Letter to the Editor

Response to “Leptin disturbance probably involved in the pathogenesis of obesity-induced depressive symptoms”

We thank Dr. Hua for his comment. Indeed, there is an increase of evidence supporting a common etiology for obesity and mood disorders, with signaling pathways involved in the maintenance of energy balance and mood stability. Leptin has many actions within the brain, including the reduction of food intake and increase of energy expenditure. Dr. Hua’s comment, linking depression with leptin, is consistent with previous studies suggesting that the association between depression and leptin seems to be mediated by increased adiposity. Morris et al. have noted that in obesity, rather than elevated serum levels of leptin, there is a resistance to its action and consequently, a decrease in leptin signals [1]. Milaneschi studies also confirm this observation [2]. However not only leptin plays a pivotal role in obesity and mood disorders as there are also signals involved: orexigenic and anorexigenic neuropeptides, metabolic factors, stress responsive hormones, cytokines, and neurotrophic factors [3].

After the onset of the menopause many signaling metabolic pathways are altered. For example, hypoestrogenism alters leptin sensitivity and consequently body fat distribution changes [4]. This would be consistent with leptin resistance observed during the climacteric, which is expressed by increased appetite, obesity and depression. Nevertheless, as already mentioned, although leptin seems to be an important peptide linking obesity to depression, there are a number of other mechanisms that might be involved in weight gain observed during the menopausal transition. Hypoestrogenism is associated with increased adiposity, increased orexigenic neuropeptides (neuropeptide Y, ghrelin and melanin-concentrating hormone) and decreased anorexigenic neuropeptides (insulin, leptin and serotonin) [5]. Therefore, estrogen deficiency exerts a multiplicity of mechanisms that seem to explain weight gain observed during female mid-life. Indeed, there is much research to carry out to completely elucidate involved mechanisms.

References


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