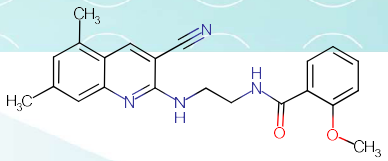
SL-72. Cystic fibrosis

ASINEX
Signature Libraries

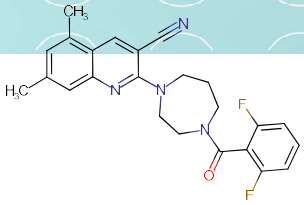
Dysfunctional cystic fibrosis transmembrane conductance regulator (CFTR) protein is responsible for the onset and development of a cystic fibrosis disease that causes persistent lung infection and limits the ability to breathe over time. Several CFTR-modulating therapies have been proposed targeting specific mutations of the CF gene. Two drugs, ivacaftorand lumacaftor/ivacaftor, have been approved by the FDA [1].

One of the most common mutations is ΔF508-CFTR which leads to the CFTR loss of function. A group headed by Dr. Kurth published a series of cyanoquinolines, small molecules that act as a corrector of ΔF508-CFTR, targeting the plasma membrane and potentiator of ΔF508-CFTR-mediated chloride channel activity. Compounds with potentiator-only, corrector-only, and dual potentiator–corrector activities were disclosed [2].

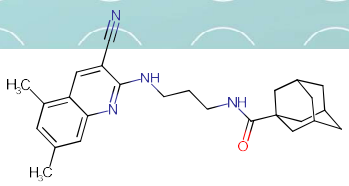
80 analogs of the reported hits were included in this library.



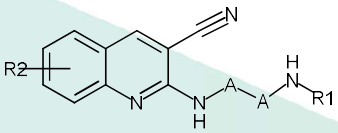
LAS 06540480



LAS 06541401



LAS 06540921



Hit Series [2]

Signature Library 72

Formats	Supplementary Information
80 compounds per plate 0.1 mg; 1 mg; 2 mg dry film/powder 0.1 µmol; 1 µmolDMSO solutions	SL#72_CFTR.sdf

References:

1. www.cff.org
2. *J. Med. Chem.*, 2012, 55 (3), pp 1242–1251 doi: 10.1021/jm201372q

Contact us:

USA:	+1 336 721 1617	mparisi@asinex.com
Japan:	+81-80-3401-9097	sota@asinex.com
Europe/Global:		lsadovenko@asinex.com