

LTL-313H datasheet

Origin	Human prostate cancer	Histopathology	High grade adenocarcinoma
Year of establishment	2009	Doubling time	13 days (subrenal capsule graft site)
Local invasion	Yes	Metastasis	Yes
Hormone Sensitivity	Androgen-dependent		

The LTL-313H tumor tissue line (Fig. 1) was developed from a patient's prostate cancer biopsy (Fig. 2, high grade prostate adenocarcinoma). When grafted under the renal capsules of NOD-SCID mice, the LTL-313H shows invasion into adjacent renal parenchyma and metastases to distant organs. Growth and prostate-specific antigen (PSA) production of the LTL-313H *in vivo* is androgen-dependent (Fig. 3). Figure 4 shows clinical course details of the patient. Viable tissues of the LTL-313H in early generations have been preserved by cryopreservation (DMSO), and can be readily recovered for grafting.

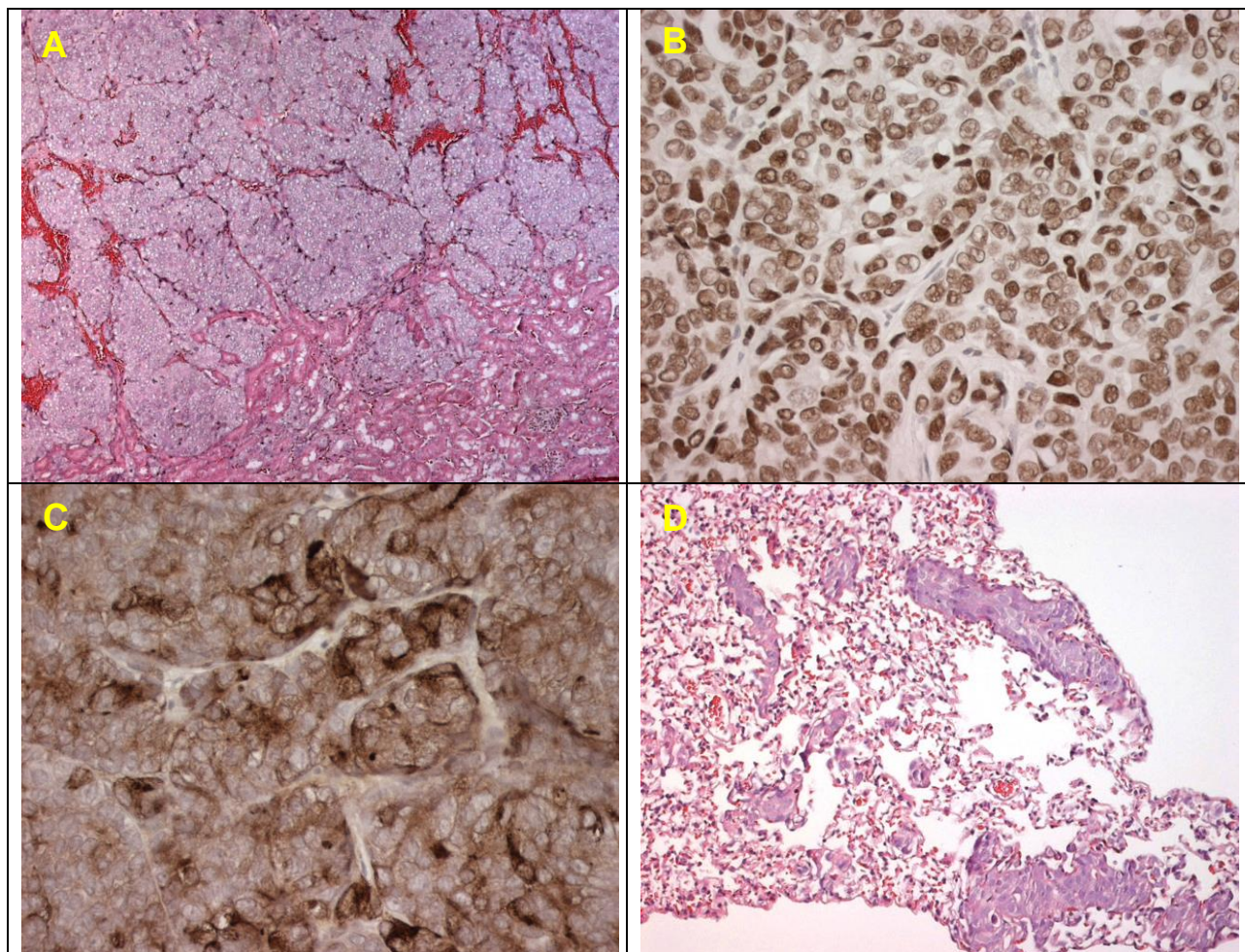


Fig. 1. (A). H&E stained LTL-313H tissue section. The tumor cells grow in solid sheets and invade host's renal parenchyma. X100 **(B, C).** The tumor cells show strong immunostaining with antibodies to human-specific **(B)** androgen receptor and **(C)** prostate-specific antigen. x400 **(D).** Lung metastases of the LTL-313H. X200

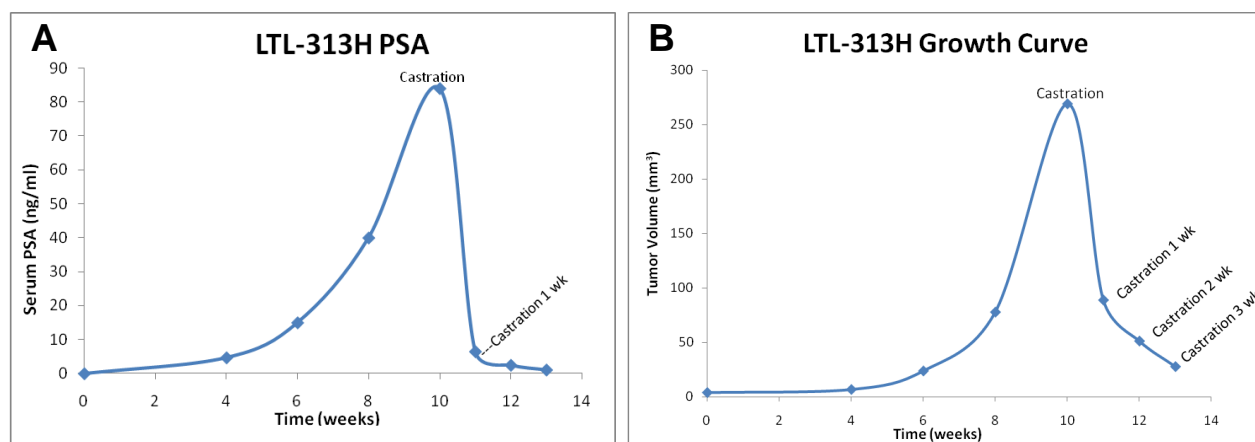
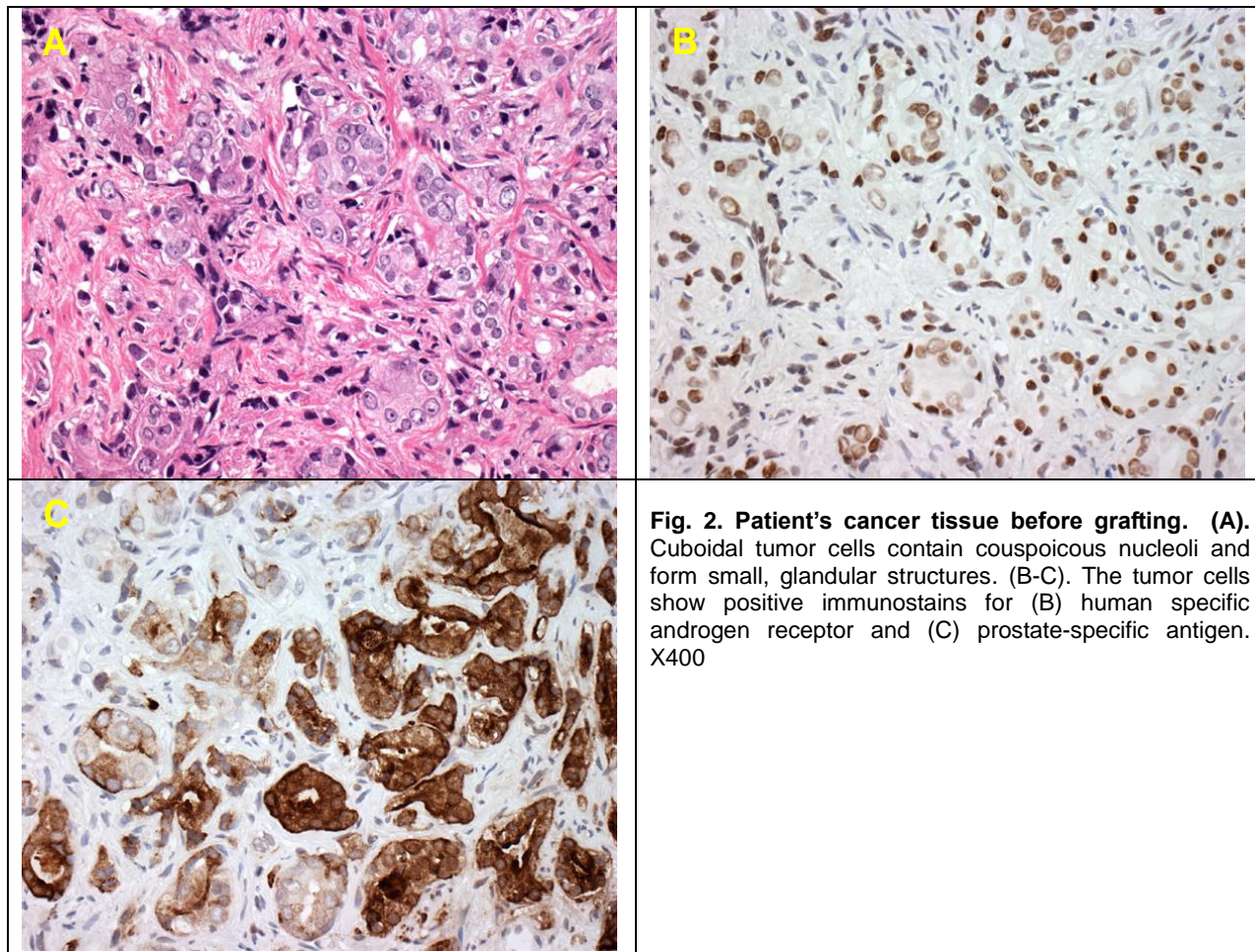


Fig. 3. LTL-313H shows androgen-dependent PSA production and growth *in vivo*: **(A)** Serum PSA levels increase in intact mice following implantation of LTL-313H xenografts under the renal capsules. Castration (androgen ablation) quickly decreases the serum PSA levels to very low concentrations. **(B)** Castration leads to major tumor shrinkage.

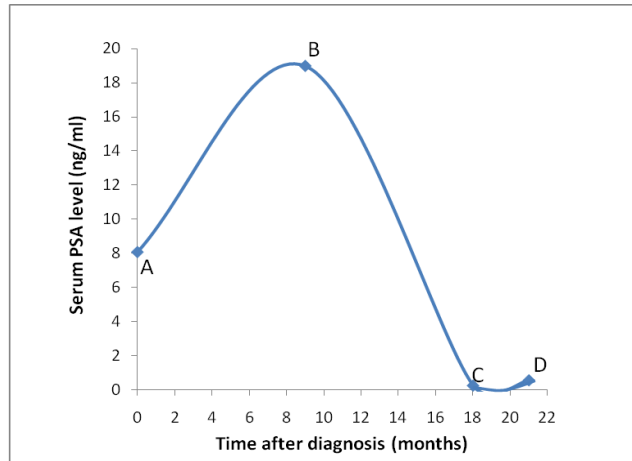


Fig. 4. Clinical course details of the patient. (A), detection of elevated blood PSA levels. **(B),** biopsy of tumor tissue used for LTL-313H development and initiation of androgen deprivation. **(C, D),** serum PSA levels remain low in response to androgen deprivation.

The LTL-313H tumor tissue line has been characterized using array CGH, next generation sequencing (NGS) and RNA microarray.

Applications

1. Pre-clinical evaluation of established and potential anticancer drugs. Examination of drug efficacy on tumor growth, cell death (apoptosis, necrosis), tissue invasion, metastasis and angiogenesis.
2. Discovery of potential therapeutic targets and/or biomarkers for drug sensitivity.
3. Study of mechanisms underlying tumor growth, progression and metastasis.

Publications

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3. Hu P, Chu GC, Zhu G, Yang H, Luthringer D, Prins G, Habib F, Wang Y, Wang R, Chung LW, Zhau HE. *Multiplexed quantum dot labeling of activated c-Met signaling in castration-resistant human prostate cancer*. *PLoS One*. 2011;6(12):e28670. Epub 2011 Dec 21.
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5. Yan Ting Chiang, Kendric Wang, Ladan Fazli, Robert Z. Qi, Martin E. Gleave, Colin C. Collins, Peter W. Gout and Yuzhuo Wang. GATA2 as a potential metastasis-driving gene in prostate cancer. *Oncotarget*. 2014 Jan 30;5(2):451-61.

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