LTL-313B datasheet

Origin	Human prostate cancer	**Histopathology**	High grade adenocarcinoma
Year of establishment	2009	**Doubling time**	11-15days (subrenal capsule graft site)
Local invasion	Yes	**Metastasis**	No
Hormone Sensitivity	Partially androgen-dependent		

The LTL-313B tumor tissue line (Fig. 1) was developed from a patient's prostate cancer biopsy (high grade prostate adenocarcinoma). When grafted under the renal capsules of NOD-SCID mice, the LTL-313B shows limited invasion into adjacent renal parenchyma but no distant metastasis. Prostate-specific antigen (PSA) production of the LTL-313B *in vivo* is androgen-dependent (Fig. 2A). LTL-313B growth is partially androgen-dependent (Fig.2B). Figure 3 shows clinical course details of the patient. Viable tissues of the LTL-313B in early generations have been preserved by cryopreservation (DMSO), and can be readily resurrect for grafting.

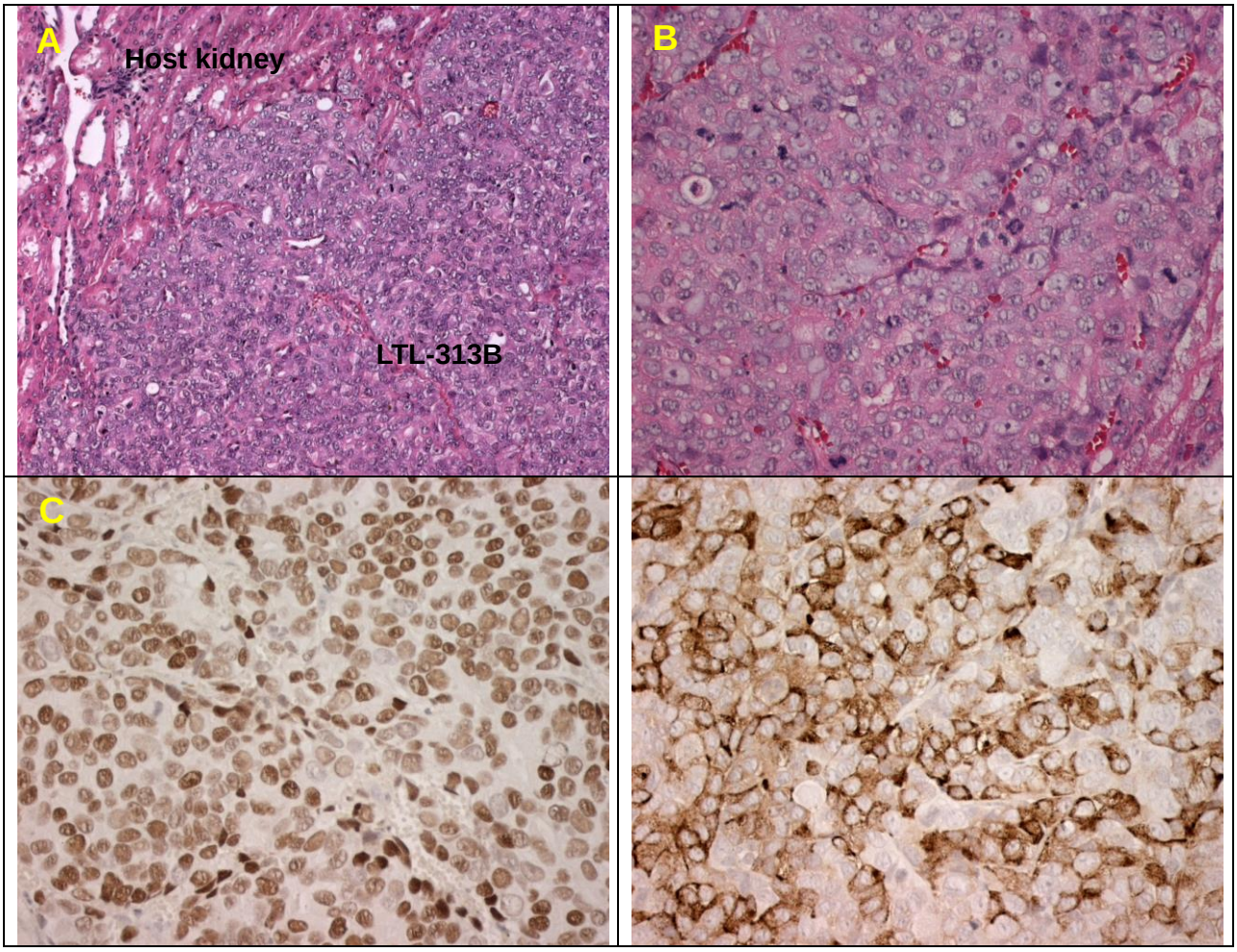

Fig. 1. (A-B). H&E stained LTL-313B tissue section. The tumor cells grow in solid sheets and show limited invasion to adjacent host kidney. **(C-D).** The tumor cells show strong immunostaining with antibodies to human-specific androgen receptor (C) and prostate-specific antigen (D) ×400

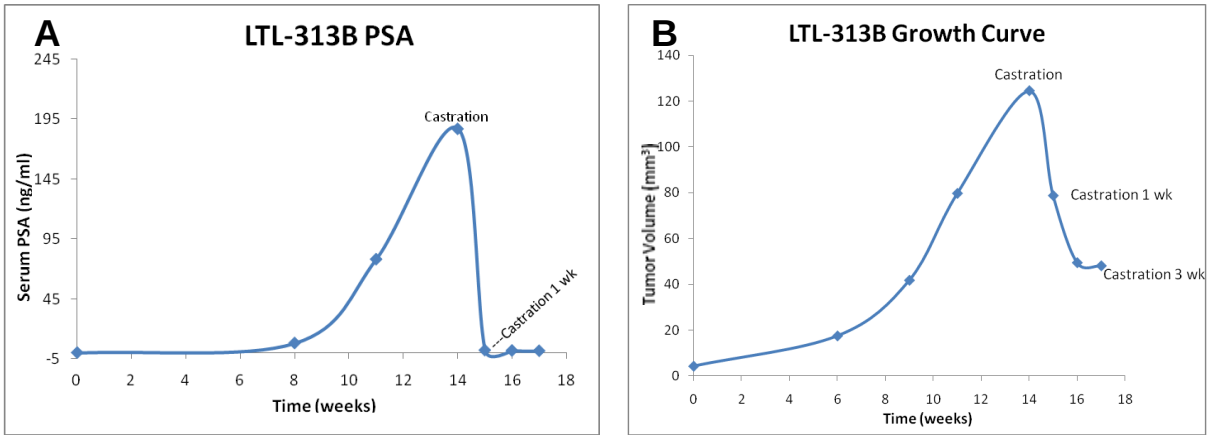

Fig. 2. LTL-313B shows androgen-dependent PSA production and growth *in vivo*: (A) Serum PSA levels increase in intact mice following implantation of LTL-313B xenografts under the renal capsules. Castration (androgen ablation) quickly decreases serum PSA to very low concentrations. **(B)** Castration leads to major tumor shrinkage.

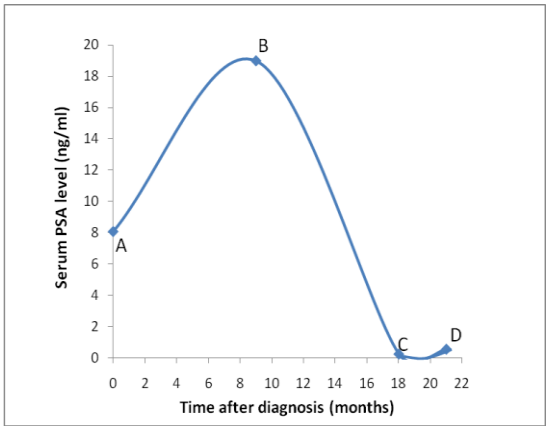

Fig. 3. Clinical course details of the patient. (A), detection of elevated blood PSA levels. (**B)**, biopsy of tumor tissue used for LTL-313B development and initiation of androgen deprivation. (**C, D)**, serum PSA levels remain low in response to androgen deprivation.

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

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 (in combination of metastatic tumor lines) and angiogenesis.

2. Discovery of potential therapeutic targets and/or biomarkers for drug sensitivity.

3. Study of mechanisms underlying tumor growth, progression and metatasis (in combination with metastatic tumor lines).

Selected publications

1. Watahiki A, Wang Y, Morris J, Dennis K, O'Dwyer HM, Gleave M, Gout PW, Wang Y. *MicroRNAs associated with metastatic prostate cancer.* PLoS One. 2011;6(9):e24950. Epub 2011 Sep 30.

2. Dong Lin, Alexander W. Wyatt, Hui Xue, Yuwei Wang, Xin Dong, Anne Haegert, Rebecca Wu, Sonal Brahmbhatt, Fan Mo, Lina Jong, Robert H. Bell, Shawn Anderson, Antonio Hurtado-Coll, Ladan Fazli, Manju Sharma, Himisha Beltran, Mark Rubin, Michael Cox, Peter W. Gout, James Morris, Larry Goldenberg, Stanislav V. Volik, Martin E. Gleave, Colin C. Collins, and Yuzhuo Wang. High Fidelity Patient-Derived Xenografts for Accelerating Prostate Cancer Discovery and Drug Development. Cancer Research 2014 Feb 15;74(4):1272-83.

3. 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.

For more information, please contact us by email: LTL@bccrc.ca or phone: (604) 675 8013