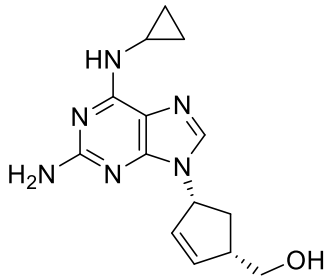
Product data sheet

MedKoo Cat#: 317150 Name: Abacavir CAS#: 136470-78-5 (free base) Chemical Formula: $C_{14}H_{18}N_6O$ Exact Mass: 286.15421 Molecular Weight: 286.34	
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:

Abacavir (ABC) is an antiretroviral medication used to prevent and treat HIV/AIDS. It is of the nucleoside analog reverse transcriptase inhibitor (NRTI) type. Viral strains that are resistant to zidovudine (AZT) or lamivudine (3TC) are generally, but not always, sensitive to abacavir. It is on the World Health Organization's List of Essential Medicines, a list of the most important medication needed in a basic health system. It is available under the trade name Ziagen and in the combination formulations abacavir/lamivudine/zidovudine, abacavir/dolutegravir/lamivudine, abacavir/lamivudine.

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
DMSO	79.0	274.15

4. Stock solution preparation table:

Concentration / Solvent Volume / Mass	1 mg	5 mg	10 mg
1 mM	3.49	17.46	34.92
5 mM	0.70	3.49	6.98
10 mM	0.35	1.75	3.49
50 mM	0.07	0.35	0.70

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. Tada K, Kobayashi M, Takiuchi Y, Iwai F, Sakamoto T, Nagata K, Shinohara M, Io K, Shirakawa K, Hishizawa M, Shindo K, Kadowaki N, Hirota K, Yamamoto J, Iwai S, Sasanuma H, Takeda S, Takaori-Kondo A. Abacavir, an anti-HIV-1 drug, targets TDP1-deficient adult T cell leukemia. Sci Adv. 2015 Apr 24;1(3):e1400203. doi: 10.1126/sciadv.1400203. PMID: 26601161; PMCID: PMC4640626.

2. Adam J, Wuillemin N, Watkins S, Jamin H, Eriksson KK, Villiger P, Fontana S, Pichler WJ, Yerly D. Abacavir induced T cell reactivity from drug naïve individuals shares features of allo-immune responses. PLoS One. 2014 Apr 21;9(4):e95339. doi: 10.1371/journal.pone.0095339. PMID: 24751900; PMCID: PMC3994040.

In vivo study

1. Kohler JJ, Hosseini SH, Green E, Fields E, Abuin A, Ludaway T, Russ R, Lewis W. Absence of mitochondrial toxicity in hearts of transgenic mice treated with abacavir. Cardiovasc Toxicol. 2010 Jun;10(2):146-51. doi: 10.1007/s12012-010-9070-2. PMID: 20379802; PMCID: PMC7704124.

2. Cardone M, Garcia K, Tilahun ME, Boyd LF, Gebreyohannes S, Yano M, Roderiquez G, Akue AD, Juengst L, Mattson E, Ananthula S, Natarajan K, Puig M, Margulies DH, Norcross MA. A transgenic mouse model for HLA-B*57:01-linked abacavir drug

tolerance and reactivity. J Clin Invest. 2018 Jul 2;128(7):2819-2832. doi: 10.1172/JCI99321. Epub 2018 May 21. PMID: 29782330; PMCID: PMC6025983.

7. Bioactivity

Biological target:

Abacavir is a potent nucleoside analog reverse-transcriptase inhibitor (NRTI).

In vitro activity

The cytotoxic effects of six NRTIs (ddI, d4T, 3TC, AZT, TDF, and ABC) were examined, all of which are current therapeutic agents for HIV-1, on ATL cell lines [ED-40515(−), ED-40515(+), SYK-11L(+), and ATL-43T] and on HTLV-1–infected cell lines (MT-2 and Hut-102). Strikingly, the clinically relevant concentration of ABC was highly toxic to all ATL- and HTLV-1–infected cell lines but not to non–HTLV-1–infected cell lines (Jurkat, H9, and SU-DHL-6) (Fig. 1, A and B, fig. S1, and table S1). Therefore, it is concluded that ABC potently and selectively kills HTLV-1–infected cells, including ATL cells, in vitro. Next, the effect of ABC on cell cycle and apoptosis in ATL cells was examined. ABC induced S/G2-phase arrest and apoptosis in ED-40515(−) cells, but not in Jurkat cells (Fig. 2, A to C, and fig. S3). This finding suggests that ABC may cause DNA damage by prematurely terminating the replication of host chromosomal DNA, thereby activating the DNA damage checkpoint to induce transient S/G2-phase arrest and apoptosis.

Sci Adv. 2015 Apr; 1(3): e1400203. https://www.ncbi.nlm.nih.gov/pmc/articles/PMC4640626/

In vivo activity

Since NRTIs exhibit tissue-specific inhibition of mitochondrial DNA (mtDNA) synthesis, the ability of ABC (Abacavir) to inhibit mtDNA synthesis in vivo was evaluated. Inbred wild-type (WT) and transgenic mice (TG) treated with ABC (3.125 mg/d p. o., 35 days) were used to define mitochondrial oxidative stress and cardiac function. Cardiac function was assessed for each group of mice. LV (left ventrical) mass and LVEDD (left ventricle end-diastolic dimension) were derived from direct echocardiographic measurements of cavity and wall thickness in each mouse to define effects of ABC and/or genetic manipulation on LV. Results showed LV mass was unchanged in all vehicle-treated TGs (including MSB, MnSOD OX, SOD2+/− KO, MCAT, and ALDH2 KO) compared to vehicle-treated WTs (Fig. 2a). Similarly, ABC had no effect on LV mass in the MnSOD OX and MCAT TGs, as expected (Fig. 2a). Surprisingly, SOD2+/− KOs were also found to have no change in LV mass following ABC, further supporting the hypothesis that oxidative stress is not associated with ABC treatment (Fig. 2a). In contrast, SOD2+/− KO previously was shown to have increased LV mass following AZT treatment for 5 weeks [3]. Any potential changes in LV mass due to the absence of ALDH2 activity was also disproven with results here that showed no change in ALDH2 KOs treated with ABC compared to WTs (Fig. 2a). LVEDD also remained unchanged (with or without ABC treatment) in WTs and all TGs, even SOD2+/− KO and ALDH2 KO (Fig. 2b). These results further support the conclusion that ABC treatment for 5 weeks has no detectable cardiotoxicity and suggests that oxidative stress is not associated with ABC treatment.

Cardiovasc Toxicol. Author manuscript; available in PMC 2020 Nov 30. https://www.ncbi.nlm.nih.gov/pmc/articles/PMC7704124/

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.