Captor Therapeutics®

CT-01 molecular target disclosure – what does this mean for project development

21st April 2022

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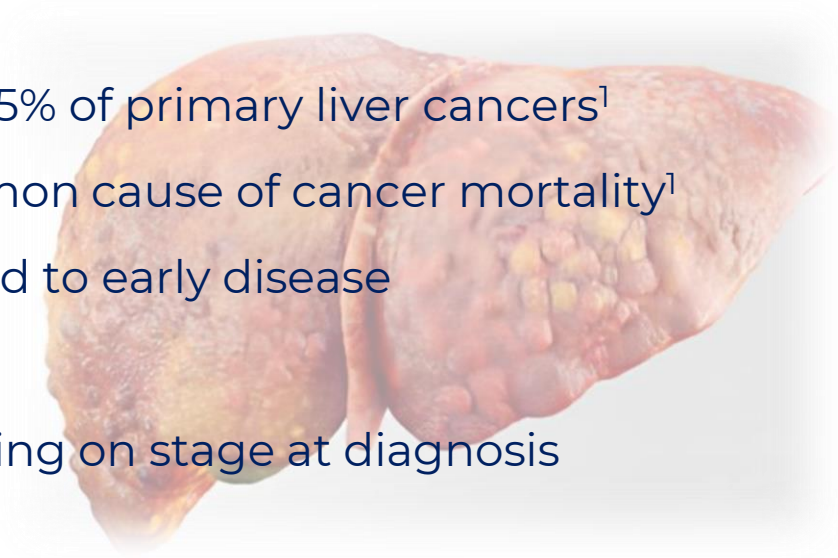
Captor Therapeutics®

DEMONSTRATING THE POTENTIAL OF CAPTOR'S TPD PLATFORM

CT-01: Addressing one of the deadliest cancers with Captor's molecular glue degrader drugs

CT-01: Addressing one of the deadliest cancers

- Hepatocellular Carcinoma (HCC) accounts for 75-85% of primary liver cancers[1]
- ~ 700 000 new cases each year, the 2nd most common cause of cancer mortality[1]
- Curative treatments (tumor resection) are restricted to early disease
- High rate of metastases
- 5-year Survival Rates[2] vary from 3% to 34% depending on stage at diagnosis



References: [1] Global Cancer Statistics 2018, [2] Data for the US, 2010-2016, ACS Cancer Facts & Figures, [3] DOI: 10.1200/JCO.2021.39.3_suppl.267

Approved drugs offer modest therapeutic benefit

- 2007 - **Sorafenib** approved as first-line treatment in HCC in 2007 – survival 2.8 months longer as compared to no drug*
- 2020 - Combination of **Atezolizumab** (TECENTRIQ®) **plus Bevacizumab** (AVASTIN®) – 5.8 months longer survival as compared to sorafenib**

In overall, a patent with unresectable liver tumor treated with standard of care**:

- on average lives for 19.2 months
- only 29.8% of patents respond to the treatment

** Llovet J et al. 2007, DOI: 10.1200/jco.2007.25.18_suppl.lba1*

*** IMBrave 150, DOI: 10.1056/NEJMoa1915745, updated: DOI: 10.1200/JCO.2021.39.3_suppl.267*

Molecular targets of CT-01 compounds

- GSPT1 is a GTPase, which forms a complex with the translation termination factor eRF1 to mediate translation termination
- Degradation of GSPT1 disrupts protein maturation leading to integrated stress response (ISR) and apoptosis

Surka CH et al. Blood. 2021 Feb 4; 137(5): 661–677

- SALL4 (Sal-like protein 4) is a transcription factor expressed in the human fetal liver and silenced in adults
- SALL4 is often reexpressed in hepatocellular carcinoma patients, which correlates with a poor prognosis

Tatetsu H et al. Gene. 2016 Jun 15;584(2):111-9

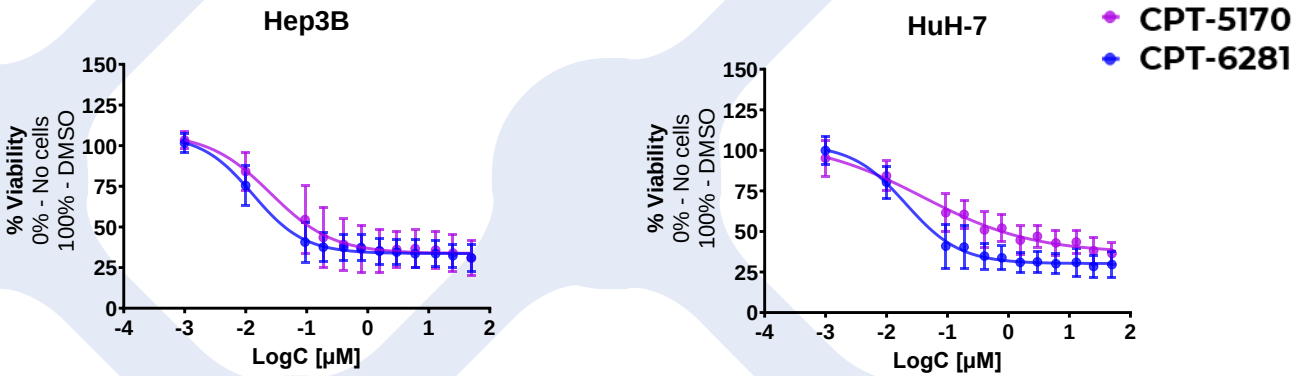
Combined degradation profile for the treatment of HCC

Another, yet undislosed neo-substrate involved in tumorgenesis

CT-01: molecular glue programme in HCC

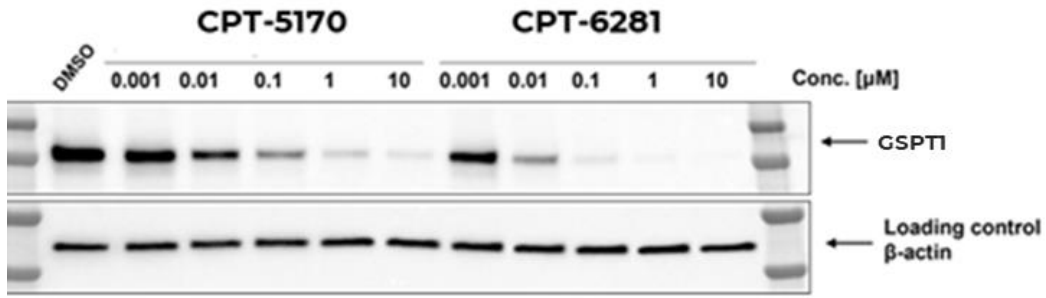
- Derived from the Captor proprietary library of molecular glues
- Active against a panel of HCC cell lines
- Selective degradation profil – does not degrade Ikaros and Aiolos

Cytotoxic effect in liver cancer cell lines



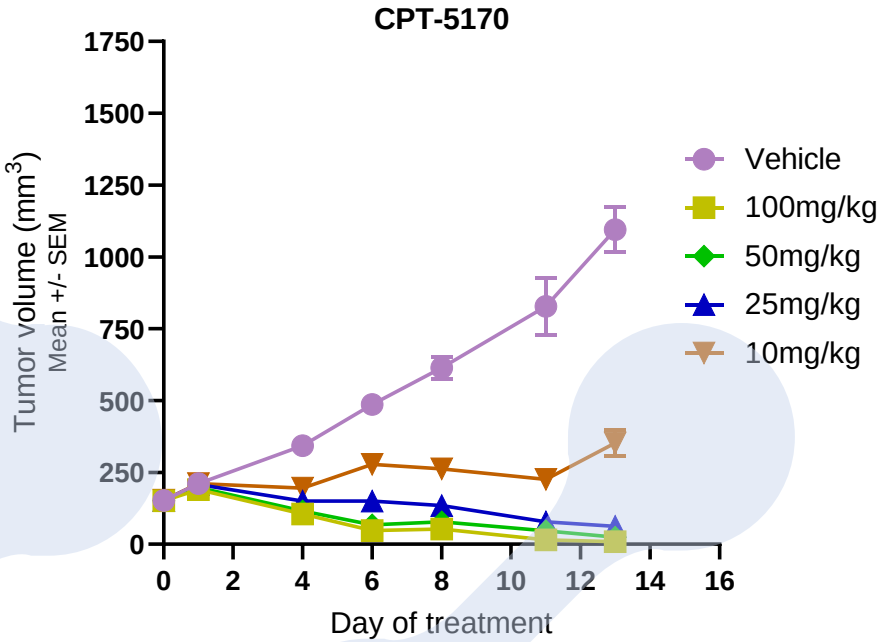
72 hours, CTG assay

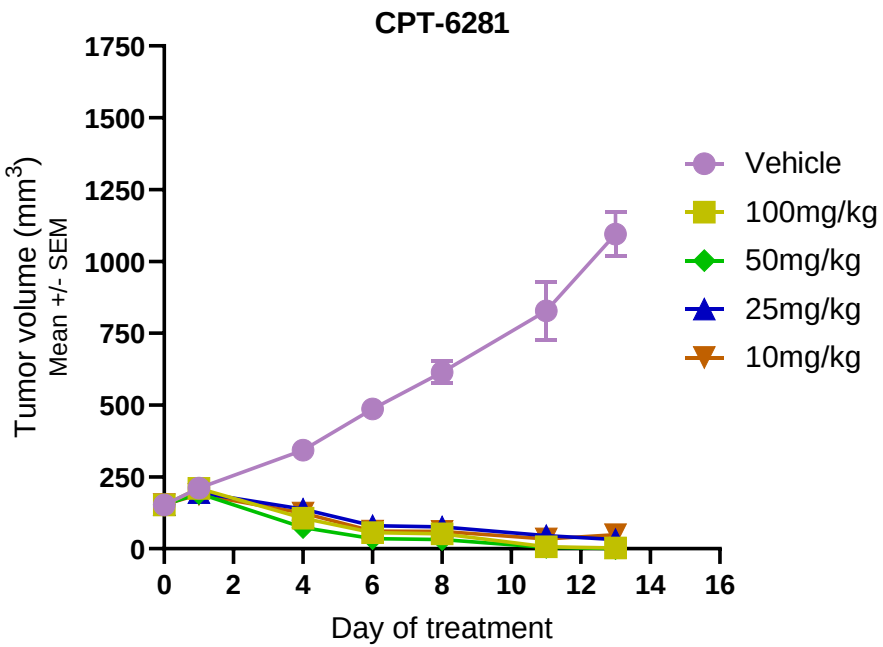
Potent degradation of GSPT1 with CT-01 compounds



HEP3B cells, 24 hours

New study: *In vivo dose response* – tumor regression



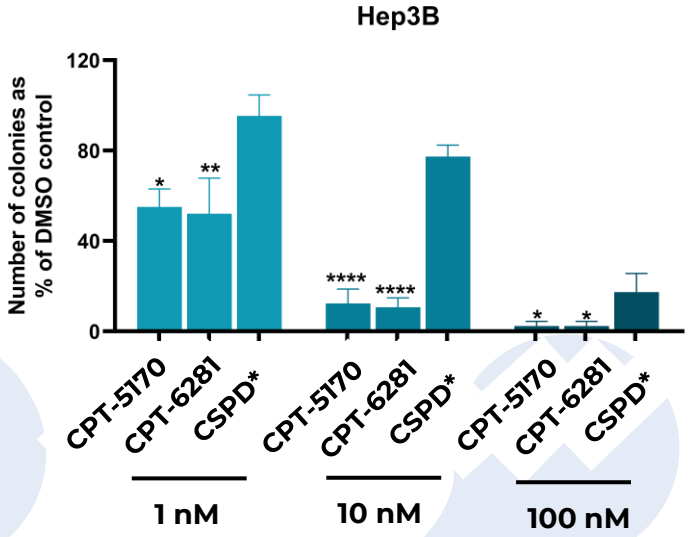


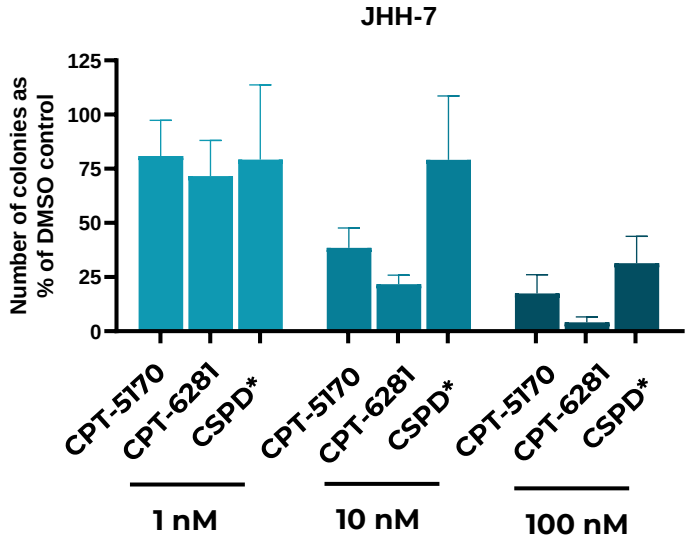
Human liver cancer model - Hep 3B2.1-7 (NSG mice)

The study performed by reputable subcontractor Covance/LabCorp

- 2 CT-01 candidates induced **tumor regression** following **oral administration**
  - Both compounds were very **well tolerated** by the animals

CT-01 compounds differentiated against CC-90009





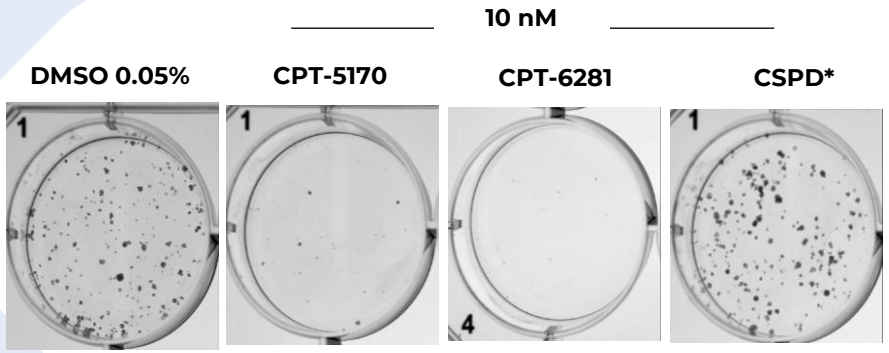


Fig. *Representative images of colony formation assay in Hep3B for DMSO control and 10 nM dose of tested compounds.*

***CSPD -** clinical-stage PoI degrader, CC-90009 (Celgene/BMS)

CT-01 highly attractive and unique product in development

- CC-90009 and MRT-2359 are GSPT1-degraders in development by Celgene (now BMS) and Monte Rosa Therapeutics, respectively
- In Phase 1 clinical trial of CC-90009, **promising antileukemic activity** was observed, **demonstrating potential of GSPT1 in cancer treatment**
- None of the degraders **in development target HCC**, whereas in CT-01 **we have demonstrated very compelling efficacy data in animal models of this disease**
- CT-01 compounds have totally different degradation profile as compared to CC-90009 and MRT-2359 which can explain the different efficacy

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Q&A session

All images by Piotr Piatek

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Thank you!

All images by Piotr Piatek

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