Metabolic alterations such as hyperglycemia and insulin resistance are associated with cognitive decline and Alzheimer's disease (AD). This study was aimed to compare the effects of AD and sucrose-induced metabolic alterations on brain mitochondrial bioenergetics and oxidative status. Three groups of experimental animals were used: wild type (wt) control mice, wt mice treated with 20% sucrose-sweetened water for 7 months and 3xTg-AD mice. Wt mice without access to sucrose-sweetened water were used as "control" group. Several parameters were evaluated: respiratory chain and oxidative phosphorylation system, calcium fluxes, mitochondria ultrastructure, ATP, hydrogen peroxide (H$_2$O$_2$) and glutathione levels, and mitochondrial aconitase, superoxide dismutase (SOD), glutathione peroxidase (GPx) and glutathione reductase (GR) activities. Sucrose intake induced several metabolic alterations including an increase in glucose, glycated haemoglobin (HBA1c) and triglycerides levels. Mitochondria isolated from 3xTg-AD and sucrose-treated mice showed an impairment in the respiratory chain and oxidative phosphorylation system, a decreased capacity to accumulate and retain calcium and ultrastructural alterations characterized by mitochondria enlargement and cristae rupture. Furthermore, an increase in GPx activity and a decrease in GR activity and vitamin E levels were observed in both groups of animals. Altogether our results show that the metabolic alterations induced by sucrose intake promote brain mitochondrial abnormalities similar to those found in 3xTg-AD mouse model of Alzheimer's disease suggesting that metabolic alterations increase the risk of developing AD. This study is supported by Fundação para a Ciência e a Tecnologia (FCT) (PTDC/SAU-NEU/103325/2008). Cristina Carvalho has a PhD fellowship from the FCT (SFRH/BD/43965/2008)