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TITLE

Diallyl trisufide (DATS) suppresses high glucose-induced cardiomyocyte apoptosis by inhibiting JNK/NFκB signaling via attenuating NADPH oxidase-derived ROS generation

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Hyperglycemia is an important risk factor for cardiovascular diseases. High glucose-induced generation of reactive oxygen species (ROS) can lead to diabetic cardiomyopathy. In our previous study, we showed that NADPH oxidase-derived ROS-induced apoptosis is mediated via the JNK-dependent activation of NF-κB in cardiomyocytes exposed to high glucose (HG). In this study, we investigated the mechanisms governing the anti-apoptotic effect of diallyl trisulfide (DATS) on HG-exposed cardiac cells both *in vitro* and *in vivo*. H9c2 cells were incubated with media containing 5.5 or 33 mM of glucose for 36hr in the presence or absence of DATS. We found that DATS treatment led to a dose-dependent decrease in ROS levels as well as protein levels of p22, gp91, phosphorylated JNK, and phosphorylated c-Jun. In addition, DATS inhibited the HG-induced activation of caspase 3 as well as the nuclear translocation of NF-κB. Similar results were observed in HG-exposed neonatal primary cardiomyocytes and streptozotocin-treated diabetic rats. Echocardiographic data showed that DATS administration led to a marked increase in fractional shortening and cardiac output. Therefore, DATS appears to suppress high glucose-induced cardiomyocyte apoptosis by inhibiting NADPH oxidase-derived ROS and its downstream JNK/NF-κB signaling.