

Resource Summary Report

Generated by [dkNET](#) on Aug 2, 2026

[y\[1\] sc\[*\] v\[1\] sev\[21\]; P{y\[+t7.7\] v\[+t1.8\]=TRiP.HMS00139}attP2/TM3, Sb\[1\]](#)

RRID:BDSC_34824

Type: Organism

Proper Citation

RRID:BDSC_34824

Organism Information

URL: <https://n2t.net/bdsc:34824>

Proper Citation: RRID:BDSC_34824

Description: Drosophila melanogaster with name y[1] sc[*] v[1] sev[21]; P{y[+t7.7] v[+t1.8]=TRiP.HMS00139}attP2/TM3, Sb[1] from BDSC.

Species: Drosophila melanogaster

Notes: Donor: Transgenic RNAi Project

Affected Gene: Prosbeta1, UAS, Sb, sc, sev, v, y

Genomic Alteration: Chromosome 1, Chromosome 3

Catalog Number: 34824

Database: Bloomington Drosophila Stock Center (BDSC)

Database Abbreviation: BDSC

Availability: available

Alternate IDs: BDSC_34824, BL34824

Organism Name: y[1] sc[*] v[1] sev[21]; P{y[+t7.7] v[+t1.8]=TRiP.HMS00139}attP2/TM3, Sb[1]

Record Creation Time: 20240911T222557+0000

Record Last Update: 20260801T195143+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.HMS00139}attP2/TM3, Sb[1].

No alerts have been found for y[1] sc[*] v[1] sev[21]; P{y[+t7.7] v[+t1.8]=TRiP.HMS00139}attP2/TM3, Sb[1].

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 [dkNET](#) .

Zhang J, et al. (2025) Nutrient status alters developmental fates via a switch in mitochondrial homeodynamics. Nature communications, 16(1), 1258.

Rai M, et al. (2021) Proteasome stress in skeletal muscle mounts a long-range protective response that delays retinal and brain aging. Cell metabolism, 33(6), 1137.

Fernández-Cruz I, et al. (2020) Rpt2 proteasome subunit reduction causes Parkinson's disease like symptoms in Drosophila. IBRO reports, 9, 65.

Tsakiri EN, et al. (2019) Proteasome dysfunction induces excessive proteome instability and loss of mitostasis that can be mitigated by enhancing mitochondrial fusion or autophagy. Autophagy, 15(10), 1757.