

Resource Summary Report

Generated by [NIF](#) on Sep 4, 2026

C57BL/6J-Unc13d^{Jinx}/Mmucd

RRID:MMRRC_016137-UCD

Type: Organism

Proper Citation

RRID:MMRRC_016137-UCD

Organism Information

URL: https://www.mmrrc.org/catalog/sds.php?mmrrc_id=16137

Proper Citation: RRID:MMRRC_016137-UCD

Description: Mus musculus with name C57BL/6J-Unc13d^{Jinx}/Mmucd from MMRRC.

Species: Mus musculus

Notes: Research areas: Cell Biology, Immunology and Inflammation, Models for Human Disease, Virology; Mutation Type: chemically induced mutation ; Collection: Beutler Mutagenetix

Phenotype: increased neutrophil cell number [MP:0000219]| increased monocyte cell number [MP:0000220]| enlarged spleen [MP:0000691]| anemia [MP:0001577]| abnormal bone marrow morphology [MP:0002397]| increased susceptibility to viral infection [MP:0002418]| abnormal macrophage physiology [MP:0002451]| abnormal cytokine secretion [MP:0003009]| thrombocytopenia [MP:0003179]| decreased cytotoxic T cell cytolysis [MP:0005079]| increased CD8-positive [MP:0008078]| alpha-beta T cell number [MP:0008523]| absent lymph node germinal center [MP:0010766]

Affected Gene: Unc13d

Catalog Number: 016137-UCD

Background: chemically induced mutation

Database: Mutant Mouse Resource and Research Center (MMRRC)

Database Abbreviation: MMRRC

Source References: [PMID:17420270](#)

Alternate IDs: MMRRC_16137-UCD, MMRRC_016137, MMRRC_16137

Organism Name: C57BL/6J-*Unc13d*^{Jinx}/Mmucd

Record Creation Time: 20230308T054939+0000

Record Last Update: 20260829T122319+0000

Ratings and Alerts

No rating or validation information has been found for C57BL/6J-*Unc13d*^{Jinx}/Mmucd.

No alerts have been found for C57BL/6J-*Unc13d*^{Jinx}/Mmucd.

Data and Source Information

Source: [Integrated Animals](#)

Source Database: Mutant Mouse Resource and Research Center (MMRRC)

Usage and Citation Metrics

We found 3 mentions in open access literature.

Listed below are recent publications. The full list is available at [NIF](#) .

Lei L, et al. (2026) Genotoxicity profiling reveals distinct platform-and cell type-specific effects in therapeutic gene editing for genetic hyperinflammation. *Cell stem cell*, 33(6), 930.

Kögl T, et al. (2024) Patients and mice with deficiency in the SNARE protein SYNTAXIN-11 have a secondary B cell defect. *The Journal of experimental medicine*, 221(7).

Weißert K, et al. (2022) Adoptive T cell therapy cures mice from active hemophagocytic lymphohistiocytosis (HLH). *EMBO molecular medicine*, 14(12), e16085.