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Overview

- Beta Cat is a “bench to bed” oncology company that:
  - Takes on only game changing therapies with high unmet need
  - Epigenetics and Cancer “stem cell” like tumors
- Company formed in 2010
- Lead Products: βC2059 and βC2528
  - βC2059 - **First in Class; Potent inhibitor of β catenin activation complex**
    - Broad potential in colorectal, hematopoietic and other cancers
  - βC2528 - **First in Class; Potent inhibitor of Lysine Specific Histone Demethylase 1**
    - Orphan drug registration strategy in sarcomas plus major tumor potential in prostate and triple negative breast
  - INDs expected in 2013
- Highly skilled executive team
- Business model provides broad, deeply experienced leadership and in-house R&D functions, with streamlined operations and capital efficiency
- Submitting with CPRIT for a Company Formation Award

Huge potential market for βC2059

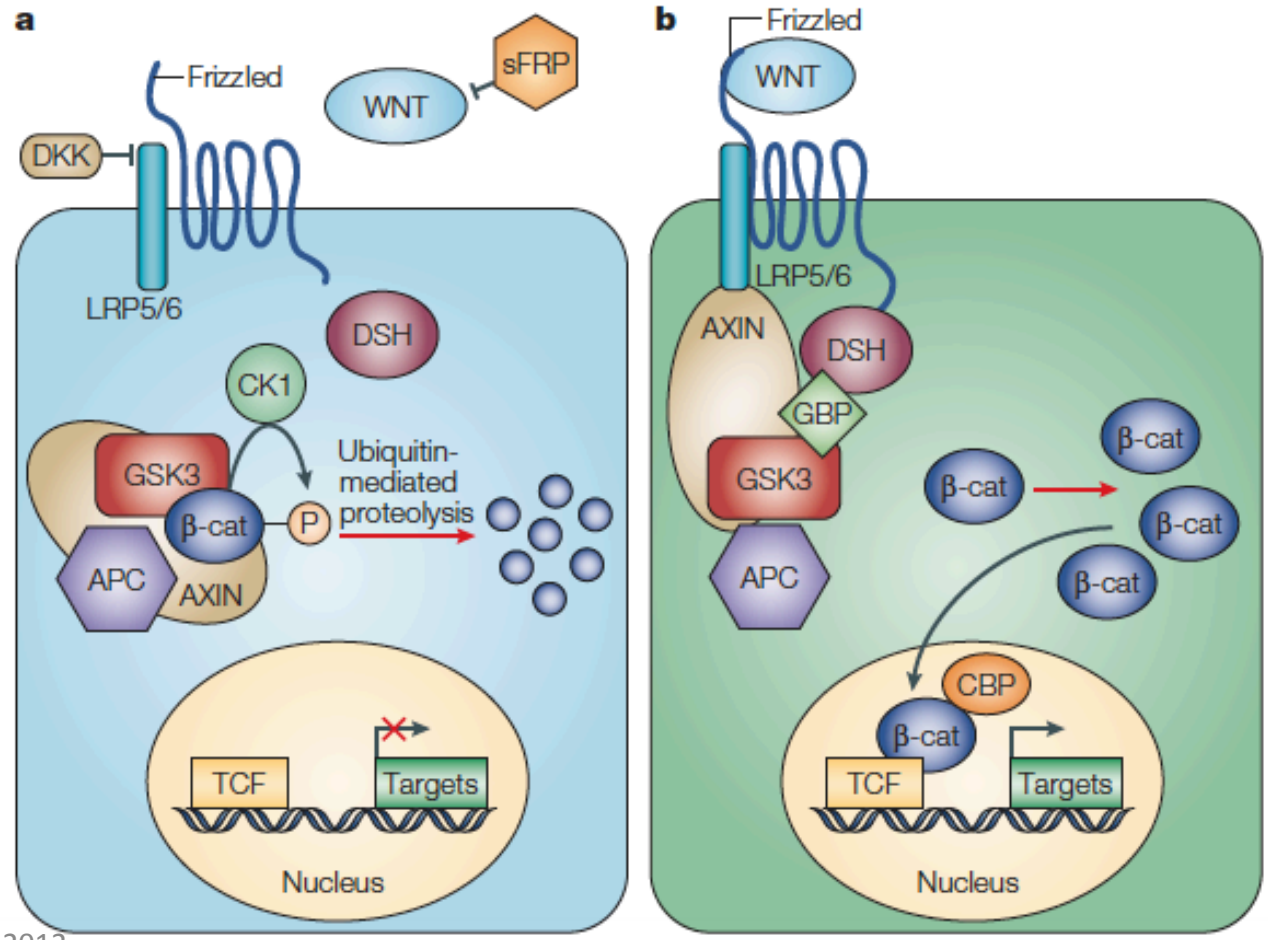
Type of Cancer	% with β Catenin activation / mutation	Annual New Patients (000) USA only
Colon	90+%	100+
Breast	40-50%	90
Lung	40%	85
Prostate	20%	55
Melanoma	30%	20
Ovarian	50%	11
Hepatocellular	30%	8
Gastric	25%	6
Hematopoietic	frequently	NA
Wilm's	50+%	0.3
Total		> 375

Mutations in the beta catenin pathway are a central driver for many cancers, but the pathway has been difficult to drug using conventional approaches, causing most pharmas to give up after many failures and move upstream to different Wnt targets

Increased β Catenin protein levels promote cancer

- The β catenin pathway is important for cancer stem cell maintenance
  - In a normal cell, β Catenin regulates cell to cell junctions, and stays in biological balance
  - In an aberrant cancer cell, it cannot be properly degraded, finding its way into the nucleus where it promotes transcription and oncogenesis.

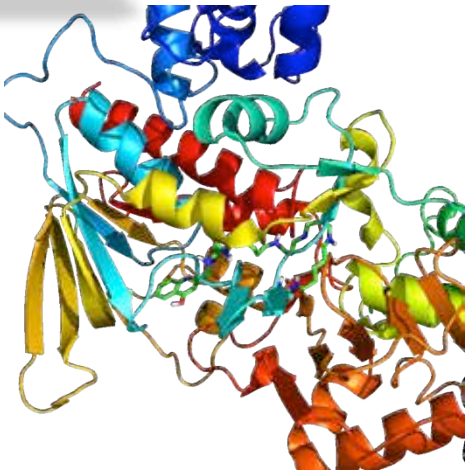
Normal Cell: Degradation of β Catenin in the cytoplasm



Cancer Cell: Accumulation of β Catenin in the cytoplasm and then in the nucleus

SP-2528 IS AN EXCITING POTENTIAL ONCOLOGY EPIGENETIC THERAPEUTIC

- Blocking LSD1 stops the demethylation of lysines in histone 3, allowing them to methylate (activate).
- The result is:
  - tumor suppressor genes (TSGs) that the cancer has silenced are reactivated
  - oncogenes the cancer has activated are stopped.



- Potential to be "the solution" in undifferentiated sarcomas (Ewing's Sarcoma) like Gleevec for CML
  - Orphan drug, fast to market registration
- Other major tumor types like prostate and breast susceptible
- Differentiates AML and increases survival *in vivo*
- *Oral, QB or BID small molecule – excellent drug-like properties*

The Value of Texas BioAlliance Support

- Informed pre-submission evaluation
  - *For both content and presentation*
    - Helps to find the right tone, emphasis, and detail
  - *We used thru the entire process*
    - Initial submission
    - Presentation
    - diligence
- Beneficial personal consulting on style and approach
  - *Presentation "boot camp" 2 ½ hour personal attention*

There are many uncontrollables in life.

One thing you can control is your preparation.

End