lling Code 4167-05-P

**DEPARTMENT OF HEALTH AND HUMAN SERVICES**

**National Institutes of Health**

**Government Owned Invention Available for License: Generating Conditional and Reverse Conditional Loss-of-Function Alleles in Mouse Casq2**

**AGENCY:** National Institutes of Health, HHS.

**ACTION:** Notice.

**SUMMARY:** The *Eunice Kennedy Shriver* National Institute of Child Health and Human Development (NICHD) is seeking potential licensees interested in further developing or utilizing these Casq2 mouse strains. As a research tool, patent protection is not being pursued for this technology. More information to access these strains can be found here: https://www.jax.org/strain/036291 and https://www.jax.org/strain/036290.

**FOR FURTHER INFORMATION CONTACT:** Inquiries related to this license opportunity should be directed to: Heather Gunas JD, MPH, Senior Technology Transfer Manager, NCI, Technology Transfer Center, Email: *gunash@mail.nih.gov* or Phone: 240-276-5530.

**SUPPLEMENTARY INFORMATION:** Cardiac calsequestrin (Casq2) plays an essential role in maintaining cardiac $Ca^{2+}$ homeostasis. Human *CASQ2* mutations are associated with catecholaminergic polymorphic ventricular tachycardia (CPVT), a rare familial arrhythmogenic disorder within a group of diseases characterized as Sudden Arrhythmic Death. The inventors have generated Casq2Flox and Casq2RevFlox mouse strains that model CPVT. The two novel strains successfully phenocopy aspects of CPVT, including stress-induced arrhythmias and reduced basal heart rates. The strains allow investigators to determine the importance of Casq2 gene function in specific tissues and at specific developmental time points. They also allow investigators to determine the efficacy of gene therapy and to address key mechanism questions. The materials are validated and fully functional.

This Notice is in accordance with 37 C.F.R. § 404.4 Authority to grant licenses.

**NIH Reference Number**: E-128-2024.

**Related Technologies**: N/A.

**Product Type**: Research Material/Tool.

**Therapeutic Area(s)**: Rare/Neglected Disease.

**Development Stage**: Validated and fully functional.

**Publications**:

- Knollmann BC, et al. *Casq2* deletion causes sarcoplasmic reticulum volume increase, premature $Ca^{2+}$ release, and catecholaminergic polymorphic ventricular tachycardia. (PMID 16932808).
- Flores DJ, et al. Conditional ablation and conditional rescue models for Casq2 elucidate the role of development and of cell-type specific expression of Casq2 in the CPVT2 phenotype. (PMID 29452352).
- Blackwell DJ, et al. The Purkinje–myocardial junction is the anatomic origin of ventricular arrhythmia in CPVT.

  (PMID 34990403).

**Patents**: N/A.

**Potential Commercial Applications**:

- Study of Casq2 function.
- Study of calcium storage in cardiac muscle and CPVT.
- Determining the efficacy of gene therapy for CPVT pies.

**Competitive Advantages**:

- Only available conditional and reverse conditional loss-of-function alleles in mouse Casq2.
- Allows the study of Casq2 gene function in specific tissues and at specific developmental points.

**Collaboration Opportunity**: NICHD seeks licensing for further developing or utilizing these Casq2 mouse strains.

Dated: July 6, 2026.

**Richard U. Rodriguez,**

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