

LTL-313D datasheet

Origin	Human prostate cancer	Histopathology	High grade adenocarcinoma
Year of establishment	2009	Doubling time	11-15 days (subrenal capsule graft site)
Local invasion	Yes	Metastasis	Yes
Hormone Sensitivity	Androgen-dependent		

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

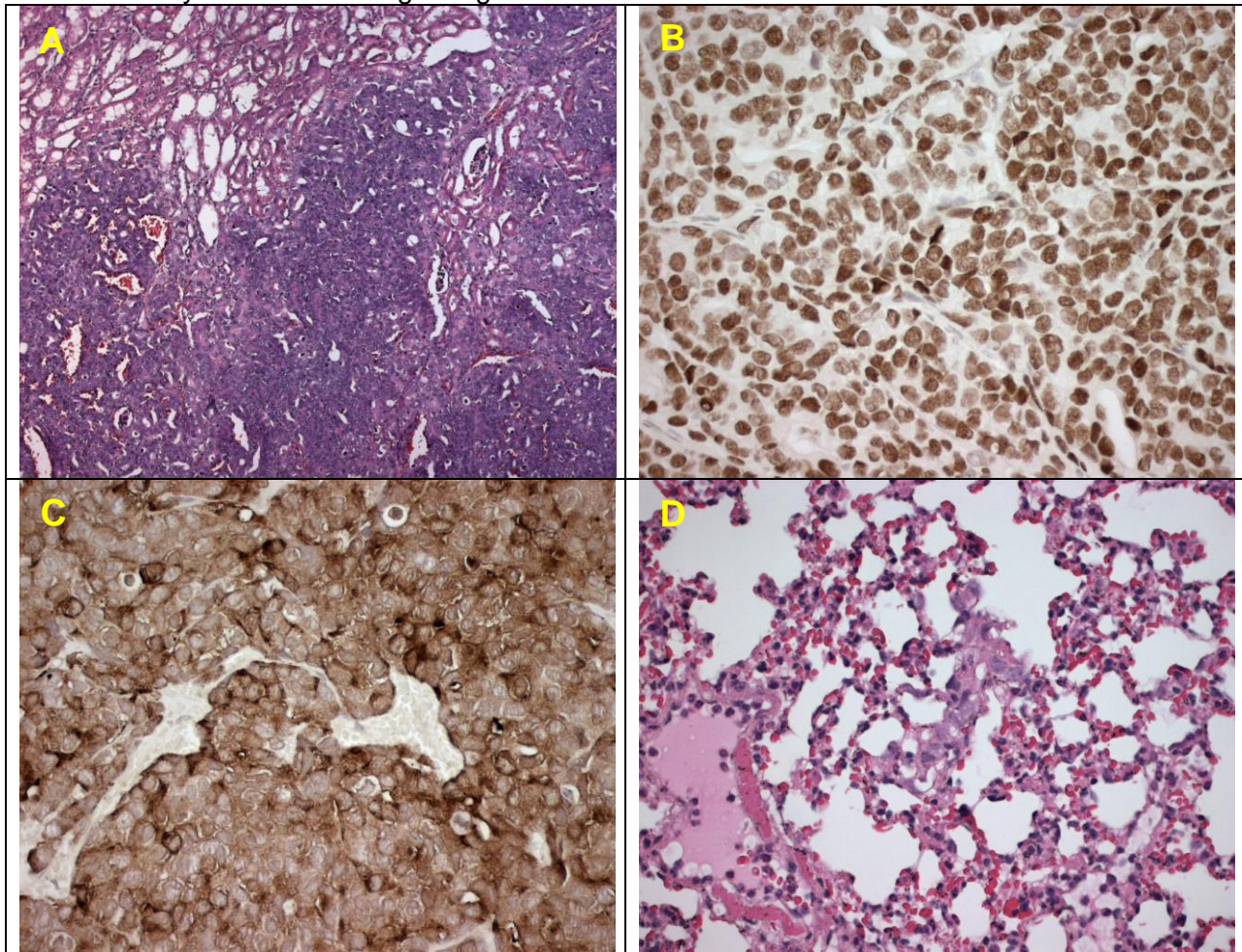


Fig. 1. (A). H&E stained LTL-313D tissue section. The tumor cells grow in solid sheets and invade host's renal parenchyma. **(B-C).** The tumor cells show strong immunostaining with antibodies to human-specific androgen receptor (B) and prostate-specific antigen (C). **(D).** Microscopic metastases of the LTL-313D cells in host lung. X400

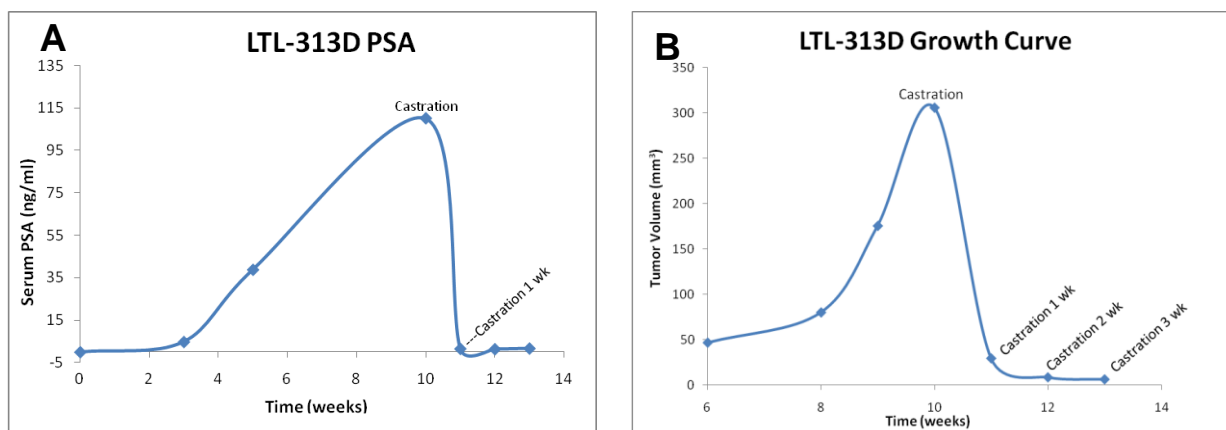


Fig. 2. LTL-313D shows androgen-dependent PSA production and growth *in vivo*: (A) Serum PSA levels increase in intact mice following implantation of LTL313D xenografts under the renal capsules. Castration (androgen ablation) quickly decreases the serum PSA to very low concentrations. (B) Castration leads to major decline in tumor volume.

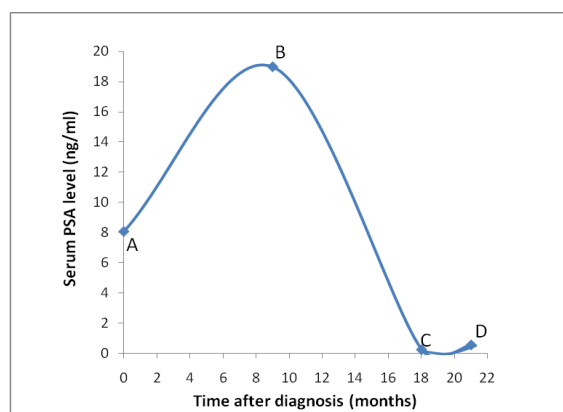


Fig. 3. Clinical course details of the patient. (A), detection of elevated blood PSA levels. (B), biopsy of tumor tissue used for LTL-313D development and initiation of androgen deprivation. (C, D), serum PSA levels remain low in response to androgen deprivation.

The LTL- 313D 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 metastasis.

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