Family 1 subject – LAD-III Lymphoblastoid Cell Line

Catalogue number: 153766

Sub-type: Images:

Contributor

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Images:

Tool details

*FOR RESEARCH USE ONLY

ools.org Name: Family 1 subject – LAD-III Lymphoblastoid Cell Line

Alternate name: Fermitin family homolog 3, MIG2-like protein, Unc-112-related protein 2

Class:

Conjugate:

Description: An EBV-transformed B lymphoblastoid cell line (LCL) derived from a Maltese subject that has Leukocyte Adhesion Deficiency-III (LAD-III) with a mutation in the kindlin-3 gene. LCLs are also available from the mother, father and sister. This cell line was derived from 'Family 1 subject' as described in Svensson et al. 2009. N.Nat Med. 2009 Mar;15(3):306-12. PMID: 19234463. The Family 1 subject has a homozygous inactivating mutation within the splice acceptor site of exon 14 in the KINDLIN3 gene. This mutation leads to an overall decrease in KINDLIN3 mRNA levels and loss of protein expression.

Purpose: Parental cell: Organism: Human Tissue: Blood

Model: Gender: Isotype: Reactivity: Selectivity: Host:

Immunogen:

Immunogen UNIPROT ID:

Sequence:

Growth properties: Suspension, lymphoblastoid cell line

Production details:

Formulation:

Recommended controls:
Bacterial resistance:
Selectable markers:
Additional notes:

Target details

Target: Kindlin-3, UniProt ID:Q86UX7

Target alternate names:

Target background:

Molecular weight:

Ic50:

Applications

Application:

Application notes:

Handling

Format: Frozen
Concentration:
Passage number:

Growth medium: RPMI-1640 + 10% FCS

Temperature: Atmosphere: Volume:

Storage medium: Storage buffer:

Storage conditions: Liquid Nitrogen

Shipping conditions: Dry ice

Related tools

Related tools: Family 1 father - LAD-III Lymphoblastoid Cell Line; Family 1 mother - LAD-III

Cancer Tools.org

Lymphoblastoid Cell Line ; Family 1 sibling - LAD-III Lymphoblastoid Cell Line

References

References: Svensson et al. 2009. Nat Med. 15(3):306-12. PMID: 19234463. ; Leukocyte adhesion deficiency-III is caused by mutations in KINDLIN3 affecting integrin activation.

