



## Exercise Biochemistry Review

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Proceedings of IBEC 2018, Beijing, China, October 23-25  
PO-273

### Exercise, myocardial insulin resistance and myostatin

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**Objective** Myostatin (MSTN) is a member of the TGF- $\beta$  superfamily and is a negative regulator of skeletal muscle growth. MSTN is also expressed in other tissues such as heart and fat. The purpose of this study was to elucidate the occurrence of myocardial IR caused by myocardial MSTN expression, and to elucidate the regulation of MSTN expression by exercise and improve myocardial IR status and myocardial interstitial fibrosis.

**Methods** The literatures on exercise, myocardial IR, and myocardial MSTN published in recent years were reviewed and summarized.

**Results** 1. Myostatin, which is rarely expressed in myocardium under normal conditions, the appropriate amount of MSTN expression has anti-cardiac hypertrophy, and excessive MSTN expression can cause various cardiovascular diseases. In the IR state, the expression of myocardial inflammatory factors is increased, and MSTN is highly expressed. MSTN acts on the myocardium through various pathways, leading to the occurrence of myocardial fibrosis. In the IR state, possible pathways for inducing high expression of MSTN include: (1) inflammatory factor TNF- $\alpha$  is elevated, and MSTN expression is induced by P38MAPK and NF-KB; (2) The transcription factor Smad-2/3/4 up-regulates the MSTN promoter activity; (3) Glucocorticoid up-regulates MSTN expression; (4) IGF-1 up-regulates MSTN expression via p38MAPK and transcription factor MEF-2; (5) AngII up-regulates MSTN via Ang1 receptor, p38MAPK and MEF-2; (6) urotensin-II and urocortin can stimulate MSTN expression; (7) The ERK-1/2-MEF-2 pathway up-regulates MSTN expression. 3. Possible pathways by which MSTN acts on the myocardium include: (1) MSTN induces Smad2/3 overexpression; (2) MSTN activates the NF-KB pathway; (3) MSTN activates miR-1 expression, inhibits HSP70, and decreases pAkt levels; (4) MSTN inhibits the PI3K-Akt pathway, leading to induction or damage of the downstream pathway. Regular endurance exercise and resistance exercise improve myocardial IR and myocardial interstitial fibrosis involved in MSTN. This effect is mainly dependent on exercise can effectively improve the body's chronic inflammatory state, down-regulate the MSTN superior inducer, resulting in a series of cascade reactions.

**Conclusions** 1. MSTN expression in myocardial tissue is dose-dependent, proper expression of MSTN in the myocardium can prevent cardiac hypertrophy, and overexpression of MSTN can lead to various cardiovascular diseases. 2. Under the hyperglycemia and inflammation state, it can induce high expression of MSTN. After high expression of myocardial MSTN, it can participate in myocardial IR and can lead to myocardial interstitial fibrosis. 3. Exercise can effectively improve the chronic inflammatory state of the body, down-regulate the level of MSTN induction, improve myocardial IR status and IR-induced myocardial interstitial fibrosis.