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Exploiting DCAF16 Mis-Splicing to Target Cancers with Oncogenic SF3B1 Mutations

Stanford researchers have discovered a technology for selectively targeting cancers with SF3B1 mutations by utilizing DCAF16-based protein degraders. This approach exploits elevated DCAF16 protein levels in these cancers to degrade therapeutic proteins, offering a novel and effective treatment strategy for myeloid cancers, chronic lymphocytic leukemia (CLL), and select solid tumors.

The current field of cancer treatment faces significant challenges in selectively targeting oncogenic mutations, particularly those involving the RNA splicing factor SF3B1. Existing therapies often lack specificity, leading to off-target effects and limited efficacy. Additionally, many cancers with SF3B1 mutations have no specific FDA-approved treatments, leaving a critical gap in effective therapeutic options.

This technology addresses these issues by leveraging DCAF16-based protein degraders to selectively target and degrade proteins in SF3B1 mutant cancers, offering a novel and precise approach to cancer therapy. This approach addresses the major problem of lack of specificity in current cancer therapies, which often results in off-target effects and limited efficacy. By exploiting elevated DCAF16 protein levels in SF3B1 mutant cancers, this technology facilitates the degradation of multiple therapeutic proteins, offering a highly selective and effective treatment strategy. Preclinical studies have demonstrated the preferential selectivity and effectiveness of these protein degraders, highlighting their potential to improve therapeutic outcomes significantly.

Stage of Development
Proof of concept

Applications

- **Targeted therapy for cancers** with SF3B1 mutations, including CLL, MDS, and select solid tumors
- **Personalized treatment strategies** by identifying and targeting specific SF3B1 mutations in patients

Advantages

- **Enhanced drug selectivity** for cancers with SF3B1 mutations, reducing off-target effects and improving therapeutic outcomes
- **Ability to degrade multiple therapeutic proteins** via DCAF16-based degraders, offering versatile treatment options
- **Increased efficacy** in SF3B1 mutant cancers
- **Novel mechanism** utilizing gain-of-function splicing alterations

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