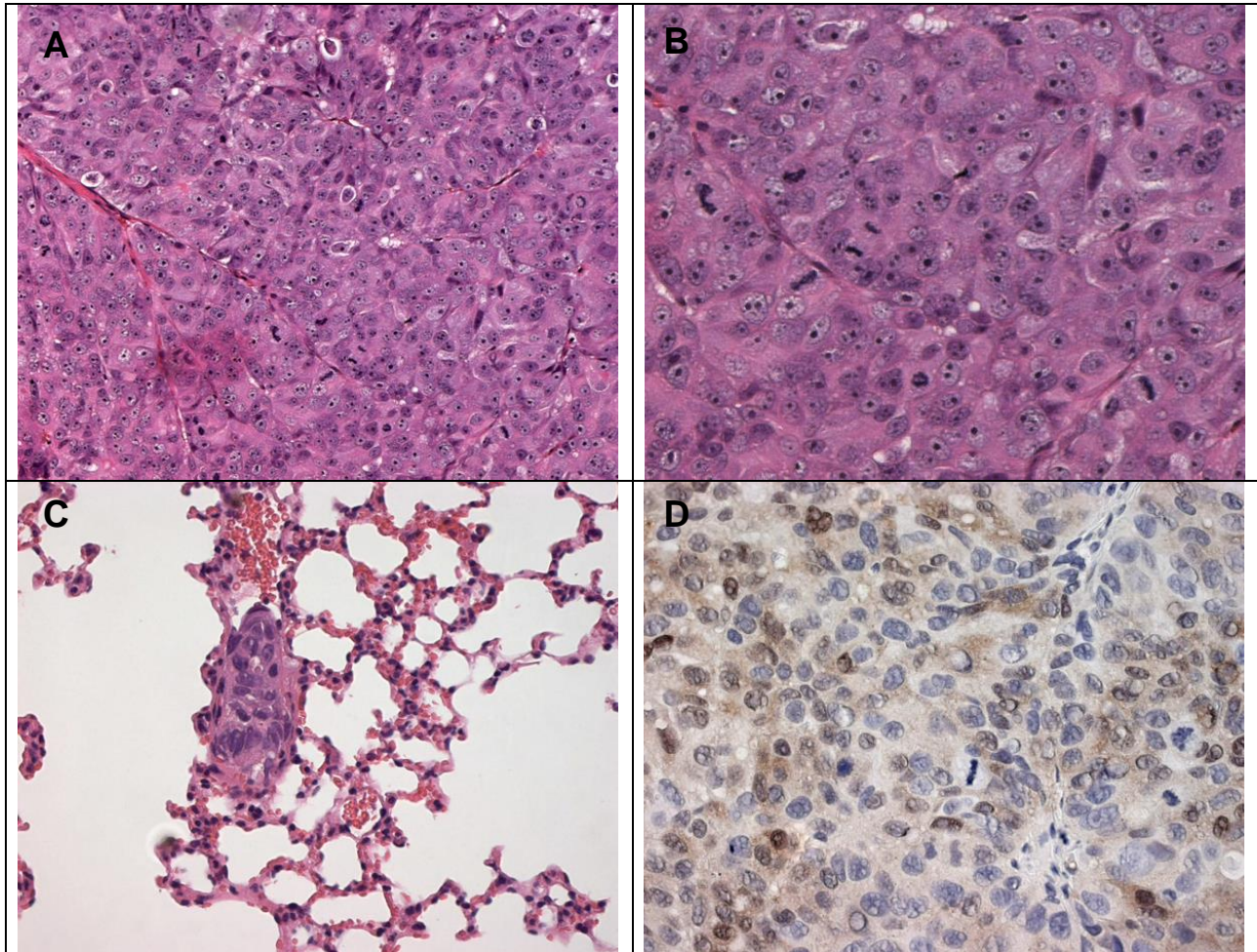
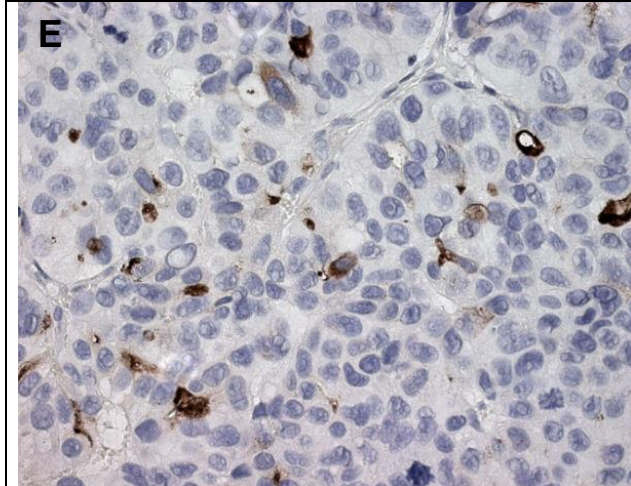


# LTL-573R datasheet

<b>Origin</b>	Human prostate cancer	<b>Histopathology</b>	Castration-resistant prostate adenocarcinoma
<b>Year of establishment</b>	2015	<b>Doubling time</b>	10-11 days
<b>Local invasion</b>	Yes	<b>Metastasis</b>	Yes
<b>Hormone Sensitivity</b>	Androgen independent		

The LTL-573R tumor tissue line (Fig. 1) is a castration-resistant subline of [LTL-573](#); it was developed by castration (androgen ablation) of mice bearing LTL-573 xenografts. The LTL-573R presents androgen-independent growth *in vivo*. When grafted under the renal capsules of NOD-SCID mice, the LTL-573R shows invasion into adjacent host kidney parenchyma and metastases to distant organs of the host. Viable tissues of the LTL-573R in early generations have been preserved by cryopreservation (DMSO), and can be readily resurrected for grafting. The LTL-573R grows well subcutaneously.





**Fig. 1. (A-B)**, an H&E stained LTL-573R tissue section showing the xenograft presents androgen-independent growth in castrated host mice. The tumor cells grow in solid sheets with high mitotic activities. (A, x200; B, x400) **(C)**, lung metastases of the LTL-573R. (x200) **(D)**, the tumor cells showed reduced nuclear AR immunoreactivity resulted from castration of the host mice. (x400) **(E)**, PSA expression was seen in scattered individual cancer cells. (x400)

### 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 or metastasis.

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