

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: 20260801T191049+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 2449 mentions in open access literature.

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

Takeuchi C, et al. (2026) Comprehensive perturbation of transcription factors in human cardiomyocytes reveals the regulatory architecture of congenital heart disease. bioRxiv : the preprint server for biology.

Meurs R, et al. (2025) MCTS2 and distinct eIF2D roles in uORF-dependent translation regulation revealed by in vitro re-initiation assays. The EMBO journal, 44(3), 854.

Kim JM, et al. (2025) Two novel genetic variants in the WFDC2 gene from patients with bronchiectasis. Respiratory research, 26(1), 108.

Chin D, et al. (2025) Genome-wide association study of Idiopathic Pulmonary Fibrosis susceptibility using clinically-curated European-ancestry datasets. medRxiv : the preprint server for health sciences.

Orgil BO, et al. (2025) Unraveling the genetic blueprint of doxorubicin-induced cardiotoxicity through systems genetics approaches. Cardio-oncology (London, England), 11(1), 53.

Jackson VE, et al. (2025) Multi-omic spatial effects on high-resolution AI-derived retinal thickness. *Nature communications*, 16(1), 1317.

Wang G, et al. (2025) Cellular SLC35B4 promotes internalization during influenza A virus entry. *mBio*, 16(5), e0019425.

Issa KHB, et al. (2025) Molecular basis for the activation of outer dynein arms in cilia. *Nature structural & molecular biology*, 32(12), 2454.

Ramzan M, et al. (2025) Carboxypeptidase D deficiency causes hearing loss amenable to treatment. *The Journal of clinical investigation*, 135(23).

Hatzikotoulas K, et al. (2025) Translational genomics of osteoarthritis in 1,962,069 individuals. *Nature*, 641(8065), 1217.

Anderson KJ, et al. (2025) Androgens mediate sexual dimorphism in Pilarowski-Bjornsson Syndrome. *medRxiv : the preprint server for health sciences*.

Ahmed S, et al. (2025) Large-scale audiometric phenotyping identifies distinct genes and pathways involved in hearing loss subtypes. *Communications biology*, 8(1), 1515.

Marton T, et al. (2025) Polymerase theta repairs persistent G1-induced DNA breaks in S-phase during class switch recombination. *Nature communications*, 16(1), 10536.

Zafeer MF, et al. (2025) Human organoids for rapid validation of gene variants linked to cochlear malformations. *Human genetics*, 144(4), 375.

Vulto-van Silfhout AT, et al. (2025) Bi-allelic loss-of-function variants in POC5 cause a syndromic retinal, endocrine, and neuromuscular ciliopathy. *Genetics in medicine : official journal of the American College of Medical Genetics*, 27(10), 101513.

Skerrett-Byrne DA, et al. (2025) Dad's Diet Shapes the Future: How Paternal Nutrition Impacts Placental Development and Childhood Metabolic Health. *Molecular nutrition & food research*, 69(22), e70261.

Wotton JM, et al. (2025) Identifying genetic determinants of outer retinal function in mice using a large-scale gene-targeted screen. *PLoS genetics*, 21(9), e1011886.

Vitry S, et al. (2025) Advancing precision ear medicine: leveraging animal models for disease insights and therapeutic innovations. *Mammalian genome : official journal of the International Mammalian Genome Society*, 36(2), 417.

Attanasio C, et al. (2025) Morphological phenotyping of the aging cochlea in inbred C57BL/6N and outbred CD1 mouse strains. *Aging cell*, 24(1), e14362.

Cornejo-Sanchez DM, et al. (2025) Mendelian non-syndromic and syndromic hearing loss genes contribute to presbycusis. *European journal of human genetics : EJHG*, 33(6), 758.