LETTER TO THE EDITOR

Effect of Rosiglitazone on early-morning plasma cortisol levels

Jolanta Kunert-Radek & Marek Pawlikowski

Chair of Endocrinology, Medical University of Lodz

Correspondence to: Professor Jolanta Kunert-Radek,
Chair of Endocrinology, Medical University of Lodz,
Sterling str 3, 91-425 Lodz, POLAND
EMAIL: neuroendo@csk.umed.lodz.pl

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The study by Catrina et al. published in this issue, showing that the long-term treatment of diabetic patients with PPAR gamma agonist rosaglitazone failed to affect the morning cortisol levels seemingly stays in opposition with our [1] and other earlier data concerning *in vitro* effects of rosiglitazone on animal pituitary tumors. However, several important points should be taken into consideration.

First, our study was performed on cells isolated from the estrogen-induced rat pituitary tumor which is an animal model of prolactinoma but cannot be considered as a model of human ACTH-secreting pituitary adenoma.

In the other paper from our laboratory [2] we studied the immunohistochemical expression of nuclear PPAR gamma receptors in human pituitary adenomas. We found the highest rate of PPAR gamma-immunopositive cell nuclei in GH-secreting and PRL-secreting adenomas, whereas ACTH-secreting adenomas presented rather low expression (albeit higher then normal anterior pituitary gland). It could mean that ACTH-secreting tumors are rather not good candidates for trials of rosiglitazone treatment.

Moreover, the study by Catrina et al. included the patients with type 2 diabetes but not the patients with Cushing's disease. It means that their data refer rather to the non-tumoral corticotrophs. It cannot be excluded that non-tumoral corticotrophs differ from tumoral cells in their reaction to PPARgamma agonists. It is worth recalling that PPARgamma expression is higher in pituitary adenomas (including ACTH-secreting) in comparison to the non-tumoral anterior pituitary gland [2,3].

Raising the problems in the letter of Catrina et al. allowed us to make our discussion richer and explain some aspects not mentioned in our paper so far.

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