PrecisION® hCFTR Recombinant Stable Cell Line

Catalog Number CYL3088 **Lot Number** See Vial

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

Background Information

The cystic fibrosis transmembrane conductance regulator (CFTR) is an ion channel that once phosphorylated by PKA is gated by ATP binding and hydrolysis (Csanády et al, 2010). When open, this channel allows chloride ion flux. The CFTR gene was cloned by Drumm et al (1990) and has been of interest as a therapeutic target since the first observation that a nucleotide substitution found in patients with cystic fibrosis encodes for a trafficking-deficient mutant channel termed ΔF508. Additional information can be found on page 2.

Product Information

Description Recombinant HEK 293 cell line expressing the human CFTR ion channel

Family Chloride, Ligand-Gated

Target CFTR

	Target Protein	Accession Number
1	CFTR	NM_000492
2	N/A	N/A
3	N/A	N/A
4	N/A	N/A

Species Human

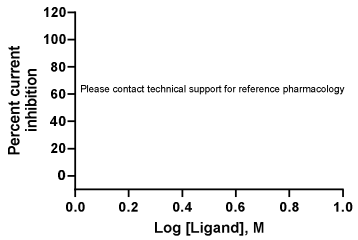
Host Cell Type HEK 293

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 hCFTR were characterized in terms of their pharmacological and biophysical properties using whole-cell patch clamp techniques.



Electrophysiology Method MPC

Reference Agonist

Reference Antagonist Glibenclamide

Antagonist IC_{50} (μM)

Passage Stability

Please contact technical support.

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

To date, at least 136 allelic variants of CFTR have been associated with patients that have cystic fibrosis. The CFTR ion channel is expressed in the respiratory tract, sweat glands, intestinal tissue, and pancreas, and decreased surface expression or function of mutant channels in these tissues is consistent with disease symptoms. To date, specific activators (Mills et al., 2010) and trafficking modulators that normalize surface expression and function toward wild-type levels have shown promise as therapies for cystic fibrosis (Brown et al 1996; Loo et al, 2009, Mills et al., 2010).

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