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High intensity interval cycling performed prior to resistance exercise stimulates autophagy signaling

### **Introduction**

High intensity interval cycling performed prior to resistance exercise potently increases AMPK activity and mRNA expression of the muscle specific E3 ligases MuRF1 and MAFbx, suggesting a link between AMPK activation and muscle breakdown (1). Autophagy, another catabolic process, is also influenced by AMPK. AMPK phosphorylates ULK1 at Ser317 resulting in the activation of autophagy and the subsequent breakdown of proteins. Thus, AMPK-mediated activation of this process may represent a pathway by which training adaptations are modulated in response to various training modes. The aim of this study was therefore to examine if autophagy signaling is activated to a higher degree by concurrent exercise compared to resistance exercise alone.

### **Methods**

Eight male subjects performed two trials in a randomized order. In the ER trial, they performed five 4-min intervals at a work rate of 85% of each subject's maximal oxygen uptake. Fifteen minutes after the last interval, subjects performed 3 warm-up sets after which they performed 10 sets of heavy-resistance exercise; 4 sets of 8–10 repetitions at ~80% 1RM, 4 sets of 10–12 repetitions at ~70% 1RM, and 2 sets to fatigue at ~60% 1RM. In the R trial, the exercise was identical except that the cycling was replaced by rest. Muscle biopsies were sampled at rest before exercise, immediately after cycling in the ER trial and after rest at the corresponding time point in the R trial, immediately after resistance exercise and at 90 and 180 minutes during recovery in both trials. Tissue samples were analyzed for the phosphorylation status of ULK1 at Ser317 using western blot.

### **Results**

After cycling in the ER trial, phosphorylation of ULK1 at Ser317 was increased by ~ 130% compared to before exercise ( $p < 0.05$ ). Phosphorylation of S317 was maintained at a similar level (+110%;  $p < 0.05$ ) immediately after resistance exercise. In the R trial, phosphorylation of Ser317 was increased by ~ 50% ( $p < 0.05$ ) compared to before exercise, but this increase was significantly smaller than that seen at the same time point in the ER trial.

### **Conclusion**

Endurance exercise-induced activation of AMPK increases phosphorylation of ULK1 at Ser317, likely resulting in increased autophagy. These findings provide a novel mechanism by which concurrent exercise may alter training adaptations compared to single mode resistance exercise.

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**References**

1. Apró et al. *Am J Physiol Endocrinol Metab.* 2015 Mar 15;308(6):E470-81