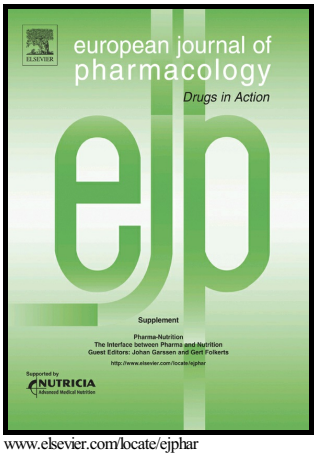

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ABSTRACT

Myocardial ischemia is one of the main causes of sudden cardiac death worldwide. Depending on the cell type and stimulus, ERK activity mediates different anti-proliferative events, such as apoptosis, autophagy, and senescence. The aim of this study was to determine the protective effect of 1,4-diamino-2,3-dicyano-1,4-bis[2-aminophenylthio] butadiene (U0126), an ERK kinase inhibitor, on myocardial ischemia/reperfusion (I/R) injury and the mechanisms involved. An I/R model was established in vivo in C57BL/6 mice and in vitro using mouse cardiomyocytes, respectively. To evaluate the protective effects of U0126 on I/R injury, we measured the myocardial infarct area, apoptosis, and autophagy. Our data indicated that pretreatment with U0126 significantly reduced the infarct area caused by I/R. Moreover, U0126 reduced the caspase-3 activity and the number of TUNEL-positive cardiomyocytes,