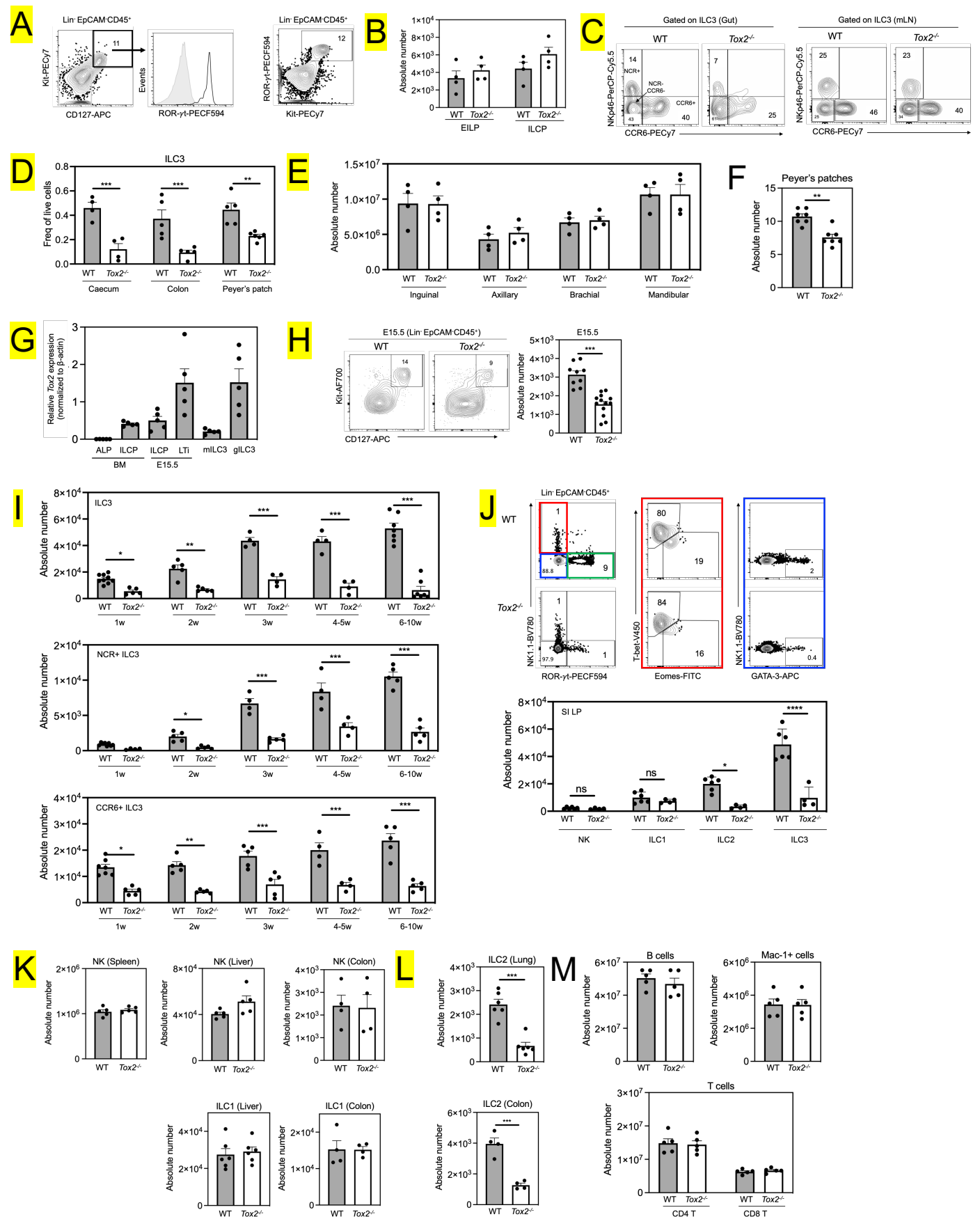


Fig 1: *Tox2* is specifically required for gut ILC3 and not ILC3 at central sites



S1: Tox2 requirements in immune cells

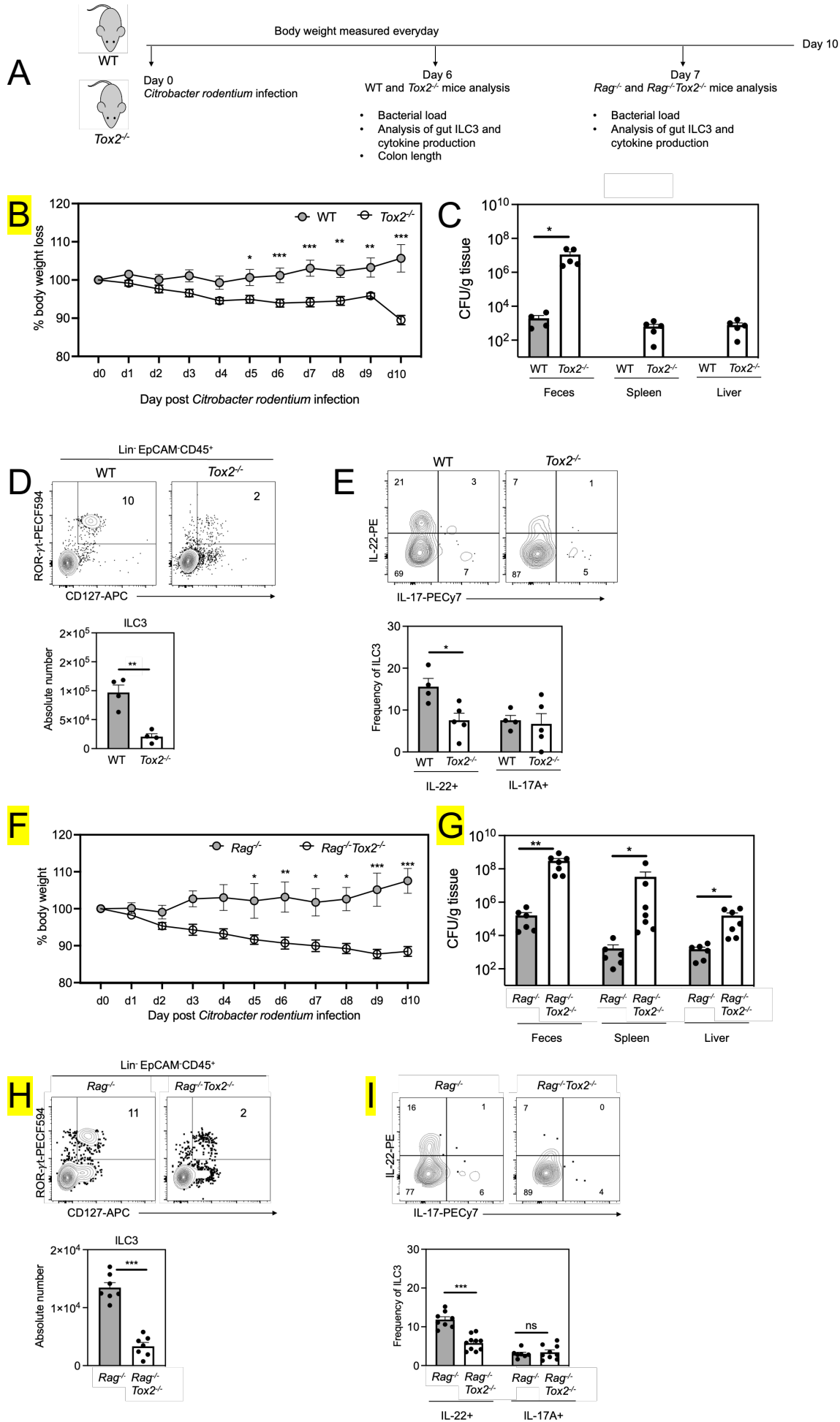
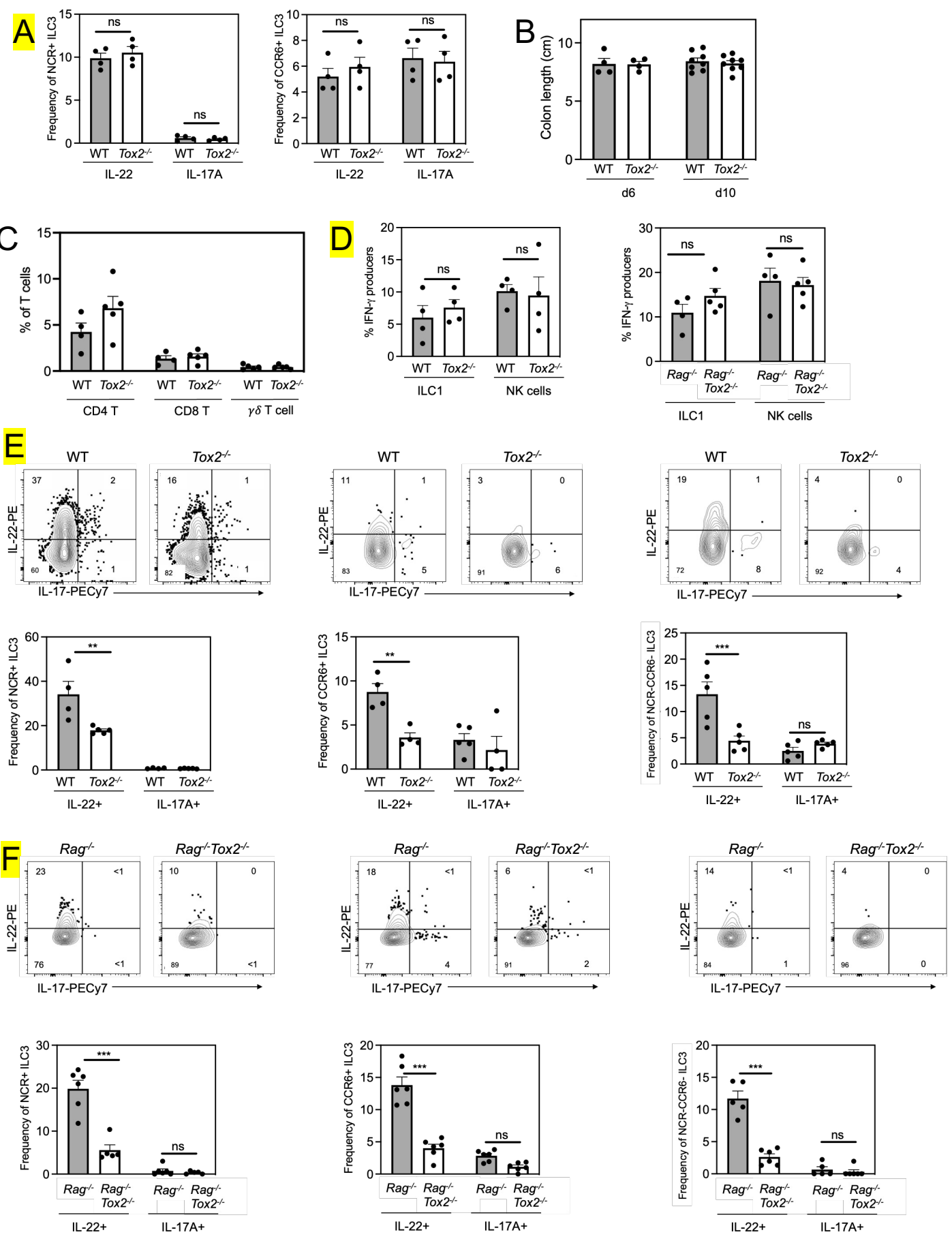


Fig 2: Tox2 ablation leads to reduced ILC3 response upon *Citrobacter rodentium* infection



S2: NCR⁺ and CCR6⁺ ILC3 subsets showed reduced ILC3 response in *Tox2*-deficient mice post *C. rodentium* infection

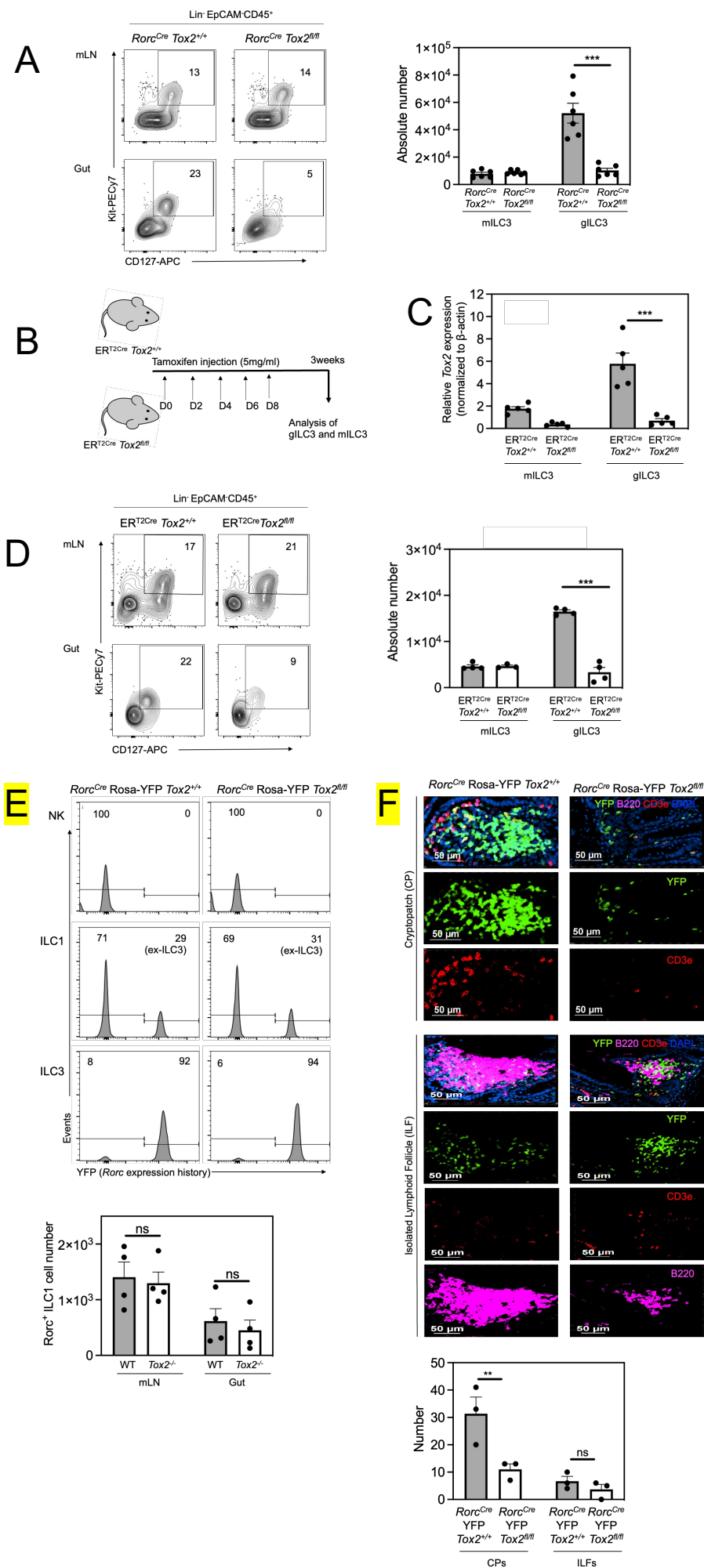
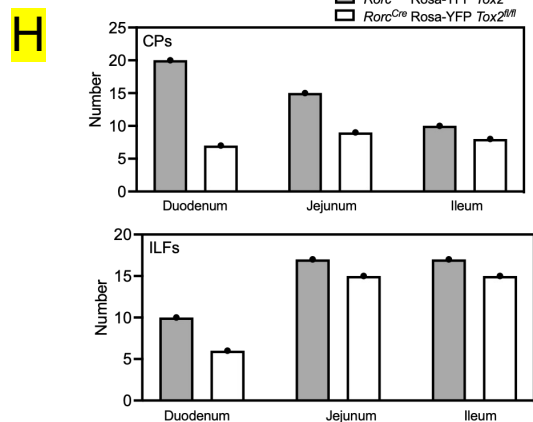
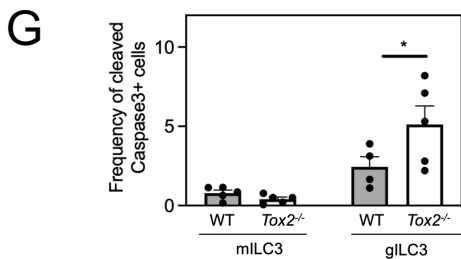
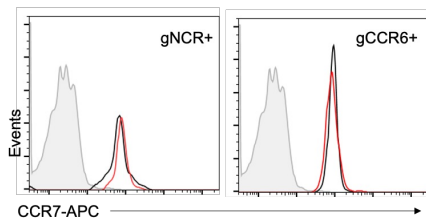
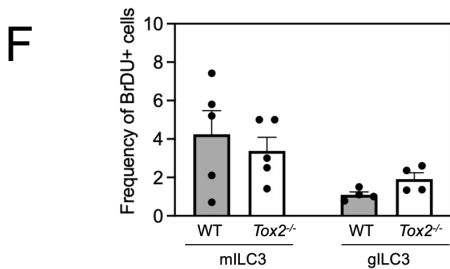
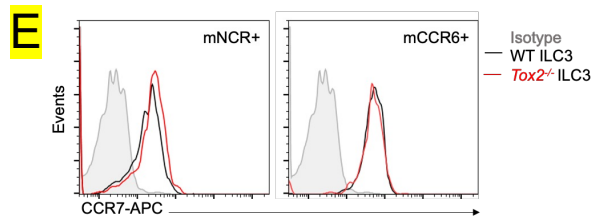
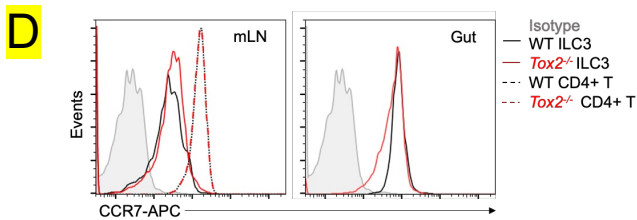
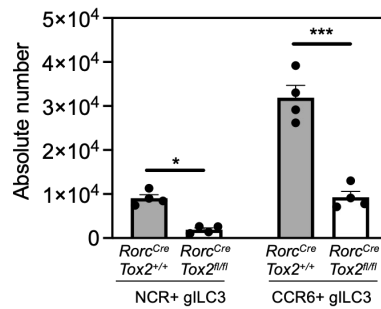
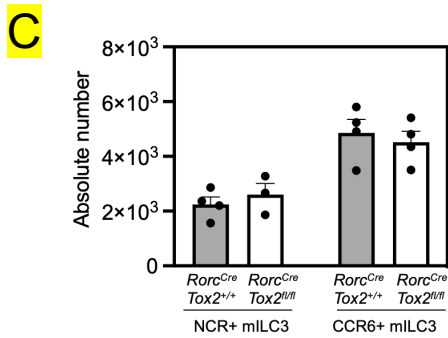
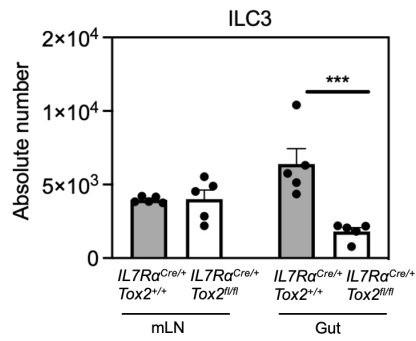
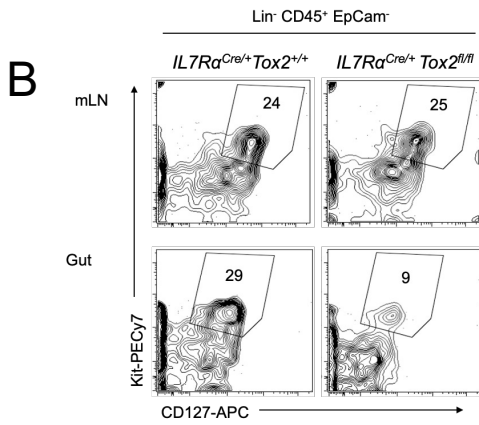
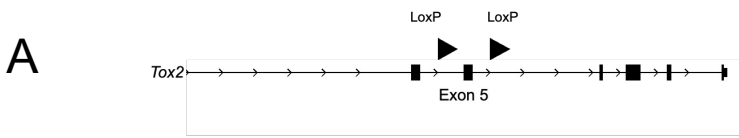


Fig 3: Tox2 is required by gut ILC3 for their persistence in gut



S3: Tox2 is not needed for homing of ILC3 into gut and proliferation of ILC3

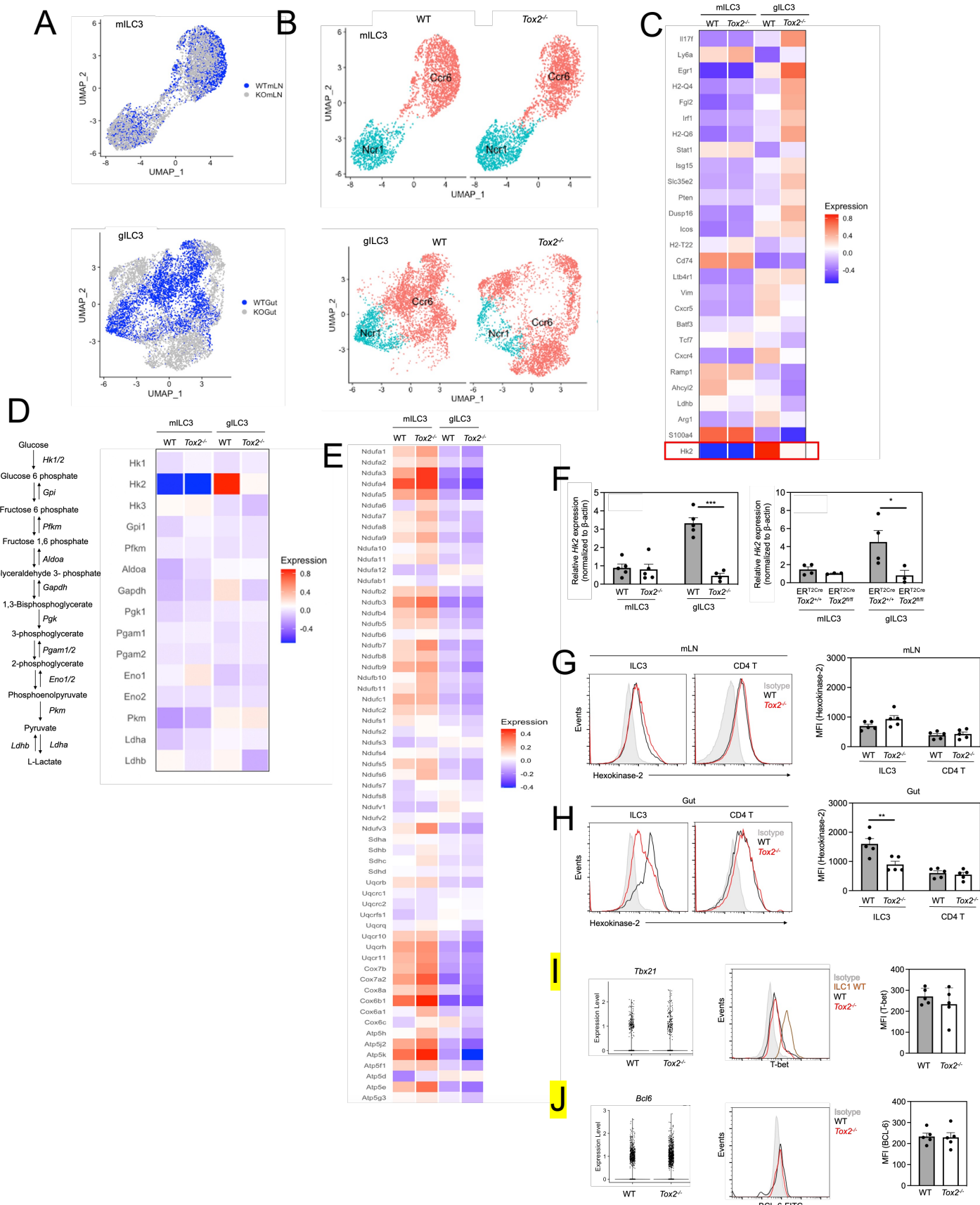
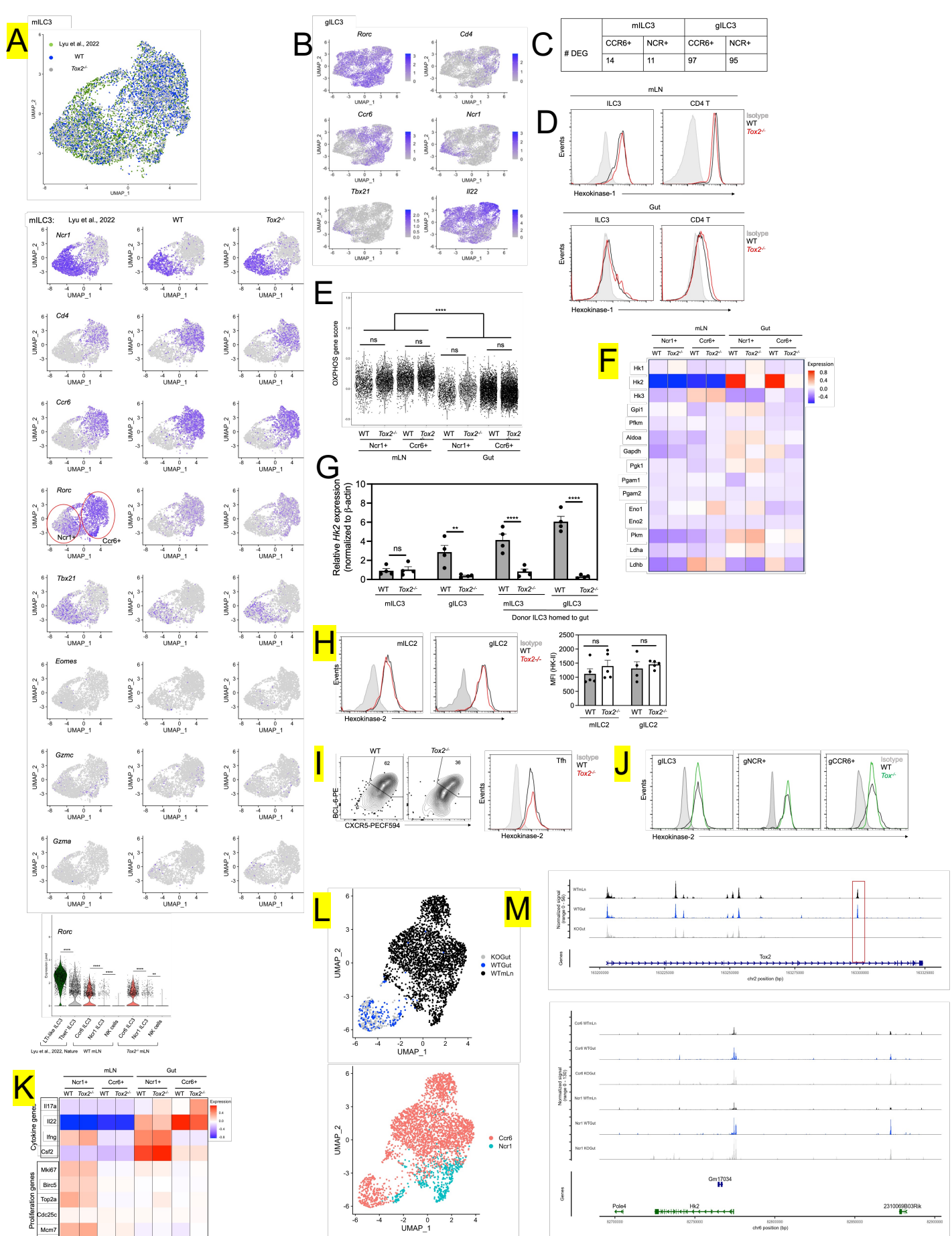


Fig 4: Gut ILC3 from *Tox2*^{-/-} mice are transcriptionally distinct from their WT counterparts



S4: Single-cell transcriptome analysis of ILC3 from mLN and gut

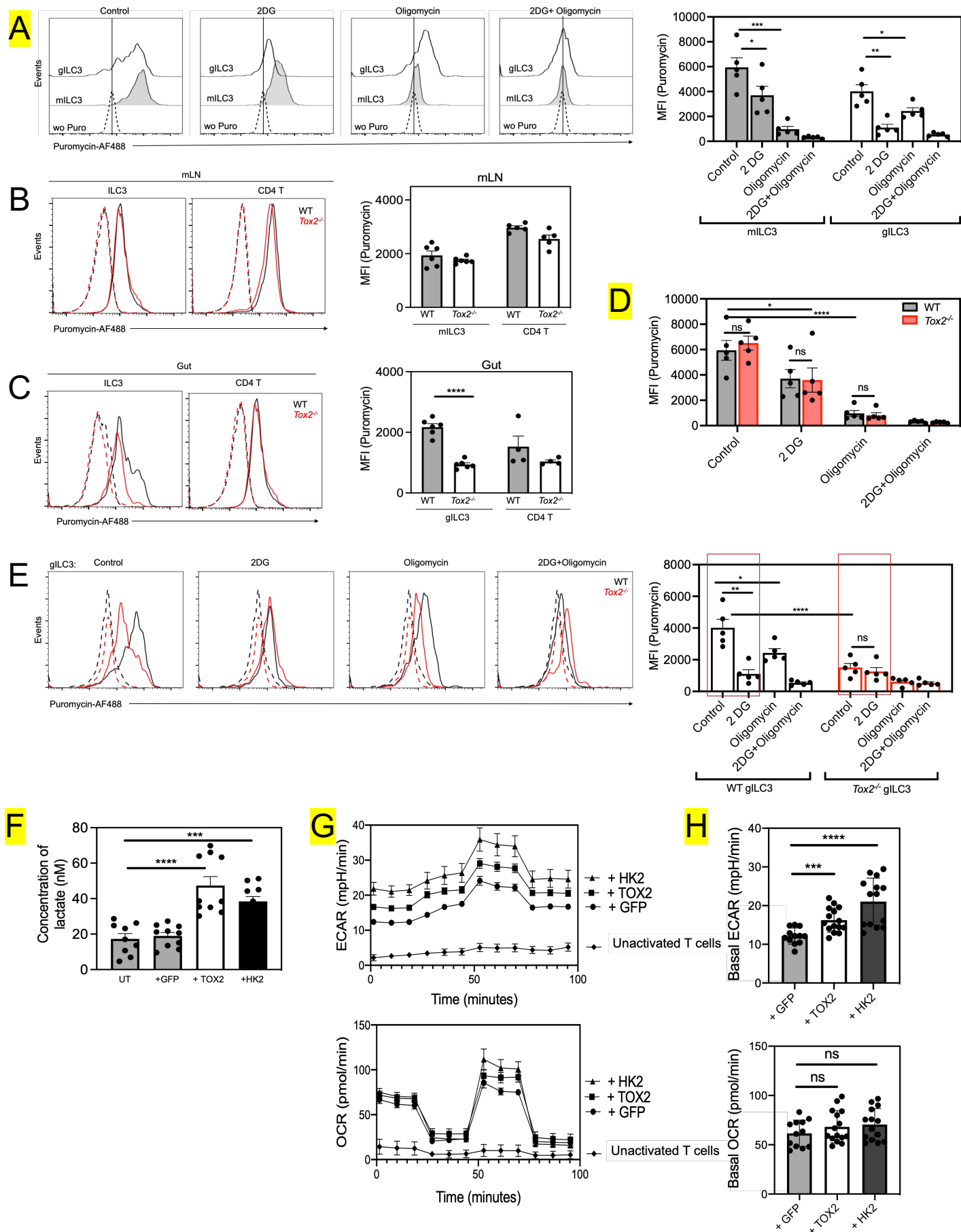
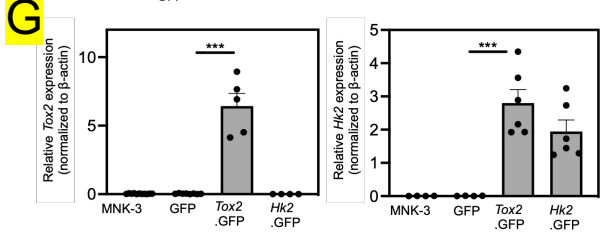
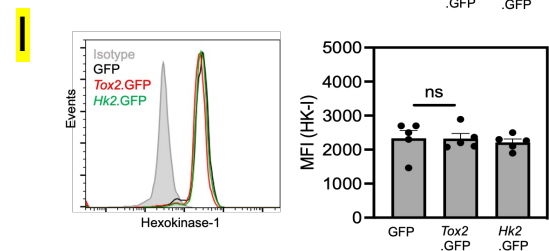
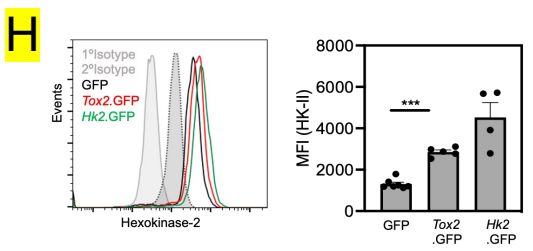
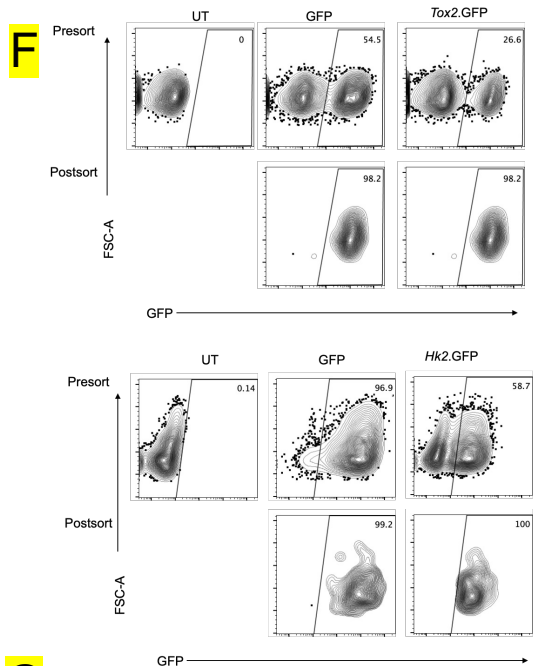
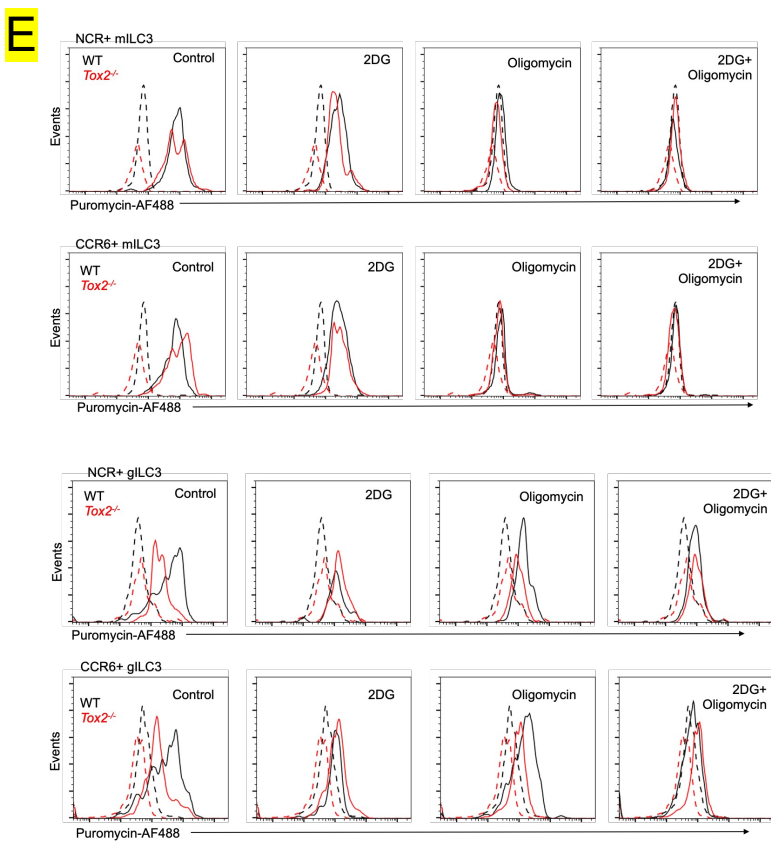
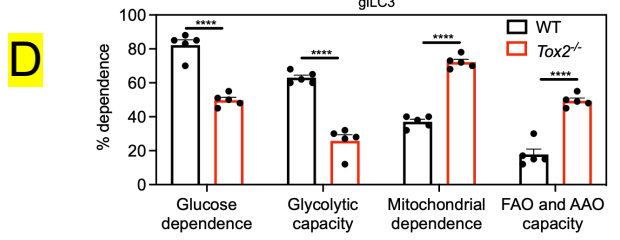
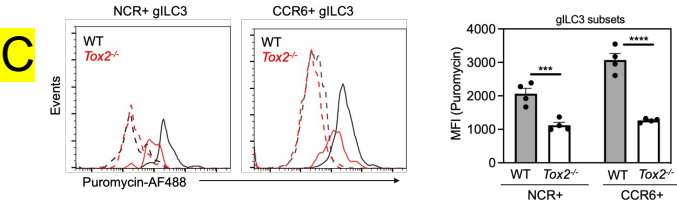
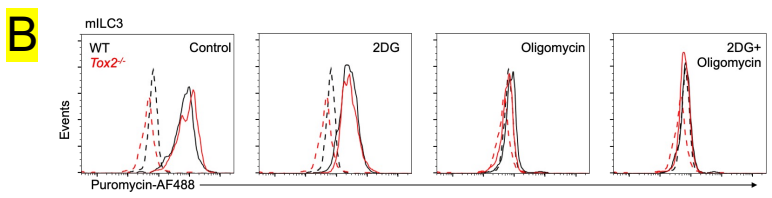
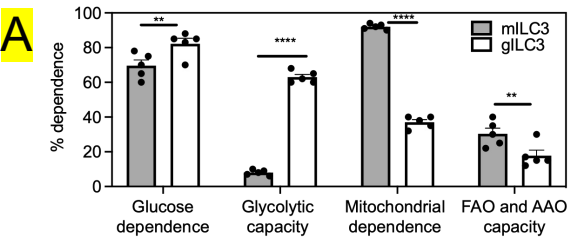


Figure 5: *Tox2* deficiency leads to attenuated protein translation in gILC3 with a decreased dependence on glycolysis



S5: Tox2 deficiency leads to reduced protein translation and decreased dependence on glucose in both ILC3 subsets in gut

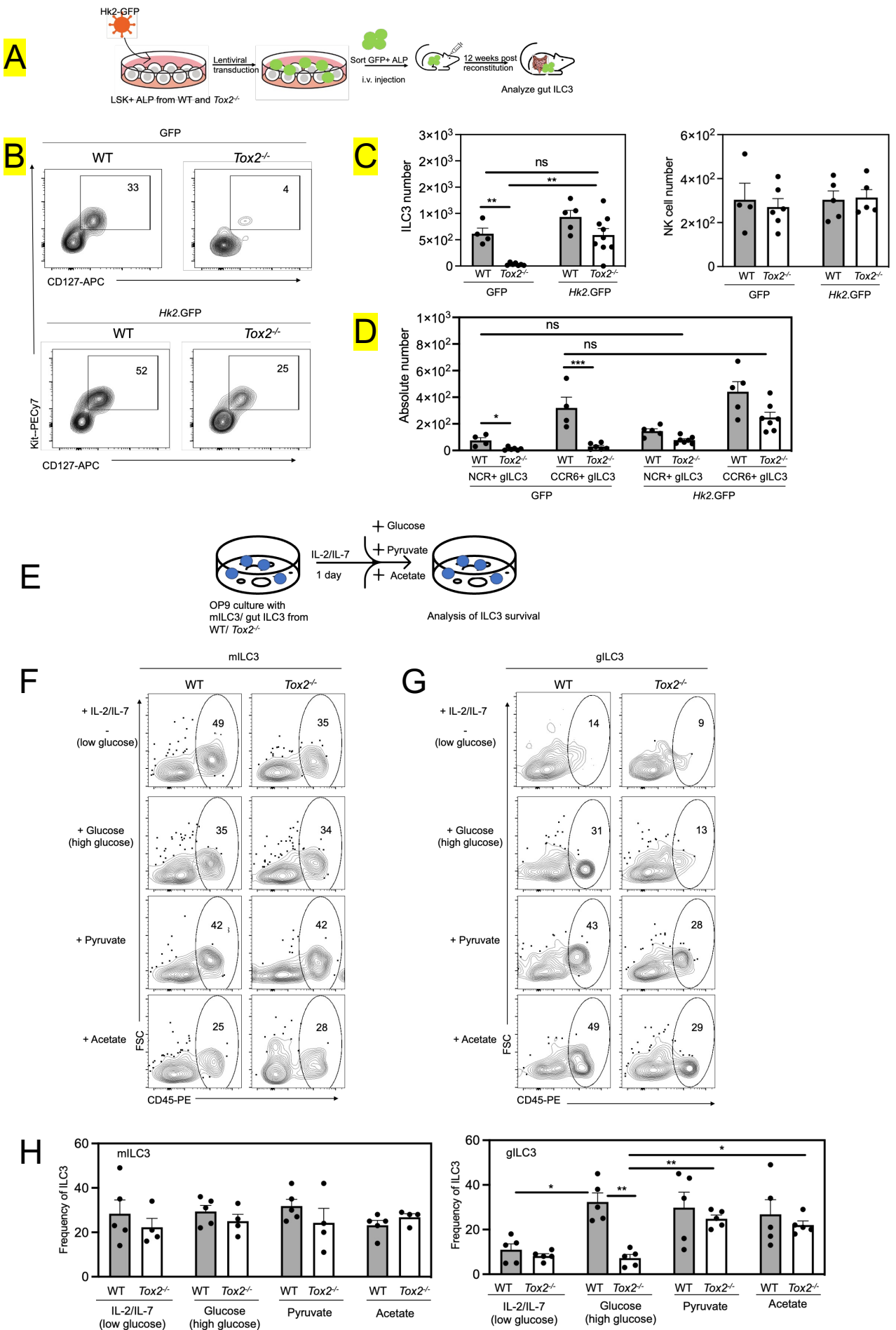


Fig 6: Bypassing and overexpression of hexokinase 2 rescued survival of *Tox2*^{-/-} gut ILC3

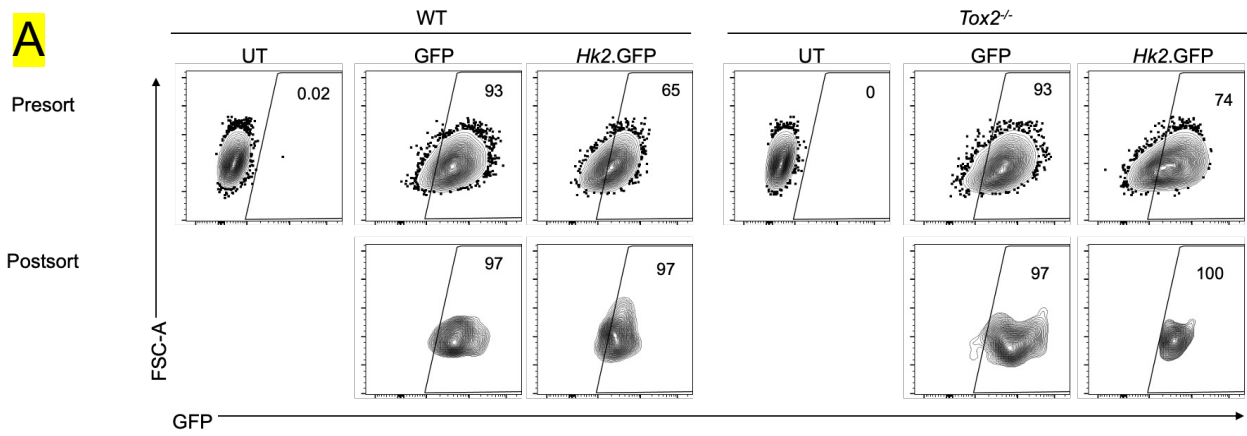
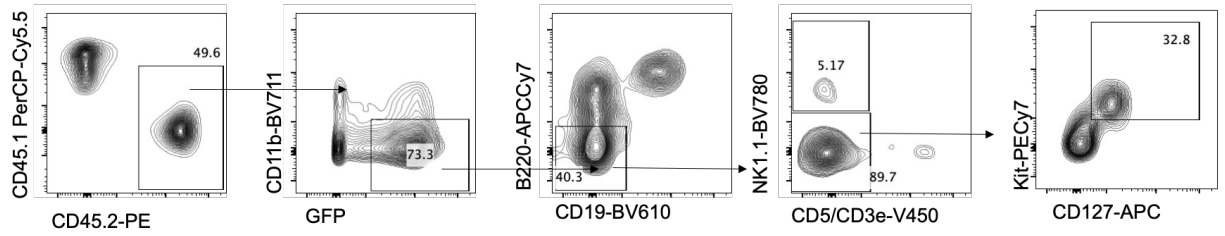
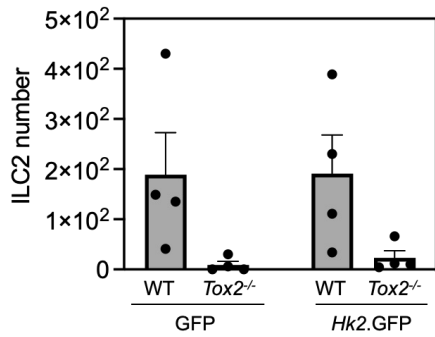
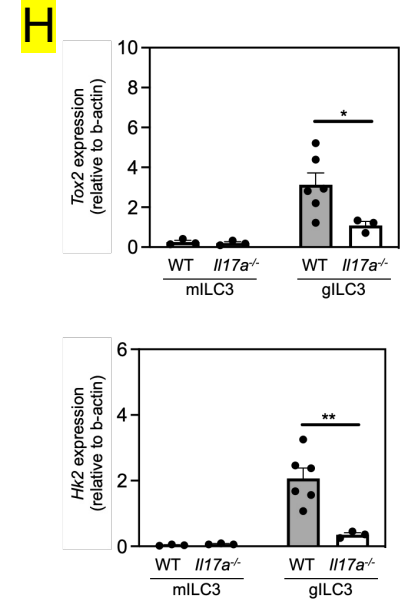
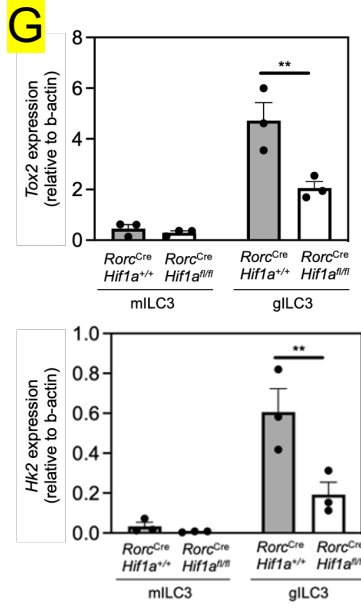
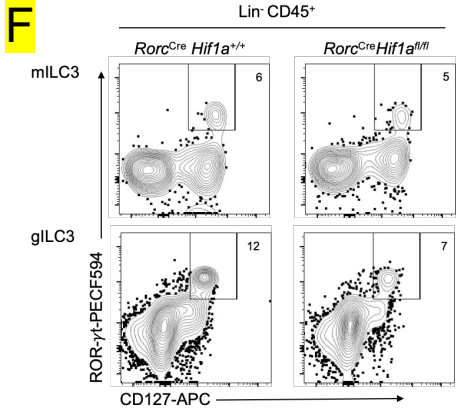
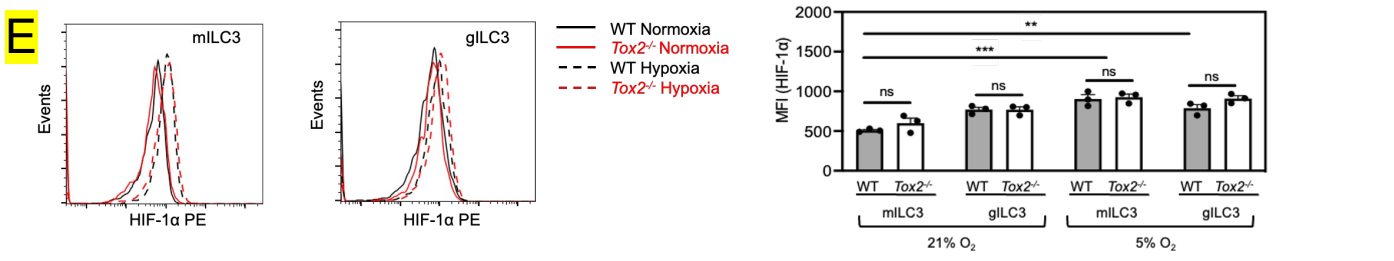
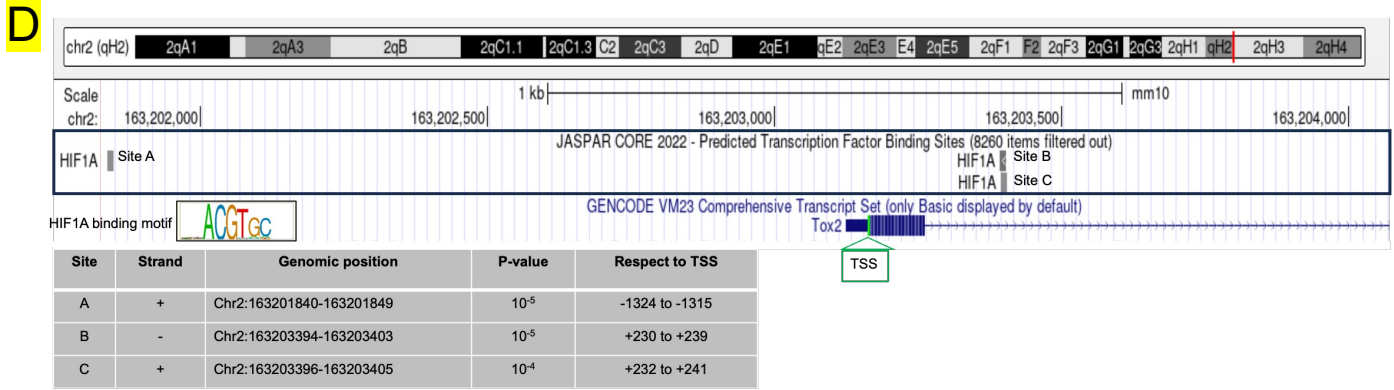
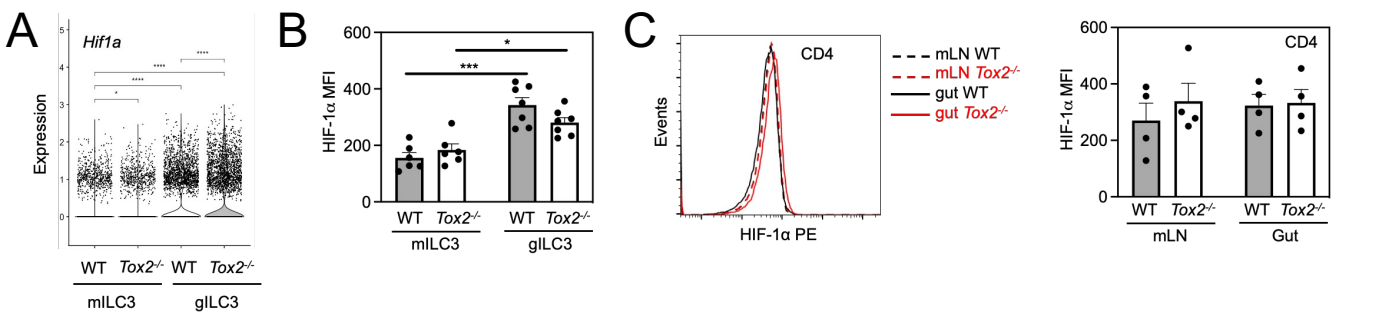
A**B****C**



Fig 7: Hypoxia and IL-17 upregulated Tox2 in gut ILC3



S7: HIF-1α and IL-17A upregulated Tox2 in gut ILC3