Figure S1. Schematic representation of doxorubicin-induced oxidative and nitrosative stress and mitochondrial Cx43 involvement. Doxorubicin administration induces an increase in ROS production, that causes both the activation of the NF-κB pathway and mitochondria-mediated apoptosis. This leads to cell death and cardiotoxicity. The inhibition of Cx43 translocation to mitochodria further increases these effects.