How challenging is to establish parameters good enough to assess the repercussions of trials to fight obesity

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Obesity is presently becoming one of the great scourges in modern societies, particularly in those living in most developed countries. And even in these places, the most developed regions are particularly more affected. On the other hand, the therapeutic and nutritional procedures used to correct disturbances of nutrition, such as those due to low calorie or protein-calorie intake, which occurs in impoverished places can potentially lead to future outcomes to body weight control resulting in obesity (1).

Of course, the adipose tissue which is the central subject of the obesity studies, due to its well-known metabolic and endocrine capacities has been classified as an organ, the adipose organ (2). Among its multiple functions, its ability to synthesize and secrete almost a hundred distinct biologically active substances, some of them classified as proteins and called adipokines (3) and some other of lipidic nature and called lipokines (4), I will focus on two of them, leptin and adiponectin. Both are produced almost exclusively in the adipose tissue. The first one has importance as a hormone that acts to regulate appetite and body weight (5) and the second one is well-known for its insulin-like and anti-inflammatory properties (6).

The subcutaneous fat depot that comprises about 75-80% of body fat mass is considered the main leptin source and, possibly, the main source of adiponectin (7). The circulating leptin levels signalize to the central nervous system (more specifically to neurons located in certain hypothalamic nuclei, such as the arcuate nucleus) the amount of fat stored in the body, since leptin levels in the blood keep a direct correlation with the body fatty mass and also acts to attenuate the drive to feed (5). In such a way, leptin is an important participant in the regulation of feeding and body weight. In relation to adiponectin, its more elevated circulating levels are found in lean subjects and tend to decrease with weight gain and with the increase in body fat while its levels tend to enhance with slimming down (6).

In obese subjects it is reported the occurrence of a state of inflammatory activity (8), although of low intensity, which can be detected through the determination of inflammatory markers, like C reactive protein (CPR) and serum amyloid A (SAA) and some pro-inflammatory cytokines (TNFα and IL6). These two cytokines come from inflammatory cells (lymphocytes and macrophages) which heavily infiltrate the adipose tissue, but are also synthesized by the adipocyte itself. Obese subjects have a high degree of adipose tissue inflammation and both inflammation and obesity work together to aggravate the metabolic state and to intensify the reduction in adiponectin production.

In this edition of AE&M, two distinct papers are published. In both, the obesity is fought by means of two different strategies. Jalalvand and cols. (Acarbose *versus trans*-chalcone: comparing the effect of two glycosidase inhibitors on obese mice) (9) show

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that the obesity in mice fed a high fat diet was treated using α-glucosidase inhibitors in order to block intestinal carbohydrate absorption and to reduce postprandial glucose levels. Svidnicki and cols. (Swim training and the genetic expression of adipokines in monosodium glutamate-treated obese rats) (10) used exercise (swimming) training to fight obesity in rats.

The first study (9) evidenced a certain degree of changes in circulating adipokine levels (leptin reduction under trans-chalcone treatment and adiponectin increase with acarbose). However, both glucosidase inhibitors did not affected significantly the body weight, although the authors suggested the occurrence of fatty mass reduction as well as regression of liver steatosis. Although data shown seemed very intriguing, this study will need more substantial analysis by investigation of some more parameters (for example, glucose tolerance, insulin resistance) and maybe a more prolonged observation and the investigation of other adipose territories (subcutaneous, retroperitoneal, epidydimal, mesenteric).

In the second paper (10), the authors showed, in obese rats pretreated with monosodium glutamate (MSG) in the neonatal period and submitted to an exercise training, that swimming did not resulted in any alteration of pro-inflammatory cytokine content and also the adiponectin mRNA expression levels which were elevated in lean rats submitted to swimming training did not change in obese trained ones. However, the authors did not make adipokine determinations in blood. They concluded that physical training did not interfere with the fatty mass accumulation in MSG rats. It must be very careful to interpret these results, since the obesity, such as was obtained here, has some unique characteristics (MSG causes hypothalamic lesions) not found in diet-induced or other common forms of human or animal obesity and, therefore, it did not serve as good obesity paradigm.

Anyway, I recommend the reading of both papers and I emphasize that the approach to understand the adipose tissue biology is still a very complex task and requires from us a careful position and critical attitude for interpretation of the results.

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REFERENCES

1. Martins VJ, Neves AP, Franco M do C, Clemente AP, Sawaya AL. Impact of nutritional recovery with linear growth on the concentrations of adipokines in undernourished children living in Brazilian slums. Br J Nutr. 2014;112(6):937-44.
2. Fruhbeck G, Gomez-Ambrosi J, Muruzabal FJ, Burrel AM. The adipocyte: a model for integration of endocrine and metabolic signaling in energy metabolism regulation. Am J Physiol Endocrinol Metab. 2001;280:E827-47.
3. Fonseca-Alaniz MH, Takada J, Alonso-Vale MIC, Lima FB. Adipose tissue as an endocrine organ: from theory to practice. J Pediatr (Rio J). 2007;83(5 Suppl):S192-203.
4. Queiroz JC, Alonso-Vale MI, Curi R, Lima FB. Control of adipogenesis by fatty acids. Arq Bras Endocrinol Metabol. 2009;53(5):582-94.
5. Ahima RS, Flier JS. Leptin. Annu Rev Physiol. 2000;62:413-37.
6. Stefan N, Stumvoll M. Adiponectin – its role in metabolism and beyond. Horm Metab Res. 2002;34(9):469-74.
7. Lefebvre AM, Laville M, Vega N, Riou JN, van Gaal L, Auwerx J, et al. Depot specific differences in adipose tissue gene expression in lean and obese subjects. Diabetes. 1998;47:98-103.
8. Lumeng CN, Saltiel AR. Inflammatory links between obesity and metabolic diseases. J Clin Invest. 2011;121:2111-17.
9. Jalalvand F, Amoli MM, Yaghmei P, Kimiagar M, Ebrahim-Habibi A. Acarbose versus trans-chalcone: comparing the effect of two glycosidase inhibitors on obese mice. Arch Endocrinol Metab. 2015;59(3):202-9.
10. Svidnicki PV, Leite NC, Vicari MR, de Almeida MC, Artoni RF, Favero GM, et al. Swim training and the genetic expression of adipokines in monosodium glutamate-treated obese rats. Arch Endocrinol Metab. 2015;59(3):210-4.