Supporting Information

Dhingra et al. 10.1073/pnas.1414665111

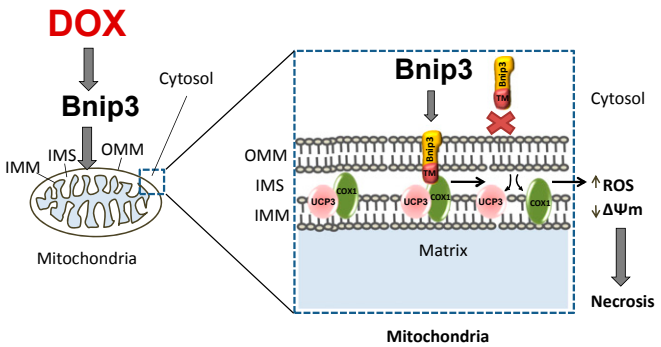

Fig. S1. Model for DOX-induced mitochondrial respiratory chain defects and necrotic cell death of cardiomyocytes. DOX results in the activation and mitochondrial targeting of Bnip3. The integration of Bnip3 into mitochondrial membranes disrupts interaction between COX1 and UCP3. This promotes ROS formation, loss of ΔΨM, mPTP opening, and necrotic cell death of cardiac myocytes. Interventions that block integration of Bnip3 into mitochondrial membranes prevents disruption of COX1-UCP3 complexes and suppresses DOX-induced mitochondrial respiratory defects, ROS production, and necrotic cell death.