LTL-331 datasheet

Origin	Human prostate cancer	**Histopathology**	High grade adenocarcinoma
Year of establishment	2010	**Doubling time**	9 days (subrenal capsule graft site)
Local invasion	Yes	**Metastasis**	Yes, microscopic
Hormone sensitivity	Androgen-dependent		

The LTL-331 tumor tissue line (Fig. 1) was developed from a patient's primary prostate cancer (high grade prostate adenocarcinoma, Fig. 2). When grafted under the renal capsules of NOD-SCID mice, the LTL-331 line produces Prostate Specific Antigen (PSA) and shows invasion into adjacent renal parenchyma and metastases to distant organs. LTL-331 xenografts are initially sensitive to castration (androgen ablation) *in vivo,* with declines in serum PSA levels and tumor volumes, but then become resistant, presenting rapid, androgen-*in*dependent growth (Fig. 3). A castration-resistant tumor subline developed from the LTL-331 is designated LTL-331R. Viable tissues of the LTL-331 in early generations have been preserved by cryopreservation (DMSO), and can be readily resurrected for grafting.

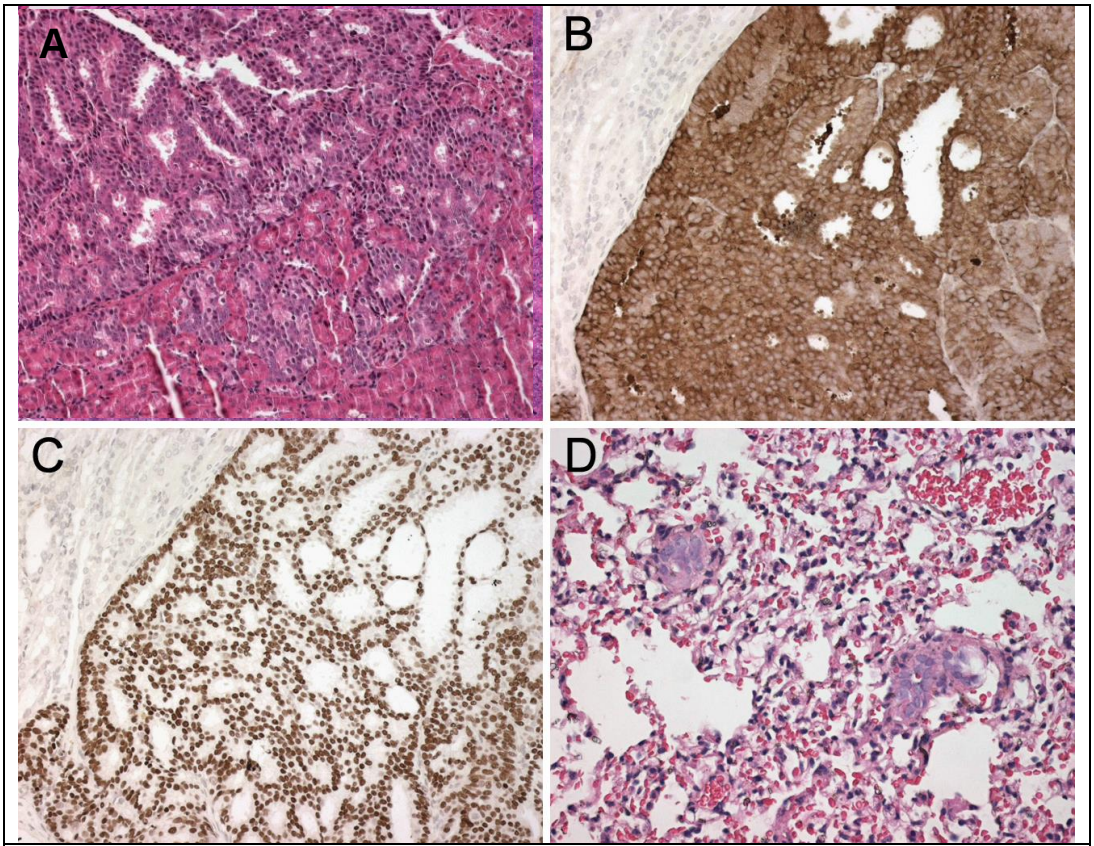

Fig. 1. (A). An H&E stained LTL-331 tissue section. The tumor cells form glandular structures and show local invasion to adjacent host's kidney. **(B, C).** The tumor cells show strong immunostaining with antibodies to human-specific PSA (B) and Androgen Receptor (C). **(D).** Microscopic metastases of LTL-331 tumor cells in host lung. X200

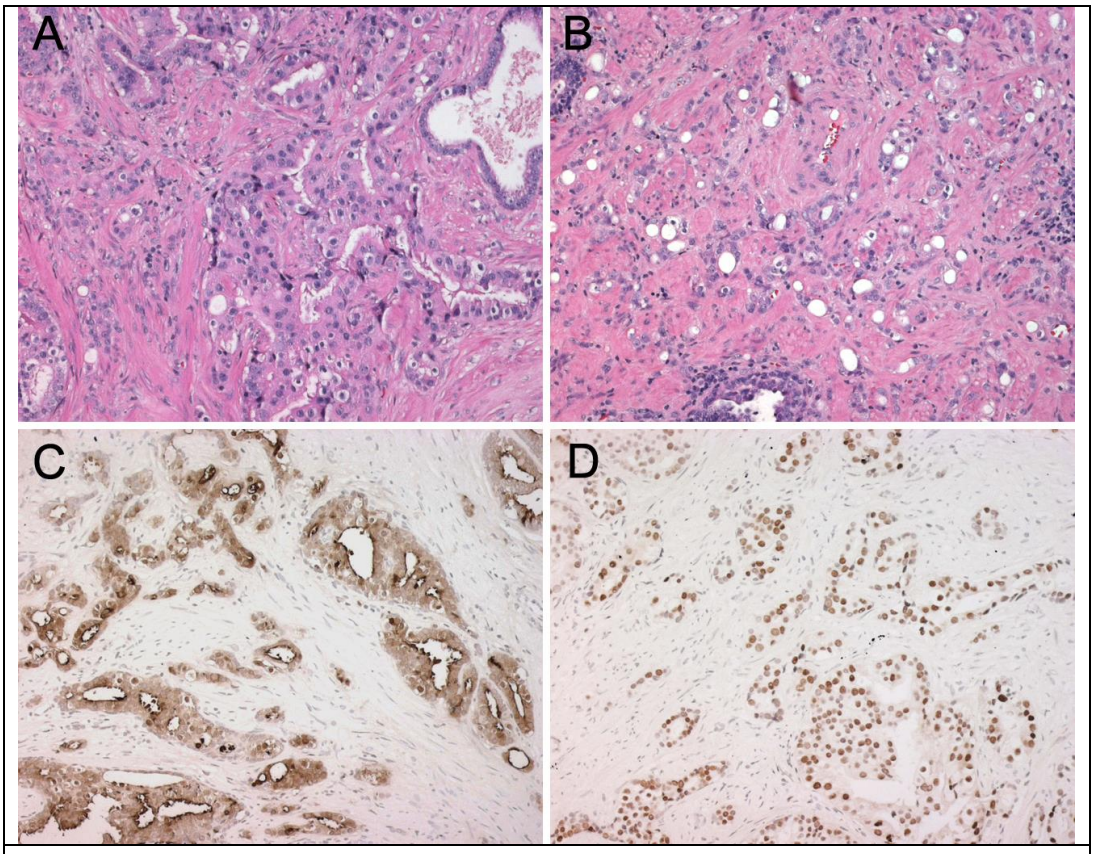

Fig 2. Patient's cancer tissue before grafting (high grade adenocarcinoma of the prostate). (A, B). The cancer cells form medium- or small-sized glandular or cribriform structures, or are present as single infiltrating cells. (C, D). They show positive immunohistochemical staining for PSA (C) and Androgen Receptor (D). x200

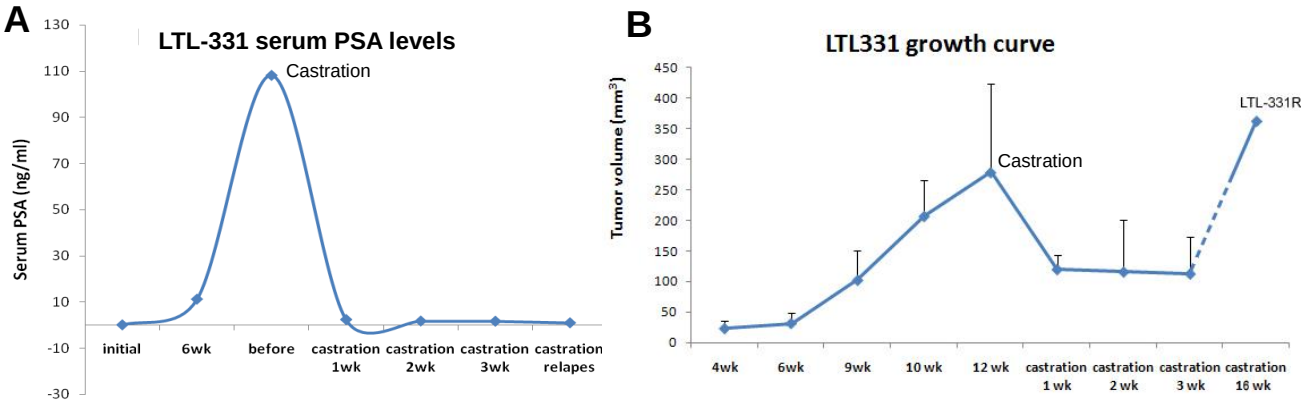

Fig. 3. (A). Serum PSA levels increase following implantation of LTL-331 xenografts under the renal capsules of intact male mice. Castration quickly decreases the serum PSA levels to exceedingly low concentrations. **(B).** The LTL-331 tumor tissue line initially responds to castration showing a major decline in tumor volume; at 16-32 weeks, it shows castration resistance, presenting rapid, androgen-independent growth without increasing serum PSA levels.

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 castration resistance, chemoresistance, tumor growth, progression/metatasis.

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