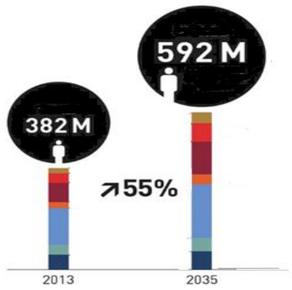

Neuroprotective proline-containing dipeptide Nooopept ameliorates NGF and BDNF deficit in pancreas and liver caused by diabetogenic toxin streptozotocine on Wistar rats

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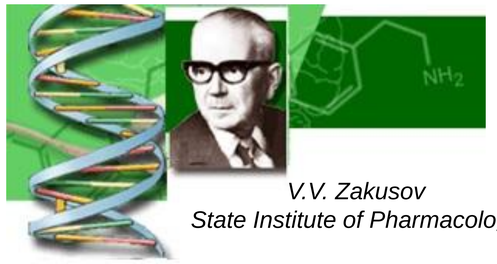

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Background

Noopept (N-phenylacetyl-L-prolylglycine ethyl ester) revealed wide spectrum of neuroprotective effects based on NGF-mimetic and antioxidant activities [1,2]. We postulated that pancreatic beta-cells and neurons are sharing many common features including the involvement of similar neurotrophic factors [3]. This hypothesis prompted us to study different neuroprotective medicines as a potential antidiabetic drugs.

Aim

The study of Noopept on the hyperglycemia and body weight loss as well as on the content of NGF and BDNF in pancreas and liver of the rats preliminary treated with diabetogenic toxin, streptozotocine (STZ).

Methods

Experiments were carried out on Wistar rats divided into 4 groups presented on figure 1. STZ was used in single doses of 40 mg/kg i.p. shown in our previous experiments to reproduce the features of D2. Noopept was administered in doses of 0.5 mg/kg for 14 days.
The measurement of NGF and BDNF content in pancreas and liver was performed 14 days after the finish of Noopept administration by Western-Blot analysis [4] using antibody against BDNF и NGF (Santa Cruz Biotechnology) with 1:1000 dilution.

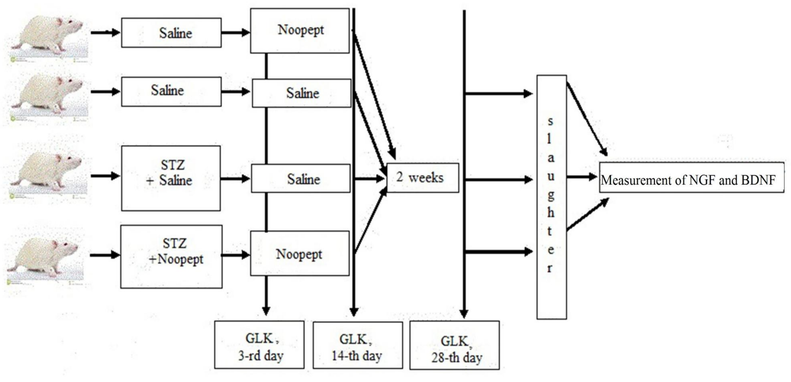
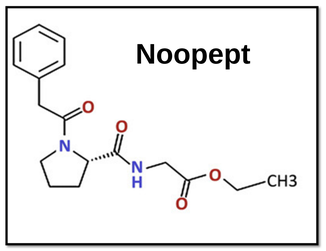
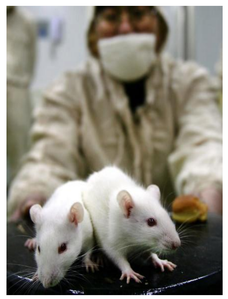


Fig.1 Design of the experiment





Results

While STZ provoked the pronounced hyperglycemia (Fig.2) and body weight loss, Noopept was shown to overcome both these effects.

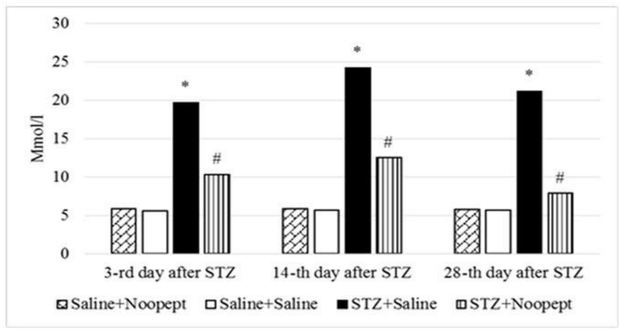


Fig.2 Blood glucose level (Mmol/l)
***** - statistical differences (P<0.05) between the STZ-treated and control groups
# - statistical differences (P<0.05) between the STZ-treated and STZ+Noopept treated groups

STZ was also shown to decrease the content of NGF and BDNF in pancreas and liver.

These data are in whole agreement with data of Ole M.S. et al. [5].

Noopept was FIRSTLY revealed to overcome NGF (Fig. 3A, Fig. 3B) and BDNF (Fig. 4A, Fig. 4B) deficits in these organs and even to increase the NGF content in the liver to the level exceeding those for intact animals.

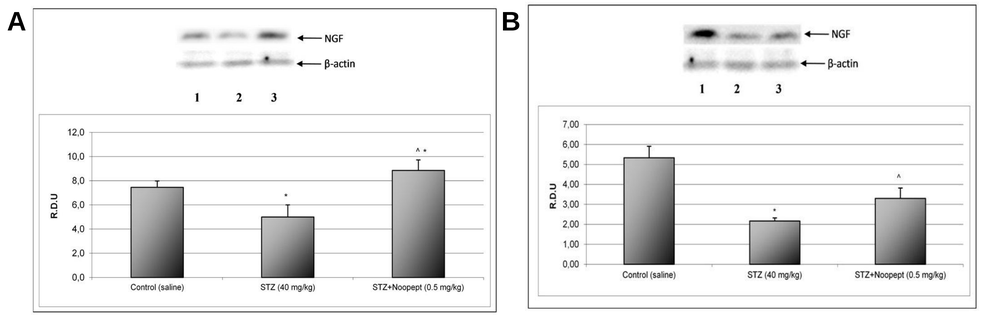


Fig.3 The content of NGF in pancreatic (A) and hepatic (B) tissues
***** - statistical differences (P<0.05) between the STZ-treated and control groups
^ - statistical differences (P<0.05) between the STZ-treated and STZ+Noopept treated groups

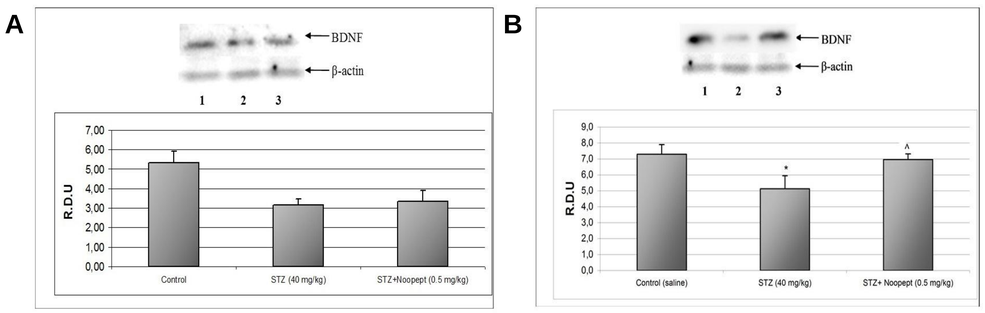


Fig.4 The content of BDNF in pancreatic (A) and hepatic (B) tissues
***** - statistical differences (P<0.05) between the STZ-treated and control groups
^ - statistical differences (P<0.05) between the STZ-treated and STZ+Noopept treated groups

Conclusion

1. Noopept was shown to overcome main metabolic effects of diabetogenic toxin, streptozotocine, in the experiments on Wistar rats: hyperglycemia, NGF and BDNF deficits in target organs of diabetes – pancreas and liver.
2. Clinical manifestation of diabetes are tightly connected with NGF and BDNF deficits. Noopept ability to ameliorate these deficits supposed to be involved in its antidiabetic effects.
3. Data obtained should be considered as an additional evidence for similarity of neurochemical mechanisms regulating the functions of neurons and pancreatic beta-cells.

References

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