



## Exercise Biochemistry Review

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### **DDAH1 knockout has a protective effect on muscle damage caused by downhill running**

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**Objective Purpose:** Downhill running can cause muscle damage, called delayed muscle damage and induced oxidative stress and inflammatory reaction, causing abnormality of skeletal muscle morphology, changing in blood biochemical indexes, and decreasing in function of skeletal muscle systolic. Asymmetric dimethylarginine (ADMA), an endogenous nitric oxide synthase (NOS) inhibitor, is degraded by dimethylarginine dimethylaminohydrolase 1 (DDAH1). There were new evidences demonstrated that DDAH1 is an important regulator of cell redox state and apoptosis. In summary, the study shown that DDAH1 is an important regulator of cell redox state and apoptosis. Emerging evidences suggests that DDAH1 controls cellular oxidative stress and apoptosis via a miR-21-dependent pathway. However, the effect and mechanism of DDAH1 on damage of skeletal muscle caused by downhill running is not clear enough. Thus, the purpose of this experiment was to determine the effect and mechanism of DDAH1 in downhill running.

**Keys:** downhill running; delayed onset muscle soreness(DOMS); eccentric exercise; skeletal muscle.

**Methods Method:** The experimental mice were 24 female C57 mice of 10 weeks old and 24 female DDAH1 hybrid knockout mice of 10 weeks old. DDAH1 KO mice used for this study was knockout of dimethylarginine dimethylaminohydrolase 1 compared with WT mice. Animals were fed standard laboratory chow and had access to water ad libitum. C57 mice were divided into 3 groups: C57 control, C57 48H, C57 120H; DDAH1 KO mice were divided into 3 groups: DDAH1 control, DDAH1 48H, DDAH1 120H. C57 and DDAH1 KO mice used for this study completed a single bout of downhill running exercise (20°, 17 m/min, 60 min), and gastrocnemius muscle, soleus muscle and quadriceps femoris muscle were collected 48 and 120 hours (H) postexercise (PE). C57 control group and DDAH1 KO control group dose not exercise. Speed on the treadmill was gradually increased from 10 to 17m/min during a 7-min warm-up period (increased of 1m/min every minute). All experiments were conducted at approximately the same time of day. Maximal grip strength was measured ifor each groups. Grip strength testing was completed to detect post-eccentric exercise injury in C57 and DDAH1 KO mice. All results were analyzed by means of methods of histological and molecular biological.

**Results Method:** The experimental mice were 24 female C57 mice of 10 weeks old and 24 female DDAH1 hybrid knockout mice of 10 weeks old. DDAH1 KO mice used for this study was knockout of dimethylarginine dimethylaminohydrolase 1 compared with WT mice. Animals were fed standard laboratory chow and had access to water ad libitum. C57 mice were divided into 3 groups: C57 control, C57 48H, C57 120H; DDAH1 KO mice were divided into 3 groups: DDAH1 control, DDAH1 48H, DDAH1 120H. C57 and DDAH1 KO mice used for this study completed a single bout of downhill running exercise (20°, 17 m/min, 60 min), and gastrocnemius muscle, soleus muscle and quadriceps femoris muscle were collected 48 and 120 hours (H) postexercise (PE). C57 control group and DDAH1 KO control group dose not exercise. Speed on the treadmill was gradually increased from 10 to 17m/min during a 7-min warm-up period (increased of 1m/min every minute). All experiments were conducted at approximately the same time of day. Maximal grip strength was measured ifor each groups. Grip strength testing was completed to detect post-eccentric exercise injury in C57 and DDAH1 KO mice. All results were analyzed by means of methods of histological and molecular biological.

**Conclusions** Conclusion: The DDAH1 knockout has a protective effect on delayed onset muscle soreness(DOMS) caused by downhill running, and accelerate the injury recovery.