Further improvement of durability and anti-tumour response rates is likely to result...

KY1055 modulates T cell function as measured by the increase of T-cell proliferation and survival.

The rationale for targeting of PD-L1 and ICOS using a bispecific antibody is based on the combination of therapies targeting, for example, different mediators of T cell function.

KY1055’s high affinity binding allows for a selective depletion of the regulatory T cell (TReg) population.

KY1055 demonstrates monotherapy efficacy in several syngeneic models that are poorly responsive to the respective monotherapies and added potency is seen when used in combination with anti-CTLA-4 and PD-1.

KY1055 depletes TReg and improves CD8+:TReg ratio in the TME.

KY1055 elicits IFNγ release by blocking PD-L1/PD-1 and driven by ICOS agonism.

KY1055 induces ICOS+ CEM cells killing by ADCC.

KY1055 bridges ICOS and PD-L1 expressing cells.

KY1055 demonstrates anti-tumour efficacy in CT26 model.

KY1055 elicits IFNγ release by blocking PD-L1/PD-1 and driven by ICOS agonism.

KY1055 demonstrates anti-tumour efficacy in CT26 model.

KY1055 is a human IgG3 antibody that binds to ICOS (1-359) on the Fab's and PS (P1-1-630) on the Fab’s Fv region with strong binding affinities.

KY1055 is a novel ICOS/PD-L1 bispecific antibody, efficiently enhances T cell activation and delivers a potent anti-tumour response in vivo.

KY1055 is a novel ICOS/PD-L1 bispecific antibody, efficiently enhances T cell activation and delivers a potent anti-tumour response in vivo.

Conclusions

KY1055 is a novel ICOS/PD-L1 bispecific antibody that binds with high affinity to PD-L1 and ICOS and modulates T cell function as measured by:

1. Increasing levels of IFNγ as a result of PD-1/PD-L1 blocking, but also as a direct effect of PD-L1 binding dependent ICOS-driven agonism.

2. Modulation of tumour-suppression loops in the TME by restraining targeting high PD-L1 expressing cancer cells.

3. Induction of anti-tumour effector T cell activation in several syngeneic models that are poorly responsive to the respective monotherapies and added potency is seen when used in combination with anti-CTLA-4 and PD-1.

4. T cell dependent further development of KY1055 for the treatment of solid tumours.

References:


Acknowledgments:

We thank our Kymab colleagues for stimulating discussions and helpful feedback.