Letter to Editor

Exercise and Adipsin: A Potential Link in Shaping Healthy Fat Depots

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Dear Editor-in-Chief

White and brown adipose tissues play distinct roles in the human body, with white adipose tissue further classified into subcutaneous and visceral fat depots. These depots serve various functions, including energy storage, thermal insulation, and organ protection [1]. Moreover, they produce adipokines, such as adipsin, which influence energy metabolism and adipose tissue homeostasis [1]. Adipsin, also known as complement factor D, is particularly interesting due to its role in maintaining adipose tissue balance [2]. Research has shown significantly increased adipsin expression in central obesity, particularly in abdominal subcutaneous adipose tissue (abdominal_{sat}) and visceral adipose tissue (abdominal_{vat}) [3]. Higher adipsin expression in abdominal_{sat} has been associated with healthier adipose tissue and even specific obesity subphenotypes like metabolically healthy obesity [3]. Exercise has long been recognized as an effective strategy to combat obesity, reshaping both SAT and VAT depots and altering their secretory profiles [4,5]. However, the effects of exercise on adipsin expression in obesity models remain understudied. Given the evidence of adipsin's role in adipose tissue homeostasis and the known impact of exercise on fat depot remodeling, it could be proposed that exercise training could increase adipsin expression in both SAT and VAT depots in obesity models. This increase may be associated with elevated circulating adipsin levels.

Potential Implications

If the hypothesis holds true, adipsin could emerge as a valuable biomarker, reflecting the health of adipose tissue by increasing SAT depots and reducing VAT depots. This finding would warrant further investigation and mechanistic studies.

Annals of Obesity & Disorders Volume 7, Issue 1 (2023) www.austinpublishinggroup.com Alizadeh H © All rights are reserved Furthermore, it's worth noting that adipsin is also expressed by human muscle tissue [6]. Skeletal muscle is recognized as an endocrine organ, releasing molecules known as myokines that mediate the health-promoting effects of exercise [7]. Therefore, a potential muscle-fat crosstalk mediated by adipsin could be explored in future studies, shedding light on its role in remodeling white fat depots.

In summary, it is postulated that exercise training may boost adipsin expression in both white fat depots and skeletal muscle. This could contribute to the remodeling of white fat depots in obesity models, suggesting that adipsin may serve as a novel adipomyokine, acting as an exercise mediator to promote health. Further research is needed to validate these hypotheses and explore their implications fully.



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Author Statements

Conflict of Interest

There is no conflict of interest to declare.

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