PrecisION® hKir6.2/SUR2A Recombinant Stable Cell Line

Catalog Number CYL3099 **Lot Number** See Vial

Contents 2 Vials, 2 x 10^6 to 4 x 10^6 in 1 mL

Background Information

Molecular cloning has identified the channel as a heteromer comprised of four pore-forming subunits, termed Kir 6.2, and four auxiliary sulphonylurea receptor subunits, Sur2A. The channel is gated by intracellular ATP and NDP while the Sur2A is regulated by both agonists, such as the potassium channel opener pinacidil, and antagonists, such as glibenclamide. Additional information can be found on page 2.

Product Information

Description Recombinant HEK 293 cell line expressing the human Kir6.2 and SUR2A ion channel subunits

Family Potassium, Inward Rectifier

Target Kir6.2/SUR2A

	Target Protein	Accession Number
1	Kir6.2	NM_000525.3
2	SUR2A	NM_005691.2
3	N/A	N/A
4	N/A	N/A

Species Human

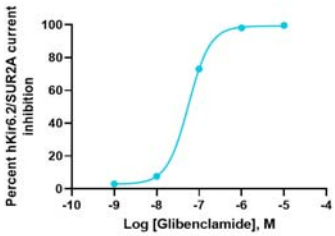
Host Cell Type HEK 293

Application Electrophysiology assay (conventional and automated patch clamp platforms)

Storage Vials are to be stored in vapor phase of liquid nitrogen

Functional Performance

HEK293 cells expressing hnKir6.2/SUR2A were characterized in terms of their pharmacological and biophysical properties using whole-cell patch clamp techniques.



Electrophysiology Method QPatch

Reference Agonist Pinacidil

Reference Antagonist Glibenclamide

Antagonist IC_{50} (μM) 0.06

Passage Stability

This cell line has been confirmed to be stable through at least 12 passages with no significant drop in assay window or change in pharmacology.

Mycoplasma Testing

This lot was tested and found to be free of mycoplasma contamination. Data available upon request.

Notes

Additional functional (pharmacological and electrophysiological) validation on multiple platforms is available upon request.

Additional Ligand Information

Control Compound	Glibenclamide
Vendor Name :	Tocris
Vendor Catalog No.	0911

Additional Background Information

In vivo the cardiac KATP channel is thought to be cardioprotective during periods of ischemic stress, as activation of the channel hyperpolarizes the cell, reducing calcium channel activity, leading to a shortening of the action potential duration.

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