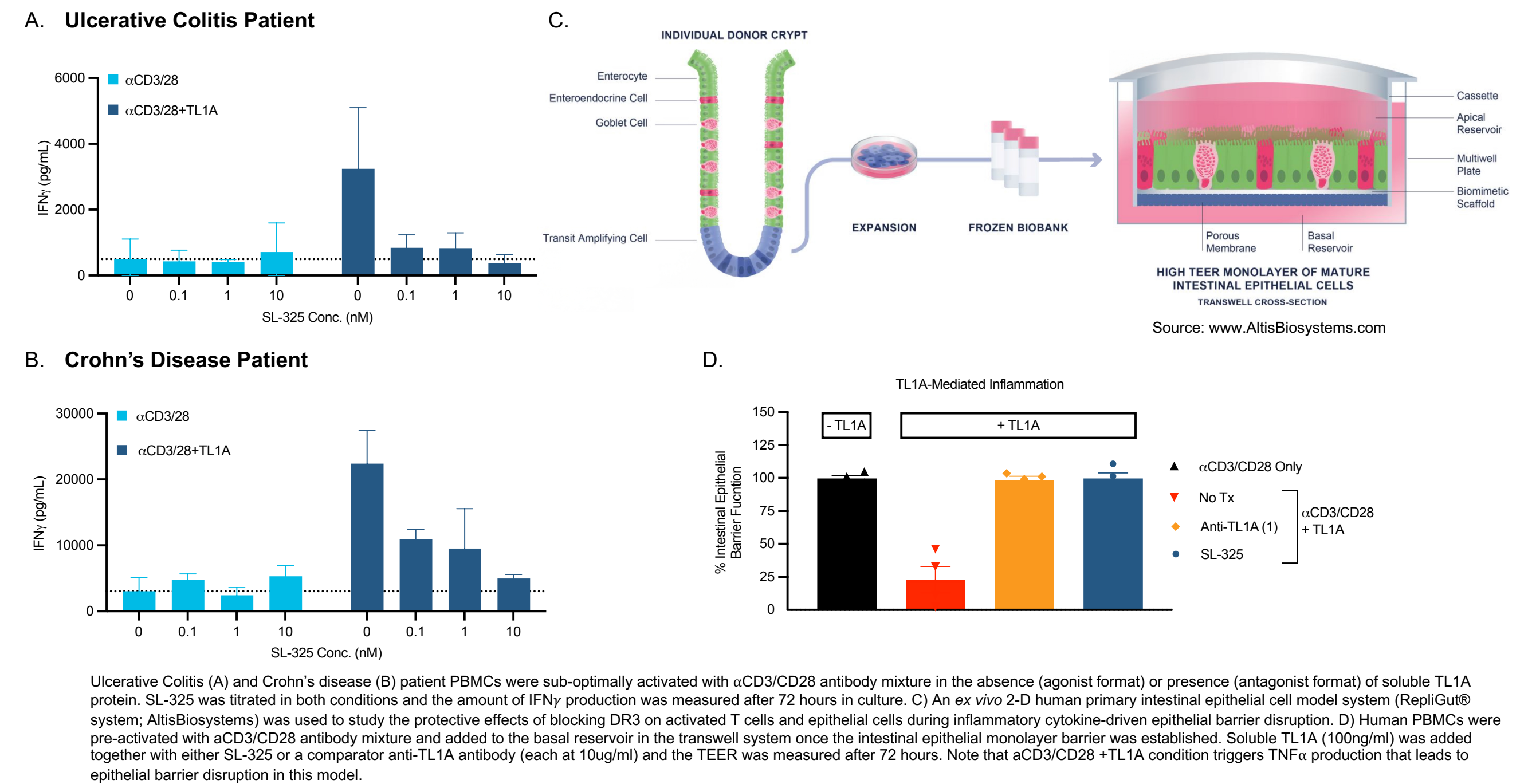
4. SL-325 Blocks Soluble and Membrane TL1A-Induced Immune Cell Activation with No Evidence of Agonistic Properties



5. SL-325 Binding to DR3 Does Not Trigger T cell Activation or Proliferation



6. SL-325 Blocks TL1A-Induced Activation of IBD Patient PBMCs and Protects Epithelial Barrier Disruption Caused by Immune Cell Activation



7. Conclusions

- SL-325 is a fully Fc-silenced humanized IgG monoclonal antibody that has demonstrated high affinity binding to human DR3 and potent antagonistic properties with no evidence of residual agonism.
- SL-325 has undergone a GLP toxicology study in non-human primates and was safe up to 100mg/kg Q2W IV dosing. Results from this study will be presented at ECCO 2025 in Berlin, Germany.
- A Phase 1 clinical trial to determine the safety and recommended Phase 2 dose of SL-325 is planned for 2H25.

