MOTS-c(human) acetate

Cat. No.: HY-P2048A

Molecular Formula: $C_{103}H_{156}N_{28}O_{24}S_2$

Molecular Weight: 2234.64

Sequence: Met-Arg-Trp-Gln-Glu-Met-Gly-Tyr-Ile-Phe-Tyr-Pro-Arg-Lys-Leu-Arg

Sequence Shortening: MRWQEMGYIFYPRKLR

MRWQEMGYIFYPRKLR (acetate salt)

Target: AMPK; GLUT

Pathway: Epigenetics; PI3K/Akt/mTOR; Membrane Transporter/Ion Channel

Storage: Sealed storage, away from moisture

Powder -80°C 2 years

-20°C 1 year

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

SOLVENT & SOLUBILITY

In Vitro

H_2O : 6.25 mg/mL (2.80 mM; Need ultrasonic)

DMSO : 4 mg/mL (1.79 mM; Need ultrasonic)

Preparing Stock Solutions	Solvent / Mass / Concentration	1 mg	5 mg	10 mg
	1 mM	0.4475 mL	2.2375 mL	4.4750 mL
	5 mM	---	---	---
	10 mM	---	---	---

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

BIOLOGICAL ACTIVITY

Description

MOTS-c(human) acetate is a mitochondrial-derived peptide. MOTS-c(human) acetate induces the accumulation of AMP analog AICAR, increases activation of AMPK and expression of its downstream GLUT4. MOTS-c(human) acetate induces glucose uptake and improves insulin sensitivity. MOTS-c(human) acetate has implications in the regulation of obesity, diabetes, exercise, and longevity[1].

IC_{50} & Target			
	AMPK	GLUT4	AICAR

In Vitro

MOTS-c inhibits the folate cycle at the level of 5Me-THF, resulting in an accumulation of AICAR [5-aminoimidazole-4-carboxamide ribonucleotide). MOTS-c also increases cellular NAD^+ levels, which are also nucleotide precursors[1]. MOTS-c is a mitochondrial signal that stimulates cellular glucose uptake while suppressing respiration. The glucose taken up in response to MOTS-c is routed to the anabolic pentose phosphate pathway (PPP), which provides carbon sources for the synthesis of purines, rather than being metabolized through glycolysis. In addition, MOTS-c increases the levels of carnitine shuttles, which transport activated fatty acids into the mitochon-dria for β-oxidation, increases the level of a β-

	oxidation intermediate, and reduces intracellular levels of essential and non-essential fatty acids, suggesting enhanced lipid utilization; myocytes that stably overexpress MOTS-c also exhibits increased glucose uptake[1]. MCE has not independently confirmed the accuracy of these methods. They are for reference only.
In Vivo	MOTS-c injections in mice show activation of skeletal muscle AMPK and increased the level of its downstream glucose transporter GLUT4. MOTS-c may also act as a potential mitochondrial signal that mediates an exercise-induced mitohormesis response, thereby stimulating physiological adaptation and increased tolerance to exercise[1]. The primary target organ of MOTS-c appears to be skeletal muscle and fat. MOTS-c levels in mice decline with age in skeletal muscle and in circulation concomitantly with the age-dependent development of insulin resistance. Restoring MOTS-c levels by systemic injections in older mice (12 mo.) successfully reverses age-dependent skeletal muscle insulin resistance[1]. MCE has not independently confirmed the accuracy of these methods. They are for reference only.

REFERENCES

[1]. Changhan Lee, et al. MOTS-c: A Novel Mitochondrial-Derived Peptide Regulating Muscle and Fat Metabolism. Free Radic Biol Med. 2016 Nov;100:182-187.

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