### Methods

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</table>

### Results

![Fig 1](image1.png)

- Percentage of CD11b+ DCs
- Percentage of DC11b
- Percentage of CD11b-F4/80+ subM2
- Percentage of CD11c

**Fig 1.** (A) Arthritis score at different time points (left panel). One-way ANOVA test. *P = 0.05, ****P<0.0001. (B) Representative pictures of CIA-CFA+CII-IFA mice receiving PBS (no ICi) or ICi. (C) Arthritis score over time based on ICi regimen.

![Fig 2](image2.png)

- Percentage of CD11b+ DCs
- Percentage of DC11b
- Percentage of CD11b-F4/80+ subM2
- Percentage of CD11c

**Fig 2.** Delineation of major immune cell subsets in spleen. (A) Gating strategy for Flow Cytometry analysis. (B) Quantitative analysis.

![Fig 3](image3.png)

- Percentage of CD4+ T cells
- Percentage of CD8+ T cells
- Percentage of CD19
- Percentage of CD11b

**Fig 3.** Delineation of major T cell subsets in spleen. (A) Gating strategy for Flow Cytometry analysis. (B) Quantitative analysis.

### Conclusion

- Compared with PD-1 inhibitor arthritis group, Th17, Th1,17, CXCR5+ CD8 T cells, Tc1, Tc1,17 were expanded in the combined ICi arthritis group.
- In contrast, TNFα+ CD4 T cells, GM-CSF+ CD8 T cells, both pro-inflammatory and anti-inflammatory Tregs were expanded in PD-1 inhibitor arthritis group.
- Together, like humans, our data suggested that immune profiles underpinning arthritis differ by ICi regimen in our in vivo system.
- We successfully generated in vivo murine model recapitulating the human arthritis-irAE settings. Our model will serve as a powerful tool for us to understand mechanisms underlying arthritis-irAE as well as formulate appropriate therapeutic strategies for arthritis-irAE.

### Future Directions

1. Since CTLA-4 monotherapy group developed arthritis rapidly on D32 after first ICi immunization, we need to analyze mice before D32.
2. Experiment needs to be repeated in order to detect pro-inflammatory and anti-inflammatory Tregs and other intracellular cytokine changes between groups of interest.

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### References