RESULTS

A Single Dose of THOR-707 Increases Intracellular T Cell Function and TCR Diversity

THOR-707, the THOR-707 single agent study, Day 17

THOR-707 Promoting the Establishment of Persistent Memory T Cell Responses Preventing CT26 Tumor Growth in Surviving Animals Challenged by Re-injection of CT26 Cells

THOR-707 Single Agent Study, Day 17

Combination Study: Overall Survival (p=0.036)

THOR-707 Does Not Induce Vascular Leak Syndrome in NHP

THOR-707 Alone or Combined with Pembrolizumab or Nivolumab Does Not Elicit Evidence of Cytokine Release Syndrome with DOX or Carbo/DOX Whole Body System

RESULTS

CONCLUSIONS

THOR-707, a Novel Not-alpha IL-2, Promotes All Key Immune System Anti-tumor Actions of IL-2 Without Eliciting Vascular Leak Syndrome (VLS)

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THOR-707 is an engineered IL-1 protein with increased PLK1 and PLK3 binding, which limits its immunostimulatory activity, and on its own it might cause a reduction of the vascular endothelium, which leads to eosinophilic recruitment and activation, resulting in the often elevated expression of the checkpoint receptor forms (27).

THOR-707 has a high affinity for PEG covalently attached to the T cell receptor (TCR) (ref. 3) to identify cell surface receptors and to determine whether the T cell receptor is biased toward IL-2Rβγ, which leads to expansion and differentiation toward a Th1 phenotype. In THOR-707, the IL-2Rα is blocked with nivolumab. This is important because IL-2Rα pathway blockage with nivolumab can cause T cell exhaustion (ref. 4).

The THOR-707-induced increase in T cell clonal diversity is also observed in mice (Day 8).