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Editorial Board Member of World Journal of Diabetes, Wei Wang, MD, PhD, Chief Physician, Professor, Director, Department of Endocrinology, Xiang'an Hospital of Xiamen University, School of Medicine, Xiamen University, Xiamen 361101, Fujian Province, China. wwang@xah.xmu.edu.cn

AIMS AND SCOPE

The primary aim of World Journal of Diabetes (WJD, World J Diabetes) is to provide scholars and readers from various fields of diabetes with a platform to publish high-quality basic and clinical research articles and communicate their research findings online.

WID mainly publishes articles reporting research results and findings obtained in the field of diabetes and covering a wide range of topics including risk factors for diabetes, diabetes complications, experimental diabetes mellitus, type 1 diabetes mellitus, type 2 diabetes mellitus, gestational diabetes, diabetic angiopathies, diabetic cardiomyopathies, diabetic coma, diabetic ketoacidosis, diabetic nephropathies, diabetic neuropathies, Donohue syndrome, fetal macrosomia, and prediabetic state.

INDEXING/ABSTRACTING

The WID is now abstracted and indexed in Science Citation Index Expanded (SCIE, also known as SciSearch®), Current Contents/Clinical Medicine, Journal Citation Reports/Science Edition, PubMed, PubMed Central, Reference Citation Analysis, China National Knowledge Infrastructure, China Science and Technology Journal Database, and Superstar Journals Database. The 2022 Edition of Journal Citation Reports® cites the 2021 impact factor (IF) for WJD as 4.560; IF without journal self cites: 4.450; 5-year IF: 5.370; Journal Citation Indicator: 0.62; Ranking: 62 among 146 journals in endocrinology and metabolism; and Quartile category: Q2.

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LETTER TO THE EDITOR

Loss of skeletal muscle mass is not specific to type 2 diabetes

Bo Zhou, Ying-Qi Jin, Lian-Ping He

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Provenance and peer review: Unsolicited article; Externally peer

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Abstract

Skeletal muscle is a massive insulin-sensitive tissue in the body. Loss of muscle mass is associated with mitochondrial dysfunction, and is often a result of diabetes. Insulin deficiency or insulin resistance can only be seen as reduced skeletal muscle mass. Diabetes is caused by insulin deficiency or insulin resistance; however, insulin resistance is not unique to diabetics. Insulin resistance also exists in many diseases.

Key Words: Diabetics; Insulin deficiency; Insulin resistance; Skeletal muscle mass

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Core Tip: Insulin resistance is present in hypertension, and in this case, loss of skeletal muscle mass occurs. At the same time, insulin resistance also results in obesity, and in this case, there is also a reduction in skeletal muscle mass. Loss of skeletal muscle mass can occur in many diseases.

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TO THE EDITOR

We read with great interest the study by Chen LY *et al*[1] which discovered that there is a relationship between loss of skeletal muscle mass and the presence of diabetic mellitus in males, but not in females. The findings have positive implications for the treatment and prevention of diabetes. Nonetheless, it appears to me that there are still some issues worth rethinking.



In the study, loss of skeletal muscle mass was shown to be associated with diabetes in men; however, the loss of skeletal muscle mass is not unique to diabetes. High insulin resistance occurs in both type 2 diabetes and high blood pressure. Insulin resistance plays a major role in the development of hypertension. Previous animal studies have also found that the spontaneously hypertensive rat manifests insulin resistance^[2]. At the same time, there is a loss of skeletal muscle mass in insulinresistant diseases. Skeletal muscle is the largest insulin-sensitive tissue in the body. Decreased muscle mass is associated with mitochondrial dysfunction and increased fat infiltration. This leads to a decrease in glucose processing capacity. Therefore, loss of skeletal muscle mass is also associated with hypertension.

In addition, insulin resistance also appears in adolescent obesity. Lipid accumulation is evident in skeletal muscles in adolescents with obesity. Intermuscular fat may impair insulin action through reducing blood flow to muscles [3,4]. Obesity is associated with biological dysfunction in skeletal muscles^[5]. Sarcopenic obesity is a symptom of obesity with loss of muscle mass and physical dysfunction. Obesity can cause several biological dysfunctions, including insulin resistance, mitochondrial dysfunction, and inflammation. These changes further aggravate skeletal muscle loss and physical dysfunction. There is a study that shows that in the early stages of juvenile obesity development, the microvasculature and prefrontal cortex exhibit impaired insulin signaling[6]. This study suggests that obesity has insulin resistance. At the same time, there is a loss of skeletal muscle mass in insulin-resistant diseases. This further suggests that skeletal muscle mass loss is not unique to diabetes.

In summary, decreased skeletal muscle mass occurs in both hypertension and obesity. Insulin resistance is not just a loss of skeletal muscle mass. Loss of skeletal muscle mass is also present in many diseases and is not a specific feature of diabetes. More research is needed to determine the relationship between reduced skeletal muscle mass and diabetes.

FOOTNOTES

Author contributions: Zhou B and He LP came up with ideas and constructs; Zhou B and Jin YQ wrote the manuscript; He LP approved the main conceptual ideas and made corrections; all authors provided final edits and approved the manuscript.

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