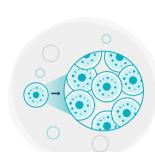
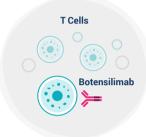


Multiple mechanisms of action



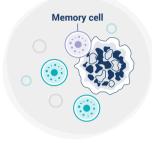
Primes and expands new T cells

to destroy cancer cells if they return, creating a durable response



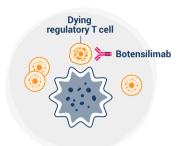
Activates existing T cells

to increase the magnitude of the immune attack against cancer



Establishes memory cells

to destroy cancer cells if they return for a durable response



Eliminates immunosuppressive regulatory T cells

that the cancer recruits to suppress the immune response

Fc Engineering?

Fcγ region The back-end is

Fc-enhanced to improve binding to activating Fcy receptors which optimizes the activity of the antibody



Variable region The front-end is optimized for

high affinity binding to
CTLA-4 and blockade of
CTLA-4 co-inhibitory signaling

the type Fcy receptors that activate immune cells. This engagement promotes a more effective immune response against cancer

Botensilimab has modifications in the Fc region that increase engagement with

Botensilimab Different?

How is



Unique mechanism of actionFc-enhanced modification builds a tighter, longer-lasting "bridge" between

antigen-presenting cells and T cells to promote optimal T cell priming and greater activation Fc-enhanced modification also improves engagement with NK cells and macrophages to

increase depletion of immuno-suppressive regulatory T cells

~40% of patients have immune cells that don't bind well to a standard Fc region because

Broader benefit

they have a low affinity FcγRIIIA; these patients have a poor response to 1st-generation CTLA-4 therapy. Botensilimab is optimized to bind well to all variants of FcγRIIIA on immune cells, expanding the potential benefit of CTLA-4 therapy to all patients.





Improved tolerability profile

avoids complement binding to prevent these serious side effects.

1st generation antibodies bind to complement, which can trigger an inflammatory response that leads to difficult-to-treat side effects. Botensilimab's Fc modification