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The pituitary independent regulation of the glucocorticoid secretion

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Adaptation to environment is a fundamental process of life and the hypothalamo-pituitary-adrenal axis is a key element of it. According to the canonical view adrenocorticotropin (ACTH) stimulates the adrenal cortex to release glucocorticoids (GC) into the blood. However several data suggest an ACTH-independent GC secretion. Our aim was to find other regulatory molecule(s). As during stress catecholamines are also released from the adrenal medulla, we proposed that adrenalin could be such a molecule. To test our hypothesis we used 10 days old Brattleboro rats as previously 24h maternal separation induced GC elevation without ACTH peak in the vasopressin (AVP) deficient animals. In fasted pups 90 min after ip injection of 3NE/kg rapid insulin we could detect hypoglycaemia together with an activation of the stress axis. In AVP-deficient pups the presence of ACTH-independent GC secretion was reproduced. In AVP-deficient rats pretreatment with a β-adrenerg antagonist (15 min, 2.5mg/kg propranolol) reduced the hypoglycaemia-induced GC elevation without affecting the ACTH levels. In vitro the GC secretion of the adrenal gland of a normal pup was enhanced by ACTH (10-10M) and propranolol (10-5M) treatment reduced both the basal and ACTH-induced secretion. Our results support the view, that adrenaline might stimulate the GC secretion from the adrenocortical cells. As the effect of the antagonist was not complette we can assume that other regulatory molecules might have an impact, too.