



**AG-490** 

**Catalog No: tcsc0108** 



## **Available Sizes**

Size: 10mg

Size: 50mg

Size: 100mg

Size: 200mg



# **Specifications**

CAS No:

133550-30-8

Formula:

 $C_{17}^{H}_{14}^{N}_{2}^{O}_{3}$ 

### **Pathway:**

JAK/STAT Signaling; Protein Tyrosine Kinase/RTK; Autophagy; JAK/STAT Signaling; Stem Cell/Wnt

**Target:** 

EGFR;EGFR;Autophagy;STAT;STAT

**Storage Buffer:** 

5% DMSO+40% PEG300+5% Tween80+50% 6.25mg/ml

**Purity / Grade:** 

>98%

**Solubility:** 

DMSO 58.0 mg/mL (197.1 mM) Ethanol 6.0 mg/mL (20.4 mM)

Water Insoluble

## **Storage Instruction:**





Powder: -20°C for 3 years In Solvent: -80°C 12 months

#### **Alternative Names:**

Tyrphostin AG 490

### **Observed Molecular Weight:**

294.3

### **Notes**

Mechanism: AG-490 blocks protein tyrosine kinases by binding to the substrate-binding site.

# **Product Description**

AG-490 is an tyrosine kinase inhibitor, inhibits EGFR and Stat-3.

IC50 & Target: EGFR and Stat-3<sup>[1]</sup>

In Vitro: AG490 inhibits the activation of Stat-3 by selectively blocking JAK2. AG490 is used to selectively inhibit JAK/Stat-3 activation. At a dose of 10  $\mu$ M, Stat-3 phosphorylation is decreased by >95% and cell viability is maintained. AG490 at a dose of 10  $\mu$ M results in >95% decrease in pStat-3 in EGF-stimulated A431 cells with no effect on Stat-3 mass<sup>[1]</sup>. AG-490 is a potent inhibitor of the JAK3/STAT, JAK3/AP-1, and JAK3/MAPK pathways and their cellular consequences. AG-490 abolishes IL-2-inducible [ $^3$ H]thymidine incorporation in a dose-dependent manner, displaying an IC<sub>50</sub> of 25  $\mu$ M. AG-490 potently inhibits IL-2-mediated proliferation in T cells, results distinct from previous studies that showed this agent induced apoptosis in ALL cells while exerting apparently no effects on the growth of mitogen-stimulated normal T cells<sup>[2]</sup>.

In Vivo: AG490 significantly inhibits the development of type 1 diabetes (T1D) (p=0.02, p=0.005; at two different time points). Monotherapy of newly diagnosed diabetic NOD mice with AG490 (1 mg/mouse) markedly results in disease remission in treated animals (n=23) in comparision to the absolute inability (0%; 0/10, p=0.003, Log-rank test) of DMSO and sustained eugluycemia is maintained for several months following drug withdrawal<sup>[3]</sup>. AG490 (1-10  $\mu$ g) significantly attenuates  $\Lambda$ -carrageenan-induced thermal hyperalgesia in a dose-dependent manner. AG490 also reduces mechanical hyperalgesia<sup>[4]</sup>.



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