

Meeting abstract

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## Electrophysiological characteristics of heart ventricular papillary muscles in diabetic histidine decarboxylase knockout and wild-type mice

Andrea Szebeni\*<sup>1</sup>, András Falus<sup>2</sup> and Valéria Kecskeméti<sup>1</sup>

Address: <sup>1</sup>Department of Pharmacology and Pharmacotherapy, Semmelweis University, 1089 Budapest, Hungary and <sup>2</sup>Department of Genetics, Cell and Immunobiology, Semmelweis University, 1089 Budapest, Hungary

Email: Andrea Szebeni\* - [szeband@pharma.sote.hu](mailto:szeband@pharma.sote.hu)

\* Corresponding author

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### Background

Diabetes-induced action potential (AP) abnormalities have been studied mainly in rats where significant prolongation of repolarization and reduced maximum rate of depolarization ( $V_{max}$ ) was detected. Histidine decarboxylase knockout (HDC-KO) mice lack endogenous histamine and they are characterized by impaired glucose tolerance. Furthermore, they have autoantibodies reactive to glutamic acid decarboxylase (GAD). These findings suggested that this model might have an increased susceptibility to autoimmune diabetes.

### Methods

A standard microelectrode technique was used to characterise the cardiac electrophysiological parameters of control and streptozotocin (STZ)-induced diabetic HDC-KO mice compared with those of wild-type animals.

### Results

With aging, blood glucose levels in HDC-KO mice were shifted towards values characteristic of diabetes. The electrophysiological changes relevant to diabetes, i.e. prolongation of repolarization and depression of  $V_{max}$  developed without any induction by STZ. In this group, STZ treatment caused no further significant AP changes.

### Conclusion

One of the likely explanations may be that in the chain of events in HDC-KO mice on the one hand and in STZ-induced diabetes on the other hand, leading to the alterations in the heart electrophysiological parameters, there is a common link. This link may be a similar shift in the expression/function of certain  $K^+$  channel populations.

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