Acute eccentric exercise is known to accelerate connective tissue degradation by increasing activities of matrix metalloproteinases (MMPs) especially those, which are suggested to be involved in basement membrane type IV collagen degradation. Previously it has been shown that prior forced shortening and lengthening contractions may result to faster and more pronounced upregulation in type IV collagen degradation capacity after repeated bout of contractions. The purpose of the present study was to investigate whether type of previous exercise prevents the changes caused by lengthening contractions in type IV collagen degradation capacity after repeated bout of contractions. More specifically to investigate the effects of repeated bout of exercise on the expression of tissue inhibitors of metalloproteinases (TIMP), which are known to inhibit MMP activity. The left TA muscles of 12 week-old male Wistar rats were subsequently unilaterally subjected to 240 forced shortening or lengthening contractions. Fused tetanic contractions were induced by electrical stimulation of the exposed common peroneal nerve (typically with 150 Hz and 3 V) for 300 ms via a stainless steel electrode. Shortening or lengthening of TA muscles were performed by either plantar or dorsiflexion of the foot at the ankle joint with rotational velocity of 500 °/s. Fifteen days later half of the both groups were subjected to second bout of exercise, forced lengthening contractions. The contralateral leg was used as a non-exercised control. Light microscopical examination of transverse sections of TA muscle showed severe skeletal muscle fiber injury 4 days after forced lengthening contractions and both types of repeated bout of exercise (shortening + lengthening or lengthening + lengthening), but only slight histopathological changes after forced shortening contractions. The contralateral leg was used as a non-exercised control. Light microscopical examination of transverse sections of TA muscle showed severe skeletal muscle fiber injury 4 days after forced lengthening contractions and both types of repeated bout of exercise (shortening + lengthening or lengthening + lengthening), but only slight histopathological changes after forced shortening contractions. MMP-2 inhibitory activity of TIMP-2 elevated remarkable 4 days after both forced shortening and lengthening contractions compared to contralateral muscle. TIMP-2 was still elevated 15d and 30d after single bout of exercise. Interestingly, the second bout of exercise (lengthening contractions) 4d after did not cause as remarkable increase as the single bout exercises. Actually, MMP-2 inhibitory activity of TIMP-2 was in the same level as at 15d (baseline level for repeated bout exercise) after single bout exercise, which indicated that the second bout of exercise did not cause changes in TIMP-2 level. Changes in MMP-2 inhibitory activity of TIMP-1 were more moderate than changes in TIMP-2 and the changes seems to be caused mostly by forced lengthening contractions. Results from previous experiment showed that the second bout of exercise does not increase MMP inhibition in the same magnitude as single bout exercise. This indicates that MMP degradation capacity is higher after repeated bout exercise than after single bout exercise, however it can not be excluded that TIMP-2 level might be increased in the contralateral muscle after repeated bout exercise.

Keywords: Eccentric Exercise, Muscle Damage