LTL-313A datasheet

Origin	Human prostate cancer	**Histopathology**	High grade adenocarcinoma
Year of establishment	2009	**Doubling time**	10-12 days (subrenal capsule graft site)
Local invasion	Yes	**Metastasis**	Yes
Hormone Sensitivity	Androgen-dependent		

The LTL-313A tumor tissue line 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-313A shows invasion into adjacent renal parenchyma (Fig. 1A) and metastases to distant organs (Fig. 1B). Growth and prostate-specific antigen (PSA) production of the LTL-313A *in vivo* is androgen-dependent (Fig. 2). Figure 3 shows clinical course details of the patient. Viable tissues of the LTL-313A in early generations have been preserved by cryopreservation (DMSO), and can be readily resurrected for grafting.

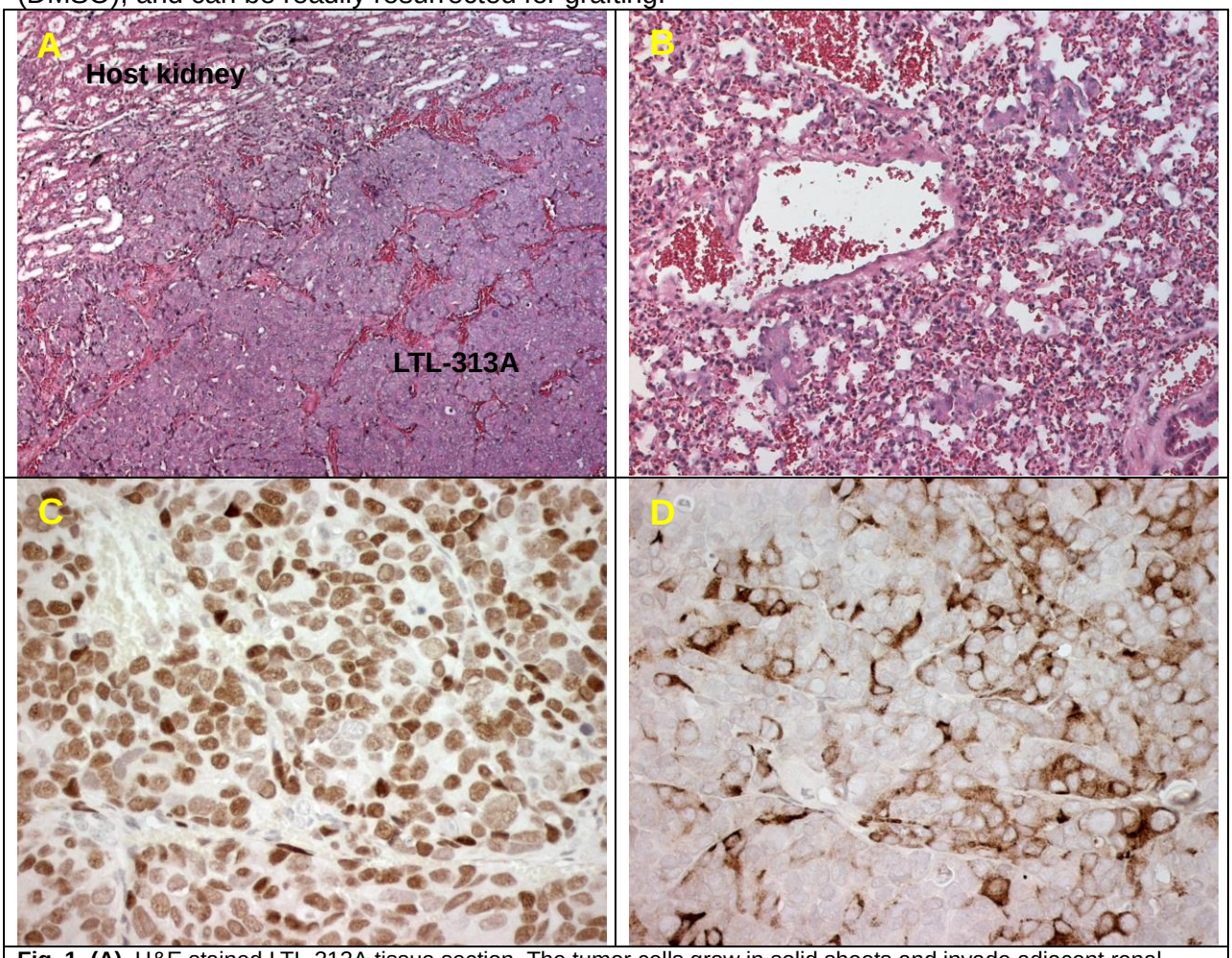

Fig. 1. (A), H&E stained LTL-313A tissue section. The tumor cells grow in solid sheets and invade adjacent renal parenchyma. X100 **(B)** Lung metastases of the LTL-313A. x200 **(C-D),** the tumor cells show strong immunostaining with antibodies to (C) human-specific androgen receptor and (D)prostate-specific antigen. x400

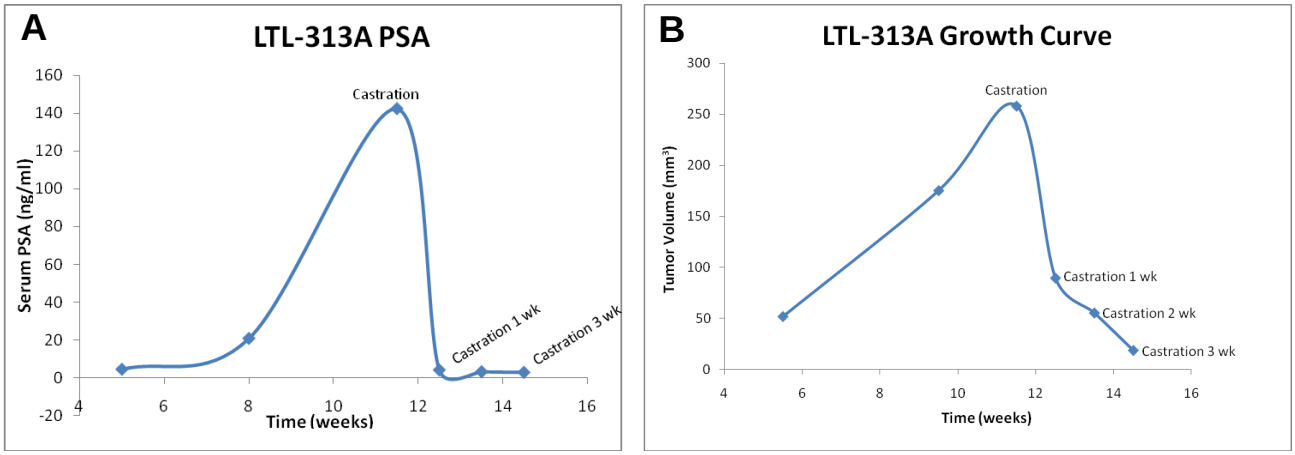

Fig. 2. LTL-313A shows androgen-dependent PSA production and growth *in vivo*: (A) Serum PSA levels increase in intact mice following implantation of LTL-313A xenografts under the renal capsules. Castration (androgen ablation) quickly decreases serum PSA levels to very low concentrations. **(B)** Castration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

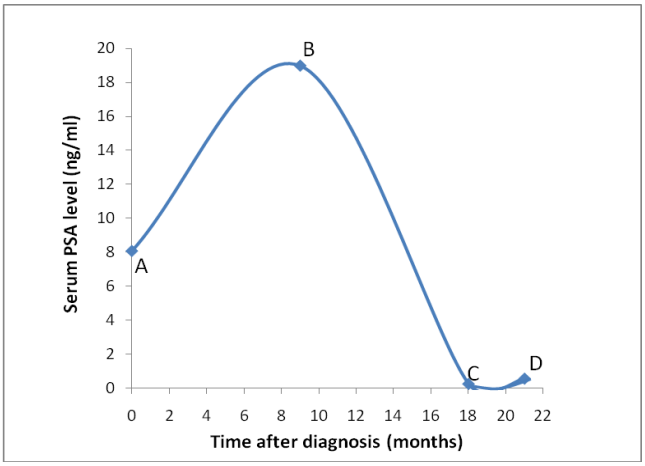

Fig. 3. Clinical course in th e patient. **(A)**, detection of elevated blood PSA levels. **(B)**, biopsy of tumor tissue used for LTL-313A development and initiation of androgen deprivation. **(C, D)**, serum PSA levels remain low in response to androgen deprivation.

Applications

1. Preclinical 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 genetic and cellular mechanisms underlying chemoresistance, tumor growth and progression/metastasis.

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