LTL-313C datasheet

Origin	Human prostate cancer	**Histopathology**	High grade adenocarcinoma
Year of establishment	2009	**Doubling time**	23 days (subrenal capsule graft site)
Local invasion	Yes	**Metastasis**	Yes
Hormone Sensitivity	Partially androgen-dependent		

The LTL-313C 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-313C shows invasion into adjacent renal parenchyma and metastases to distant organs. Prostate-specific antigen (PSA) production of the LTL-313C *in vivo* is androgen-dependent (Fig. 2A). However, growth of LTL-313C *in vivo* is partially androgen-dependent (Fig. 2B). Figure 3 shows clinical course details of the patient. Viable tissues of the LTL-313C in early generations have been preserved by cryopreservation (DMSO), and can be readily resurrected for grafting.

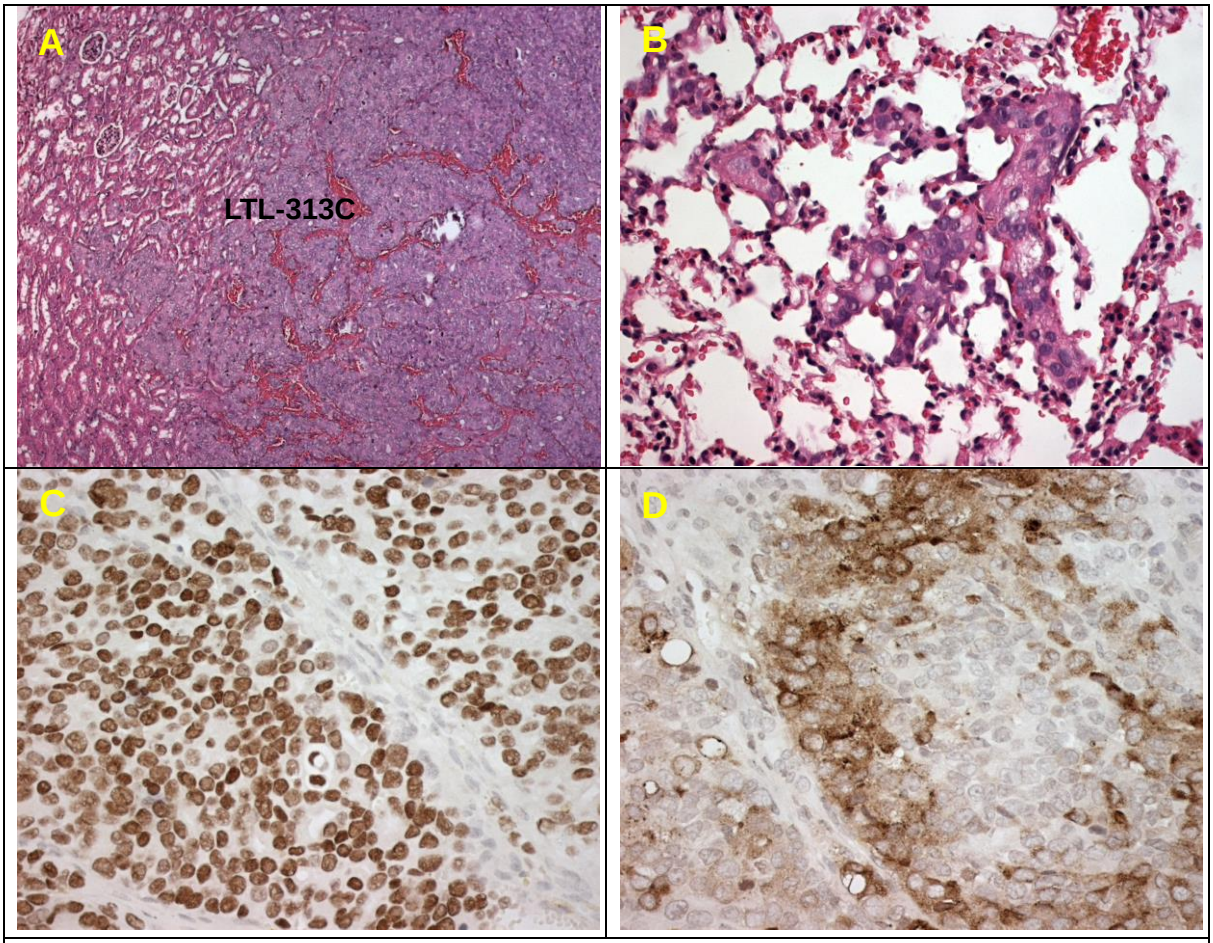

Fig. 1. (A). H&E stained LTL-313C tissue section. The tumor cells grow in solid sheets and invade host's renal parenchyma.x100 **(B).** Lung metastases of the LTL-313C. ×400 **(C-D).** The tumor cells show strong immunostaining with antibodies to human-specific androgen receptor (C) and prostate-specific antigen (D).

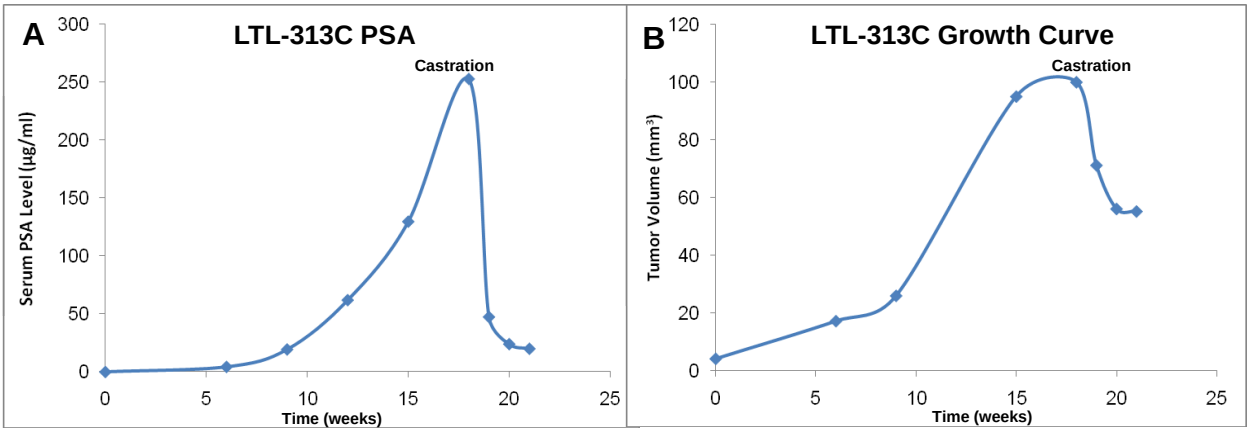

Fig. 2. LTL-313C shows androgen-dependent PSA production and growth in vivo: **(A)** Serum PSA levels increase in intact mice following implantation of LTL-313C xenografts under the renal capsules. Castration (androgen ablation) quickly decreases serum PSA levels to low concentrations. **(B)** Castration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

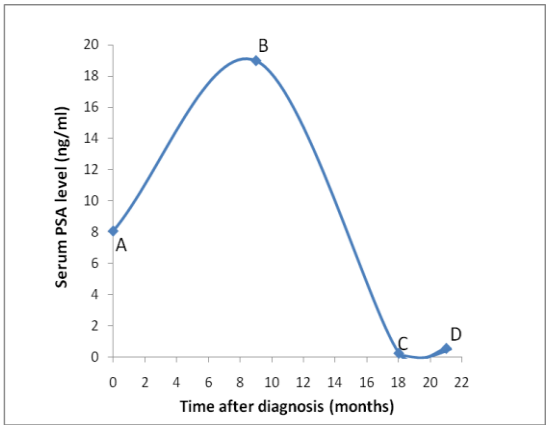

Fig. 3. Clinical course of the patient. (A), detection of elevated blood PSA levels. (**B)**, biopsy of tumor tissue used for LTL-313C development and initiation of androgen deprivation. (**C, D)**, serum PSA levels remain low in response to androgen deprivation.

The LTL- 313C tumor line has been characterized using array CGH 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 metatasis.

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