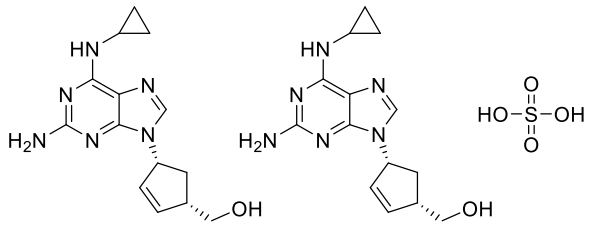
Product data sheet

MedKoo Cat#: 562587 Name: Abacavir sulfate CAS#: 188062-50-2 (sulfate) Chemical Formula: $C_{28}H_{38}N_{12}O_6S$ Molecular Weight: 670.75	
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 sulfate is a nucleoside reverse transcriptase inhibitor analog of guanosine. It acts by decreasing HIV viral loads, retarding or preventing the damage to the immune system, and reducing the risk of developing AIDS.

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	0.15	0.22

4. Stock solution preparation table:

Concentration / Solvent Volume / Mass	1 mg	5 mg	10 mg
1 mM	1.49 mL	7.45 mL	14.91 mL
5 mM	0.30 mL	1.49 mL	2.98 mL
10 mM	0.15 mL	0.75 mL	1.49 mL
50 mM	0.03 mL	0.15 mL	0.30 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. 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. Collado-Diaz V, Andujar I, Sanchez-Lopez A, Orden S, Blanch-Ruiz MA, Martinez-Cuesta MA, Blas-García A, Esplugues JV, Álvarez Á. Abacavir Induces Arterial Thrombosis in a Murine Model. J Infect Dis. 2018 Jun 20;218(2):228-233. doi: 10.1093/infdis/jiy001. PMID: 29346575.

7. Bioactivity
Biological target:

Abacavir (1592U89) is a commonly used nucleoside analogue with potent antiviral activity against HIV-1.

In vitro activity

In vitro culture of PBMC with abacavir results in the outgrowth of abacavir-reacting CD8+ T cells, which release IFNγ and are cytotoxic. How this immune response is induced and what is recognized by these T cells is still a matter of debate. The conditions required to develop an abacavir-dependent T cell response in vitro is analyzed. The abacavir reactivity was independent of co-stimulatory signals, as neither DC maturation nor release of inflammatory cytokines were observed upon abacavir exposure. Abacavir

induced T cells arose in the absence of professional APC and stemmed from naïve and memory compartments. These features are reminiscent of allo-reactivity. Screening for allo-reactivity revealed that about 5% of generated T cell clones (n = 136) from three donors were allo-reactive exclusively to the related HLA-B*58：01. The addition of peptides which can bind to the HLA-B*57：01-abacavir complex and to HLA-B*58：01 during the induction phase increased the proportion of HLA-B*58：01 allo-reactive T cell clones from 5% to 42%. In conclusion, abacavir can alter the HLA-B*57：01-peptide complex in a way that mimics an allo-allele ('altered self-allele') and create the potential for robust T cell responses.

Reference: PLoS One. 2014 Apr 21;9(4):e95339. https://www.ncbi.nlm.nih.gov/pmc/articles/pmid/24751900/

In vivo activity

Pretreatment with ABC intrascrotally (at doses of 2.5, 5, and 7.5 µg/mL) or orally (dose range, 100–200 mg/kg) neither altered blood flow (measured as the vascular wall shear rate) nor resulted in the formation of thrombi (measured as the time to occlusion; data not shown). Likewise, in the absence of any pretreatment, superfusion with 25 or 50 mM of ferric chloride did not affect the aforementioned parameters. However, when the concentration of ferric chloride in the superfusion was augmented to 75 mM, there was a rapid formation of thrombi and a subsequent reduction in wall shear rate characteristic of a cessation of blood flow (Supplementary Figure 1). Likewise, the arterioles became occluded when the 25 mM concentration of ferric chloride was perfused over the cremaster of animals intrascrotally pretreated with ABC. This occlusion developed more rapidly as the dose of ABC increased (Figure 1A and 1B) and was accompanied by a parallel dose-dependent reduction in the arterial wall shear rate (Figure 1D). The oral administration of ABC (mean plasma concentration [±SEM], 5.3 ± 0.8 and 16.2 ± 1.9 µg/mL following 100 and 200 mg/kg, respectively) reproduced the effects of locally administered ABC (Figure 1B). Superfusion with 25 mM of ferric chloride had no effects in mice pretreated intrascrotally with one of the other NRTIs analyzed (TDF, ddI, FTC, or 3TC), even at concentrations (7.5 µg/mL) substantially higher than those considered clinically appropriate (Figure 1C). In contrast, in animals pretreated with a clinical concentration of diclofenac or rofecoxib—2 well-known vascular injurious agents—25 mM of ferric chloride led to the formation of arterial thrombi in a similar way to that produced by the 2 highest doses of ABC evaluated (Figure 2A).

Reference: J Infect Dis. 2018 Jun 20;218(2):228-233. https://academic.oup.com/jid/article-lookup/doi/10.1093/infdis/jiy001