Resource Summary Report

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University of Wisconsin-Madison Advanced Genome Editing Laboratory

RRID:SCR_021070
Type: Tool

Proper Citation

University of Wisconsin-Madison Advanced Genome Editing Laboratory (RRID:SCR_021070)

Resource Information

URL: https://biotech.wisc.edu/age/

Proper Citation: University of Wisconsin-Madison Advanced Genome Editing Laboratory (RRID:SCR_021070)

Description: Provides services and expertise to unlock genome editing tools to advance your research. Routinely generates new genome edited models, particularly mouse, rats, swine, and cell lines, as well as supports in vivo editing, novel preclinical therapeutic strategies, pooled lentiCRISPR screening, and other applications.

Abbreviations: AGEL

Synonyms: University of Wisconsin Madison UW-Genome Editing & Animal Models, UW-Genome Editing & Animal Models, University of Wisconsin Madison Advanced Genome Editing Laboratory, UW Biotechnology Center Advanced Genome Editing Laboratory, Genome Editing and Animal Models Core, University of Wisconsin Madison Genome Editing Core Facility, Transgenic Animal Facility

Resource Type: access service resource, core facility, service resource

Keywords: USEDit, generating genome edited models, mouse, rats, swine, ABRF, ABRF, CRISPR, gene editing, genome editing, lentiCRISPR

Funding: NCI P30 CA014520

Resource Name: University of Wisconsin-Madison Advanced Genome Editing Laboratory

Resource ID: SCR_021070

Alternate IDs: ABRF_1148

Alternate URLs: https://coremarketplace.org/?FacilityID=1148

Record Creation Time: 20220129T080353+0000

Record Last Update: 20260905T133420+0000

Ratings and Alerts

No rating or validation information has been found for University of Wisconsin-Madison Advanced Genome Editing Laboratory.

No alerts have been found for University of Wisconsin-Madison Advanced Genome Editing Laboratory.

Data and Source Information

Source: SciCrunch Registry

Usage and Citation Metrics

We found 14 mentions in open access literature.

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

Na B, et al. (2026) Development of a Genetically Engineered Porcine Model of Rhabdoid Tumor Predisposition Syndrome Type 1 (RTPS-1). Cancers, 18(12).

Kemna EI, et al. (2026) Evolution-guided engineering of an ancient nitrogenase interface enhances enzyme activity and stability. bioRxiv : the preprint server for biology.

Heaton AR, et al. (2026) Divergent role of CD8 T cells with distinct metabolic phenotypes during curative radio-immunotherapy in hot versus cold tumors. bioRxiv : the preprint server for biology.

Bernau K, et al. (2026) Development and characterization of an inducible Tensin1 deficient transgenic murine model. Scientific reports, 16(1).

Gowda AM, et al. (2026) Loss of phospholamban phosphorylation blunts Ca2+ handling during the cellular adrenergic response. Journal of molecular and cellular cardiology, 216, 20.

Yamamoto S, et al. (2026) Deficiency of the collagen endocytic receptor MRC2 accelerates mouse lung fibroblast proliferation. American journal of respiratory cell and molecular biology, 74(6), 768.

Veith AC, et al. (2025) The draft genome of the Wisconsin Miniature SwineTM, a valuable biomedical research tool. G3 (Bethesda, Md.), 15(6).

Zheng J, et al. (2025) Preventing the phosphorylation of RyR2 at canonical sites reduces Ca2+ leak and promotes arrhythmia by reactivating the INa current. Nature cardiovascular

research, 4(8), 976.

Veith AC, et al. (2025) Retrospective analysis and decentralized distribution to improve the lifecycle of Ah receptor research assets. Biochemical pharmacology, 236, 116874.

Braun MM, et al. (2024) Ca+2 and N??-lysine acetylation regulate the CALR-ATG9A interaction in the lumen of the endoplasmic reticulum. Scientific reports, 14(1), 25532.

English LA, et al. (2024) F-BAR proteins CIP4 and FBP17 function in cortical neuron radial migration and process outgrowth. bioRxiv : the preprint server for biology.

Zheng J, et al. (2024) Ablation of three major phospho-sites in RyR2 preserves the global adrenergic response but creates an arrhythmogenic substrate. bioRxiv : the preprint server for biology.

McLean DT, et al. (2023) Single-cell RNA sequencing of neurofibromas reveals a tumor microenvironment favorable for neural regeneration and immune suppression in a neurofibromatosis type 1 porcine model. Frontiers in oncology, 13, 1253659.

Rubinstein CD, et al. (2021) Assessment of Mosaicism and Detection of Cryptic Alleles in CRISPR/Cas9-Engineered Neurofibromatosis Type 1 and TP53 Mutant Porcine Models Reveals Overlooked Challenges in Precision Modeling of Human Diseases. Frontiers in genetics, 12, 721045.