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KMUP-3 suppressed high glucose-induced apoptosis in cultured cardiomyocytes

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Background: Diabetic cardiomyopathy increases the risk for the development of heart failure independent of coronary artery disease and hypertension. Hyperglycemia-induced oxidative stress and apoptosis have been implicated in the pathogenesis of diabetic cardiomyopathy. Our recent studies indicated that KMUP-3 can induce autophagy in cardiomyocytes. Aims: This study aimed to investigate whether KMUP-3's promotion of autophagy activity can prevent high glucose-induce (HG) cardiac injury. Methods: primary cultures of neonatal rat cardiomyocytes were treated with high glucose and used as a diabetic cardiomyopathy model. Results: The results showed that KMUP-3 treatment attenuated HG-induced cell death by MTT assay. Additionally, KMUP-3 also inhibited HG-induced apoptosis, with associated increase of Bcl-2 protein, and decrease of Bax protein and caspase-3 cleavage. Microtubule-associated protein I light chain 3-II (LC3-II) is the key protein associated with autophagy. As expected, KMUP-3 pretreatment dose-dependently reduced the HG-induced decrease of LC3-II, Atg7, and phosphor-AMPK expression. In conclusion, KMUP-3 inhibited high glucose-induced apoptosis in rat cardiomyocytes through activating autophagy.