| Group          | Product | Genome Modification(s) | Phase    | Trial Number        | Condition          | Description                                                                                                                                                                                                 | Ref. |
|----------------|---------|------------------------|----------|---------------------|--------------------|-------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------|------|
| Fate Therapeutics | FT500   | None                   | I        | NCT03841110 (Recruiting) | Solid tumors       | Used in combination with checkpoint blockade therapy against solid tumors. Patients undergo preparative lymphodepletion regimen followed by infusion of FT500 and immune checkpoint inhibitor. Goals of the study are to measure patients with dose limiting toxicities. |      |
| Fate Therapeutics | FT500   | None                   | N/A      | NCT04106167 (Recruiting) | Solid tumors       | Measure long term survival and safety data from patients who participated in the parent FT500 trial (NCT03841110)                                                                                      |      |
| Fate Therapeutics | FT516   | Engineered to include a high-affinity, non-cleavable CD16 (hnCD16) Fc receptor | I        | NCT04023071 (Recruiting) | Hematologic Malignancies | Measure incidence of subjects with dose limiting toxicities. Administered in combination with monoclonal antibodies such as rituximab (anti-CD20) or Obinutuzumab (anti-CD20) or as a monotherapy. |      |
| Fate Therapeutics | FT538   | CD38-less + hnCD16 + IL15/R | Preclinical | -        | Multiple Myeloma | Engineered with knock-out of CD38 receptor and knock-in of high-affinity, non-cleavable CD16 receptor, and fused IL-15 receptor. To be used in combination with daratumumab (anti-CD38), monoclonal antibody. |      |
| Fate Therapeutics | FT596   | CAR19 + hnCD16 + IL15/R | Preclinical | -        | Hematologic Malignancies | Engineered to include a NK cell-specific anti-CD19 CAR, high-affinity, non-cleavable CD16 receptor, and fused IL-15 receptor. Used in combination with rituximab (anti-CD20) monoclonal antibody. |      |
| Kaufman         |         | None                   | Preclinical | -        | HIV/AIDS           | iPSC-NK cells to target HIV infected CD4+ T cells                                                                                                                                                    | 94   |
| Kaufman         |         | Engineered to include a recombinant receptor with CD4 extracellular domain and CD3z signaling chain | Preclinical | -        | HIV/AIDS           | iPSC-NK cells engineered with a recombinant CD4z receptor to target HIV infected CD4+ T cells                                                                                                             | 98   |
| Kaufman         |         | Anti-meso CAR with NKG2D, 2B4, CD3zeta signaling domains | Preclinical | -        | Ovarian cancer xenograft model | Insertion of anti-meso CAR with NK specific signaling domains using piggyback transposons to increase antigen binding efficiency and anti-tumor activity                                                                 | 120  |
| Kaufman         |         | Site directed mutagenesis (S197P) in CD16a receptor | Preclinical | -        | -                  | Site directed mutagenesis in the CD16a receptor (S197P) using a sleeping beauty transposon to prevent receptor cleavage and shedding upon NK activation (SKOV3 in vitro model)                      | 121  |
| Kaufman         |         | Deletion of CISH gene using CRISPR | Preclinical | -        | -                  | Site directed mutagenesis in the CD16a receptor (S197P) using a sleeping beauty transposon to prevent receptor cleavage and shedding upon NK activation (K562 in vitro model)                      | 122  |
| Kaufman         |         | Recombinant CD64/16A receptor | Preclinical | -        | -                  | CISH gene (encoding for CIS regulatory element) was deleted using CRISPR/Cas9 to overcome negative regulation of IL-15 by CIS (K562 and MOLM-13 in vitro models)                                                                 | 123  |
| Walcheck        |         | Site directed mutagenesis (S197P) in CD16a receptor | Preclinical | -        | -                  | Insertion of a recombinant receptor with the extracellular domain of CD64, and the intracellular and transmembrane domain of CD16a using a sleeping beauty transposon to prevent CD16 receptor cleavage and shedding upon NK activation (SKOV3 in vitro model) | 124  |
| Kaufman         |         | Engineered to include a high-affinity, non-cleavable CD16 (hnCD16) Fc receptor | Preclinical | -        | -                  | Engineered to include a high-affinity, non-cleavable CD16 receptor to improve ADCC capabilities in hematologic malignancies and solid tumors                                                                 | 126  |