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Meeting abstract

Electrophysiological characteristics of heart ventricular papillary muscles from histidine decarboxylase knockout and wild-type mice: effects of rosiglitazone

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Background

Thiazolidinediones (troglitazone, rosiglitazone), synthetic peroxisome proliferator-activated receptor agonists, act as insulin sensitizers but beyond their antidiabetic actions improve cardiac function in experimental animals.

Aim and methods

Our first aim was to characterise the electrophysiological parameters of right ventricular papillary muscles from histidine decarboxylase knockout (HDC KO) mice compared with those of wild-type (WT) by standard microelectrode technique. Furthermore we investigated the effects of rosiglitazone (1, 3, 30 μM) on transmembrane action potentials.

Results

In KO mice statistically significant prolongation of action potential duration (APD) and decrease in maximum rate of rise of depolarisation phase (V_{max}, dV/dt) can be observed. Rosiglitazone caused a concentration-dependent shortening of APD in both types of mice but reduced V_{max} only in WT mice.

Conclusion

The most important difference in the electrophysiological parameters (APD, V_{max}) between HDC KO and WT mice could be due to the fact that HDC KO mice are more susceptible for hyperglycaemia. The results also suggest that rosiglitazone might act on K^+ channels and this effect might take part in the protective effect of rosiglitazone in ischemia/reperfusion injury observed in rats, but further, direct ionic current measurements need to support this explanation.

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