LTL-418 datasheet

Origin	Human prostate carcinoma	**Histopathology**	Adenocarcinoma
Year of establishment	2011	**Doubling time**	17.5 days (subrenal capsule graft site)
Local invasion	Yes, limited	**Metastasis**	No
Hormone Sensitivity	Androgen-dependent		

The LTL-418 tumor tissue line (Fig. 1) was developed from a patient's high-grade prostate adenocarcinoma (Fig. 2). When grafted under the renal capsules of NOD-SCID mice, the LTL-418 shows invasion into adjacent host kidney parenchyma. No metastasis was observed in host. Growth and prostate-specific antigen (PSA) production of the LTL-418 *in vivo* is androgen-dependent (Fig. 3). Viable tissues of the LTL-418 in early generations have been preserved by cryopreservation (DMSO), and can be readily resurrected for grafting.

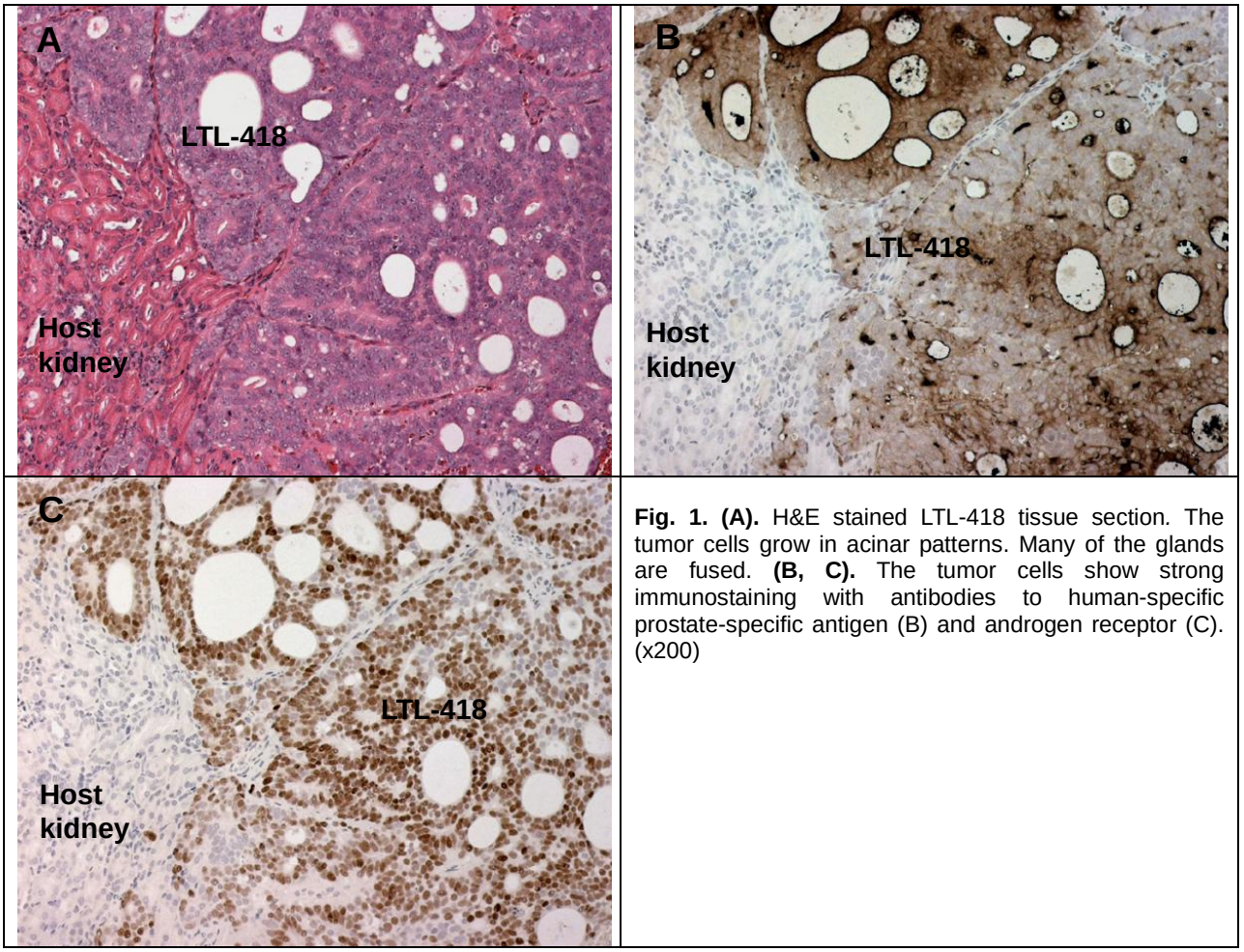

Fig. 1. (A). H&E stained LTL-418 tissue section. The tumor cells grow in acinar patterns. Many of the glands are fused. **(B, C).** The tumor cells show strong immunostaining with antibodies to human-specific prostate-specific antigen (B) and androgen receptor (C). (x200)

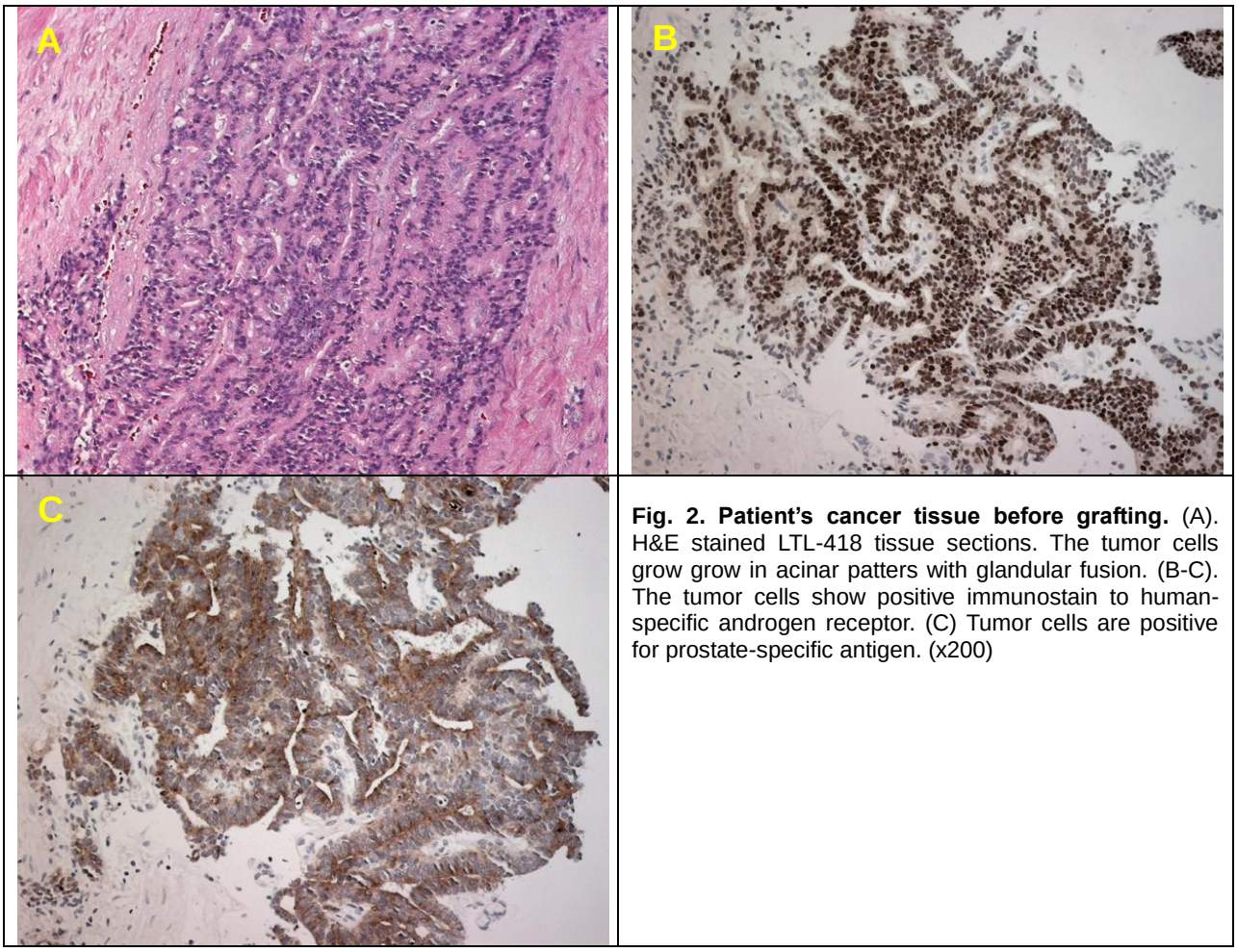

Fig. 2. Patient's cancer tissue before grafting. (A). H&E stained LTL-418 tissue sections. The tumor cells grow grow in acinar patters with glandular fusion. (B-C). The tumor cells show positive immunostain to human-specific androgen receptor. (C) Tumor cells are positive for prostate-specific antigen. (x200)

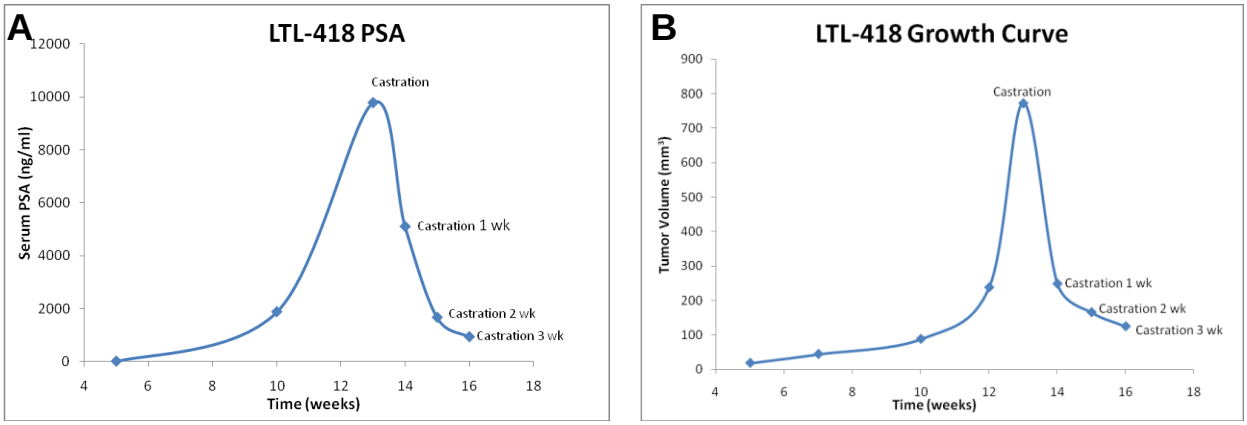

Fig. 3. LTL-418 shows androgen-dependent PSA production and growth *in vivo*: (A) Serum PSA levels increase in intact mice following implanation of LTL-418 xenografts under the renal capsules. Castration (androgen ablation) quickly decreases serum PSA levels to very low concentrations. **(B)** Castration leads to major tumor shrinkage.

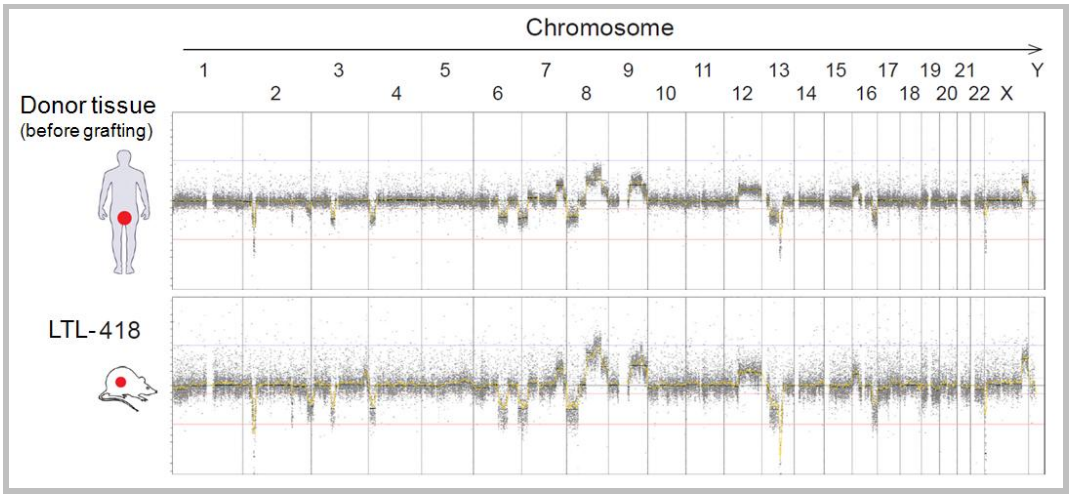

Fig. 4. Global aCGH profiles of LTL-418 and patient's cancer tissue before grafting. The chromosomal copy number profiles in the LTL-418 closely resembled the patient's cancer tissue, suggesting conservation of gross genome structure.

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 with 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 (in combination with metastatic tumor lines).

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