Improves stability, cell selectivity, and specificity.

Demonstrates better proteolytic resistance.

Increases half-life and serum stability.

Reduces toxicity to normal cells.


Each year, over 8,000 cases of chronic myeloid leukemia (CML) are diagnosed in the United States. Current first-line treatment utilizes tyrosine kinase inhibitors (TKIs) that demonstrate high potency against CML. This treatment is limited, however, by mutations in Bcr-Abl (a gene fusion found in the majority of patients with CML) that confer resistance.

A novel, stapled, mutant three-cell penetrating peptide for Bcr-Abl inhibition prevents the oncogenic function of Bcr-Abl without triggering resistance-causing mutations. The peptide inhibitor consists of a modified Bcr coil that preferentially interacts with Bcr-Abl and prevents dimerization. The coil has a leukemia specific cell penetrating peptide and a hydrocarbon staple to ensure the peptide will only bind to and inhibit Bcr-Abl cancer cells. This causes cancer cell death while leaving non-cancer cells healthy and unaffected. The smaller peptide is also easier to deliver and demonstrates increased cell permeability.

- Proof of concept demonstrated in K562 Bcr-Abl positive leukemia cells.

- Animal studies required.

**STAGE OF DEVELOPMENT**

**IP PROTECTION**

**LEARN MORE**

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