

# Capivasertib

Catalog No: tcsc1284



## Available Sizes

**Size:** 5mg

**Size:** 10mg

**Size:** 50mg

**Size:** 100mg



## Specifications

**CAS No:**

1143532-39-1

**Formula:**

$C_{21}H_{25}ClN_6O_2$

**Pathway:**

PI3K/Akt/mTOR;Autophagy

**Target:**

Akt;Autophagy

**Purity / Grade:**

>98%

**Solubility:**

DMSO :  $\geq 21.5$  mg/mL (50.13 mM)

**Alternative Names:**

AZD5363

**Observed Molecular Weight:**

428.92

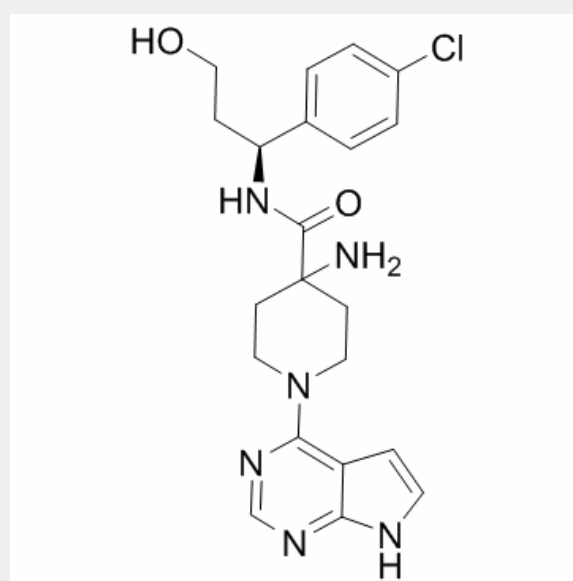
## Product Description

Capivasertib (AZD5363) is a potent **pan-AKT** kinase inhibitor with **IC<sub>50</sub>** of 3, 7 and 7 nM for **Akt1, Akt2** and **Akt3**, respectively.

IC50 & Target: IC50: 3 nM (Akt1), 7 nM (Akt2), 7 nM (Akt3), 6 nM (p70S6K), 7 nM (PKA), 60 nM (ROCK2)<sup>[1]</sup>

**In Vitro:** Capivasertib, a novel pyrrolopyrimidine-derived compound, inhibits all AKT isoforms with a potency of 10 nM or less and inhibits phosphorylation of AKT substrates in cells with a potency of approximately 0.3 to 0.8  $\mu$ M. Capivasertib inhibits phosphorylation of these substrates with an IC<sub>50</sub> value of 0.06 to 0.76  $\mu$ M in the 3 cell lines. Capivasertib effectively inhibits phosphorylation of S6 and 4E-BP1 in these cell lines, whereas it increases phosphorylation of AKT at both ser<sup>473</sup> and thr<sup>308</sup>. In BT474c cells, Capivasertib induces FOXO3a nuclear translocation with EC<sub>50</sub> value of 0.69  $\mu$ M; a concentration of 3  $\mu$ M is sufficient to almost completely localize FOXO3a to the nucleus. AZD5363Capivasertibinhibitor MK-2206 is much less active (IC<sub>50</sub>>30  $\mu$ M)<sup>[1]</sup>.

**In Vivo:** Oral dosing of Capivasertib (AZD5363) to nude mice causes dose- and time-dependent reduction of PRAS40, GSK3 $\beta$ , and S6 phosphorylation in BT474c xenografts (PRAS40 phosphorylation EC<sub>50</sub> ~0.1  $\mu$ M total plasma exposure), reversible increases in blood glucose concentrations, and dose-dependent decreases in 2[<sup>18</sup>F]fluoro-2-deoxy-D-glucose (<sup>18</sup>F-FDG) uptake in U87-MG xenografts. Chronic oral dosing of Capivasertib caused dose-dependent growth inhibition of xenografts derived from various tumor types, including HER2<sup>+</sup> breast cancer models that are resistant to trastuzumab. Capivasertib also significantly enhances the antitumor activity of docetaxel, lapatinib, and trastuzumab in breast cancer xenografts<sup>[1]</sup>.



All products are for RESEARCH USE ONLY. Not for diagnostic & therapeutic purposes!