# SAH-SOS1A TFA

**Cat. No.:** HY-P2265A

**Molecular Formula:** $C_{102}H_{160}N_{27}F_3O_{30}$

**Molecular Weight:** 2301.55

**Sequence:** Arg-Arg-Phe-Phe-Gly-Ile-Aaa-Leu-Thr-Asn-Aaa-Leu-Lys-Thr-Glu-Glu-Gly-Asn (Covalent bridge:Aaa7-Aaa11)

RRFFGI{Aaa}LTN{Aaa}LKTEEGN (Covalent bridge:$Aaa_7$-$Aaa_{11}$) (TFA salt)

**Sequence Shortening:** RRFFGI{Aaa}LTN{Aaa}LKTEEGN (Covalent bridge:Aaa7-Aaa11)

**Target:** Ras

**Pathway:** GPCR/G Protein

**Storage:** Sealed storage, away from moisture and light, under nitrogen

Powder -80°C 2 years

-20°C 1 year

* In solvent : -80°C, 6 months; -20°C, 1 month (sealed storage, away from moisture and light, under nitrogen)

## SOLVENT & SOLUBILITY

**In Vitro**

$H_2O$ : 33.33 mg/mL (14.48 mM; Need ultrasonic)

| Preparing Stock Solutions | Solvent / Mass / Concentration | 1 mg | 5 mg | 10 mg |
|---|---|---|---|---|
| | 1 mM | 0.4345 mL | 2.1724 mL | 4.3449 mL |
| | 5 mM | 0.0869 mL | 0.4345 mL | 0.8690 mL |
| | 10 mM | 0.0434 mL | 0.2172 mL | 0.4345 mL |

Please refer to the solubility information to select the appropriate solvent.

## BIOLOGICAL ACTIVITY

**Description**

SAH-SOS1A TFA is a peptide-based SOS1/KRAS protein interaction inhibitor. SAH-SOS1A TFA binds to wild-type and mutant KRAS (G12D, G12V, G12C, G12S, and Q61H) with nanomolar affinity ($EC_{50}$=106-175 nM). SAH-SOS1A TFA directly and independently blocks nucleotide association. SAH-SOS1A TFA impairs KRAS-driven cancer cell viability and exerts its effects by on-mechanism blockade of the ERK-MAPK phosphosignaling cascade downstream of KRAS[1].

**$IC_{50}$ & Target**

| | | | |
|---|---|---|---|
| KRAS-SOS1 | KRas G12C<br>140 nM (EC50) | KRas G12D<br>109 nM (EC50) | KRas G12V<br>154 nM (EC50) |
| KRas G12S<br>155 nM (EC50) | KRas Q61H<br>175 nM (EC50) | K-Ras WT<br>106 nM (EC50) | |

**In Vitro**

SAH-SOS1A TFA (0.625-40 μM; 24 hours) dose-responsively impairs the viability of cancer cells bearing G12D, G12C, G12V,

G12S, G13D, and Q61H mutations with IC50 values in the 5- to 15-μM range. Cancer cells expressing wild-type KRAS, such as HeLa and Colo320-HSR cells, are similarly affected[1].
SAH-SOS1A TFA (5-40 μM; 4 hours) dose-responsively inhibits MEK1/2, ERK1/2, and AKT phosphorylation[1].
MCE has not independently confirmed the accuracy of these methods. They are for reference only.

Cell Viability Assay[1]

| | |
|---|---|
| Cell Line: | Panc 10.05 cells bearing the KRAS G12D mutation |
| Concentration: | 0.625-40 μM |
| Incubation Time: | 24 hours |
| Result: | Dose-responsively impaired the viability of cancer cells bearing KRAS G12D. |

Western Blot Analysis [1]

| | |
|---|---|
| Cell Line: | Panc 10.05 cells |
| Concentration: | 5-40 μM |
| Incubation Time: | Indicated doses for 4 h, followed by 15-min stimulation with EGF |
| Result: | Dose-responsively inhibited MEK1/2, ERK1/2, and AKT phosphorylation. |

**In Vivo**

SAH-SOS1A (0.2 μL of 10 mM solution; injection; 48 hours; abdomens of D. melanogaster $Ras85D^{V12}$/ActinGS) treatment notably decreases the phosphorylation state of ERK1/2[1].
MCE has not independently confirmed the accuracy of these methods. They are for reference only.

## REFERENCES

[1]. Leshchiner ES, et al. Direct inhibition of oncogenic KRAS by hydrocarbon-stapled SOS1 helices. Proc Natl Acad Sci U S A. 2015;112(6):1761-1766.



Tel: 609-228-6898 Fax: 609-228-5909 E-mail: tech@MedChemExpress.com
Address: 1 Deer Park Dr, Suite Q, Monmouth Junction, NJ 08852, USA