corresponding to the distance between the lamellae which centers around 220 Å. At swelling of heart mitochondria (90 mOsm) the ordering sharply decreases and the interference peak becomes negligible. Hereby it was proved that under conditions of low-amplitude swelling of heart mitochondria the enzymes of respiration system and ATP-synthesis system function as supercomplex and these functional changes are accompanied by the changes of inner mitochondrial membrane ultrastructure.

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S2.P5

Adaptive reprogramming of brain mitochondrial biology during preconditioning prevents the sporadic Alzheimer's disease-like phenotype

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Brief episodes of sublethal hypoxia reprogram brain response to face subsequent lethal stimuli by triggering adaptive and pro-survival mechanisms — a phenomenon denominated by hypoxic preconditioning (HP). Notably, HP effectively prevents sporadic Alzheimer's disease (sAD)-related pathological features including cognitive decline and cerebral hypometabolism in the sAD rat model induced by the intracerebroventricular administration of streptozotocin (STZ). However, a deeper knowledge on the protective molecular mechanisms underlying brain tolerance is still required. Given the importance of mitochondria in determining cell fate, the present study was devoted to monitor the structural and functional alterations of brain mitochondria in response to a well-established protocol of HP induced by the cyclic exposure to moderate hypoxia (2 h of 10% O_2) with intervening 24 hour reoxygenation periods, during 3 consecutive days. Several parameters related with mitochondrial bioenergetic function, biogenesis, and fusion and fission machinery were evaluated in the cortex and hippocampus of rats immediately, 6 and 24 h after the last hypoxic session. HP induced a decrease in respiratory state 2 and an increase in ADP/O ratio in brain cortical and hippocampal mitochondria. Immediately after the last hypoxic episode, a significant increase in the protein levels of nuclear respiratory factor-1 (NRF-1), and mitochondrial transcription factor A (TFAM) was observed. 24 h after the last hypoxic episode, a shift in the mitochondrial fusion–fission balance towards fusion occurred, as evidenced by the significant increase in optic atrophy protein 1 (OPA1) protein levels and a decrease in dynamin-like protein 1 (DRP1) protein levels in the brain cortex, and the significant reduction in the fission protein 1 levels in the hippocampus. Consistently, the electron microscopy analysis revealed HP generated mitochondria with an elongated phenotype. Overall, these results indicate that HP enhances mitochondrial bioenergetic function, probably due to a coordinated interplay between mitochondrial biogenesis and fusion/fission events, increasing brain tolerance. This work is supported by Alzheimer's Association (NIRG-13-282387). Sónia C. Correia has a post-doctoral fellowship from the Fundação para a Ciência e a Tecnologia (SFRH/BPD/84163/2012).

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S2.P6

Effects of ocean acidification and warming on the mitochondrial physiology of Atlantic cod

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The Atlantic cod (*Gadus morhua*) is an economically important marine fish species exploited by both fishery and aquaculture, especially in the North Atlantic and Arctic oceans. Ongoing climate changes are happening faster in the high latitude oceans with a higher increase of temperature and a steeper decrease in water pH due to anthropogenic CO_2 than in the temperate regions threatening the existence of the Atlantic cod in the areas of its maximum exploitation. In this study, we investigated the mitochondrial physiology of two life-stages of cod under the sea water temperatures and pCO_2 conditions forecasted for the year 2100 in the North Atlantic (+5 °C, 1000 μatm CO_2). In embryos, the metabolism during development showed to be sensitive to rising temperatures with a general increase in respiratory activity until 9 °C (5 °C over the natural range) and a drop in activity at 12 °C mainly caused by a dramatic decrease in Complex I activity, which was not compensated by Complex II. In the adults, already well known for their metabolic plasticity, mitochondria from liver and heart are not affected by either increasing temperature or pCO_2. However, in heart mitochondria of animals that were reared under warm hypercapnia (10 °C + 1000 μatm CO_2), we found OXPHOS to exploit already 100% of the ETS capacity. This suggests that a further increase in temperature or pCO_2 might lead to a mismatch in the ATP demand/production and consequently decrease heart performances. The different mitochondrial plasticities of the two life-stages reflect the sensitivity range at population level and thus can provide a more realistic reading frame of the potential survival of the North Atlantic cod population under climate change.

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S2.P7

Insulin induces cristae remodeling by decreasing complex I and increasing UCP1 expression in rat brown adipose tissue

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Brown adipose tissue (BAT) has an important role in maintaining energy balance throughout mitochondrial uncoupling e.g. thermogenesis. Insulin is one of the major hormones involved in BAT physiology, but its role is still controversial. Recent evidence pinpoints mitochondrial dysfunction in brown adipocyte as an underlying cause of decreased insulin sensitivity and thus disturbed bioenergetics. The mitochondrial function or dysfunction ensuring that changes in complexes were coordinated with the activation of mitochondrial remodeling pathway was characterized by changes in inner membrane morphology and organization. Considering that cristae remodeling is pivotally involved in mitochondrial