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Letter

The Gravitostat theory: Body fat is lost but is fat-free mass preserved?

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Based on their previous fundamental experiments conducted in animals and clearly showing the effects of hypergravity on body mass and body fat, supporting then the Gravitostat hypothesis [1,2], Ohlsson and colleagues recently confronted their findings in humans, artificially increasing the body weight (weight loading) of 69 adults with moderate obesity [3]. Using a proof of concept translational randomized clinical trial, the authors, in line with their previous results obtained in rodents [1,2], observed a significant reduction of their participants' body weight and especially fat mass loss, suggesting the existence of a weight loading dependent homeostatic regulation of body weight in human (the Gravitostat theory).

This study by Ohlsson and colleagues is definitely an outstanding work and proof of concept, bringing back the light on the main regulating factors of energy metabolism: body composition and purely body mass, as main and crucial actors when it comes to energy homeostasis and the regulation of body weight. While their discussion mainly focuses on the adaptations of body weight and body fat to these 3 weeks of overload, we would like to emphasize that the other side of the coin, i.e. fat free mass, may also be involved in this Gravitostat-mediated regulation.

Although the unchanged FFM in response to the intervention is briefly mentioned by the authors, it might be, according to our interpretation, considered as a central result of their experiment. Indeed, despite the short-term induced weight loss, the authors did not observe any significant change of FFM, i.e. no expected decline in FFM, and most importantly they even depicted a non-significant but positive increase in their heavy load group (+0.12 kg, some participants reaching +0.81 kg). Although not statistically significant, this slight and counter intuitive increase might become clinically significant if sustained over time. This striking result is of particular relevance since, as usually observed and described in the literature, FFM is always significantly reduced in response to dietary restriction-induced weight loss of similar duration, questioning the relevance of caloric restriction in the specific situation of sarcopenic obesity [4,5]. Some studies also reported a reduced FFM in response to weight loss interventions integrating physical exercise [6–8], leading to an adverse decline of the patients' basal metabolic rate (which then usually favors weight regain), just like bariatric surgery [9].

Some recent results effectively point out a mean reduction of FFM of −0.6 kg, −1.1 kg and −0.9 kg in response to three weeks of resistance, endurance or combined training in adults with overweight/obesity (with a mean weight loss of 3.5 kg, 6.9 and 3.3 kg respectively) [8]. In that context, the "not decreased" FFM observed in the present study might be considered as a main result, proposing overloading and hypergravity as an efficient strategy to induce body fat loss while keeping fat free mass and ultimately preventing the patients from a reduction of resting metabolic rate, and of a lower energy cost of movement. Another strong argument on the possible involvement of the Gravitostat on FFM regulatory action may also come from the opposite situation, i.e. space flights, where a lower body gravity regularly induces body fat gain together with a significant FFM loss. The mechanisms of changes in FFM may involve weight perception systems through neural signals integration, but it is also possible that fat mass or other tissue secretory products may also favorably promote muscle maintenance in this situation.

Declaration of Competing Interest

None.

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