Effect of dehydroepiandrosterone (DHEA) treatment on oxidative energy metabolism in rat liver and brain mitochondria. A dose-response study.

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Abstract
OBJECTIVES: Effects of treatment with dehydroepiandrosterone (DHEA) on oxidative energy metabolism in rat liver and brain mitochondria were examined. DESIGN AND METHODS: Young adult rats were administered DHEA (0.1, 0.2, 1.0 or 2.0 mg/kg body weight) by subcutaneous route for 7 consecutive days. RESULTS: DHEA treatment resulted in general, in stimulation of state 3 respiration rates without having any uncoupling effect on ADP/O ratios. The stimulation of state 3 respiration rate for a given substrate was dose dependent in a tissue-specific manner. Parallel increases in the contents of cytochromes aa(3) and b were also noted. DHEA treatment stimulated the glutamate dehydrogenase (GDH) and succinate DCIP reductase (SDR) activities. Under the treatment conditions, mitochondrial ATPase activity was also stimulated. CONCLUSIONS: Treatment with DHEA significantly stimulated oxidative energy metabolism in liver and brain mitochondria.

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Treatment with dehydroepiandrosterone (DHEA) stimulates oxidative energy metabolism in the cerebral mitochondria. A comparative study of effects in old and young adult rats.

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Abstract
The content of the neurosteroids, dehydroepiandrosterone (DHEA) in the brain decreases with aging. Also the oxidative energy metabolism is known to decrease with aging. Hence we examined the effects of treatment with DHEA (0.2 or 1.0 mg/kg body weight for 7 days) on oxidative energy metabolism in brain mitochondria from old and young adult rats. State 3 respiration rates in brain mitochondria from old animals were considerably lower than those in young adults. Treatment with DHEA stimulated state 3 and state 4 respiration rates in both the groups of the animals in a dose-dependent manner. In the old rats following DHEA treatment, the state 3 respiration rates became comparable to or increased beyond those of untreated young adults. In contrast to the old rats, stimulatory effect of DHEA treatment was of greater magnitude in the young adults. However, at higher dose (1.0 mg) the effect declined. Cytochrome a3 content in the brain mitochondria from old rats was significantly low but the content of cytochrome b was unchanged while the content of cytochromes c+c1 had increased. Treatment with DHEA increased the content of cytochrome a3 and b in old as well as in young adult animals. Higher dose of DHEA (1.0 mg) had adverse effect on the content of cytochrome c+c1. DHEA treatment stimulated ATPase activity in a dose-dependent manner in young adult rats whereas in the old rats the effect on ATPase activity was marginal. Dehydrogenases activities were somewhat lower in the old rats. DHEA treatment stimulated mitochondrial dehydrogenases activities in both the groups. Results of our studies suggest that judicious use of DHEA treatment can improve oxidative energy metabolism parameters in brain mitochondria from young adult as well as old rats.

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