Generation of Novel Prostate Cancer Cell Lines that Employ the Alternative Lengthening of Telomeres (ALT) Telomere Maintenance Mechanism

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Technology description

Unmet Need

Instead of telomerase, approximately 10% of cancers maintain telomeres via a DNA template dependent mechanism called Alternative Lengthening of Telomeres (ALT). The ALT pathway is dependent on homologous recombination and tightly linked to recurrent cancer-associated somatic inactivating mutations of ATRX or DAXX , genes encoding chromatin remodeling proteins . This translates to 1.4 million new cases and 820,000 cancer deaths globally due to ALT mediated cellular immortalization. In addition, therapies targeting telomerase may cause cancer cells to activate the ALT pathway, further driving up this number. The discovery of the ALT pathway opened up new avenues of cancer study. However, additional research tools are still needed to identify unique aspects of the ALT pathway and to develop relevant drug therapies.

Technology Overview

ATRX was specifically knocked out in a genetically well characterized prostate cancer cell line. These new cell lines employ the Alternative Lengthening of Telomeres (ALT) pathway. These new isogenic cell lines can be used to further characterize and elucidate the basic biology of cancers harboring ALT, and determine which unique molecular features of ALT may be exploited to pharmacologically target these tumors.

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