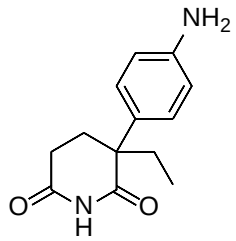
Product data sheet

MedKoo Cat#: 100051 Name: Aminoglutethimide CAS#: 125-84-8 Chemical Formula: $C_{13}H_{16}N_2O_2$ Exact Mass: 232.1212 Molecular Weight: 232.28	
Product supplied as:	Powder
Purity (by HPLC):	≥ 98%
Shipping conditions	Ambient temperature
Storage conditions:	Powder: -20°C 3 years; 4°C 2 years.
	In solvent: -80°C 3 months; -20°C 2 weeks.

1. Product description:
Aminoglutethimide is an anticancer drug that belongs to the family of drugs called nonsteroidal aromatase inhibitors. Aminoglutethimide is used to decrease the production of sex hormones (estrogen in women or testosterone in men) and suppress the growth of tumors that need sex hormones to grow. Aminoglutethimide is marketed under the tradename Cytadren by Novartis around the world. It blocks the production of steroids derived from cholesterol and is clinically used in the treatment of Cushing's syndrome and metastatic breast cancer.

2. CoA, QC data, SDS, and handling instruction
SDS and handling instruction, CoA with copies of QC data (NMR, HPLC and MS analytical spectra) can be downloaded from the product web page under "QC And Documents" section. Note: copies of analytical spectra may not be available if the product is being supplied by MedKoo partners. Whether the product was made by MedKoo or provided by its partners, the quality is 100% guaranteed.

3. Solubility data

Solvent	Max Conc. mg/mL	Max Conc. mM
DMF	10.0	43.05
DMSO	15.0	64.58
DMSO:PBS (pH 7.2) (1:1)	0.50	2.15
Ethanol	8.50	36.59

4. Stock solution preparation table:

Concentration / Solvent Volume / Mass	**1 mg**	**5 mg**	**10 mg**
1 mM	4.31 mL	21.53 mL	43.05 mL
5 mM	0.86 mL	4.31 mL	8.61 mL
10 mM	0.43 mL	2.15 mL	4.31 mL
50 mM	0.09 mL	0.43 mL	0.86 mL

5. Molarity Calculator, Reconstitution Calculator, Dilution Calculator
Please refer the product web page under section of "Calculator"

6. Recommended literature which reported protocols for in vitro and in vivo study
In vitro study

1. Shirakawa H, Katsuki H, Kume T, Kaneko S, Akaike A. Aminoglutethimide prevents excitotoxic and ischemic injuries in cortical neurons. Br J Pharmacol. 2006 Apr;147(7):729-36. doi: 10.1038/sj.bjp.0706636. PMID: 16474421; PMCID: PMC1751506.

In vivo study

1. Mutsuga M, Asaoka Y, Imura N, Miyoshi T, Togashi Y. Aminoglutethimide-induced lysosomal changes in adrenal gland in mice. Exp Toxicol Pathol. 2017 Sep 5;69(7):424-429. doi: 10.1016/j.etp.2017.04.004. Epub 2017 Apr 11. PMID: 28410883.

7. Bioactivity
Biological target: Aminoglutethimide (BA-16038, NSC-330915) is an aromatase inhibitor with an IC50 of 10 μM.

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In vitro activity

The effect of aminoglutethimide on cell death induced by overactivation of glutamate receptors in CNS neurons was investigated. In dissociated rat cerebrocortical cell cultures, long-term treatment with aminoglutethimide (10-1000 microM) attenuated NMDA receptor-mediated glutamate cytotoxicity but produced no significant effect on glutamate-induced increases in intracellular Ca2+. Brief as well as long-term pretreatment with aminoglutethimide (30-1000 microM) prevented NMDA receptor-dependent ischemic neuronal injury in organotypic cerebrocortical slice cultures, which was associated with suppression of glutamate release during the ischemic insult. These results indicate that aminoglutethimide, irrelevant to its actions on neurosteroid synthesis, protects CNS neurons from excitotoxic and ischemic injuries.

Reference: Br J Pharmacol. 2006 Apr;147(7):729-36. https://www.ncbi.nlm.nih.gov/pmc/articles/PMC1751506/

In vivo activity

Histopathological changes induced by 5-day administration of AG (aminoglutethimide) in mice were investigated. Cytoplasmic vacuoles of various sizes and single cell necrosis were found in zona fasciculata cells in AG-treated mice. Some vacuoles were positive for adipophilin, whereas others were positive for lysosome-associated membrane protein-2 on immunohistochemical staining, indicating they were enlarged lipid droplets and lysosomes, respectively. Electron microscopy revealed enlarged lysosomes containing damaged mitochondria and lamellar bodies in zona fasciculata cells, and they were considered to reflect the intracellular protein degradation processes, mitophagy and lipophagy. These results showed that AG induces excessive lipid accumulation and mitochondrial damage in zona fasciculata cells, which leads to an accelerated lysosomal degradation in mice.

Reference: Exp Toxicol Pathol. 2017 Sep 5;69(7):424-429.
https://www.sciencedirect.com/science/article/abs/pii/S0940299316302238?via%3Dihub

Note: The information listed here was extracted from literature. MedKoo has not independently retested and confirmed the accuracy of these methods. Customer should use it just for a reference only.