

Data Sheet

Product Name:	MOTS-c(human) (acetate)	
Cat. No.:	CS-0129833	
Molecular Formula:	C ₁₀₃ H ₁₅₆ N ₂₈ O ₂₄ S ₂	
Molecular Weight:	2234.64	
Target:	AMPK; GLUT	MRWQEMGYIFYPRKLR (acetate salt)
Pathway:	Epigenetics; Membrane Transporter/Ion Channel; PI3K/Akt/mTOR	
Solubility:	DMSO : 4 mg/mL (1.79 mM; Need ultrasonic)	

BIOLOGICAL ACTIVITY:

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]. *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 mitochondria 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]. *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].

References:

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

CAIndexNames:

L-Arginine, L-methionyl-L-arginyl-L-tryptophyl-L-glutamyl-L- α -glutamyl-L-methionylglycyl-L-tyrosyl-L-isoleucyl-L-phenylalanyl-L-tyrosyl-L-prolyl-L-arginyl-L-leucyl-L-leucyl-

SMILES:

[MRWQEMGYIFYPRKLR (acetate salt)]

Caution: Product has not been fully validated for medical applications. For research use only.

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