NaHCO3 INGESTION INCREASES PH IN BLOOD BUT DOES NOT ATTENUATE EIAH OR ENHANCE PERFORMANCE

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The exact causes of Exercise Induced Arterial Hypoxemia (EIAH) are not yet known. In addition to this the ergogenic effects of Sodium Bicarbonate (NaHCO3) are somewhat uncertain. Earlier studies on the ergogenic effects of NaHCO3 have neglected to investigate the occurrence of EIAH among their subject, something that could explain the conflicting results, EIAH cannot be over looked since reportedly 50% of well trained athletes experience EIAH. One possible ergogenic effect of NaHCO3 would be to attenuate EIAH through an increase in blood pH in a subject. This has been shown previously by means of intravenous infusion during maximal rowing. A possible reason for the conflicting results regarding ergogenic effects of NaHCO3 could be because of subjects experiencing different severity of arterial desaturation during maximal exercise.

The aim of the study was to examine the effect of oral intake of NaHCO3 on EIAH and performance in trained cyclists. Seven male cyclists (age 23.7 (22-27) years, VO2peak 64 (60-72) ml min-1 (kg body mass) -1 volunteered for the study. The subjects performed two maximal exercise tests to exhaustion 48 hours apart in a counter balanced cross over double blind fashion. Subjects received 0.3 g kg BW-1 CaCO3 and 0.3 g kg BW-1 NaHCO3 in the placebo and bicarbonate trial respectively.

Free flowing arterialized capillary blood was sampled at rest and exhaustion and analyzed for pH, O2 Saturation, pO2, pCO2, HCO3-, cBASE (Ecf) and blood lactate. Ventilatory variables were measured continuously throughout the test V'O2, V'CO2, V'E, V'E/VO2, RER and HR. In addition pulse oximetry was used to evaluate O2 saturation. At rest pH and PCO2 was elevated (p<0.05) in the bicarbonate trial compared to the placebo trial. At exhaustion in the bicarbonate trial pH, blood lactate, RER, HCO3-, CBASE (Ecf) was significantly elevated (p<0.05) when compared to the placebo trial. O2 saturation from blood samples at exhaustion in the bicarbonate trial showed a trend towards improving (p=0.061). No difference was seen between the two trials in PO2, VO2peak, V'Emax, V'E/VO2max, HRmax or performance.

During exercise, bicarbonate ingestion increased blood pH but did not improve arterial saturation or performance. The increase in blood pH achieved by ingestion of bicarbonate was not as large as the increase achieved by intravenous infusion in another. Even with the larger increase in blood pH in those studies, there was only a small improvement in performance. One possible explanation for the performance improvement with bicarbonate infusion in that study was a reduced V'E that could effect respiratory muscle work and thereby work capacity. The bicarbonate ingestion in the present study did not reduce ventilation. This could possible be achieved with higher doses of NaHCO3, which would most likely result in increased frequency of gastrointestinal distress among subjects. This highlights the inherent difficulties of oral supplementation of NaHCO3 to attenuate EIAH.

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