editorial

Overeating and Thermogenesis Do we Still Care?

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Classic matters in the area of eating physiology are now researched with the epignosis of the new discoveries in the area of obesity research, neuroendocrinology and neurophysiology. Some of the old beliefs tend to become old fashioned if they do not seem to have special interest in combination with the new data. Other long standing results however become even more interesting because they still hide well kept secrets that even the newly found appetite regulating hormones didn't manage to reveal.

The study area on thermogenesis during/after overeating is classical for obesity researchers. Rubner[1] was one of the first who offered evidence about the increased heat production during eating. The role of *adaptive thermogenesis* in unsuccessful weight-loss intervention was emphasized in order to explain the so called "yoyo phenomenon", or *body weight instability*. The possibility that some individuals with a genetic tendency for obesity fail to increase thermogenesis during overfeeding was one of the main challenges for obesity prevention and treatment.

James A. Levine et al[2] suggested that as humans overeat, activation of Nonexercise Activity Thermogenesis (NEAT) dissipates excess energy to preserve leanness and that failure to activate NEAT may result in ready fat gain. The "miraculous effect" that some humans appear to resist fat gain with overeating whereas others readily store excess fat, started from subjective observations and was confirmed by a small number of clinical studies that document a several fold interindividual variation in fat accumulation with overfeeding.

Except from the daily calorie consumption and expenditure, the number of meals in relation to the body thermogenesis was studied. With the use of this concept, it was revealed that 4 small meals produced greater thermogenesis than one large meal in dogs.[3] Increased O2 consumption was found in rats during voluntary overeating.[4] This phenomenon appears to be mediated by the sympathetic nervous system, since it is blocked by the beta-blocker propranolol administration. De Luca et al[5] showed the role played by the cerebral cortex on thermogenesis in rats during voluntary eating; a significantly higher colonic temperature, brown adipose tissue temperature, and rate of O2 consumption was observed in comparison to control animals. They declared the hypothesis that the cerebral cortex could be involved in the metabolic responses for reduction of body weight to the "set-point".

James A. Levine et al[6] tested the leptin responses to overfeeding and showed the relationship with body fat and NEAT. They measured plasma leptin concentrations and adipose tissue leptin messenger ribonucleic acid together with the components of energy expenditure in 16 nonobese humans before and after overfeeding to assess the relationship between leptin responses to overfeeding and the changes in NEAT. Adipocyte leptin expression was up-regulated with overfeeding, and leptin concentrations increased. Leptin concentrations correlated with body fat before and after overfeeding. Changes in leptin with overfeeding were strongly related to changes in body fat, but not to changes in NEAT. Changes in NEAT correlated inversely with fat gain. They concluded that leptin does not seem to mediate activation of NEAT with overfeeding in nonobese humans; rather, leptin directly reflects body fat mass and fat mass gain.

A very interesting experimental study by Seale et al[7], presents that Prdm16 gene determines the thermogenic program of subcutaneous white adipose tissue in mice. This conclusion they came up to was based on their results that Prdm16 transgenic mice displayed increased energy expenditure, limited weight gain, and improved glucose tolerance in response to a high-fat diet. They describe that there was a significant increase in the levels of thermogenic genes in the absence of morphological brown-like adipose development in the transgenic mice. Recently, Ono K et al[8] suggest that the intragastric administration of capsiate, a transient receptor potential channel agonist, triggers thermogenic sympathetic responses and presents a dietary-dependent regulation of energy metabolism and control of obesity.

It is a general truth that energy cannot get lost; energy that is not expended will be stored in the body. As the digestibility of foods is not affected by intake level or subject (pathology of digestion, bariatric surgery), energy storage during overfeeding can be calculated as the difference between energy intake and energy expenditure. This means that if leptin cannot explain the thermogenesis effect and its role in obesity then some other pathways are still hidden. Obesity- prone persons and obesity- resistant persons are two categories multi-stud-