low toxicity profile and to a long history of safe use. Besides known to have a null or very weak estrogenic activity in estrogen receptor assays *in vitro*, parabens were demonstrated to affect testosterone levels and sperm counts in adult rodents and to decrease the number of elongated spermatids. The aim of the present study was to evaluate the effect of methyl-, ethyl-, propyl-, butylparaben and the main metabolite (*p*-hydroxybenzoic acid) on mitochondria isolated from rat testis. The results obtained demonstrate that the metabolite does not affect state 3 and state 4 respiration, although paraben toxicity increases with the length of alkyl grouping from methyl to *n*-butyl. Mitochondrial membrane potential was decreased by propyl and butylparaben but it was not affected by methyl or ethylparaben. We also investigated the ability of parabens to induce the MPT pore, as defined by the massive release of Ca^{2+} by mitochondria in the suspension, following repeated Ca^{2+} pulses. Specifically, we measured the total mitochondrial Ca^{2+}accumulation necessary to open the MPT pore in the presence of different parabens. Increased susceptibility to the mitochondrial permeability transition was correlated with the length of alkyl grouping from methylparaben to *n*-butylparaben. The effect on respiratory complexes II–III, IV and V were also investigated but no significant alterations were observed in the range of concentrations used (0–0.25 mM). From these results, we conclude for the first time that parabens can interfere with testis mitochondrial function. Therefore, inhibition of testis mitochondrial function could interfere negatively with the male reproductive capacity.

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S10.14 Skeletal muscle UCP3 gender dimorphism in high-fat-diet-induced insulin resistance in aged rats

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We investigated whether the gender dimorphism found in mitochondrial function and oxidative stress leads to differences in the development of high-fat-diet-induced insulin resistance in rat skeletal muscle. Fifteen-month-old male and female rats were fed with a high-fat diet (HFD) for 14 weeks. Oral glucose tolerance test was performed. Serum glucose, insulin and adipokine levels were measured. Oxygen consumption, H_2O_2 production and COX, GPx, GRd and Mn-SOD activities were determined in gastrocnemius muscle mitochondria. Catalase activity, TBARS, protein carbonyl groups, UCP3 and GLUT4 levels were measured in muscle homogenate. Control male rats showed a more marked insulin resistance status than females. HFD induced an increase in both muscle mitochondrial H_2O_2 production and in oxidative damage, together with a decrease in the Mn-SOD activity in both genders. However, HFD fed female rats showed a less marked insulin resistance profile than males, and higher mitochondrial oxidative capacity and UCP3 and GLUT4 protein levels. These results point to a gender dimorphism in the insulin resistance status and in the response of skeletal muscle to HFD feeding which could be related to a more detrimental effect of age in male rats.

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S10.15 The high-fat-diet effect on rat liver mitochondrial biogenesis is sex-dependent

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The aim of this study was to investigate the sex-related differences in rat liver mitochondrial biogenesis in response to a high-fat-diet-induced oxidative stress. Ten-week-old male and female Wistar rats were fed with a pelleted standard diet (control group), or with a cafeteria-diet (HFD group) for 26 weeks. HFD rats had free access to a variety of highly palatable foods: cookies, pork liver paté, fresh bacon, chocolate, ensaïmada (a typical Majorcan pastry) and pelleted standard chow. Body weight was assessed once a month; food and energy intake and whole body respirometry were analyzed at the end of the dietary treatment. Mitochondria oxidative capacity, superoxide dismutase and glutathione peroxidase activities, glutathione levels and oxidative damage markers were measured to confirm the high-fat-diet-induced oxidative stress status. Akt and TFAM protein levels, as markers of mitochondrial biogenesis and differentiation, were also analyzed. Liver mitochondria of female rats showed a higher hydrogen peroxide production and an enhanced antioxidant capacity than those of males. The response to the HFD seems to be different between genders. Thus, female rats could counteract better the oxidant effect of the HFD than males, maintaining higher levels of mitochondrial differentiation than males.

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S10.16 Ca^{2+}-induced reactive oxygen species production in alpha-glicerophosphate supported mitochondria

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Alpha-glycerophosphate dehydrogenase (α-GPDH) is localized on the outer surface of mitochondrial inner membrane and plays a role in the cytosolic-mitochondrial shuttle of reducing equivalents. Earlier we have shown that oxidation of alpha-glycerophosphate (α-GP) is able to induce reverse electron transport (RET) in brain mitochondria and RET is associated with an accelerated Reactive Oxygen Species (ROS) production. In the present study the effects of Ca^{2+} were studied on the H_2O_2 production in brain mitochondria respiring on α-GP. H_2O_2 formation was measured by the Amplex method. It is shown that in the presence of ADP micromolar concentrations of Ca^{2+} can stimulate α-GPDH-dependent ROS production. ADP prevented opening of mitochondrial permeability transition pore (mPTP) and *via* decreasing the mitochondrial membrane potential ($\Delta\Psi m$) inhibited the RET mediated ROS production. Elevation of calcium concentration up to 5 μM stimulated ROS production and elevated mitochondrial $\Delta\Psi m$. Higher calcium concentrations decreased $\Delta\Psi m$ and also decreased H_2O_2 formation. Blocking the Ca^{2+} uniporter with Ru360 prevented the depolarizing effects of high [Ca^{2+}] and maintained high ROS production. These results show that alpha-glycerophosphate shuttle combined with cytosolic calcium elevation can increase ROS production in brain mitochondria.

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