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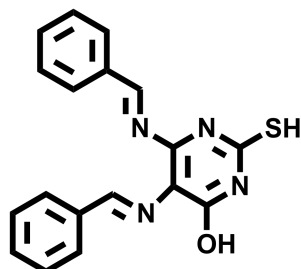
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## NHEJ inhibitor– SCR7

**Chemical Name:** 5,6-bis((E)-benzylideneamino)-2-mercaptopyrimidin-4-ol



|                   |   |
|-------------------|---|
| Molecular Weight: | 334.39  |
| Formula:          | C <sub>18</sub> H <sub>14</sub> N <sub>4</sub> OS       |
| Purity:           | ≥ 98%   |
| CAS#:             | 1533426-72-0  |
| Solubility:       | DMSO up to 50 mM  |
| Storage           | Powder: 4°C 1 year<br>DMSO: 4°C 3 month<br>-20°C 1 year |

### Biological Activity:

SCR7 is a potent and selective inhibitor of non-homologous end joining (NHEJ). It inhibits joining of DSBs in cell-free DNA repair system, blocks Ligase IV-mediated joining by interfering with its DNA binding but not that of T4 DNA Ligase or Ligase I, thereby leading to accumulation of DSBs within the cells, culminating into cytotoxicity. SCR7 inhibits NHEJ in a Ligase IV-dependent manner within cells, and activates the intrinsic apoptotic pathway. More importantly, SCR7 impedes tumor progression in mouse models, and when co-administered with DSB-inducing therapeutic modalities it enhances their sensitivity significantly. In addition, SCR7 can promote the efficiency of HDR 4–5-fold for CRISPR editing in both human and mouse cell lines.

### How to Use:

**In vitro:** SCR7 was used at 20-150 μM final concentration in vitro and in cellular assays for anti-cancer. SCR7 was used at 1 μM final concentration in CRISPR editing.

**In vivo:** SCR7 was intraperitoneally (IP) dosed to mice at 20 mg/kg once per day.

### Reference:

1. Srivastava M, et al. An inhibitor of nonhomologous end-joining abrogates double-strand break repair and impedes cancer progression. (2012) Cell. 151(7):1474-87.
2. Chu VT, et al. Increasing the efficiency of homology-directed repair for CRISPR-Cas9-induced precise gene editing in mammalian cells. (2015) Nat Biotechnol. In press.

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