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Jornal de Pediatria

Platelet and leptin in obese adolescents

Dear Editor,

I would like to compliment Jornal de Pediatria for publishing the article entitled "Platelet and leptin in obese adolescents,"[1] which addresses an interesting subject, although often ignored by Brazilian journals.

While reading the article, the description in the methods section that adolescents were in "(...) pubertal stage as assessed by means of Tanner" caught my attention. This is in all probability a typing error, since it would not be possible to compare groups in different Tanner stages. Previous studies published in the literature show that the increase in testosterone observed in pre-pubertal boys is preceded by peaks of leptin secretion, which return to baseline levels once puberty is reached; girls, on the other hand, present a progressive increase in leptin concentrations in the beginning of puberty.[2] So, the process of sexual maturation is marked by a distinct pattern of leptin secretion, which also correlates its doses with those of sexual hormones.

Another aspect that deserves to be highlighted is that the authors did not find differences in the levels of leptin according to gender in non-obese groups. The difference in leptin concentrations in adults according to gender is well documented in the literature,[3] and sexual dimorphisms have also been observed in newborns, as reported in my article previously published in this journal.[4] Also, the distribution of obese and non-obese adolescents according to gender was not completely clear in the study by Foschini et al.,[1] an information that could possibly explain the fact that only the group of obese adolescent girls presented higher concentrations of leptin when compared to obese adolescent boys. What was the distribution of body fat and lean body mass according to gender for the groups studied? Sexual dimorphism seems to be correlated with body composition, i.e., a higher body fat percentage in women and the inhibitory action of testosterone on leptin secretion in men.[5]

Discussions on platelet and leptin concentrations found in obese adolescents in such an important publication for pediatricians is undoubtedly a great opportunity to disseminate knowledge on the complex mechanism of hormonal interaction involved in the pathogenesis of obesity.

References

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Authors' reply

Dear Editor,

We are sincerely grateful to Dr. Inês Pardo for her observations and suggestions; they will be most helpful in improving future investigations. Below we present some comments on the issues raised in her letter.

1. Indeed, there was a typing error in the methods section of our article[1] while describing the population assessed. The mistake however does not appear in the abstract of the article, where the term "post-pubertal" was used.

The excerpt "aged 15-19 years and pubertal stage as assessed by means of Tanner" should read "aged 15-19 years and at post-pubertal stage according to Tanner criteria." For this study,[1] the post-pubertal stage classification adopted as the inclusion criteria was stage 5, both for boys (Tanner[2]) and for girls (Marshall & Tanner[3]).

2. It is well established that cortisol is a potent immunosuppressant[4,5]; therefore, the main motivations of the study were to shed some light into the role played by leptin and cortisol in immune cell count and to establish a possible relationship between these hormones in obese adolescents. In our study, the non-obese group was considered as a control group, so as to allow our objectives to be reached.

We understand that factors such as gender, body composition and several hormones, including gonadotrophic and somatotrophic hormones, can influence the concentrations as well as the function of immune cells and other hormones. Nevertheless, taking into consideration the tests carried out in our

study, it is necessary to say that a broader discussion of such factors lay beyond the scope of the study. We believe that such suggestions are vital for the performance of further investigations, especially in view of the small number of studies describing in detail the pathophysiology of obesity in adolescents of both genders and the interface among multiple triggers in this chronic degenerative disease.

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