LTL-310F datasheet

Origin	Human prostate cancer	**Histopathology**	High grade adenocarcinoma
Year of establishment	2010	**Doubling time**	26.20±6.00 days
Local invasion	Yes, limited	**Metastasis**	No
Hormone Sensitivity	Androgen-dependent		

The LTL-310F tumor tissue line (Fig. 1) was developed from a patient's primary prostate cancer (high grade prostate adenocarcinoma). When grafted under renal capsules of NOD-SCID mice, the LTL-310F shows invasion into adjacent renal parenchyma but no distant metastasis. The LTL-310F xenografts are initially sensitive to castration (androgen ablation) *in vivo,* with declines in serum PSA levels and tumor volumes, and then become resistant, presenting a rapid, androgen-*in*dependent growth (Fig. 2). A castration-resistant tumor subline developed from the LTL-310F is designated LTL-310FR. Viable tissues of the LTL-310F in early generations have been preserved by cryopreservation (DMSO), and can be readily resurrect for grafting. The LTL-310 has been characterized using array CGH, next generation sequencing (NGS) and RNA microarray.

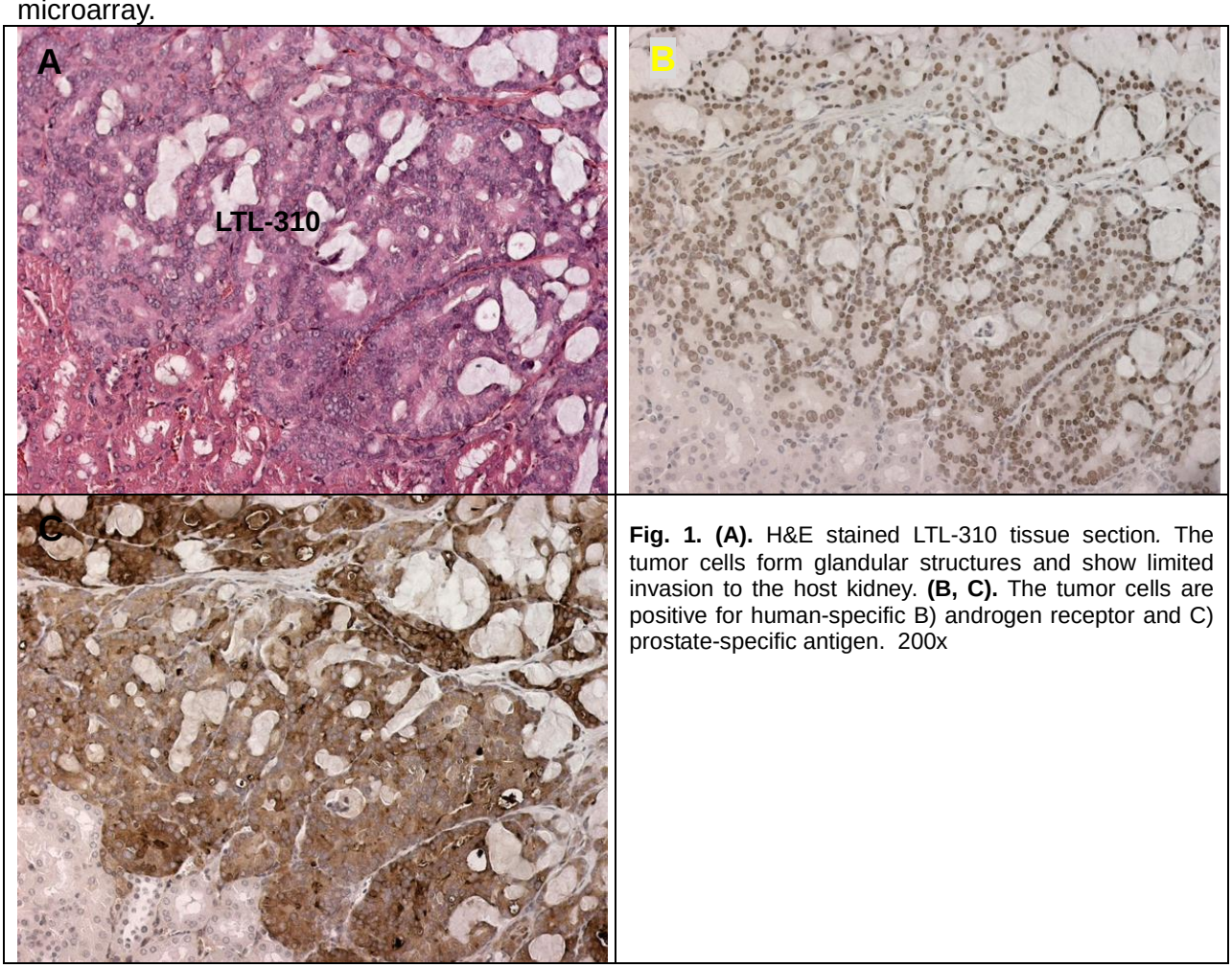

Fig. 1. (A). H&E stained LTL-310 tissue section. The tumor cells form glandular structures and show limited invasion to the host kidney. **(B, C).** The tumor cells are positive for human-specific B) androgen receptor and C) prostate-specific antigen. 200x

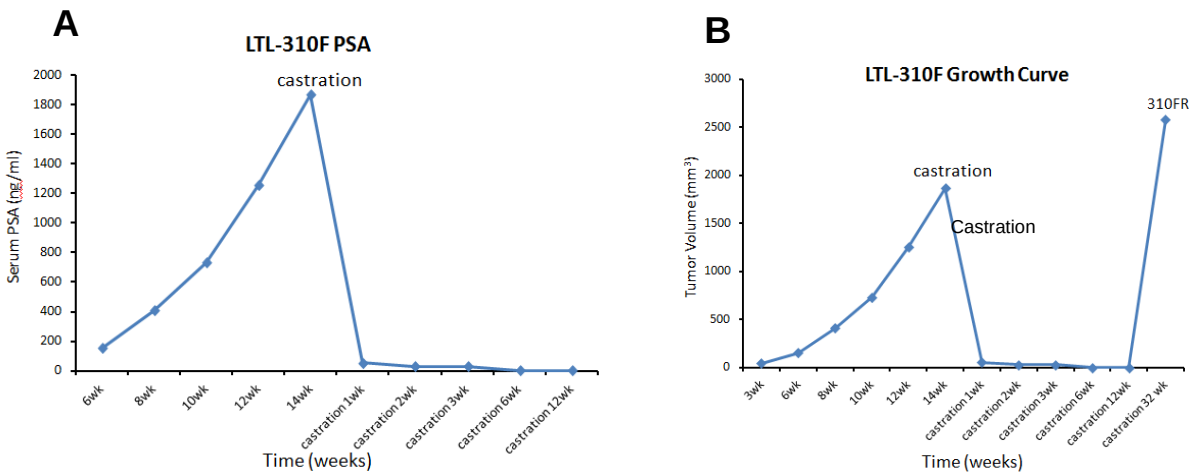

Fig. 2. (A). Serum PSA levels increase following implantation of LTL-310F xenografts under the renal capsules of intact male mice. Castration quickly decreases the serum PSA levels to exceedingly low concentrations. **(B).** The LTL-310F tumor tissue line initially responds to castration, showing a major decline in tumor volume; at 12 weeks after castration, it shows castration resistance, presenting rapid, androgen-independent growth without increasing serum PSA levels.

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.

Publications

1. 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. Full Text

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