Stabilized NADH improves the physical and mental performance in highly conditioned athletes

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Introduction

NADH (nicotinamide adenine dinucleotide hydride) is one of the most important coenzymes present in every living cell. NADH catalyzes more than thousand metabolic reactions the most important of which is the trigger for ATP production. Furthermore, it plays a decisive role in cell regulation and DNA repair as well as stimulator of the cellular immune system. Due to his high redox potential; NADH has an enormous antioxidant capacity. The content of NADH in organ and tissue reflects the need for it. The highest concentration is found in heart cells (90 mcg/g tissue), brain (50 mcg/g tissue) and muscles (50 mcg/g tissue). The organs with the highest amount of energy (heart and brain) contain the highest level of NADH. In an aging organism and in any patient with chronic diseases a certain NADH deficiency and ATP deficiency could be detected. This leads to a decline in the availability of energy of the cells and organs. In vitro as well as in vivo studies showed that the cellular energy metabolism and ATP production can be improved by exogenous NADH. Based on this finding, a study protocol was developed to find out whether the stabilized, orally absorbable form of NADH (ENADAlert) has an energy and performance increase effect.

Method

In a double-blind, placebo-controlled cross-over study, fourteen highly conditioned athletes (18-49 yrs, VO2 max > 55 ml/kg/min) with a constant training and nutritional program were investigated. They received ENADAlert (sublingual form of NADH) or an identically looking placebo tablet. The daily dosage was 30 mg of NADH which was applied for four weeks. After that, a six-week wash-out phase took place and then, in a cross-over design the placebo subject received NADH and the NADH subject received placebo. The maximal aerobic capacity was determined on a treadmill including spiroergometry. As parameter for ventilation, the oxygen uptake (VO2 in ml/min) and the carbon dioxide exhalation (VCO2 in ml/min) were measured. In addition, the heart frequency as well as the lactate levels in capillary blood was determined. The respiratory ratio (RQ) was calculated. The subjects were examined in a standard treadmill with a long-term endurance test in which a steady state at 7% of the individual VO2 maxes over 14 minutes to place. For the entire test period, the parameters for ventilation VO2 and VCO2 were determined. The data of the cross-over design were evaluated by non-parametric statistical procedures (Mann-Whitney-Wilcoxon-test). Approvability of P<0.05 was regarded as statistical significant.

Results

Subjects received 30 mg/day over a period of four weeks. Neither side-effects nor changes in all clinical chemical and hematological parameters were observed. No drop-outs did occur. Changes in the training condition or well-being have not been found in the diary. Under NADH, a reduced uptake of oxygen and 6.2% (base is 0.07; 42.8 vs. 40.2ml/kg/min) could ascertain this treatment effect. This reduction of oxygen consumption could also be found by using the RQ in the aerobic transition phase (VO2 values around 3000ml). If the individual vales for VCO2 and VO2 per breath stroke are inserted in a scattergram and evaluated, a coefficient could be calculated which differs in the subjects taking NADH, than in the subjects taking placebo. An O2 sparing effect of 5.9% was found under supplementation with NADH. The heart frequency and lactate level in the blood were identical between the placebo and the NADH group. However, in the endurance trial under aerobic steady state condition, a 14% lower lactate level was found in the NADH group. (p =0.07, 1.67 vs.1.43
mmol/l). The additional gain in energy supply is most likely due to an increase of the ATP production. ENADAAlert™, the stabilized, sublingually absorbed form of NADH can increase muscular energy in athletes by an average of 7%. The decrease in lactate levels after intake of NADH implies that athletes can perform longer under aerobic physiological conditions.

Discussion

**In vitro** studies have shown that NADH does influence the metabolism of a cell, in particular the production of NADH. In a double-blind, placebo-controlled, FDA approved clinical trial it has been demonstrated that ENADA/NADH can improve the energy level of subjects suffering from chronic fatigue syndrome (CFS). In another study, it was found that patients suffering from chronic fatigue syndrome do show an ATP deficiency in their muscle tissue after physical exercise as measured by nuclear magnetic resonance. The question this study has answered was: Can highly conditioned athletes from whom one would assume that they have a maximum reserve in energy, still gain an increase? In order to achieve this, NADH must enter the cell and must reach the target in the cell where ATP is produced. ENADA, the stabilized, orally absorbable form of NADH, is absorbed in the intestinal tract. NADH penetrates the intestinal mucosa by passive diffusion. ENADA also passes the blood-brain-barrier as shown by studies performed in rats. 20 minutes after oral application of ENAD/NADH to rats, an increase of NADH in the rat brain court was detected by laser induced NADH fluorescence. There is also evidence that NADH must penetrate the cell membrane and possibly also the mitochondrial membrane. When pheochromacytoma cells (PC12 cells) are incubated with NADH in the culture medium, an increase in mitochondrial membrane potential can be detected. This finding provides indirect evidence of a higher energetic state of the cell. Even more convincing evidence could be obtained in isolated single hart cells. If these cells are incubated with NADH, a dosage-dependent increase in ATP concentration in the cell can be found. From this observation one can deduce that NADH must pass the cell membrane to induce ATP production. This is the most likely mechanism by which ENADA/NADH leads to an increase in energy state in muscle tissue. The reduction of the oxygen consumption and a change in the CO₂/O₂ scattergram indicates an improved cellular bioavailability of NADH and due to this to an increased ATP supply. This mechanism of action is supported by the reduction of the lactate level in the blood under NADH treatment. If highly conditioned athletes gain on average 7% more muscular energy by NADH, one can calculated on the basis of well-known parameters that healthy non-athletic individuals may achieve a gain in energy of 25%. As a constant decline in energy production is observed in elderly people, the energy level of these individuals could be increased considerably by supplementing with ENADA/NADH. Finally, an application in the field of so-called mitochondrial diseases such as Parkinson’s and Alzheimer’s disease could be considered. In those ailments, the energy production in the mitochondria is disturbed and reduced. In a number of controlled clinical trials it could be shown that ENADA improve symptoms of patients with Parkinson’s disease and Alzheimer’s disease. Therefore ENADA the stabilized, orally absorbable form of NADH may be helpful for a number of bioenergetic related ailments.

References