



Diabetes, Insulin Resistance, Fetuin-B and Exercise Training

Ayoub Saeidi, Anthony C. Hackney, Seyed Morteza Tayebi, Mehdi Ahmadian, Hassane Zouhal

► To cite this version:

Ayoub Saeidi, Anthony C. Hackney, Seyed Morteza Tayebi, Mehdi Ahmadian, Hassane Zouhal. Diabetes, Insulin Resistance, Fetuin-B and Exercise Training. *Annals of Applied Sport Science*, 2019, 7 (2), pp.1-2. 10.29252/aassjournal.7.2.1 . hal-02178302

HAL Id: hal-02178302

<https://univ-rennes.hal.science/hal-02178302>

Submitted on 10 Jul 2020

HAL is a multi-disciplinary open access archive for the deposit and dissemination of scientific research documents, whether they are published or not. The documents may come from teaching and research institutions in France or abroad, or from public or private research centers.

L'archive ouverte pluridisciplinaire **HAL**, est destinée au dépôt et à la diffusion de documents scientifiques de niveau recherche, publiés ou non, émanant des établissements d'enseignement et de recherche français ou étrangers, des laboratoires publics ou privés.



LETTER TO EDITOR

Diabetes, Insulin Resistance, Fetuin-B and Exercise Training

¹Ayoub Saeidi, ²Anthony C. Hackney, ³Seyed Morteza Tayebi*, ⁴Mehdi Ahmadian, ⁵Hassane Zouhal*

¹Exercise Biochemistry Division, Faculty of Physical Education and Sport Science, University of Mazandaran, Babolsar, Iran. ²Department of Exercise & Sport Science, University of North Carolina, Chapel Hill, NC, United States. ³Core Research of Health Physiology and Physical Activity, Department of Exercise Physiology, Faculty of Sport Science, Allameh Tabataba'i University, Tehran, Iran. ⁴Department of Physical Education and Sport Sciences, Aliabad Katoul Branch, Islamic Azad University, Aliabad Katoul, Iran. ⁵Movement, Sport and Health Sciences laboratory (M2S), University of Rennes 2, Rennes, France.

Submitted 25 December 2018; Accepted in final form 20 January 2019.

DEAR EDITOR

Diabetes represents a major global public health threat and, together with obesity, constitutes an important contributor to a decline in life expectancy (1). The pathophysiology of type 2 diabetes is complex. In addition to impaired insulin secretion from pancreatic cells, a reduced insulin sensitivity is found to play a predominant role in the pathogenesis of the disease (2). Several circulating proteins are involved in the regulation of insulin sensitivity; such as, adiponectin, retinol binding protein 4, and fetuin-A (3). The fetuin family consists of a set of orthologous plasma proteins found in humans, sheep, pigs, cows and rodents. Fetuin-A has been identified as a major protein during fetal life and is also involved in important functions such as inhibition of insulin receptor tyrosine kinase activity, protease inhibitory activity and the development of associated regulation of calcium metabolism and osteogenesis. Furthermore, fetuin-A is a key component in the recovery phase of an acute inflammatory

response. There is a second protein of the fetuin family, called fetuin-B, which is found at least in human and rodents. Fetuin-B was discovered in mice prone to diabetes. Based on domain homology, overall conservation of cysteine residues and chromosomal assignments of the corresponding genes in these species, fetuin-B is a unambiguously paralogue of fetuin-A (3, 4). Recently, fetuin-B has also been identified as a novel adipokine/hepatokine which is significantly increased in hepatic steatosis and which mediates impaired insulin action, as well as glucose intolerance (4). Meex et al. demonstrated that secretion of fetuin-B by isolated hepatocytes is augmented in mice with hepatic steatosis as compared to control littermates and it impairs glucose homeostasis in both humans and rodents (4). Most interestingly, short hairpin RNA-induced knockdown of plasma fetuin-B improved glucose tolerance in mice as compared to controls without any effect on body weight (4). Previous studies have shown that Fetuin-B is in high levels

*. Corresponding Authors:

Hassane Zouhal, Professor

Movement, Sport and Sciences laboratory (M2S). UFR-STAPS, University of Rennes 2, Avenue Charles Tillon, CS 24414, 35044 Rennes Cedex, France. **E-mail:** hassane.zouhal@univ-rennes2.fr

Seyed Morteza Tayebi, Assistant Professor

Core Research of Health Physiology and Physical Activity, Department of Exercise Physiology, Faculty of Sport Science, Allameh Tabataba'i University, Shahid Hemmat West, Dehkadeh-y-Olympic, P.O. Box: 1489684511. **E-mail:** tayebism@gmail.com

in patients with Type 2 diabetes. In an experimental trial, patients were found to have high levels of Fetuin-B only if they were also pre-diabetic or diabetic. Moreover, another study demonstrated that fetuin-B levels are increased in women with gestational diabetes mellitus compared with healthy pregnant control women (5). Also Fetuin-B protein has been shown to impair the action of insulin in the body (5). This evidence collectively provides a clear and causal link between the development of non-alcoholic fatty liver disease (NAFLD) and the development of Type 2 diabetes. If we can develop a drug that can block this protein perhaps we may be able to prevent the development of diabetes in patients with NAFLD (5, 6).

On the other hand, exercise training is well noted as one of the most effective behavioral factors in the prevention of diabetes and inflammation. Numerous studies have examined

the effect of exercise training on diabetics, obese patients, and cardiovascular patients, confirming that exercise training is able to reduce insulin resistance, obesity, inflammatory markers and adipokines such as leptin, retinol binding protein 4, resistin (associated with insulin resistance), while at the same time increasing adiponectin (7-11). The aforementioned points highlight the positive effect of exercise training on obesity, diabetes and inflammation risk factors (12); and, in turn, suggest the effect of exercise training on fetuin-B research is highly warranted – and as of yet remains unexamined. The study of fetuin-B and exercise may provide new insight and important understanding about the connections between, and prevention of diabetes, insulin resistance, and related diseases. We encourage exercise immunology and physiology researchers to examine the theoretical constructs present here and test the premise put forward.

REFERENCES

1. Olshansky SJ, Passaro DJ, Hershow RC, Layden J, Carnes BA, Brody J, et al. A potential decline in life expectancy in the United States in the 21st century. *New England Journal of Medicine*. 2005;352(11):1138-45. [DOI:10.1056/NEJMs043743] [PMID]
2. Stumvoll M, Goldstein BJ, van Haeften TW. Type 2 diabetes: principles of pathogenesis and therapy. *The Lancet*. 2005;365(9467):1333-46. [DOI:10.1016/S0140-6736(05)61032-X]
3. Olivier E, Soury E, Ruminy P, Husson A, Parmentier F, Daveau M, et al. Fetuin-B, a second member of the fetuin family in mammals. *Biochemical Journal*. 2000;350(2):589-97. <https://doi.org/10.1042/bj3500589> [DOI:10.1042/0264-6021:3500589] [PMID] [PMCID]
4. Meex RC, Hoy AJ, Morris A, Brown RD, Lo JC, Burke M, et al. Fetuin B is a secreted hepatocyte factor linking steatosis to impaired glucose metabolism. *Cell metabolism*. 2015;22(6):1078-89. [DOI:10.1016/j.cmet.2015.09.023] [PMID]
5. Kralisch S, Hoffmann A, Lössner U, Kratzsch J, Blüher M, Stumvoll M, et al. Regulation of the novel adipokines/hepatokines fetuin A and fetuin B in gestational diabetes mellitus. *Metabolism*. 2017;68:88-94. [DOI:10.1016/j.metabol.2016.11.017] [PMID]
6. Ghanbari-Niaki A, Saeidi A, Aliakbari-Beydokhti M, Ardeshiri S, Kolahdouzi S, Chaichi MJ, et al. Effects of Circuit Resistance Training with *Crocus Sativus* (Saffron) Supplementation on Plasma Viscosity and Fibrinogen. *Annals of Applied Sport Science*. 2015;3(2):1-10. [DOI:10.18869/acadpub.aassjournal.3.2.1]
7. Lakka TA, Laaksonen DE. Physical activity in prevention and treatment of the metabolic syndrome. *Applied physiology, nutrition, and metabolism*. 2007;32(1):76-88. [DOI:10.1139/h06-113] [PMID]
8. Bassuk SS, Manson JE. Epidemiological evidence for the role of physical activity in reducing risk of type 2 diabetes and cardiovascular disease. *Journal of applied physiology*. 2005;99(3):1193-204. [DOI:10.1152/jappphysiol.00160.2005] [PMID]
9. Pedersen BK. The anti-inflammatory effect of exercise: its role in diabetes and cardiovascular disease control. *Essays in biochemistry*. 2006;42:105-17. [DOI:10.1042/bse0420105] [PMID]
10. Tayebi SM, Hasanzadeh P, Saeidi A, Fadaei MR. Intense Circuit Resistance Training along with *Zataria multiflora* Supplementation Reduced Plasma Retinol Binding Protein-4 and Tumor Necrosis Factor- α in Postmenopausal Females. *Jundishapur Journal of Natural Pharmaceutical Products*. 2018;13(2):e38578. [DOI:10.17795/jjnpp.38578]
11. Tayebi SM, Saeidi A, Fashi M, Pouya S, Khosravi A, Shirvani H, et al. Plasma retinol-binding protein-4 and tumor necrosis factor- α are reduced in postmenopausal women after combination of different intensities of circuit resistance training and *Zataria* supplementation. *Sport Sciences for Health*. 2019. [DOI:10.1007/s11332-019-00544-2]
12. McMurray RG, Hackney AC. Interactions of metabolic hormones, adipose tissue and exercise. *Sports medicine*. 2005;35(5):393-412. [DOI:10.2165/00007256-200535050-00003] [PMID]