Cardiac fibroblast death by ischemia/reperfusion is partially inhibited by IGF-1 through both PI3K/Akt and MEK-ERK pathways

Vivar, Raúl

Humeres, Claudio

Varela, Marcelo

Ayala, Pedro

Guzmán, Nancy

Olmedo, Ivonne

Catalán, Mabel

Boza, Pía

Muñoz, Claudia

Díaz Araya, Guillermo

Cardiac fibroblast (CF) death by ischemia/reperfusion (I/R) has major implications for cardiac wound healing. Although IGF-1 has well-known cytoprotective effects, no study has been done on CF subjected to simulated I/R.Simulated ischemia of neonate rat CF was performed in a free oxygen chamber in an ischemic medium; reperfusion was done in normal culture conditions. Cell viability was evaluated by trypan blue assay, and apoptosis by a FACS flow cytometer; p-ERK-1/2 and p-Akt levels were determined by western blot.We showed that simulated I/R triggers CF death by necrosis and apoptosis. IGF-1 partially inhibits I/R-induced apoptosis. PD98059 and LY294002 neutralize the preventive effects of IGF-1.Conclusion: IGF-1 partially inhibits CF apoptosis induced by simulated I/R by PI3K/Akt- and MEK/ERK1/2-dependent signaling pathways.