letter to editor

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Chronic Stress: Glucocorticoids and Metabolic Disturbances

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Defense mechanisms including behavioral and physiological adaptations are evoked in response to a potentially dangerous situation and are associated to the activation of hypothalamus-pituitary-adrenals (HPA) and sympatho-adrenal axes. Stress response affects different biological systems and includes increase in glucocorticoids serum levels (cortisol in humans and corticosterone in rodents). However, the persistent activation of hypothalamic-pituitary-adrenals axis, with the subsequent increase in glucocorticoid serum levels, is associated with metabolic disturbances such as the metabolic syndrome [1].

Anxiety is an important and prevalent mental disorders widespread around the world [2-3]. Frequently anxiety disorder is associated with activation of HPA axis resulting in increased serum glucocorticoid levels [4-5]. Glucocorticoid treatment has been shown to interfere in the pituitary response to thyrotropin releasing hormone (TRH), inducing a decrease in thyrotropin (TSH) secretion [6]. Thyroid hormone metabolism is also affected by glucocorticoids. In humans there are some studies suggesting a correlation between thyroidal disturbances and anxiety disorders [7-9]. In fact, Toyota et al., described a decrease in mRNA expression and type 2 iodothyronine deiodinase (D2) activity induced by glucocorticoids on the vascular bed; since D2 catalyzes the conversion of the pro-hormone T4 into T3, the biological active thyroid hormone, this decrease may affect vascular function [10].

Our previews studies demonstrated alterations in endocrine system and metabolic dysfunctions in a model of anxiety disorder in rats, including increases in the corticosterone and leptin serum levels [11]. We also detected abnormalities in thyroid function and regulation, since T3 serum levels were decreased while the TSH and T4 serum levels were normal. Body weight and food intake were unchanged, notwithstanding epididymal and retroperitoneal fat depots mass were around twice that of controls. Total cholesterol and triglycerides serum levels were increased in this rodent model of anxiety. Glucose tolerance test response and insulin serum levels were unchanged nevertheless fasting glycemia was elevated. Oxygen consumption was decreased as well as the BAT D2 activity [11]. Our data clearly associate anxiety with endocrine and metabolic dysfunctions mainly deriving from a HPA axis disturbance, and support a possible relationship between chronic anxiety and the appearance of metabolic diseases. Taken together, these data suggest that other studies should be done in order for us to enhance knowledge between chronic anxiety, increased serum glucocorticoids, and metabolic dysfunctions relationship.

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