Technology Overview

Prostate cancer is the second most common male cancer, affecting cells of the prostate glands. About 90% of these early stage prostate cancers are dependent on androgens for growth, so inhibitors of the androgen receptor (AR) are often used as a treatment. However, prolonged use of androgen deprivation therapy (ADT) transforms many hormone sensitive prostate cancers into lethal castration-resistant prostate cancer (CRPC), which is not entirely dependent on the presence of androgens to grow. While improved targeting of androgen receptors is now possible, which can slow progression of CRPC, the disease remains uniformly lethal. Therefore, a need exists for new therapeutics targeted for lethal AR-deficient prostate cancers. Using a several types of human and mouse model samples, Hutch researchers discovered a “molecular brake” known as 4EBP1 which is inactivated in AR-resistant tumors and propagates a growth promoting cell-signaling pathway. By introducing 4EBP1 mimics, they discovered that these compounds killed AR-low, but not AR high, cells as well as significantly reduced the growth and survival of AR-low prostate cancers in mice. Therefore, they have identified a druggable link between the AR and protein synthesis that could be targeted in AR-low advanced prostate cancers.

Applications

- Therapeutic treatments for androgen low and resistant prostate cancers
- Reveals processes where the androgen receptor regulates protein synthesis

Potential Advantages

- Highlights druggable targets when the androgen receptor is low/absent

Market Overview: Prostate cancer is the fifth leading cause of death worldwide. Over 1.2 million new cases and 359,000 deaths were reported in 2018.

Investigator Overview

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