Resource Summary Report

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C. elegans Gene Knockout Consortium

RRID:SCR_003000
Type: Tool

Proper Citation

C. elegans Gene Knockout Consortium (RRID:SCR_003000)

Resource Information

URL: http://celeganskoconsortium.omrf.org

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Description: THIS RESOURCE IS NO LONGER IN SERVCE, documented September 2, 2016. The mission of the C. elegans Gene Knockout Consortium is to facilitate genetic research of this important model system through the production of deletion alleles at specified gene targets. We choose targets based on investigator requests. Strains produced by the consortium are freely available with no restrictions to any investigator. At one time, our capacity dictated that we restrict requests to five per lab. This restriction no longer holds. Investigators are encouraged especially to register requests for functionally related groups of genes. Consortium strains are distributed by the C. elegans Genetic Center (CGC). In most cases, when you use the Consortium web site to request an existing allele, your request is forwarded automatically to the CGC. However, if you indicate that an existing allele is not satisfactory for your research, (for whatever reason), you may request that we generate another allele for the same target. Any information generated by the Consortium is entered into the official C. elegans data repository, WormBase.

Abbreviations: C. elegans Gene Knockout Consortium

Synonyms: C. elegans Gene Knockout Consortium

Resource Type: biomaterial supply resource, material resource, organism supplier

Keywords: gene, locus, knockout, genetic, research, model, allele, target, strain, deletion allele, gene target

Availability: THIS RESOURCE IS NO LONGER IN SERVICE

Resource Name: C. elegans Gene Knockout Consortium

Resource ID: SCR_003000

Alternate IDs: nif-0000-30230

Record Creation Time: 20220129T080216+0000

Record Last Update: 20260905T132456+0000

Ratings and Alerts

No rating or validation information has been found for C. elegans Gene Knockout Consortium.

No alerts have been found for C. elegans Gene Knockout Consortium.

Data and Source Information

Source: SciCrunch Registry

Usage and Citation Metrics

We found 30 mentions in open access literature.

Listed below are recent publications. The full list is available at PRECISE-TBI .

Ballestriero F, et al. (2018) Correction: Antinematode Activity of Violacein and the Role of the Insulin/IGF-1 Pathway in Controlling Violacein Sensitivity in Caenorhabditis elegans. PloS one, 13(12), e0210026.

Jensen VL, et al. (2015) Formation of the transition zone by Mks5/Rpgrip1L establishes a ciliary zone of exclusion (CIZE) that compartmentalises ciliary signalling proteins and controls PIP2 ciliary abundance. The EMBO journal, 34(20), 2537.

Ballestriero F, et al. (2014) Antinematode activity of Violacein and the role of the insulin/IGF-1 pathway in controlling violacein sensitivity in Caenorhabditis elegans. PloS one, 9(10), e109201.

Manil-Ségalen M, et al. (2014) Interactions between endosomal maturation and autophagy: analysis of ESCRT machinery during Caenorhabditis elegans development. Methods in enzymology, 534, 93.

Milton AC, et al. (2013) The NF-Y complex negatively regulates Caenorhabditis elegans tbx-2 expression. Developmental biology, 382(1), 38.

Song BM, et al. (2013) Recognition of familiar food activates feeding via an endocrine serotonin signal in Caenorhabditis elegans. eLife, 2, e00329.

Zheng C, et al. (2013) Histone methylation restrains the expression of subtype-specific genes during terminal neuronal differentiation in Caenorhabditis elegans. PLoS genetics, 9(12), e1004017.

Lecroisey C, et al. (2013) ZYX-1, the unique zyxin protein of Caenorhabditis elegans, is involved in dystrophin-dependent muscle degeneration. Molecular biology of the cell, 24(8), 1232.

Baylis HA, et al. (2012) Genetic analysis of IP3 and calcium signalling pathways in C. elegans. Biochimica et biophysica acta, 1820(8), 1253.

Ródenas E, et al. (2012) Dissection of the NUP107 nuclear pore subcomplex reveals a novel interaction with spindle assembly checkpoint protein MAD1 in Caenorhabditis elegans. Molecular biology of the cell, 23(5), 930.

Leroy M, et al. (2012) Pathogen-induced Caenorhabditis elegans developmental plasticity has a hormetic effect on the resistance to biotic and abiotic stresses. BMC evolutionary biology, 12, 187.

Howe K, et al. (2012) WormBase: Annotating many nematode genomes. Worm, 1(1), 15.

Williams CL, et al. (2011) MKS and NPHP modules cooperate to establish basal body/transition zone membrane associations and ciliary gate function during ciliogenesis. The Journal of cell biology, 192(6), 1023.

Huang L, et al. (2011) TMEM237 is mutated in individuals with a Joubert syndrome related disorder and expands the role of the TMEM family at the ciliary transition zone. American journal of human genetics, 89(6), 713.

Hao L, et al. (2011) The retrograde IFT machinery of C. elegans cilia: two IFT dynein complexes? PloS one, 6(6), e20995.

Huang L, et al. (2011) Three-generation experiment showed female C57BL/6J mice drink drainage canal water containing low level of TCDD-like activity causing high pup mortality. The Journal of toxicological sciences, 36(6), 713.

Fridolfsson HN, et al. (2010) UNC-83 coordinates kinesin-1 and dynein activities at the nuclear envelope during nuclear migration. Developmental biology, 338(2), 237.

Au C, et al. (2009) SMF-1, SMF-2 and SMF-3 DMT1 orthologues regulate and are regulated differentially by manganese levels in C. elegans. PloS one, 4(11), e7792.

Gontijo AM, et al. (2009) Mutations in genes involved in nonsense mediated decay ameliorate the phenotype of sel-12 mutants with amber stop mutations in Caenorhabditis elegans. BMC genetics, 10, 14.

Kulkarni M, et al. (2008) E1 ubiquitin-activating enzyme UBA-1 plays multiple roles throughout C. elegans development. PLoS genetics, 4(7), e1000131.