

Resource Summary Report

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International Mouse Phenotyping Consortium (IMPC)

RRID:SCR_006158

Type: Tool

Proper Citation

International Mouse Phenotyping Consortium (IMPC) (RRID:SCR_006158)

Resource Information

URL: <http://www.mousephenotype.org/>

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(RRID:SCR_006158)

Description: Center that produces knockout mice and carries out high-throughput phenotyping of each line in order to determine function of every gene in mouse genome. These mice will be preserved in repositories and made available to scientific community representing valuable resource for basic scientific research as well as generating new models for human diseases.

Abbreviations: IKMC, IMPC

Synonyms: KOMP, KOMP-CSD, KOMP-Regeneron, IMPC - International Mouse Phenotyping Consortium, International Mouse Phenotyping Consortium, IMPC, International Mouse Phenotyping Consortium (IMPC), EUCOMM, IKMC

Resource Type: biomaterial supply resource, material resource

Defining Citation: [PMID:27626380](#), [PMID:24652767](#), [PMID:24197666](#), [PMID:25127743](#), [PMID:25343444](#), [PMID:24642684](#), [PMID:21677750](#), [PMID:22968824](#), [PMID:22940749](#), [PMID:22991088](#), [PMID:25992600](#), [PMID:22566555](#), [PMID:23519032](#), [PMID:22211970](#), [PMID:24194600](#), [PMID:26147094](#), [PMID:24634472](#), [PMID:24932005](#), [PMID:25093073](#), [PMID:24046361](#), [PMID:24033988](#), [PMID:23315689](#), [PMID:22926223](#), [PMID:21185382](#), [PMID:21737429](#), [PMID:19933761](#), [PMID:19689210](#), [PMID:17905814](#), [PMID:17218247](#), [PMID:16933996](#), [PMID:16254554](#), [PMID:15908916](#), [PMID:15340423](#), [PMID:15340424](#), [PMID:28650954](#), [PMID:28650483](#), [PMID:29026089](#), [PMID:29348434](#), [PMID:29352221](#), [PMID:29396915](#), [PMID:29626206](#), [PMID:22566555](#)

Keywords: phenotype, phenotyping, gene, knockout mouse, knockout, genome, function, gene function, mouse model, mutation, embryonic stem cell, genotype, disease, anatomy, procedure, image, experimental protocol, annotation, genotype-phenotype, FASEB list

Funding: NIH Office of the Director UM1 OD023222

Availability: Free, Freely available

Resource Name: International Mouse Phenotyping Consortium (IMPC)

Resource ID: SCR_006158

Alternate IDs: nlx_151660

Alternate URLs: <https://www.mousephenotype.org/data/documentation/data-access>

Record Creation Time: 20220129T080234+0000

Record Last Update: 20260821T042703+0000

Ratings and Alerts

No rating or validation information has been found for International Mouse Phenotyping Consortium (IMPC).

No alerts have been found for International Mouse Phenotyping Consortium (IMPC).

Data and Source Information

Source: [SciCrunch Registry](#)

Usage and Citation Metrics

We found 2527 mentions in open access literature.

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

Hernández-Martínez R, et al. (2026) AXIN1 and AXIN2 regulate the WNT-signaling landscape to promote distinct mesoderm programs. *Developmental cell*, 61(7), 1572.

Vicencio-Jimenez S, et al. (2026) Abnormal Hearing Phenotypes in "Ignorome" Knockout Mice as Predictors of Cognitive Dysfunction. *Genes, brain, and behavior*, 25(1), e70045.

Bandesh K, et al. (2026) Deep single-cell decoding of human pancreatic islets reveals T2D β -cell gene expression defects. *The EMBO journal*, 45(11), 3978.

Galter I, et al. (2026) EchoVisuALL: From Echocardiography to Gene Discovery. *bioRxiv* : the preprint server for biology.

Parenti I, et al. (2026) Pathogenic variants in the cohesin loader subunit MAU2 underlie a distinct Cornelia de Lange Syndrome subtype. *Nature communications*, 17(1).

Sukseree S, et al. (2026) Comparative Genomics Reveals Recurrent Loss of Autophagy-Related 9B (ATG9B) in Amniotes. *Genes*, 17(6).

Ursachi VC, et al. (2026) Phosphodiesterase inhibitors as emerging therapeutics for skeletal disorders: A comprehensive review of mechanisms and repurposing potential. *Bone reports*, 29, 101905.

Inglebert Y, et al. (2026) The calcium-binding protein S100 β regulates synaptic plasticity in the visual cortex. *iScience*, 29(5), 115793.

Chen N, et al. (2026) Genetic and embryonic transcriptome analyses reveal the molecular and developmental basis of Mayer-Rokitansky-Küster-Hauser syndrome. *Journal of medical genetics*, 63(2), 113.

Coppola MR, et al. (2026) Zebrafish as a model for Catel-Manzke syndrome-identification and characterization of the zebrafish TGDS ortholog. *The FEBS journal*, 293(8), 2248.

Wilson R, et al. (2026) International Mouse Phenotyping Consortium Portal: facilitating investigation of gene function and providing insights into human disease. *Nucleic acids research*, 54(D1), D1133.

Hernandez-Beeftink T, et al. (2026) Contribution of dominant and recessive model effects to the genetic architecture of Idiopathic Pulmonary Fibrosis. *medRxiv : the preprint server for health sciences*.

Moulin TC, et al. (2026) Conserved NT5C2 links context-specific behaviors with psychiatric and metabolic risk. *Behavioral and brain functions : BBF*, 22(1), 7.

Ogan BM, et al. (2026) FAM83H Regulates Postnatal T Cell Development Through Thymic Stroma Organization. *European journal of immunology*, 56(1), e70126.

Saad C, et al. (2026) Amino adipate-semialdehyde synthase, a potential target for substrate reduction therapy in glutaric aciduria type 1. *Scientific reports*, 16(1).

Yi P, et al. (2026) Genetic link between metabolic syndrome and coronary artery disease: Insights from genome-wide cross-trait analysis. *Global medical genetics*, 13(1), 100092.

Ibarra-Soria X, et al. (2026) Large-scale mouse mutagenesis identifies novel genes affecting vertebral anatomy. *Mammalian genome : official journal of the International Mammalian Genome Society*, 37(1), 34.

Pollo P, et al. (2026) New approaches to meta-analyze differences in skewness, kurtosis, and correlation. *PLoS biology*, 24(2), e3003653.

Laurent K, et al. (2026) Epigenetic Legacy: The Role of Sperm miRNAs in the Paternal Inheritance of Diabetes and Obesity Development. *Diabetes/metabolism research and reviews*, 42(3), e70157.

Wang F, et al. (2026) Microglial HVCN1 Deficiency Improves Movement and Survival of SOD1G93A ALS Mice by Enhancing Microglial Migration and Neuroprotection. *Advanced science (Weinheim, Baden-Wurttemberg, Germany)*, 13(13), e12149.