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A Direct Molecular Inhibitor Of Retinoblastoma Protein Inactivation

Tech ID: 22897 / UC Case 2012-681-0

BACKGROUND

The Retinoblastoma tumor suppressor protein (Rb) negatively regulates cell proliferation and the cell cycle through inhibition of E2F transcription factors. E2F transcription factors are a group of genes that are involved in cell cycle regulation and synthesis.

Cancer cells almost invariably have genetic alterations of Rb pathway components that alter normal cell cycle function.

In the past many of the efforts in controlling the effects of Rb mutations has focused on design of therapeutic Cdk inhibitors. Several small molecule ATP-competitive inhibitors have been designed that efficiently inhibit Cdk activity, but new therapeutics are always necessary.

TECHNOLOGY DESCRIPTION

Researchers at UCSC have invented peptides that prevent inactivation of the Rb protein with molecules. These molecules are intended to inhibit E2F release even when Rb is phosphorylated by kinases.

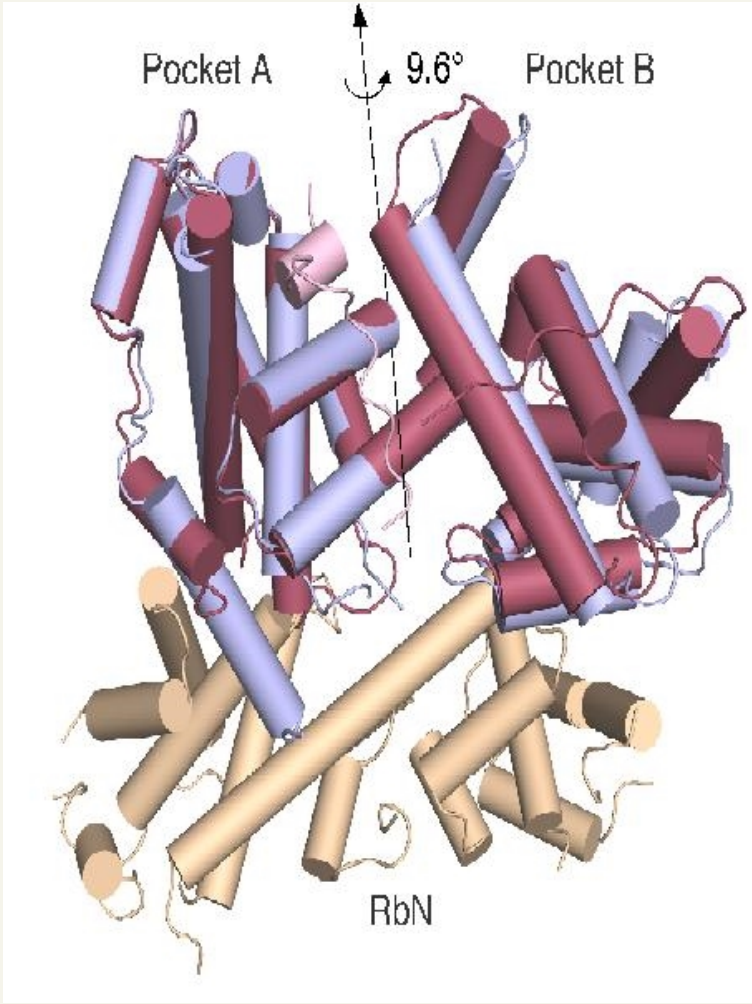


Image 1: Structural comparison of inactive Rb (gold and blue) and active Rb (red). Phosphorylation induces an interdomain interaction that allosterically distorts the structure of the E2F-binding site.

Some of the peptides include D-amino acids rather than more typical L-amino acids.

CONTACT

Jeff M. Jackson
jjackso6@ucsc.edu
tel: .

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OTHER INFORMATION

KEYWORDS

Retinoblastoma, Tumor Suppressor

Protein, Cell Cycle Inhibition,

Transcription Factors, Cell Cycle

Regulation, Protein Targeting, D-amino acids

CATEGORIZED AS

- ▶ Medical
 - ▶ Disease: Cancer
 - ▶ Therapeutics

RELATED CASES

2012-681-0

APPLICATIONS

Cancer therapeutics

INTELLECTUAL PROPERTY INFORMATION

Country	Type	Number	Dated	Case
United States Of America	Issued Patent	9,573,977	02/21/2017	2012-681
United States Of America	Issued Patent	9,365,621	06/14/2016	2012-681
Patent Cooperation Treaty	Published Application	WO 2013/155360	10/17/2013	2012-681

RELATED MATERIALS

► [Structures of inactive retinoblastoma protein reveal multiple mechanisms for cell cycle control](#) - 06/01/2012