Abstract of Presentation

Presentation Title:

Analysis of AVP functions via V1a and V1b receptors with knockout mice

Abstract:

The neurohypophyseal peptide [Arg^8]-vasopressin (AVP) is involved in diverse functions such as the regulation of body fluid homeostasis, vasoconstriction, and ACTH release. These physiological effects are mediated by three subtypes of AVP receptors, designated as V1a, V1b and V2, all of which belong to G protein-coupled receptors. Studies with mutant mice lacking the V1a or V1b vasopressin receptor revealed that AVP stimulates ACTH from the anterior pituitary cells, and insulin and glucagon from the pancreatic cells via the V1b receptor and that blockade of the AVP/V1b signal results in the decreased plasma levels of ACTH, and insulin. Blockade of V1a receptor result in decreased BP, blood volume, aldosteron and plasma renin activity. These findings with mutant mice indicate that antagonists specific for the receptors could affect various physiological functions *in vivo*.