

## Aldosterone mineralocorticoid receptors expression in male Ay mice

N.S. Logvinenko\*, L.E. Katkova, G.S. Baturina  
*Institute of Cytology and Genetics SB RAS, Novosibirsk, Russia*  
\* e-mail: ninlo@bionet.nsc.ru

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*Motivation and Aim:* Mice with melanocortin type of obesity, heterozygous for the dominant lethal mutation Agouti-yellow, (Ay-mice) are a convenient model for studying molecular mechanisms of obesity development. Obesity and complications associated with it often lead to the chronic renal failure, which is one of the main causes of death in the late stages of the syndrome. However, the basic molecular characteristics of the mineralocorticoid system at this type of genetically determined obesity have not been adequately studied. Therefore, the aim of this work was to study the expression of mineralocorticoid receptor and aldosterone level in Ay mice.

*Methods and Algorithms:* Using the real-time PCR method the mRNA level of the mineralocorticoid receptors (MR) in the hypothalamus, kidneys, heart and adipocytes in adult male mice of the standard C57Bl/6J and the Ay mice at the age of 29–30 weeks was investigated. At this age, Ay mice show a non-dietary type of obesity. The level of aldosterone in the blood of both lines was studied using the enzyme immunoassay method (Mouse Aldosterone (ALD) ELISA kit).

*Results:* We have detected a higher level of MR mRNA in the kidney cortex and in the heart left ventricle in C57Bl/6J mice, compared to the line Ay ( $p \leq 0.05$ ). Perhaps it is due to a more active aldosterone-dependent protein regulation in these target tissues in control animals. No significant differences in MR mRNA level in the pituitary glands and adipocytes in mice of both lines have been identified. We have shown that there was no difference in the plasma level of aldosterone in the blood of both lines ( $124.1 \pm 25.1$  and  $102.8 \pm 16.5$  pg/ml in the male Ay and C57Bl/6J, respectively,  $p \geq 0.05$ ).

*Conclusion:* We suggest that the absence of significant differences in the expression of mineralocorticoid receptors in adipocytes and the pituitary gland and in the level of aldosterone in blood of male Ay and C57Bl/6J is due to the gender peculiarities of the mineralocorticoid system participation in the development of melanocortin type of obesity.

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