MDNA132 was generated from yeast display screening of a large number of mutations at key sites required for IL13 binding to IL13Rα1, IL13Rα2, and IL4Rα.

MDNA132 has enhanced affinity for IL13Rα2 (10X lower $K_D$), and greatly decreased affinity towards IL13Rα1 (365X higher $K_D$).

Further *in vitro* characterization of MDNA132 is ongoing.

MDNA132 has 16,000,000 X greater specificity than IL13 towards the cancer antigen IL13Rα2 vs ubiquitous IL13Rα1.

MDNA132 is available for license for use as an attractively differentiated targeting domain for inclusion in CAR-T constructs.

**MDNA132 TO TARGET CANCER**

- IL13Rα2 has been validated as a target to treat glioblastoma; an IL13-toxin fusion reaching phase III clinical trials (*PMID: 20511192)*.

- IL13Rα2 is now a targeted antigen for CAR-T cells using a mutated IL13 (zetakine, Mustang Bio), showing early signs of efficacy in phase I trials (*Brown et. Al., ASGCT meeting, May 2016)*.

- MDNA132 has superior targeting compared to IL13 and the zetakine, with lower affinity to the ubiquitously expressed IL13Rα1, while retaining sub-picomolar affinity to IL13Rα2.

- MDNA132 is a differentiated solid tumor targeting asset with solid IP.

**IL13Rα2 AS A CANCER TARGET**

- IL13Rα1 is a widely expressed receptor for IL13 and IL4. IL13Rα2 is a decoy receptor for the IL-13 cytokine, that acts to dampen IL13-mediated responses in inflamed tissues.

- IL13Rα2, a cancer testis antigen, is highly expressed at the surface of certain tumor cells minimally on normal cells.

- IL13Rα2 is recognized as a major glioblastoma marker (*PMID: 18172271*) and tumor suppressor gene (*PMID:24723564*).

- IL13Rα2 is increasingly recognized as a target and late-stage aggressiveness factor in basal-like breast (*PMID: 26208975*) and colorectal cancers (*PMID:22505647*).