

# Dofequidar

## Catalog No: tcsc2042



### Available Sizes

**Size:** 5mg

**Size:** 10mg

**Size:** 50mg

**Size:** 100mg



### Specifications

**CAS No:**

129716-58-1

**Formula:**

$C_{30}H_{31}N_3O_3$

**Pathway:**

Membrane Transporter/Ion Channel

**Target:**

P-glycoprotein

**Purity / Grade:**

>98%

**Solubility:**

10 mM in DMSO

**Observed Molecular Weight:**

481.59

## Product Description

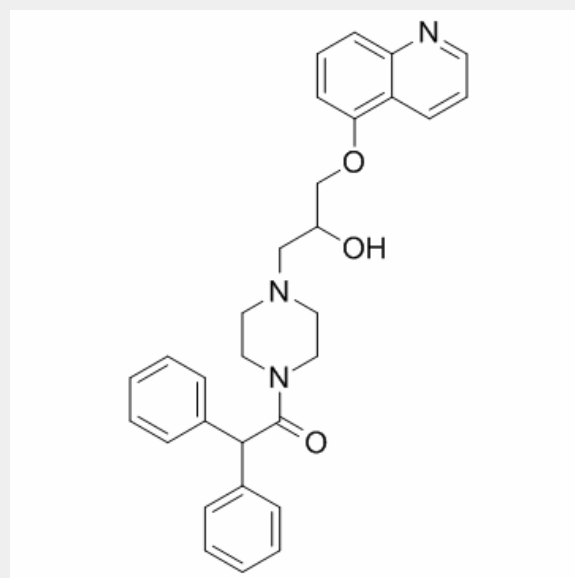
Dofequidar(MS-209) is a novel quinoline compound, which can reverse P-glycoprotein (P-gp)-mediated MDR.

IC50 value:

Target: P-gp

in vitro: MS-209 at 3 microM effectively overcame docetaxel resistance in MDR cancer cells, and this concentration was achieved in blood plasma for > 7 h without serious toxicity [1]. MS-209 restored chemosensitivity of SBC-3 / ADM cells to VP-16, ADM, and VCR in a dose-dependent manner in vitro [2]. MS-209 strongly reversed drug resistance to adriamycin (ADM) and vincristine (VCR) in acquired MDR tumor cell lines, 2780AD and KB-C1. In addition, MS-209 enhanced the cytotoxic effect of ADM and VCR on various human and murine cell lines. Particularly in 4-1St cells, which are extremely resistant to ADM and VCR, MS-209 at a concentration of 3 microM enhanced the cytotoxicity of ADM and VCR, 88- and 350-fold, respectively [3].

in vivo: Treatment with docetaxel alone at the maximal tolerated dose (MTD) showed an apparent antitumor activity to an intrinsically resistant HCT-15 tumor xenograft, and MS-209 additionally potentiated the antitumor activity of docetaxel. Against a MCF-7/ADM tumor xenograft expressing larger amounts of P-gp, docetaxel alone at the MTD showed no antitumor activity, whereas the MTD of docetaxel combined with MS-209 greatly reduced MCF-7/ADM tumor growth [1]. Intravenous injection with SBC-3 or SBC-3 / ADM cells produced metastatic colonies in the liver, kidneys and lymph nodes in natural killer (NK) cell-depleted severe combined immunodeficiency (SCID) mice, though SBC-3 / ADM cells more rapidly produced metastases than did SBC-3 cells. Treatment with VP-16 and ADM reduced metastasis formation by SBC-3 cells, whereas the same treatment did not affect metastasis by SBC-3 / ADM cells. Although MS-209 alone had no effect on metastasis by SBC-3 or SBC-3 / ADM cells, combined use of MS-209 with VP-16 or ADM resulted in marked inhibition of metastasis formation by SBC-3 / ADM cells to multiple organs [2]. MS-209 administered orally, together with ADM, enhanced the antitumor activity of ADM on Colon 26 and 4-1St tumors implanted subcutaneously (SC) in mice; the antitumor effect of ADM plus MS-209 was higher than that of ADM alone at the maximum tolerated dose (MTD) [3].



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