Technical Data Sheet

MOTS-c

Product Information

Alternate Names:	N/A
Size:	10.0mg
Format/Appearance:	Lyophilized, white/off-white powder
Sequence:	NH2 - Met - Arg - Trp - Gln - Glu - Met - Gly - Tyr - Ile - Phe - Tyr - Pro - Arg - Lys - Leu - Arg - COOH
Purity:	>98%
Recommended Diluent:	Bacteriostatic Water

Description

MOTS-c is a peptide of 16 amino acids expressed by a mitochondrial gene. Research by Pinchas Cohen and his colleagues at the Leonard Davis School of Gerontology at the University of Southern California provides evidence that mitochondria play a key role in signaling and in energy production.

MOTS-c is known to regulate metabolic functions throughout the body, including turning glucose into usable energy. The first studies on MOTS-c were conducted on obese mice. They showed that the peptide helped boost glucose metabolism even when the mice were fed a high fat diet. These preliminary studies show evidence for improved control over blood sugar levels for those with type 2 diabetes and obesity.

Cohen's research also shows that skeletal muscle is the major target tissue of MOTS-c. The skeletal muscle enhances insulin sensitivity and increases glucose uptake in myocytes (muscle cells) by activating the AMPK pathway and at the same time without increasing insulin. He also went on to say that it is fair to call MOTS-c an exercise-mimetic, meaning it imitates exercise on the body. Exercise also increases muscle glucose uptake without stimulating insulin.

Indications and Benefit

- Promotes fatty acid metabolism in the liver
- Protects against age and diet dependent insulin resistance and obesity
- Helps accelerate weight loss through glucose regulation

- Improves exercise capacity
- Helps prevent osteoporosis by promoting osteoblast formation

Preparation and Storage

Prior to reconstitution, MOTS-c should be stored at -20C, protected from light. After reconstitution, store at 4C protected from light.

Clinical Research and Related Publications

Kasai, T., Bandow, K., Suzuki, H., Chiba, N., Kakimoto, K., Ohnishi, T., Kawamoto, S.-, Nagaoka, E., & Matsuguchi, T. (2009). Osteoblast differentiation is functionally associated with decreased AMP kinase activity. Journal of Cellular Physiology, 221(3), 740-749. https://doi.org/10.1002/jcp.21917

Kim, S. -. J., Miller, B., Mehta, H. H., Xiao, J., Wan, J., Arpawong, T. E., Yen, K., & Cohen, P. (2019). The mitochondrial-derived peptide MOTS-c is a regulator of plasma metabolites and enhances insulin sensitivity. Physiological Reports, 7(13), https://physoc.onlinelibrary.wiley.com/doi/10.14814/phy2.14171. https://doi.org/10.14814/phy2.14171

Lee, C., Kim, K. H., & Cohen, P. (2016). MOTS-c: A novel mitochondrial-derived peptide regulating muscle and fat metabolism. Free Radical Biology and Medicine, 100, 182-187. https://doi.org/10.1016/j.freeradbiomed.2016.05.015

Lee, C., Zeng, J., Drew, B. G., Sallam, T., Martin-Montalvo, A., Wan, J., Kim, S.-J., Mehta, H., Hevener, A. L., de Cabo, R., & Cohen, P. (2015). The Mitochondrial-Derived Peptide MOTS-c Promotes Metabolic Homeostasis and Reduces Obesity and Insulin Resistance. Cell Metabolism, 21(3), 443-454. https://doi.org/10.1016/j.cmet.2015.02.009

López-Otín, C., Galluzzi, L., Freije, J. M. P., Madeo, F., & Kroemer, G. (2016). Metabolic Control of Longevity. Cell, 166(4), 802-821. https://doi.org/10.1016/j.cell.2016.07.031

Ming, W., Lu, G., Xin, S., Huanyu, L., Yinghao, J., Xiaoying, L., Chengming, X., Banjun, R., Li, W., & Zifan, L. (2016). Mitochondria related peptide MOTS-c suppresses ovariectomy-induced bone loss via AMPK activation. Biochemical and Biophysical Research Communications, 476(4), 412-419. https://doi.org/10.1016/j.bbrc.2016.05.135

MOTS-c improves osteoporosis by promoting osteogenic differentiation of bone marrow mesenchymal stem cells via TGF-β/Smad pathway. (2018). MOTS-c Improves Osteoporosis by Promoting Osteogenic Differentiation of Bone Marrow Mesenchymal Stem Cells via TGF-β/Smad Pathway, https://www.europeanreview.org/article/16247. https://doi.org/10.26355/eurrev_201811_16247

Muoio, D. M. (2014). Metabolic Inflexibility: When Mitochondrial Indecision Leads to Metabolic Gridlock. Cell, 159(6), 1253-1262. https://doi.org/10.1016/j.cell.2014.11.034

Olson, K. A., Schell, J. C., & Rutter, J. (2016). Pyruvate and Metabolic Flexibility: Illuminating a Path Toward Selective Cancer Therapies. Trends in Biochemical Sciences, 41(3), 219-230. https://doi.org/10.1016/j.tibs.2016.01.002

Ramanjaneya, M. (2018). MitochondrialDerived Peptide MOTSc promotes hepatic fatty acid metabolism and regulation by metformin. Qatar Foundation Annual Research Conference Proceedings Volume 2018 Issue 2, https://www.qscience.com/content/papers/10.5339/qfarc.2018.HBPD728. https://doi.org/10.5339/qfarc.2018.hbpd728

Reynolds, J. C., Lai, R. W., Woodhead, J. S. T., Joly, J. H., Mitchell, C. J., Cameron-Smith, D., Lu, R., Cohen, P., Graham, N. A., Benayoun, B. A., Merry, T. L., & Lee, C. (2019). Mitochondrial-Encoded Peptide MOTS-c is an Exercise-Induced Regulator of Aging Metabolic Homeostasis and Physical Capacity. BioRxiv, https://www.biorxiv.org/content/10.1101/2019.12.22.886432v3. https://doi.org/10.1101/2019.12.22.886432

Shah, M., Kola, B., Bataveljic, A., Arnett, T. R., Viollet, B., Saxon, L., Korbonits, M., & Chenu, C. (2010). AMP-activated protein kinase (AMPK) activation regulates in vitro bone formation and bone mass. Bone, 47(2), 309-319. https://doi.org/10.1016/j.bone.2010.04.596

Zarse, K., & Ristow, M. (2015). A Mitochondrially Encoded Hormone Ameliorates Obesity and Insulin Resistance. Cell Metabolism, 21(3), 355-356. https://doi.org/10.1016/j.cmet.2015.02.013