

Effect of Molecular Hydrogen Saturated Alkaline Electrolyzed Water on Disuse Muscle Atrophy in Gastrocnemius Muscle

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Abstract

The objectives of this paper were to determine the level of oxidative stress in atrophied gastrocnemius, and to verify the effect of molecular hydrogen (H_2) saturated alkaline electrolyzed water (HSW) on gastrocnemius atrophy by modifying the redox status, indicated by 8-hydroxy-2'-deoxyguanosine (8-OHdG), malondialdehyde (MDA), and superoxide dismutase (SOD)-like activity. Female Wistar rats were divided into four groups: (1) the control (CONT); (2) the Hindlimb unloading (HU, for 3 weeks) given purified normal water (HU-NW); (3) the HU given alkaline electrolyzed reduced water (HU-AEW); and (4) the HU given HSW (HU-HSW). We showed that 8-OHdG, but not MDA, significantly increased by 149% and 145% in HU-NW and HU-AEW, respectively, when compared with CONT. In contrast, there was a trend toward suppression in 8-OHdG levels (increased by 95% compared with CONT) by treatment of HSW, though this effect was not prominent. Additionally, SOD-like activity significantly increased in both HU-NW (184%) and HU-AEW (199%) when compared with CONT. This result suggests the elevation of $O_2^{\cdot -}$ in the atrophied gastrocnemius. However, upregulation of SOD-like activity in the HU-HSW was increased by only 169% compared with CONT, though this difference is too small to detect statistical significance. HU led to 13% and 15% reduction of gastrocnemius wet weights in HU-NW and HU-AEW, respectively, compared with CONT. And the reduction of gastrocnemius wet weights in HU-HSW was attenuated by 7% compared with CONT. The gastrocnemius wet weights in the HU-HSW group were significantly greater than those in the HU-AEW, but not statistically significant with HU-NW. These results indicate that HU causes an increase in oxidative stress, but,

in this experimental protocol, continuous consumption of HSW during HU does not demonstrate successful attenuation of oxidative stress and HU-mediated gastrocnemius atrophy.

Journal

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