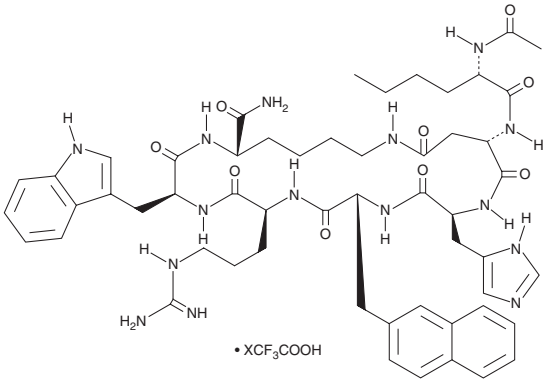
# PRODUCT INFORMATION

## SHU9119 (trifluoroacetate salt)

*Item No. 24152*

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| **CAS Registry No.:** | 168482-23-3 |
| **Formal Name:** | N-acetyl-L-norleucyl-L-α-aspartyl-L-histidyl-3-(2-naphthalenyl)-D-alanyl-L-arginyl-L-tryptophyl-L-lysinamide, (2→7)-lactam, trifluoroacetate salt |
| **MF:** | $C_{54}H_{71}N_{15}O_9 \bullet XCF_3COOH$ |
| **FW:** | 1,074.2 |
| **Purity:** | ≥95% |
| **Supplied as:** | A lyophilized powder |
| **Storage:** | -20°C |
| **Stability:** | ≥4 years |

*Information represents the product specifications. Batch specific analytical results are provided on each certificate of analysis.*

### Laboratory Procedures

SHU9119 (trifluoroacetate salt) is supplied as a lyophilized powder. A stock solution may be made by dissolving the SHU9119 (trifluoroacetate salt) in water. The solubility of SHU9119 (trifluoroacetate salt) in water is approximately 1 mg/ml. We do not recommend storing the aqueous solution for more than one day.

### Description

SHU9119 is an agonist of melanocortin receptor 1 (MC1R) and antagonist of MC4R ($IC_{50}$s = 1.2 and 2.9 nM, respectively, for displacement of melanocortin).[1] It induces cAMP formation in HEK293 cells expressing human MC1R ($EC_{50}$ = 1.11 nM), but inhibits cAMP formation in cells expressing human MC4R.[1] In rats, SHU9119 (24 nmol, i.c.v. per day for seven days) increases food intake, body weight, fat mass, and lean mass, with concomitant increases in blood glucose, insulin, and leptin levels *via* disrupted melanocortin signaling.[2] Similarly, mice treated with SHU9119 (5 nmol/day, i.c.v.) exhibit food intake-independent increases in body weight and fat mass consequent to MC4R inhibition and subsequent brown adipose tissue dysfunction.[3]

### References


1. Yang, Y., Chen, M., Lai, Y., *et al.* Molecular determinants of human melanocortin-4 receptor responsible for antagonist SHU9119 selective activity. *J. Biol. Chem.* **277(23)**, 20328-20335 (2002).
2. Nogueiras, R., Wiedmer, P., Perez-Tilve, D., *et al.* The central melanocortin system directly controls peripheral lipid metabolism. *J. Clin. Invest.* **117(11)**, 3475-3488 (2007).
3. Kooijman, S., Boon, M.R., Parlevliet, E.T., *et al.* Inhibition of the central melanocortin system decreases brown adipose tissue activity. *J. Lipid Res.* **55(10)**, 2022-2032 (2014).





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