

## **The Effect of 28 Days of Beta-Alanine Supplementation on Repeated-Sprint Ability.**

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**Purpose:** The purpose of this study was to determine the effect of 28 days of beta-alanine supplementation on the magnitude of fatigue, mean power output (MPO), peak power output (PPO), and blood lactate accumulation during a repeated-sprint cycling protocol (10 x 6-second sprints interspersed with 30-seconds of recovery) in active, college-aged males and females. **Methods:** This study was a double-blind placebo controlled study with 9 male and 9 female subjects. Participants performed 10 x 6-second sprints interspersed with 30- seconds of passive recovery on an electromagnetically braked cycle ergometer with a standardized resistance of 70 Nm·kg<sup>-1</sup>. Two familiarization trials were performed and then the pre-supplement baseline trial was performed. Each session was separated by 48 hours minimally. Subjects were randomly assigned to either a placebo group or supplement group based on gender and fatigue from the pre-testing trial. Subjects then underwent 28 days of supplementation with either beta-alanine (6.4 g/day) or placebo. At the conclusion of supplementation, subjects again performed the repeated-sprint protocol. A 2-way ANOVA with repeated measures on one factor (time: pre to post) was used to assess the differences for mean fatigue, mean MPO, mean PPO, mean RPE, and mean delta blood lactate ( $\Delta$  HLA) values. The alpha level for all analyses was set at  $p \leq 0.05$ . **Results:** There were no significant differences for mean fatigue (group:  $p = .538$ , time\*group:  $p = .431$ ), mean  $\Delta$  HLA (group:  $p = .231$ , time\*group:  $p = .092$ ), mean MPO (time:  $p = .139$ , time\*group:  $p = .214$ ), mean PPO (time:  $p = .131$ , time\*group:  $p = .495$ ), mean RPE (time:  $p = .102$ , time\*group:  $p = .976$ ). **Conclusion:** The present study showed that after 4 weeks of beta-alanine supplementation (6.4 g/day) repeated-sprint ability (fatigue, PPO, MPO) was not augmented, nor was the ability to buffer HLA through increased muscle carnosine. This lack of difference may reflect the impact of other mechanisms of fatigue (e.g. PCr depletion; central fatigue) or could be attributed to a lack of adequate buffering by carnosine.