



HERG CDNA with Native PolyA Tail

WARF: P05401US

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The Wisconsin Alumni Research Foundation (WARF) is seeking commercial partners interested in developing the full-length cDNA of HERG1a.

Overview

The human *ERG* gene encodes a potassium channel that is expressed in the heart. Mutations in the HERG channel are a common cause of long QT syndrome, a disorder associated with delayed cardiac repolarization, prolonged electrocardiographic QT intervals, and the development of ventricular arrhythmias and sudden death. Because unintended block of HERG channel activity by drugs can also lead to potentially fatal arrhythmias, HERG expression systems are a useful early screen in drug development.

The Invention

UW-Madison researchers have developed the full-length cDNA of HERG1a. This cDNA has been cloned and expressed in *Xenopus* oocytes, human embryonic kidney cells (HEK293) and Chinese hamster ovary cells. It is similar to a previously cloned cDNA, except that a polyadenylation signal has been removed from its vector sequence. The removal of this sequence does not affect HERG expression levels in heterologous systems.

Applications

- Testing lead compounds and drugs for their potential to block activity of the HERG-1 cardiac potassium channel

Key Benefits

- May be used to develop methods for genetic screening of individuals carrying mutations in *HERG* genes
- May enable development of gene therapies for individuals with certain cardiac arrhythmias associated with dysfunctional HERG proteins

Additional Information

For More Information About the Inventors

- [Gail Robertson](#)

Tech Fields

- [Drug Discovery & Development : Preclinical testing](#)
- [Research Tools : Cell lines](#)

For current licensing status, please contact Jennifer Gottwald at jennifer@warf.org or 608-960-9854

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