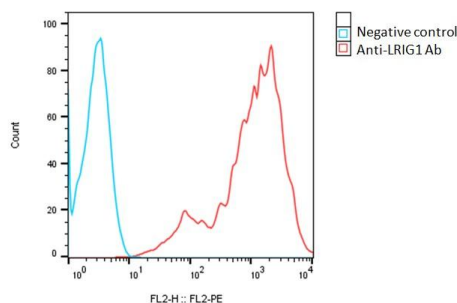


SPECIFICATIONS

Catalog Number	C3076
Cell Line Name	Human LRIG1-CHO-K1 stable cell line
Accession Number	AAI40908.1
Host Cell	Adherent CHO-K1
Quantity	Two vials of frozen cells (2x10 ⁶ per vial)
Culture Medium	DMEM with 10% FBS, 4µg/ml puromycin
Freezing Medium	90% FBS and 10% DMSO
Storage	Liquid nitrogen

DATA

Detection of human LRIG1 expression on human LRIG1-CHO-K1 stable cells using a monoclonal antibody specific for human LRIG1, followed by staining with PE-anti mouse antibody.


BACKGROUND

LRIG1, also known as Leucine-rich repeats and immunoglobulin-like domains protein 1, is a transmembrane protein that plays crucial roles in various cellular processes like regulating cell proliferation, differentiation, survival, and apoptosis. It functions as a negative regulator of various receptor tyrosine kinases (RTKs), including EGFR (Epidermal Growth Factor Receptor), PDGFR (Platelet-Derived Growth Factor Receptor), and MET (Hepatocyte Growth Factor Receptor). By inhibiting RTK signaling, LRIG1 helps to maintain tissue homeostasis and suppresses tumorigenesis. LRIG1 is widely expressed in various tissues, including epithelial tissues such as skin, lung, intestine, and the central nervous system. Dysregulation of LRIG1 expression is associated with several cancers. In many cases, LRIG1 acts as a tumor suppressor by inhibiting RTK signaling and suppressing cell proliferation. Reduced expression of LRIG1 has been observed in cancers such as glioma, breast cancer, ovarian cancer, and squamous cell carcinoma of the head and neck. Conversely, LRIG1 overexpression has been reported in certain cancers like renal cell carcinoma and glioblastoma, suggesting context-dependent roles in tumorigenesis. Strategies aimed at restoring LRIG1 expression or enhancing its activity could help inhibit aberrant RTK signaling and suppress tumor growth. Additionally, LRIG1-based therapies may enhance the efficacy of existing cancer treatments, such as RTK inhibitors. Further research into LRIG1 function and its role in cancer biology is warranted to fully exploit its therapeutic potential.

References

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