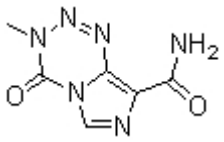
Product Introduction

Temozolomide

Temozolomide is a DNA damage inducer.

Technical Data:

Molecular Weight (MW):	194.15	
Formula:	$C_6H_6N_6O_2$	
Solubility (25°C) * <1 mg/ml means slightly soluble or insoluble:	DMSO 39 mg/mL	
	Water 5 mg/mL	
	Ethanol <1 mg/mL	
Purity:	>98%	
Storage:	3 years -20℃Powder	
	6 months-80℃in DMSO	
CAS No.:	85622-93-1	

Biological Activity

Methazolastone causes formation of DNA alkali-labile sites which are present in similar amounts and repaired at a similar rate in L-1210 and L-1210/BCNU. In L-1210 but not in L-1210/BCNU methazolastone induces an arrest of cells in SL-G2-M phases. Methazolastone induces a similar amount of DNA ALS which is also repaired at a similar rate in L-1210 and L-1210/BCNU cell lines. Methazolastone induces a similar amount of DNA ALS which is also repaired at a similar rate in L-1210 and L-1210/BCNU cell lines. [1] Methazolastone sensitivity of both chemo-sensitive and resistant cells (D54-R and U87-R) is enhanced significantly under hyperoxia. Both Methazolastone and hyperoxia are associated with increased phosphorylation of ERK p44/42 MAPK (Erk1/2), but to a lesser extent in D54-R cells, suggesting that

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Erk1/2 activity may be involved in regulation of hyperoxia and Methazolastone-mediated cell death. Hyperoxia enhances Methazolastone toxicity in GBM cells by induction of apoptosis, possibly via MAPK-related pathways. [2] Methazolastone induces in monocytes the DNA damage response pathways ATM-Chk2 and ATR-Chk1 resulting in p53 activation. [3] Chronic Methazolastone exposure results in acquired Methazolastone-resistance and elevates miR-21 expression. [4] Methazolastone treatment triggers endoplasmic reticula (ER) stress with increased expression of GADD153 and GRP78 proteins, and deceases pro-caspase 12 protein. Methazolastone induces autophagy through mitochondrial damage- and ER stress-dependent mechanisms to protect glioma cells. [5]

After a daily i.p. dose of 40 mg/kg for 5 consecutive days (days 1-5 after tumor transplant), methazolastone increases life-span by 86% in L-1210 and 22% in L-1210/BCNU. In L-1210/BCNU no effect is seen after 100 μM or 200 μM treatment; only 400 μM methazolastone produced an accumulation of cells in premitotic phase but much less than in L-1210. In L-1210/BCNU the maximum accumulation of cells in SL-G2-M is, after 48 hours-72 hours, approximately 30% as compared to 23% in untreated cells. Cells accumulates in SL-G2-M occurred too when L- 1210 leukemia-bearing mice are treated i.v. with methazola stone (40 mg/kg). No such effect is seen on L-1210/BCNU cells from mice given the same drug dose. [1]

Methazolastone is a second-generation alkylating agent.

References

[1] Catapano CV, et al. Cancer Res. 1987, 47(18), 4884-4889.

[2] Sun S, et al. J Neurooncol. 2012.

[3] Bauer M, et al. PLoS One. 2012, 7(6):e39956.

[4] Wong ST, et al. Anticancer Res. 2012, 32(7), 2835-2841.

[5] Lin CJ, et al. PLoS One. 2012, 7(6), e38706.

[6] Gori JL, et al. Cancer Gene Ther. 2012.

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