elicera THERAPEUTICS

Fighting cancer with next generation cell and gene therapies

iTANK *:

A universal CAR/TCR T-cell enhancement technology platform for durable CD8+ killer T-cell bystander antitumor responses

Preclinical data have demonstrated:

BROAD ATTACK ON MULTIPLE TARGETS

- Combined induction of an immunologically "hot" tumor microenvironment with epitope spread and activation of endogenous T-cells
- Applicable both in blood cancer and solid tumors

EFFICIENT AND DURABLE CAR T-CELLS

- Improved function and less exhaustion of CAR/TCR T-cells

SAFE

- No added toxicity

UNIVERSAL

- Universally compatible with CAR/TCR T-cell therapies

PROOF-OF-CONCEPT

- Data published in Nature Biomedical Engineering in April 2022

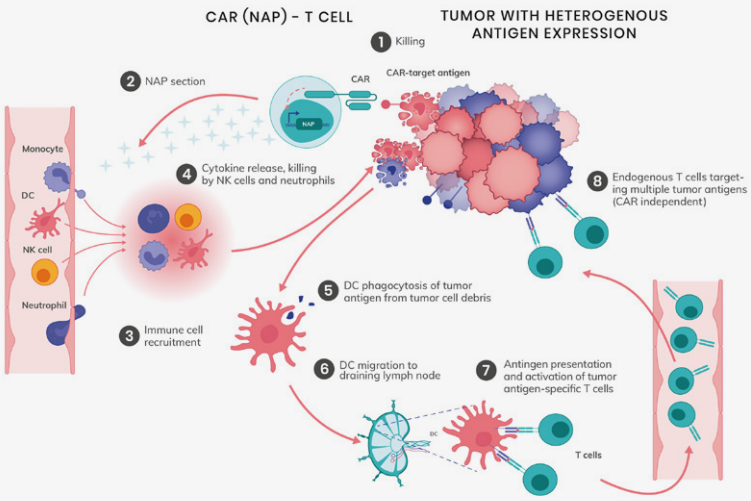
iTANK meets the following challenges in solid tumors:

1. ANTIGEN HETEROGENEITY

By generating a multi-targeted attack on tumor cells through activation of endogenous CD8+ killer T-cells.

2. HOSTILE TUMOR MICROENVIRONMENT

By inducing the formation of an immunologically 'hot' microenvironment that supports dendritic cell maturation and bystander immune responses.



Data recently published in Nature Biomedical Engineering** show that:

- CAR T-cells expressing a pluripotent pro-inflammatory neutrophil-activating protein (NAP) from Helicobacter pylori trigger endogenous bystander T-cell responses against solid cancers.
- In several mouse tumor models, CAR (NAP) T-cells lead to slower tumor growth and higher survival rates than conventional mouse CAR T-cells, regardless of target antigen, tumor type and host haplotype
- In tumors with heterogeneous antigen expression, NAP secretion induced the formation of an immunologically 'hot' microenvironment that supported dendritic cell maturation and bystander responses, as indicated by epitope spreading and infiltration of cytotoxic CD8+ T-cells targeting tumor-associated antigens other than the CAR-targeted antigen
- CAR T-cells armed with NAP neither increased off-tumor toxicity nor hampered the efficacy of CAR T-cells, and hence may have advantageous translational potential.

* iTANK: immunoTherapies Activated with NAP for efficient Killing
** DOI number: 10.1038/s41551-022-00875-5

If you are interested in licensing or co-developing TCR/CAR T-cells enhanced with the iTANK-platform, please contact:

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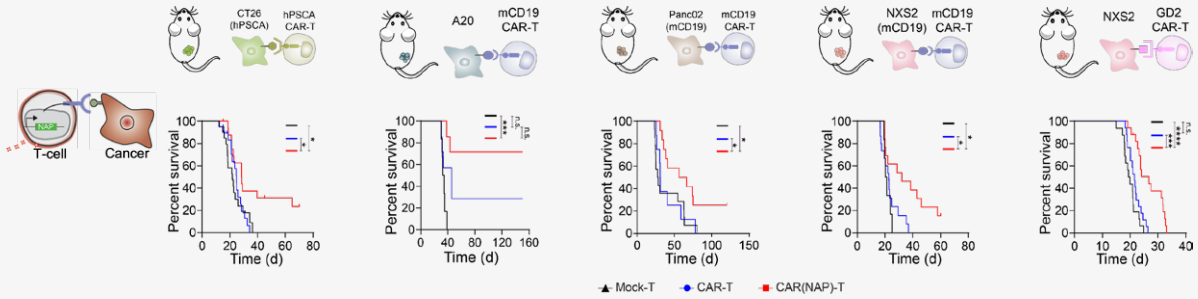
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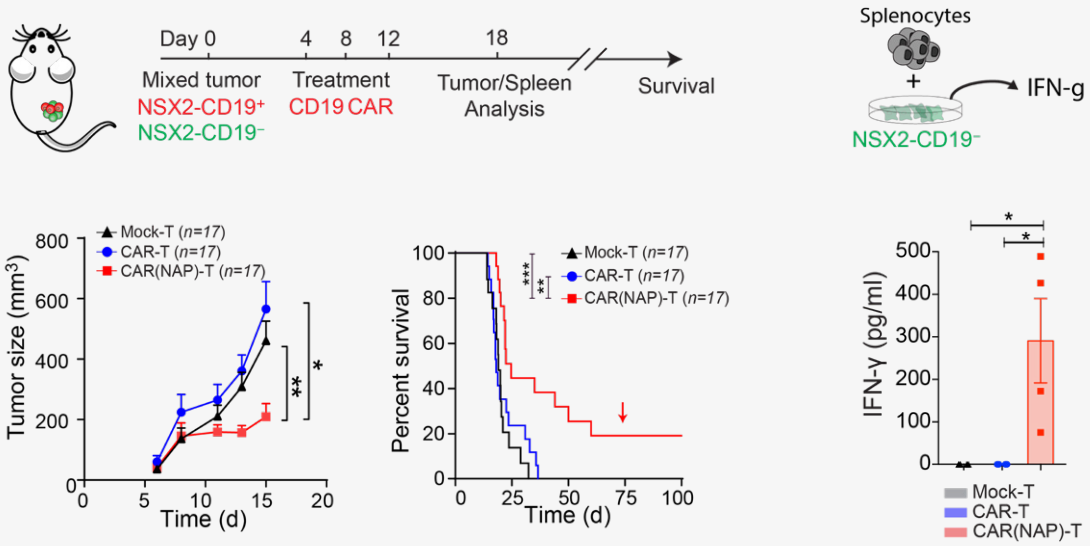
CAR(NAP)-T: Effective in multiple cancer models

Different Tumors	Colon cancer	Lymphoma	Pancreatic cancer	Neuroblastoma
Different Mouse	Balb/c		A/J	C57bl/6
Different CAR	PSCA	CD19		GD2



CAR(NAP)-T treatment showed superior efficacy against several different tumors regardless of the CAR-targets, the tumor types, and mouse haplotypes.

CAR(NAP)-T: Induce bystander immune responses



Mice harboring mixed tumors (1:1 mixture of CD19+ and CD19- tumor cells) were treated with either conventional CAR-T or iTANK-armed CAR(NAP)-T.

CAR(NAP)-T showed enhanced tumor growth inhibition, and prolonged mice survival.

Survived mice are able to reject CD19-negative tumor re-challenge, indicating the establishment of bystander immunity.

Endogenous T cells (splenocytes) from survived mice are able to recognize and react against CD19-negative tumor cells, confirming the establishment of bystander immunity.

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