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Letter

The gravitostat theory: More data needed

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We thank Drs Thivel and Boirie for bringing up additional aspects of our recent randomized clinical trial published in EClinicalMedicine [1] on the hypothetical body weight regulating system that we previously coined "the gravitostat" [2]. As pointed out by Thivel and Boirie, [3] in several preclinical studies, we found that activation of the gravitostat by increased loading decreases fat mass substantially, while there is little or no effect on fat free mass in obese experimental animals [2,4,5]. In our recent proof-of-concept randomized clinical trial we found that increased weight loading for 3 weeks reduces body weight and body fat mass, but not fat free mass, also in obese humans [1]. It is possible that the difference in effect on fat mass and non-fat mass is more pronounced after increased loading than in other fat-reducing models such as caloric restriction as suggested by Thivel and Boirie [3]. However, we have not yet directly compared the effects of increased weight loading and caloric restriction on fat mass and fat free mass in the same clinical or preclinical study.

The exact mechanisms by which weight loading generates a signal that suppresses fat mass rather than fat free mass remains to be elucidated. However, a large part of the fat free mass consists of muscle, and it may seem intuitive that the body, in response to increased loading, would avoid losing muscle mass that could help carrying the increased weight.

Thus, activation of the gravitostat primarily protects against obesity by suppressing body fat mass but not fat free mass. Further studies are clearly warranted to determine why activation of the gravitostat preferentially reduces fat mass in obese subjects as suggested by Thivel & Boirie [3].

Declaration of competing Interest

None

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