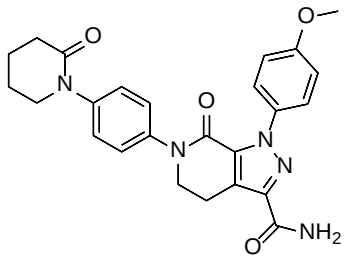
Product data sheet

MedKoo Cat#: 300170 Name: Apixaban CAS#: 503612-47-3 Chemical Formula: $C_{25}H_{25}N_5O_4$ Exact Mass: 459.1907 Molecular Weight: 459.50	
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:

Apixaban, also known as BMS-56224701, is an anticoagulant for the treatment of venous thromboembolic events. Apixaban is a direct factor Xa inhibitor. Apixaban was approved in 2012. Apixaban is indicated for the following: (1) To lower the risk of stroke and embolism in patients with nonvalvular atrial fibrillation. (2) Deep vein thrombosis (DVT) prophylaxis. DVT's may lead to pulmonary embolism (PE) in knee or hip replacement surgery patients. (3) Treatment of both DVT and PE. (4) To reduce the risk of recurring DVT and PE after initial therapy.

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	3.0	6.53
DMSO	34.0	73.99
DMSO:PBS (pH 7.2) (1:1)	0.50	1.09

4. Stock solution preparation table:

Concentration / Solvent Volume / Mass	1 mg	5 mg	10 mg
1 mM	2.18 mL	10.88 mL	21.76 mL
5 mM	0.44 mL	2.18 mL	4.35 mL
10 mM	0.22 mL	1.09 mL	2.18 mL
50 mM	0.04 mL	0.22 mL	0.44 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. Guasti L, Squizzato A, Moretto P, Vigetti D, Ageno W, Dentali F, Maresca AM, Campiotti L, Grandi AM, Passi A. In vitro effects of Apixaban on 5 different cancer cell lines. PLoS One. 2017 Oct 12;12(10):e0185035. doi: 10.1371/journal.pone.0185035. PMID: 29023465; PMCID: PMC5638249.
2. Torramade-Moix S, Palomo M, Vera M, Jerez D, Moreno-Castaño AB, Zafar MU, Rovira J, Diekmann F, Garcia-Pagan JC, Escolar G, Cases A, Diaz-Ricart M. Apixaban Downregulates Endothelial Inflammatory and Prothrombotic Phenotype in an In Vitro Model of Endothelial Dysfunction in Uremia. Cardiovasc Drugs Ther. 2021 Jun;35(3):521-532. doi: 10.1007/s10557-020-07010-z. PMID: 32651897.

In vivo study

1. Shi G, Yang X, Pan M, Sun J, Ke H, Zhang C, Geng H. Apixaban attenuates ischemia-induced myocardial fibrosis by inhibition of Gq/PKC signaling. Biochem Biophys Res Commun. 2018 Jun 7;500(3):550-556. doi: 10.1016/j.bbrc.2018.04.071. Epub 2018 Apr 24. PMID: 29654769.

2. Bushi D, Chapman J, Wohl A, Stein ES, Feingold E, Tanne D. Apixaban decreases brain thrombin activity in a male mouse model of acute ischemic stroke. J Neurosci Res. 2018 Aug;96(8):1406-1411. doi: 10.1002/jnr.24253. Epub 2018 May 14. PMID: 29761540.

7. Bioactivity

Biological target: Apixaban (BMS 562247-01) is an inhibitor of Factor Xa with Ki of 0.08 nM and 0.17 nM in human and rabbit, respectively.

In vitro activity

Macro (HUVEC) and microvascular (HMEC) endothelial cells (ECs) were exposed to serum from uremic patients or healthy donors, in absence and presence of apixaban (60 ng/ml). Changes in surface VCAM-1 and ICAM-1, intracellular eNOS, reactive oxygen species (ROS), and von Willebrand Factor (VWF) production by immunofluorescence, reactivity of the extracellular matrix (ECM) towards platelets, and intracellular signaling were evaluated. ECs exposed to uremic serum triggered dysregulation of all the parameters. Presence of apixaban resulted in decreased expression of VCAM-1 (178 ± 14 to 89 ± 2% on HMEC and 324 ± 71 to 142 ± 25% on HUVEC) and ICAM-1 (388 ± 60 to 111 ± 10% on HMEC and 148 ± 9% to 90 ± 7% on HUVEC); increased eNOS (72 ± 8% to 95 ± 10% on HMEC); normalization of ROS levels (173 ± 21 to 114 ± 13% on HMEC and 165 ± 14 to 127 ± 7% on HUVEC); lower production of VWF (168 ± 14 to 92 ± 4% on HMEC and 151 ± 22 to 99 ± 11% on HUVEC); and decreased platelet adhesion onto ECM (134 ± 22 to 93 ± 23% on HMEC and 161 ± 14 to 117 ± 7% on HUVEC). Apixaban inhibited p38MAPK and p42/44 activation in HUVEC (139 ± 15 to 48 ± 15% and 411 ± 66 to 177 ± 57%, respectively) ($p < 0.05$ vs control for all parameters). Anti-FXa strategies, such as apixaban, prevented ED caused by the uremic milieu, exhibiting anti-inflammatory and antioxidant properties and modulating the reactivity of the ECM.

Reference: Cardiovasc Drugs Ther. 2021 Jun;35(3):521-532. https://link.springer.com/article/10.1007%2Fs10557-020-07010-z

In vivo activity

As an inhibitor of FXa (prothrombin), Apixaban inhibits prothrombin conversion into thrombin leading to thrombin deficiency in vivo. Myocardial fibrosis-bearing mice induced by continuous myocardial ischemia (MI) had higher levels of thrombin. Orally administration of apixaban significantly abrogated fibrosis condition and thrombin levels. Thrombin induced collagen deposition by activation of the Par-1-coupled Gq/PKC signaling. Genetic ablation of Gq or pharmacological inhibition of PKC effectively blunted thrombin-induced collagen deposition in cardiac fibroblasts. Moreover, administration of PKC inhibitor or Gq antagonist obviously blocked MI-induced myocardial fibrosis in mice. To conclude, apixaban attenuates MI-induced myocardial fibrosis by inhibition of thrombin-dependent Par-1/Gq/PKC signaling axis.

Reference: Biochem Biophys Res Commun. 2018 Jun 7;500(3):550-556.
https://www.sciencedirect.com/science/article/abs/pii/S0006291X18308404?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.