产品名称：**Naquotinib**
产品别名：**ASP8273**

生物活性：					
Description	Naquotinib (ASP8273) is an orally available, mutant-selective and irreversible EGFR inhibitor; with IC50s of 8-33 nM toward EGFR mutants and 230 nM for EGFR.				
IC_{50} & Target	EGFR	$EGFR^{T790M}$	$EGFR^{L858R/T790M}$	$EGFR^{L858R}$	$EGFR^{Exon\ 19\ deletion/T790M}$
	230 nM (IC_{50})				
In Vitro	In assays using endogenously EGFR-dependent cells, Naquotinib inhibits the growth of PC-9(del ex19), HCC827(del ex19), NCI-H1975(del ex19/T790M) and PC-9ER(del ex19/T790M) with IC50s of 8-33 nM[1]. Naquotinib selectively inhibits phosphorylation of EGFR and its down-stream signal pathway, ERK and Akt from 10nM in HCC827 and NCI-H1975 while inhibitory effects are only detected at 1000nM in A431.In NCI-H1650 (del ex19), Naquotinib inhibits cell growth with an IC50 value of 70nM while other EGFR-TKIs are only partially effective[2].				
In Vivo	Oral Naquotinib treatment dose dependently induces tumor regression in NCI-H1975 (L858R/T790M), HCC827 (del ex19) and PC-9 (del ex19) xenograft models. Dosing schedules does not affect the efficacy of Naquotinib. In an NCI-H1975 xenograft model, complete regression of tumor is achieved after 14-days of Naquotinib treatment. Complete regression is maintained in 50% of mice more than 85 days after cessation of Naquotinib treatment[2].				
References	[1]. Sakagami H, et al. ASP8273, a novel mutant-selective irreversible EGFR inhibitor, inhibits growth of non-small cell lung cancer (NSCLC) cells with EGFR activating and T790M resistance mutations. [abstract]. In: Proceedings of the 105th Annual Meeting of the American Association for Cancer Research; 2014 Apr 5-9; San Diego, CA. Philadelphia (PA): AACR; Cancer Res 2014;74(19 Suppl):Abstract nr 1728. doi:10.1158/1538-7445.AM2014-1728 [2]. Konagai S, et al. ASP8273 selectively inhibits mutant EGFR signal pathway and induces tumor shrinkage in EGFR mutated tumor models. [abstract]. In: Proceedings of the 106th Annual Meeting of the American Association for Cancer Research; 2015 Apr 18-22; Philadelphia, PA. Philadelphia (PA): AACR; Cancer Res 2015;75(15 Suppl):Abstract nr 2586. doi:10.1158/1538-7445.AM2015-2586				