

In cells treated with low glucose (LG, 5 mM), LDH release 12h after hypoxia in A7r5 and H9c2 (80 ± 2 and $56.6 \pm 2.3\%$, respectively) was greater than in high glucose-treated cells (HG, 22 mM) (35.2 ± 2 and $26.2 \pm 0.7\%$, respectively), indicating the protective effect of HG. HG-treatment for 48h decreased the number of TUNEL-positive cells. These protective effects of HG was significantly abolished by 1h pretreatment before hypoxic insult with a PKC inhibitor chelerythrin (3, 10 μ M) in both H9c2 and A7r5 cells. The treatment with 4 β -phorbol 12-myristate 13-acetate (PMA, 10 nM), a PKC activator, mimicked the protective effect of HG. In conclusion, the present results suggest that PKC may play a role in the protective effect of HG against hypoxic cell death, although it remains to be further investigated which isotype of PKC is associated.

[PB1-5] [10/20/2000 (Fri) 15:30 - 16:30 / [Hall B]]

High Glucose Protects Cardiomyocyte against Hypoxic Injury through the Enhancement of Mitochondrial function

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Mitochondria is known as an essential organelle for cellular survival by regulating energy production, Ca²⁺ homeostasis and reactive oxygen species(ROS) generation. Recently it has been reported that high glucose enhances mitochondrial function, producing a cytoprotective effect against various insults. However, it is not known about the effect of high glucose on the function of cardiac mitochondria. Previously we demonstrated that high glucose (HG) can produce cytoprotective effect against hypoxic injury in H9c2 cardiomyocyte. In the present study, we investigated whether HG modulates mitochondrial function and if so, whether this is related to HG-induced cardioprotective effect.

H9c2 cell line was treated with low glucose(LG, 5.5mM) or high glucose(HG, 22mM) for 48h before hypoxia, and then exposed to hypoxic condition (glucose-free, serum-free, 85% N₂, 5% CO₂, 10% H₂). The signal of 2',7'-dichloro-fluorescein diacetate(DCF-DA) was measured for reactive oxygen species(ROS) production, MTT for mitochondrial redox potential and rhodamine123 for mitochondrial membrane potential (MMP).

In LG-treated cells, ROS generation was markedly increased by hypoxic insult, while much less in HG-treated cells. The signal of MTT was significantly decreased in LG-treated cells but not much in HG-treated cells. The magnitude of decrease in MMP by hypoxic insult was greater in HG-treated cells than that in cells with LG. Taken together, these results suggest that HG enhances mitochondrial function with a resultant reduction of hypoxia-induced oxidative stress, and that this may be involved in HG-induced cardioprotective effect against hypoxia-induced cell death.

[PB2-1] [10/20/2000 (Fri) 15:30 - 16:30 / [Hall B]]

Structure Activity Relationship of phenylpropanoids on Anti-inflammatory and Analgesic Activity

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To evaluate structure-activity relationship of phenylpropanoids in anti-inflammatory and analgesic activity, phenylpropanoid, such as cinnamic acid, p-coumaric acid, caffeic acid, ferulic acid, sinapinic acid and chlorogenic acid, were studied in carrageenan and phorbolmyristate acetate (PMA)-induced edema and acetic acid-induced writhing syndrome. Sinapinic acid among six phenylpropanoids has the most effect anti-inflammatory activity : $39.0 \pm 2.7\%$ at carrageenan-