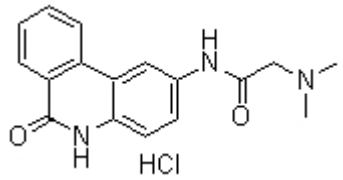
Product Introduction

PJ34 HCl

PJ34 HCl is the hydrochloride salt of PJ34, which is a PARP inhibitor with EC50 of 20 nM and is equally potent to PARP1/2.

Technical Data:

Molecular Weight (MW):	331.8	
Formula:	C17H17N3O2.HCl	
Solubility (25°C) * <1 mg/ml means slightly soluble or insoluble:	DMSO 66 mg/mL	
	Water 66 mg/mL	
	Ethanol <1 mg/mL	
Purity:	>98%	
Storage:	3 years -20℃Powder	
	6 months-80℃in DMSO	
CAS No.:	344458-15-7	

Biological Activity

PJ34 is a potent, phenanthridinone PARS inhibitor, which is approximately 10,000 times more potent than the prototypical PARS inhibitor 3-aminobenzamide. PJ34 inhibited peroxynitrite-induced cell necrosis with EC50 of 20 nM. PJ34 provides cardioprotection by decreasing myocardial infarct size and enhancing postischemic regional and global functional recovery. [1]

PJ34 suppresses the development of clinical signs of EAE in MBP-immunized PLSJL mice. PJ34 exerted

therapeutic effects at the onset of EAE that are associated with reduced CNS inflammation and the maintenance of neurovascular integrity. PJ34 partially inhibits the expression of TNF-α and ICAM-1 in the Spinal Cord Tissues of MBP-Immunized Mice.[2] PJ34 provides significant, dose-dependent, anti-inflammatory effects in a variety of local inflammation models. PJ34 dose-dependently suppresses neutrophil infiltration and nitric oxide (but not KC and IL-1β) production in peritonitis. In a model of systemic endotoxemia, PJ34 pretreatment significantly reduces plasma levels of TNF-α, IL-1β and nitrite/nitrate (breakdown products of nitric oxide) production. PJ34 treatment (oral gavage) induces a significant suppression of the inflammatory response in dextran sulfate colitis, multiple low dose streptozotocin diabetes and cyclophosphamide-accelerated autoimmune diabetes in the non-obese diabetic mice, and reduces the degree of mononuclear cell infiltration into the iris in an endotoxin-induced uveitis model. [3]

Water-soluble PARP1/2 inhibitor with >10,000-fold potentcy vs. 3-aminobenzamide (prototypical PARP inhibitor). Potential uses in cardiovascular diseases (stroke, cerebral ischemia, & myocardial ischemia).

References

[1] Garcia Soriano F, et al. Nat Med, 2001, 7(1), 108-113.
[2] Scott GS, et al. J Pharmacol Exp Ther, 2004, 310(3), 1053-1061.
[3] Mabley JG, et al. Inflamm Res, 2001, 50(11), 561-569.

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