Number of Figures: 1

Figure 1. STING-Activating CDNs Drive T Cell Priming

Figure 2. Development of Clinical Compound ADU-S100 ($100)

Figure 3. STING Activation Can Be Modulated to Induce Local Versus Systemic Immune Activation

Figure 4. CDB+ T Cells Are Necessary for Anti-Tumor Immunity Elicted by Immunogenic, but Not Ablative, Doses of ADU-S100

Figure 5. Combination of ADU-S100 and pdp-1 Enhances Non-Injected Tumor Control in a CD8+ T Cell-Derived Manner in 4T1 Mammary Carcinoma Tumor-Bearing Mice

Figure 6. The Resistance of CT26 Pten+ Tumors to pdp-1 Was Overcome by Combining with ADU-S100

Figure 7. Combination of ADU-S100 and pdp-1 Enhances Non-Injected Tumor Control and Induced Durable Immunity

Figure 8. Combination of ADU-S100, pdp-1, and cTLLA4 Enhance Anti-Tumor Immunity in B16.F10 Melanoma Tumor-Bearing Mice

CONCLUSIONS

- The magnitude of tumor-specific CDB+ T cell responses is dependent on the dose of ADU-S100.
- Lower immunogenic dosing regimens result in local STING activation and adaptive immune responses.
- More aggressive ablative dosing regimens, while effective in clearing injected tumor cells, result in failure to kill recrudescing tumor outgrowth.
- Immune doses of ADU-S100 combine effectively with checkpoint inhibitors pdp-1 and cTLLA4 in multiple tumor models including a coelomic carcinoma.
- Together, these results identify immune correlates of STING-mediated anti-tumor efficacy in mice and illustrate the potential of combining ADU-S100 with checkpoint inhibitors for the treatment of human cancer.