Background

- Ad5/3-E2F-d24-hTNFa-IL2 (TILT-123, Figure 1) is designed to enhance adoptive T-cell therapies (ACT) and checkpoint inhibitors
- ACT (TILs, CAR-Ts, and TCRs) only work in hematological cancers and melanoma, not in other solid tumors
- TILT-123 could improve ACT of solid tumors by
  a) inducing danger signaling (oncolysis);
  b) recruiting T cells into tumor (TNFa);
  c) enhancing T-cell proliferation and differentiation (IL2)

Aims

- To study if TILT-123 enhances ACTs and PD-1 checkpoint inhibitor antibodies
- To investigate systemic immunological and antitumor effects with local treatment
- To replace the need for toxic preconditioning chemotherapy and postconditioning systemic recombinant IL2

Results

Adenovirus coding for TNFa and IL2 enhances ACT and stimulates cell graft trafficking into tumors.

Local injection with TILT-123 induces systemic antitumor effects in distant non-injected tumors.

Conclusions

- TILT-123 is a potential enhancer for ACT and checkpoint blockade
- TILT-123 spreads to distant tumors and induces systemic antitumor effects
- Toxic preconditioning with chemotherapeutics and postconditioning with systemic IL2 could be replaced with TILT-123
- A clinical trial with TILT-123 TIL therapy will start in 2019

References

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