# PRODUCT INFORMATION

## CD532

*Item No. 17054*

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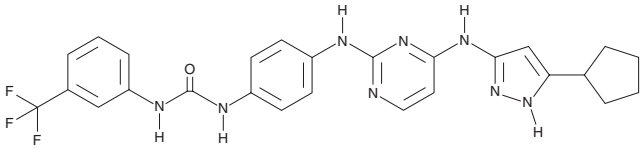
| **CAS Registry No.:** | 1639009-81-6 |
| **Formal Name:** | N-[4-[[4-[(5-cyclopentyl-1H-pyrazol-3-yl)amino]-2-pyrimidinyl]amino]phenyl]-N′-[3-(trifluoromethyl)phenyl]-urea |
| **MF:** | $C_{26}H_{25}F_3N_8O$ |
| **FW:** | 522.5 |
| **Purity:** | ≥98% |
| **UV/Vis.:** | $\lambda_{max}$: 274 nm |
| **Supplied as:** | A solid |
| **Storage:** | -20°C |
| **Stability:** | ≥4 years |



*Information represents the product specifications. Batch specific analytical results are provided on each certificate of analysis.*

### Laboratory Procedures

CD532 is supplied as a solid. A stock solution may be made by dissolving the CD532 in the solvent of choice, which should be purged with an inert gas. CD532 is soluble in the organic solvent ethanol.

### Description

CD532 is an inhibitor of Aurora A kinase activity ($IC_{50}$ = 48 nM) and the protein-protein interaction between N-Myc and Aurora A kinase.[1] It also inhibits several cyclin-dependent kinases (CDKs), FGFRs, MEKs, and PDGFRs, as well as FLT3, KIT, and RET at 10 µM.[2] CD532 induces degradation of N-Myc in SK-N-BE(2) neuroblastoma cells ($EC_{50}$ = 223 nM).[1] It prevents S-phase entry in SK-N-BE(2) cells when used at a concentration of 1 µM.[1] CD532 (25 mg/kg) reduces tumor growth and increases survival in a *MYCN*-amplified SMS-KCN neuroblastoma mouse xenograft model.

### References


1. Gustafson, W.C., Meyerowitz, J.G., Nekritz, E.A., *et al*. Drugging MYCN through an allosteric transition in Aurora kinase A. *Cancer Cell* **26(3)**, 414-427 (2014).
2. Lee, J.K., Phillips, J.W., Smith, B.A., *et al*. N-Myc drives neuroendocrine prostate cancer initiated from human prostate epithelial cells. *Cancer Cell* **29(4)**, 536-547 (2016).





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