

Identification of Proteins Interacting with the Mitochondrial Phosphate Carrier During Necrosis Due to a Myocardial Infarction.

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During necrosis due to cardiac ischemia/reperfusion, there is excess reactive oxygen production, calcium overload, and permeabilization of the inner mitochondrial membrane, thus dissipating the electrochemical gradient and resulting in ATP depletion, increased ROS production, mitochondrial swelling, and rupture. This phenomenon of mitochondrial permeability increase is mediated by a proposed mitochondrial permeability transition (MPT) pore spanning the inner and outer mitochondrial membranes. In order to identify components interacting with a proposed element of the mitochondrial pore, a phosphate carrier, we cloned the domains of the phosphate carrier to ras. Using a yeast-two hybrid model, we began screening a cDNA library fused with a membrane localization signal. Interaction between the phosphate carrier and proposed interacting elements localizes ras to the membrane and allows yeast to grow. These clones provide additional tools to study mitochondrial permeability during necrosis, and the yeast-two hybrid model should generate components involved in the MPT pore.