# Product data sheet

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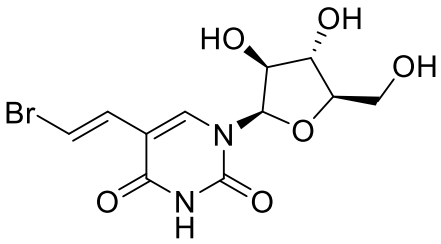
|---|---|
| MedKoo Cat#: 328023<br>Name: Sorivudine<br>CAS#: 77181-69-2<br>Chemical Formula: $C_{11}H_{13}BrN_2O_6$<br>Exact Mass: 347.9957<br>Molecular Weight: 349.137 |  |
| Product supplied as: | Powder |
| Purity (by HPLC): | ≥ 98% |
| Shipping conditions | Ambient temperature |
| Storage conditions: | Powder: -20°C 3 years; 4°C 2 years. |
| | In solvent: -80°C 3 months; -20°C 2 weeks. |

**1. Product description:**
Sorivudine, also known as Bravavir; ARYS-01; BV-araU; BVAU; JA-001; SQ-32756; YN-72; DRU-0136, is a DNA polymerase inhibitor potentially for the treatment of herpes zoster (shingles). Sorivudine: a promising drug for the treatment of varicella-zoster virus infection. Sorivudine provides a unique nucleoside analog with significantly enhanced both in vitro as in vitro activity toward VZV and enhanced oral bioavailability, as compared with existing antivirals.

**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 | 250.0 | 716.07 |

**4. Stock solution preparation table:**

| Concentration / Solvent Volume / Mass | 1 mg | 5 mg | 10 mg |
|---|---|---|---|
| 1 mM | 2.86 mL | 14.32 mL | 28.64 mL |
| 5 mM | 0.57 mL | 2.86 mL | 5.73 mL |
| 10 mM | 0.29 mL | 1.43 mL | 2.86 mL |
| 50 mM | 0.06 mL | 0.29 mL | 0.57 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. Machida H, Nishitani M, Suzutani T, Hayashi K. Different antiviral potencies of BV-araU and related nucleoside analogues against herpes simplex virus type 1 in human cell lines and Vero cells. Microbiol Immunol. 1991;35(11):963-73. doi: 10.1111/j.1348-0421.1991.tb01618.x. PMID: 1663576.
2. Machida H, Watanabe Y. Inhibition of DNA synthesis in varicella-zoster virus-infected cells by BV-araU. Microbiol Immunol. 1991;35(2):139-45. doi: 10.1111/j.1348-0421.1991.tb01541.x. PMID: 1653394.

In vivo study

1. Ijichi K, Ashida N, Machida H. Effect of 1-beta-D-arabinofuranosyl-E-5-(2-bromovinyl)uracil against herpes simplex virus type 1 infection in immunosuppressed mice. Antimicrob Agents Chemother. 1990 Dec;34(12):2431-3. doi: 10.1128/AAC.34.12.2431. PMID: 1965108; PMCID: PMC172077.

**7. Bioactivity**
Biological target:

Sorivudine (BV-araU) is a synthetic pyrimidine nucleoside antimetabolite drug that derives its antiviral activity from selective conversion by a specific thymidine kinase present in certain DNA viruses to nucleotides.

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In vitro activity

The aim of this study was to explore the inhibitory effect of BV-araU on DNA synthesis in human embryonic lung cells infected with varicella-zoster virus (VZV) or herpes simplex virus type 1 (HSV-1) and it was compared with that of acyclovir. A human embryonic lung fibroblast cell line, HAIN-55 HSV-1 and VZV were used in this study. Cellular uptake of [3H]thymidine and its incorporation into DNA was markedly stimulated by the infection with VZV or HSV-1, suggesting that the incorporation was mainly due to viral DNA synthesis. DNA synthesis in VZV-infected cells was dose-dependently suppressed by BV-araU More than 98% of inhibition was observed by treatment with 0.3 ƒÊg/ml of BV-araU or 1 ƒÊg/ml of acyclovir for HSV-infected cells. BV-araU showed three times more inhibitory activity than acyclovir, an observation similar to that seen in cells pretreated with drugs for 1 hr. More than 50% in-hibition was caused by BV-araU at a concentration as low as 3 ƒÊg/ml, for VZV-infected cells. Thus, inhibition of DNA synthesis in VZV-infected cells by BV-araU was found in all experiments at about one-thousandth of the inhibitory concentration of acyclovir, regardless of the duration of drug pretreatment. The results suggest that the antiviral action of BV-araU against VZV and HSV-1 is based on the inhibition of DNA synthesis in herpesvirus-infected cells.

Reference: Microbiol Immunol. 1991;35(2):139-45. https://pubmed.ncbi.nlm.nih.gov/1653394/

In vivo activity

In this study, we tested the efficacy of BV-araU in immunosuppressed animals and demonstrated its therapeutic efficacies against infection with two HSV-1 strains with different degrees of virulence in cyclophosphamide-treated, immunosuppressed mice (CYP mice) of different ages. Oral and i.p. doses of BV-araU up to 800 mg/kg given twice daily for 7 days did not cause death in uninfected CYP mice (data not shown), as with uninfected normal mice (5,Sa). No toxic signs were observed, except that body weight gain was slightly suppressed in a group that received 800 mg of BV-araU i.p. Changes in virus titers in several organs from CYP mice and normal mice were examined. High virus titers were found only in adrenal gland, spinal cord, and brain tissues, but low levels of the infective virus were found transiently in other organs from normal mice after i.p. infection with 1.5 x 105 PFU of strain WT-51. Virus titers in the gastrointestinal tracts of drug-treated mice did not increase for 6 days, and infective virus disappeared at 9 days in about half of the drug-treated mice. The virus titers of placebo-treated mice gradually increased until the mice died at 8 days postinfection. , This warrants clinical testing for treatment of herpesvirus infections of immunocompromised patients.

Reference: Antimicrob Agents Chemother. 1990 Dec;34(12):2431-3. https://pubmed.ncbi.nlm.nih.gov/1965108/

*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.*