LTL-636 datasheet

Origin	Primary human lung cancer	**Histopathology**	Squamous cell carcinoma
Year of establishment	2006	**Doubling time**	6 days (sub-renal)
Local invasion	Yes	**Metastasis**	No
Drug sensitivity	Not determined		

The LTL-636 was developed from a patient's primary lung cancer(Squamous cell carcinoma. Stage T2N1M0). Histopathologically, it closely resembles the patient's tumor (Figs 1, 2). When grafted under the renal capsules of SCID mice, the LTL-636 shows local invasion into adjacent host kidney parenchyma. No metastasis was observed.

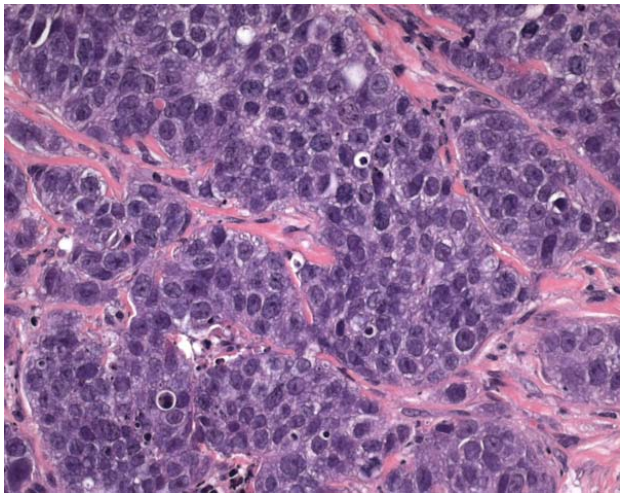

Fig. 1. H&E stained LTL-636 tissue sections.

The LTL-636 is a poorly differentiated squamous cell carcinoma with similar histopathological characteristics of the original patient's cancer (Fig. 2). (x400)

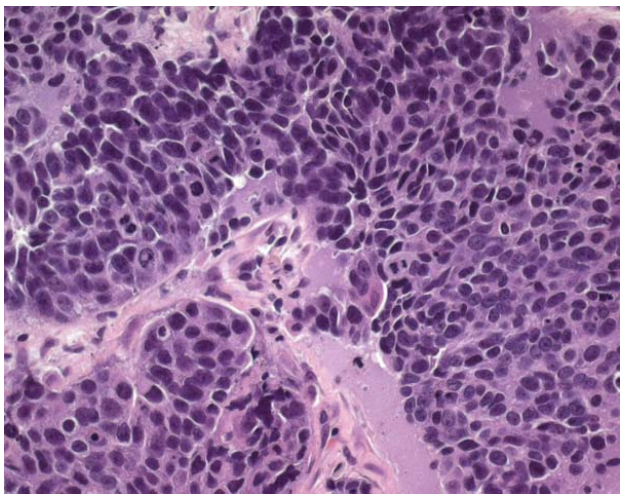

Fig. 2. Patient's cancer tissue before grafting.

Showing a poorly differentiated squamous cell carcinoma with stratification and focal keratinization

Genetic and epigenetic characteristics

The LTL-636 tissue line has been characterized using array CGH and Affymetrix chips. Some of the genes with potential therapeutic application are listed below.
Tissue microarrays containing LTL-636 tissue are available for screening potential molecular targets.

LTL-636 gene expression profile

Genes	Expression in LTL-636	Current stage in drug development
ERCC1	++	Clinical
RRM1	++++	Clinical
PTEN	+++	Clinical
BRCA1	+++	Clinical
EGFR	++	Clinical
HER (erb-B)	+++	Clinical
KRAS	+++	Clinical
P27	++++	Clinical
MRP2	/	Clinical
FasL	-	Clinical
bTubIII (tubulins)	/	Clinical
VEGFR-1	/	Clinical
VEGFR-2	/	Clinical
VEGFR-3	/	Clinical
PDGFR	++++	Clinical
CD117 (cKIT)	/	Clinical
RET	+	Clinical
CSF-1R	/	Clinical
CTLA-4	/	Clinical
CD28	-	Pre-clinical
TLR9	/	Pre-clinical
IGF1R	+++	Pre-clinical
ACVRL1 (ALK1)	-	Pre-clinical
FAK	/	Pre-clinical
Aurora Kinase (AK)	+++	Pre-clinical
mTOR	/	Pre-clinical
c-Met	/	Pre-clinical
Bcl-2	/	Pre-clinical
COX-2	++++	Pre-clinical
PCK alpha	++	Pre-clinical

Applications

1. Pre-clinical evaluation of existing and potential anticancer drugs. Examination of drug efficacy on tumor growth, cell death (apoptosis, necrosis), tissue invasion, and angiogenesis.

2. Discovery of potential therapeutic targets and/or biomarkers for drug sensitivity.

3. Study of mechanisms underlying tumor growth and progression.

References

1. Wang et al., Lab Invest (2005) 85, 1392-1404

2. Cutz et al, Clin. Cancer Res. 12(13): 4043-4054 (2006).

3. Lin et al, Cancer Res. 68 p.4352-4359 (2008)

For more information, please contact us by email: LTL@bccrc.ca or phone: 1-604-675-8013