Resource Summary Report

Generated by NIF on Sep 9, 2026

y[1] sc[*] v[1] sev[21]; P{y[+t7.7] v[+t1.8]=TRiP.HMS02849}attP2

RRID:BDSC_44553
Type: Organism

Proper Citation

RRID:BDSC_44553

Organism Information

URL: https://n2t.net/bdsc:44553

Proper Citation: RRID:BDSC_44553

Description: Drosophila melanogaster with name y[1] sc[*] v[1] sev[21]; P{y[+t7.7] v[+t1.8]=TRiP.HMS02849}attP2 from BDSC.

Species: Drosophila melanogaster

Notes: May be segregating TM3, Sb[1]. Donor: Transgenic RNAi Project

Affected Gene: mew, UAS, sc, sev, v, y

Genomic Alteration: Chromosome 1, Chromosome 3

Catalog Number: 44553

Database: Bloomington Drosophila Stock Center (BDSC)

Database Abbreviation: BDSC

Availability: available

Alternate IDs: BDSC_44553, BDSC:44553, BL44553

Organism Name: y[1] sc[*] v[1] sev[21]; P{y[+t7.7] v[+t1.8]=TRiP.HMS02849}attP2

Record Creation Time: 20240911T222724+0000

Record Last Update: 20260905T140559+0000

Ratings and Alerts

No rating or validation information has been found for y[1] sc[*] v[1] sev[21]; P{y[+t7.7] v[+t1.8]=TRiP.HMS02849}attP2.

No alerts have been found for y[1] sc[*] v[1] sev[21]; P{y[+t7.7] v[+t1.8]=TRiP.HMS02849}attP2.

Data and Source Information

Source: Integrated Animals

Source Database: Bloomington Drosophila Stock Center (BDSC)

Usage and Citation Metrics

We found 4 mentions in open access literature.

Listed below are recent publications. The full list is available at NIF .

Chang YC, et al. (2024) Identification of secretory autophagy as a mechanism modulating activity-induced synaptic remodeling. Proceedings of the National Academy of Sciences of the United States of America, 121(16), e2315958121.

Leroy C, et al. (2024) Linking Basement Membrane and Slit Diaphragm in Drosophila Nephrocytes. Journal of the American Society of Nephrology : JASN, 35(9), 1208.

Delaney M, et al. (2024) Actin Cytoskeleton and Integrin Components Are Interdependent for Slit Diaphragm Maintenance in Drosophila Nephrocytes. Cells, 13(16).

Hernandez SJ, et al. (2023) An altered extracellular matrix-integrin interface contributes to Huntington's disease-associated CNS dysfunction in glial and vascular cells. Human molecular genetics, 32(9), 1483.