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THE EFFECT AND MECHANISM OF B-BLOCKER, BISOPROLOL, ON THE HYPERTROPHY OF CARDIOMYOCYTES OF NEONATAL RATS INDUCED BY HIGH GLUCOSE LEVELS

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Objective To study the effect of β -blocker, bisoprolol on the hypertrophy of cardiomyocytes of neonatal rats induced by high glucose levels, and investigate the mechanism of PKC signal transduction pathway on the structure, function alterations of cardiomyocytes cultured in high glucose levels.

Methods Using cultured neonatal cardiac myocytes as a model, groups were divided into: control group (glucose level 5 mmol/l); high glucose group (glucose level 25.5 mmol/l); high glucose (glucose level 25.5 mmol/l)+bisoprolol (10 mmol/l); high glucose (glucose level 25.5 mmol/l)+bisoprolol (40 mmol/l); high glucose (glucose level 25.5 mmol/l)+NF- κ B inhibitor (BAY11-7082, 5 mmol/l). The cardiomyocytes were cultured for 48 h in different groups after adding corresponding treat factors, the cellular contracting frequency was counted, the cardiomyocytes' diameters were measured and the expression of PKC α , PKC β_2 , p-PKC α , p-PKC β_2 , NF- κ B, TNF α and c-fos were measured by western blot and RT-PCR.

Results Compared with control group, neonatal cardiomyocytes cultured in high glucose concentration showed an increased pulsatile frequency, cellular diameters and higher expression of PKC α , PKC β_2 , p-PKC α , p-PKC β_2 , NF- κ B, TNF α and c-fos, β -blocker, bisoprolol, and NF- κ B inhibitor BAY11-7082 could reverse these changes induced by high glucose concentration.

Conclusions High glucose levels could induce hypertrophy of cardiomyocytes, β -blocker, bisoprolol can reverse the effect of high glucose on the cardiac myocytes; it may have a protective effect on diabetic cardiomyopathy via PKC/NF- κ B/c-fos passageway.