DO NOT PUBLISH The Use of Deubiquitinase Inhibitors to Modulate p53 in Cancer

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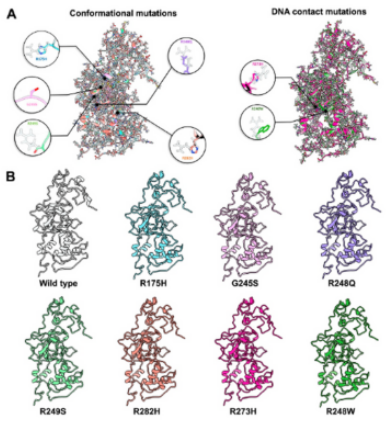

Molecular models of frequent hotspot mutations in p53

Technology Summary

P53 is a tumor suppressor protein with roles such as cell cycle arrest, DNA repair, and apoptosis. The invention uses deubiquitinase (DUB) inhibitors to modulate p53 structure and function. DUB inhibitors may convert the inactive p53 to an active conformation, capable of binding and repairing the damaged DNA. This method discusses the screening for an agent that treats cancer comprising a mutated p53. The treatment can be done by administering a therapeutic agent to the patient that targets the mutated p53 protein or the polynucleotide responsible for encoding the mutated p53.

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Application & Market Utility

The lack of knowledge in understanding the three-dimensional structure of p53 presents a barrier to understanding the molecular properties of p53 for pharmacological applications. The DNA-binding domain of p53, a stable central region of the protein, is frequently altered in cancer. P53's proteolytic activity depends on the DNA source. When bound to mismatched DNA or single-stranded DNA, p53's self-cleavage events can trigger apoptotic signaling cascades. Elucidating p53's 3D structure may identify new targets for cancer therapeutics and the creation of new cancer therapies.

Next Steps

There are plans to conduct future biochemical assessments and studies aimed at delineating the inactive to active transition occurring for the p53 dimers

TECHNOLOGY READINESS LEVEL 3

Seeking

Investment | Licensing | Research

Keywords

- P53
- Cancer
- Pharmacological
- Therapeutic agent
- Polynucleotide

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