

## PrecisION<sup>®</sup> hKv4.3/hKChIP2 Recombinant Stable Cell Line

**Catalog Number** CYL3069

**Lot Number**

See Vial

**Contents** 2 Vials, 2 x 10<sup>6</sup> to 4 x 10<sup>6</sup> in 1 mL

### Background Information

Kv4 subunits are the main pore-forming proteins responsible for the fast transient outward currents observed in the CNS (rat brain), where they have been described as 'A' currents (IA) (Serodio and Rudy, 1998) and in mammalian heart, where they are known as Ca<sup>2+</sup>-independent fast outward currents (I<sub>to,f</sub>) (Nerbonne, 2000). Although in human heart the predominant subunit responsible for I<sub>to,f</sub> is Kv4.3, in order to reconstitute all the properties of the native current co-expression with an auxiliary protein KChIP is required. Additional information can be found on page 2.

### Product Information

**Description** Recombinant HEK 293 cell line co-expressing the human Kv4.3 (voltage-gated potassium channel) and the human Kv channel interacting protein KChIP2

**Family** Potassium, Voltage-Gated

**Target** Kv4.3/KChIP2

	Target Protein	Accession Number
1	Kv4.3	NM_004980
2	KChIP2	NM_173192
3	N/A	N/A
4	N/A	N/A

**Species** Human

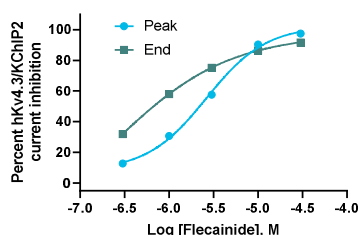
**Host Cell Type** HEK 293

**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 hKv4.3/hKChIP2 were characterized in terms of their pharmacological and biophysical properties using whole-cell patch clamp techniques.



**Electrophysiology Method** QPatch

**Reference Agonist**

**Reference Antagonist** Flecainide

**Antagonist IC<sub>50</sub> (μM)** 0.23

### 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** Flecainide

**Vendor Name :** Sigma-Aldrich

**Vendor Catalog No.** F6777

### Additional Background Information

These small molecular weight Ca<sup>2+</sup>-binding proteins typically increase cell surface expression of the channel complex, accelerate the rate of decay of the current at depolarized potentials, and increase the rate of recovery from inactivation at hyperpolarized potentials, i.e. the kinetics then more closely resembles native I<sub>to</sub> than if Kv4.3 subunits were expressed alone (Wang et al., 2002 and Deschenes et al., 2002). The predominant KChIP found in heart is KChIP2 (Rosati et al., 2001 and Deschenes et al., 2002) and can exist as various isoforms (Decher et al., 2004). The isoform co-expressed with Kv4.3 subunits in this cell line is KChIP2b (Decher et al., 2004) but will simply be referred to as KChIP2 in this document. In the heart, I<sub>to,f</sub> is primarily responsible for the 'notch' during phase 1 of the cardiac action potential. Since it is an early repolarizing current it is of crucial importance in shaping the final cardiac action potential waveform (Nerbonne, 2000). In the human heart the density of I<sub>to,f</sub> is highest in the epicardium and lowest in the endocardium; regional differences controlled by the level of KChIP2 expression (Rosati et al., 2001). These regional differences significantly contribute to the transmural voltage gradient across the myocardial wall, necessary for normal ventricular activity (Sanguinetti, 2002). Abolishing KChIP2 expression has been shown in mice to markedly affect this gradient with the consequence of

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