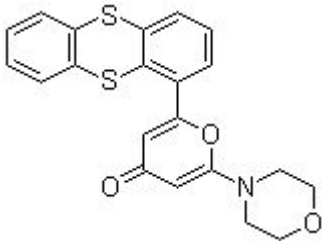


Product Introduction

KU-55933 (ATM Kinase Inhibitor)

KU-55933 is a potent and specific ATM inhibitor with IC_{50}/K_i of 12.9 nM/2.2 nM, and is highly selective for ATM as compared to DNA-PK, PI3K/PI4K, ATR and mTOR.

Technical Data:

Molecular Weight (MW):	395.49	
Formula:	C ₂₁ H ₁₇ N ₃ O ₃ S ₂	
Solubility (25°C)	DMSO 33 mg/mL	
* <1 mg/ml means slightly soluble or insoluble:	Water <1 mg/mL	
	Ethanol <1 mg/mL	
Purity:	>98%	
Storage:	3 years -20°C Powder	
	6 months -80°C in DMSO	
CAS No.:	587871-26-9	

Biological Activity

KU-55933 inhibits DNA-PK and PI3K with IC_{50} of 2.5 μ M and 16.6 μ M, respectively. Besides, KU-55933 also prevents the activity of mTOR with IC_{50} of 9.3 μ M. KU-55933 is active at the cellular level in ablating a well-characterized ATM-dependent phosphorylation event. KU-55933 has a dose-dependent effect in inhibiting this ATM-dependent phosphorylation event with IC_{50} of 300 nM. KU-58050 does not prevent the ATM-dependent phosphorylation of p53 serine 15 until a dose of 30 μ M. Addition of KU-55933 has no appreciable effects on UV-induced phosphorylation of H2AX on serine 139, NBS1 on serine 343, CHK1 on

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serine 345, and SMC1 on serine 966. In stark contrast to the UV responses, KU-55933 ablates the ionizing radiation-induced phosphorylation of these ATM substrates. KU-55933 sensitizes HeLa cells to a range of ionizing radiation doses. [1] KU-55933 inhibits the phosphorylation of Akt induced by growth factors in cancer cells. KU-55933 suppresses the proliferation of cancer cells. Furthermore, suppression of ATM by KU-55933 improves survival, probably via prevention of downstream activation of TAp63 α . [2]

Suppression of ATM-dependent STAT3 activation by KU-55933 enhances TRAIL-mediated apoptosis through up-regulation of surface DR5 expression, whereas suppression of both STAT3 and NF- κ B appears to be involved in down-regulation of cFLIP accompanied by an additional increase in apoptotic levels. The ATM inhibitor KU-55933 affects TRAIL-mediated apoptosis more strongly than the JAK2 inhibitor, AG490, or overexpression of STAT3 β . [3]

References

[1] Hickson I, et al. Cancer Res. 2004, 64(24), 9152-9159.

[2] Soleimani R, et al. Aging. 2011, 3(8), 782-793.

[3] Ivanov VN, et al. Cancer Res. 2009, 69(8), 3510-3519.



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