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Sci Rep. 2018 Jul 4;8(1):10128. doi: 10.1038/s41598-018-28510-x.

Stimulating fermentation by the prolonged acceleration of gut transit protects against decompression sickness

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Affiliations PMID: 29973647 PMCID: PMC6031626 DOI: 10.1038/s41598-018-28510-x Free PMC article

Abstract

Massive bubble formation after diving can lead to decompression sickness (DCS). Gut fermentation at the time of a dive exacerbates DCS due to endogenous hydrogen production. We sought to investigate whether medium-term stimulation of fermentation as a result of polyethylene glycol (PEG)-induced acceleration of bowel transit before diving exacerbates DCS in rats. Seven days before an experimental dry dive, 60 rats were randomly divided in two groups: an experimental group treated with PEG (n = 30) and an untreated control group (n = 30). Exhaled hydrogen was measured before the dive. Following hyperbaric exposure, we assessed for signs of DCS. After anaesthetisation, arterial blood was drawn to assay inflammatory cytokines and markers of oxidative stress. PEG led to a significant increase in exhaled H₂ (35 ppm [10-73] compared with control 7 ppm [2-15]; p = 0.001). The probability of death was reduced in PEG-treated rats (PEG: 17% [95% CI 4-41] vs control: 50% [95% CI 26-74]; p = 0.034). In addition, inflammatory markers were reduced, and the antioxidant activity of glutathione peroxidase was significantly increased (529.2 U.I⁻¹ [485.4-569.0] versus 366.4 U.I⁻¹ [317.6-414.8]; p = 0.004). Thus, gut fermentation might have a positive effect on DCS. The antioxidant and neuroprotective properties of the fermentation by-products H₂ and butyrate may explain these results.

Figures

02/07/2023, 19:39

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