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Physiological and neurocognitive signals involved in the nutritional responses to exercise in children and adolescents: Implication in pediatric obesity

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Human eating behavior, its control and regulation, rest on a constant peripheral-central cross-talk associated with a permanent neurocognitive integration and treatment of external stimuli, influenced by everyone's dietary status. Lately, the scientific literature clearly suggested that physical activity and acute exercise might interfere with these physiological and neurocognitive signals regulating energy intake, offering then potential new directions to better control energy balance and then prevent or treat obesity. While these effects of exercise have been deeply studied for the last 30 years in adults, results in pediatric populations remain recent. Our team has been among the first to propose intensive exercise has a potential "corrector" of the impaired energy intake regulation in obese adolescents, isolating its anorexigneic effect in obese but not lean youth. For the last couple of years, we have been working on the identification of the physiological (peripheral-central cross talk between the hypothalamus and some gastro-peptides, adipokines and myokines) and neurocognitive (neural responses to food cues) signals who might be responsible for this post-exercise "transient anorexia" observed in obese children and adolescents. While the role of some peptides such as PYY3-36 and GLP-1 have been identified, our late results also clearly show reduced neural activation after exercise in obese but not lean adolescents, leading to a decrease of their spontaneous energy consumption. Not only do we try to identify these pathways after acute exercise, our current researches focus on the effect of chronic physical activity programs on these regulations in youth with obesity.

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