



## Hesperadin

**Catalog No: tcsc0213** 

Available Sizes
Size: 5mg
Size: 10mg
Size: 50mg
Size: 100mg
Specifications
CAS No: 22513-13-1
Formula: C <sub>29</sub> H <sub>32</sub> N <sub>4</sub> O <sub>3</sub> S
Pathway: Cell Cycle/DNA Damage;Epigenetics;Autophagy
Target: Aurora Kinase;Aurora Kinase;Autophagy
Purity / Grade: -98%
Solubility: .0 mM in DMSO

## **Observed Molecular Weight:**

516.65

## **Product Description**

Hesperadin is an ATP-competitive inhibitor of **aurora B** kinase with an  ${
m IC}_{50}$  of 250 nM.



IC50 & Target: IC50: 250 nM (Aurora B)[1]

In Vitro: Hesperadin also inhibits other kinases such as AMPK, Lck, MKK1, MAPKAP-K1, CHK1, and PHK at 1 μM drug concentration. Hesperadin causes polyploidy in HeLa cells. Hesperadin-treated HeLa cells show alignment and segregation defects, but sister chromatid separation is intact. Hesperadin causes defects in mitosis and cytokinesis. Hesperadin inhibits Aurora B. Immunofluorescence microscopy reveals that Hesperadin-treated cells in which chromosomes are stretched toward opposite poles, i.e., which have entered anaphase, fail to assemble a central spindle and to properly localize the human centralspindlin subunits CYK-4 and MKLP1<sup>[1]</sup>. Hesperadin inhibits multiple human clinical isolates of influenza A and B viruses with single to submicromolar efficacy, including oseltamivir-resistant strains. Mechanistic studies reveal that hesperadin inhibits the early stage of viral replication by delaying the nuclear entry of viral ribonucleoprotein complex, thereby inhibiting viral RNA transcription and translation as well as viral protein synthesis<sup>[2]</sup>. Hesperadin inhibits cell cell proliferation due to appearance of multiple mitotic defects caused by Aurora B activity reduction and elimination of checkpoint proteins--such as hBUBR1 and CENP-E--from kinetochores of mitotic chromosomes<sup>[3]</sup>.

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