American Journal of Biomedical Science & Research

www.biomedgrid.com

ISSN: 2642-1747

Research Article

The Effect of Resveratrol Supplements on Sarcopenic Obesity Mice Skeletal Muscle

Jia Ping Wu[1]* and Dan Guo[1]

[1]*Department of Medical Technology, Shaoguan University Medical College, Guangdong Province, China*

***Corresponding author:** Jia Ping Wu, Department of Medical Technology, Shaoguan University Medical College, Guangdong Province, China

To Cite This Article: *Jia Ping Wu* and Dan Guo. The Effect of Resveratrol Supplements on Sarcopenic Obesity Mice Skeletal Muscle. Am J Biomed Sci & Res. 2023 18(2) AJBSR.MS.ID.002445,* DOI: 10.34297/AJBSR.2023.18.002445

Received: February 27,2023; **Published:** March 09, 2023

Abstract

Aim: The aim of this study is to determine resveratrol effects on sarcopenic obesity in C57BL/6J mice skeletal muscle due to type 2 diabetes mellitus-induced obesity in the elderly. Mice (2 years old) feed a high-oil-fat diet (HFD) for 2 weeks and then injected with streptozotocin (STZ, 30mg/kg) to induce diabetes mellitus and continued HFD fed for 7 weeks to become obese sarcopenia. High- (200 mg/kg), middle- (150 mg/kg), and low-dose (50mg/kg) resveratrol was fed for 5 weeks after STZ injection. Body weights, fat weights, and skeletal muscle weights were determined. Oxidative stress and pro-inflammatory protein expression were detected by western blotting. Body weight increase was observed HFD-diet after STZ injection in C57BL/6J mice. We found that oxidative stress and pro-inflammation were reduced, and protein expression levels of Bad, caspase 9, IL-6, and TNF-alpha were decreased. Resveratrol ameliorated DM-induced inflammation. Resveratrol supplementary intake may reverse age-related sarcopenia obesity and obese sarcopenia.

Keywords: Diabetes Mellitus, Skeletal, Sarcopenia, Resveratrol, Obesity

Abbreviations: TNF-α: Tumor Necrosis Factor-α; IL-6: Interleukin-6; FOXO: Forkhead Box Protein; T2DM: Type-2 Diabetes Mellitus; T1DM: type-1 Diabetes Mellitus; PPARα: Peroxisome Proliferator-Activated Receptor-Alpha; IR: Insulin Resistance; IMAT: Inter- and Intramuscular Adipose Tissue; HFD: High-Fat Diet; STZ: Streptozotocin

Introduction

Sarcopenia obesity is the only independent association of obesity with sarcopenia. Sarcopenia with obesity in individuals due to metabolic changes [1]. Sarcopenic is defined as the combined effects of type-2 diabetes (T2DM) and obesity. Nevertheless, sarcopenic obesity is a common trait for older adults suffering from aging, diabetes mellitus, physical inactivity, insulin resistance, low-grade inflammation, and hormonal changes [2]. On the other hand, the prevalence of sarcopenic obesity among adults remains controversial. With age, increased amounts of adipose tissue often accompany sarcopenia. In other words, the increased prevalence of obesity observed in older individuals has been characterized by a mismatch between mass and fat mass [3]. Thus, the operative definition of sarcopenic obesity and obese sarcopenia is still under discussion and creates difficulties in the mechanisms of sarcopenia and pre-sarcopenia. Sarcopenic obesity and obese sarcopenia are a conjunction of two diseases between obesity and sarcopenia termed [4]. Sarcopenic obesity and obese sarcopenia are negatively associated with the incidence of sarcopenia in diabetes mellitus. In this study, the risk of both sarcopenia obesity and obesity are led to Type 1 diabetes mellitus (T1DM) and Type 2 diabetes mellitus (T2DM) outcomes than obesity alone. That is because sarcopenic obesity and obese sarcopenia are dual conditions in which obesity and sarcopenia are synergistically present. Diabetes mellitus (DM) has a bidirectional relationship with sarcopenic obesity and obese sarcopenia [5]. A bidirectional association between T1DM and T2DM [6]. Greatly, sarcopenic obesity is a common cause of T2DM, but obese sarcopenia is T1DM. Recently, some reports have reported resveratrol supplementary intake will help type 2 diabetes mellitus (T2DM) to live. In this study, we use resveratrol supplementary intake to treat sarcopenic obesity and obese sarcopenia. Resveratrol is a natural polyphenol (flavonoids and non-flavonoids) found in grapes, peanuts, and medicinal plants [7]. Resveratrol supplementation improves vascular endothelial dysfunction and reversed oxidative stress-induced pro-inflammation. The effects of resveratrol on diabetes mellitus (DM)-induced inflammation are not investigated yet. This study aimed to investigate the efficacy of resveratrol on skeletal muscle function in T1DM and T2DM-induced sarcopenic obesity mice

models to examine the possible mechanisms of resveratrol. The Foxo1a/Foxo3a transcription factors integrate longevity-related mechanisms that play a protective role in sarcopenia and obesity by DM [8]. Foxo1a/Foxo3a signals to ameliorate TNF-alpha-induced inflammation in sarcopenic obesity through resveratrol supplementary intake availability [9]. Resveratrol supplementary intake may be a crucial role in aging and longevity. Resveratrol supplementation may ameliorate skeletal muscle injury in C57BL/6J mice by inhibiting oxidative stress and inflammation via reversed-induced inflammation.

Material and Methods

Diabetes Mellitus (DM) C57BL/6J mic

Purchased forty C57BL/6J (n=40) mice 6 weeks years-old age from the National Science Council Animal Center and used them according to the Helsinki Declaration Guidelines. C57BL/6J mice have lived for 2 years. C57BL/6J mice were divided into 5 groups. The C57BL/6J mice were housed in a room at 24 ± 2 ℃ with a 12 h/12h light-dark cycle. The humidity was approximately 40%. One group recruited 8 mice of C57BL/6J mice for a 2-year-old fed a normal diet, and other groups recruited 32 mice of C57BL/6J mice fed an HFD for 7 weeks and resveratrol intraperitoneally injected (IP) for 2 weeks. After 2 weeks of HFD feed, C57BL/6J mice were injected with intraperitoneally injected streptozotocin (STZ) to observe diabetes mellitus (DM) C57BL/6J mice. Resveratrol (low-, mid-, and high-dose) was fed after STZ-induced DM continuing after 5 weeks (Figure 1A). After, sacrificing all mice to determine aging-related protein expression. 8 mice were housed in per cage in an environmentally controlled room. Use committee-approved animal care and experiments.

Hematoxylin-eosin (H&E) staining

Cross-sections of skeletal muscle were cut 10 um thick and placed on slides. For assessment of skeletal muscle sections, tissue cross-sections were incubated for 5 min at room temperature to stain H&E staining. Stained sections were rinsed with PBS and air-dried before mounting. After gently rinsing with water, the slides were dehydrated through graded alcohols for 15 min and cleaned in xylene, then covered slip.

Western Blotting

Skeletal muscle tissue was cut into small pieces, resuspended in PBS buffer, and homogenized in Polytron homogenate. Skeletal muscle samples were then centrifuged twice at 12,000 rpm at room temperature for 30 minutes. The supernatant recovered and is considered total cytoplasmic cytolytic fluid. Separate the use of gel electrophoresis protein samples. Remove the deposited glue and remove the angularly separated adhesive by removing it. SDS-PAGE, using 10 to 12.5% polyacrylamide gel. The samples were electrophoresed at 150 V for 3.5 hours. The protein was electrophoresed on PVDF or nitrocellulose membrane (Millipore, USA, Immobilon PSQ PVDF membrane, 0.45 microns) using a Bio-Red laboratory instrument presented by Transmembrane Impression Cell Unit (Nobel Drive Hercules, California, USA)) For 2 hours, equilibrated in 25 mM Tris-HCl for 15 min, pH 8.3, containing 192 mm glycine and 20% (v / v) methanol. Blocked the PVDF in 5% skimmed milk at room temperature for 2 hours and then blocked in the buffer containing 0.1% (v / v) fetal bovine serum at room temperature for two hours. For the absence of a methanol transfer buffer, it is essential for the complete balance of the separation gel to achieve this which will result in twisting within the coated gel. Monoclonal primary antibody: The incubation was carried out at room temperature for 2 hours. The primary antibodies of IL-6, TNF-alpha, Fox1a, Fox3a, Caspase-3, Caspase 9, Bad, Bcl-2, and α-Tubulin (Santa Cruz, CA, USA) were detected. The membrane was washed in the 5 ml blotting buffer for 5 minutes after incubated with a second Horseradish Peroxidase-Conjugated Antibody: anti-rabbit (1: 1000), anti-goat (1: 1000), or anti-mouse (1: 1000) IgG at room temperature for 1 hour in the secondary antibody (Santa Cruz, CA, USA). ECL chemiluminescence (Sigma-Aldrich, Borem, Belgium) was used to detect proteins. The antibody binds to 100 mM Tris-HCl, pH 7.5, buffer.

Statistical analysis

Western blotting band images' intensity was signaled hybridization by FUJIFILM Imagine Program. The quantification of bands was carried out by scanning. All statistical analysis results were performed by Sigma Stat 10 software. Results were expressed as mean ± S.E.M. Statistical analysis was assessing groups, two-way ANOVA was utilized with turkey's post hoc test was used when indicated and it was expressed *$p<0.05$, **$p<0.01$ significant difference compared to the control group. #$p<0.05$, ##$p<0.01$ significantly different compared to the STZ-HFD group.

Results

In T2DM-induced sarcopenia obesity mice, resveratrol decreased Oxidative Stress and impairs skeletal muscle repair

Sarcopenic obesity is an age-related decline in skeletal muscle tissue mass and function. C57BL/6J (2-year-old) fed high-fat feed diet (HFD) 2 weeks high body weight. The accumulation of adipocytes has been identified in skeletal muscle loss and poor regenerative capacity in sarcopenic obesity C57BL/6J mice myoblasts. T2DM-induced sarcopenic obesity has been established by STZ-HFD. Insulin is a potent inhibitor of lipolysis from adipocytes (Figure 1A). Resveratrol supplementation has observed a significant decrease in the middle dose between high- and low-dose after STZ-injected C57BL/6J mice fed HFD (Figure 1B). Sarcopenic obesity describes age-related declines in muscle mass and fat increased. In this response, we found skeletal-muscle glucose metabolism disorders caused by obesity. Therefore, we could find body weight increased in high-fat-diet mice in all groups. Sarcopenic obesity has been implicated as both T2DM and obesity cause. High-dose resveratrol intake was observed in body weight decreased after 5 weeks (Figure 1C). Resveratrol can reduce insulin resistance by skeletal muscle synthase as our results in Figure 1D. Thus, we proved

that resveratrol supplementation elevated skeletal muscle mass. The HFD consequence of T2DM mice was observed as significant skeletal muscle mass loss ($p<0.05$) (Figure 1E). A condition of body weight increases, and muscle mass loss is referred to as sarcopenic obesity. At the same time, sarcopenic obesity was observed lower the activity of skeletal muscle oxidative stress. However, T2DM is resulting in the combination effects of diabetes and sarcopenia. Resveratrol supplementary intake reduced oxidative stress in STZ-HFD sarcopenic obesity mice by Bcl-2 protein increases (Figure 2). In the T2DM-induced sarcopenic obesity C57BL/6J mice model, resveratrol intake promotes Bcl-2 activation. The results found by middle-and high-dose resveratrol intake, Bcl-2 protein expression level increases as well as decreases Bad levels by western blotting (Figure 2A). The protein expression level of Bcl-2 was increased by STZ-HFD-MRSV ($p<0.05$) and STZ-HFD-HRSV group ($p<0.01$) (Figure 2B). Resveratrol intake-dependent inhibition of oxidative stress through caspase-3 and caspase-9 protein expression levels by western blotting (Figure 2C). Low protein expression levels were expressed in caspase-3 and -9 by STZ-HFD-LRSV, STZ-HFD-MRSV, and STZ-HFD-HRSV ($p<0.05$) (Figure 2D). These results showed resveratrol may improve nutrient availability and DNA protection of skeletal muscle cell role. Therefore, we suggested that resveratrol supplementation intake has anti-oxidative stress and skeletal muscle impairment in longevity for protection in T2DM-induced sarcopenic obesity in C57BL/6J mice.

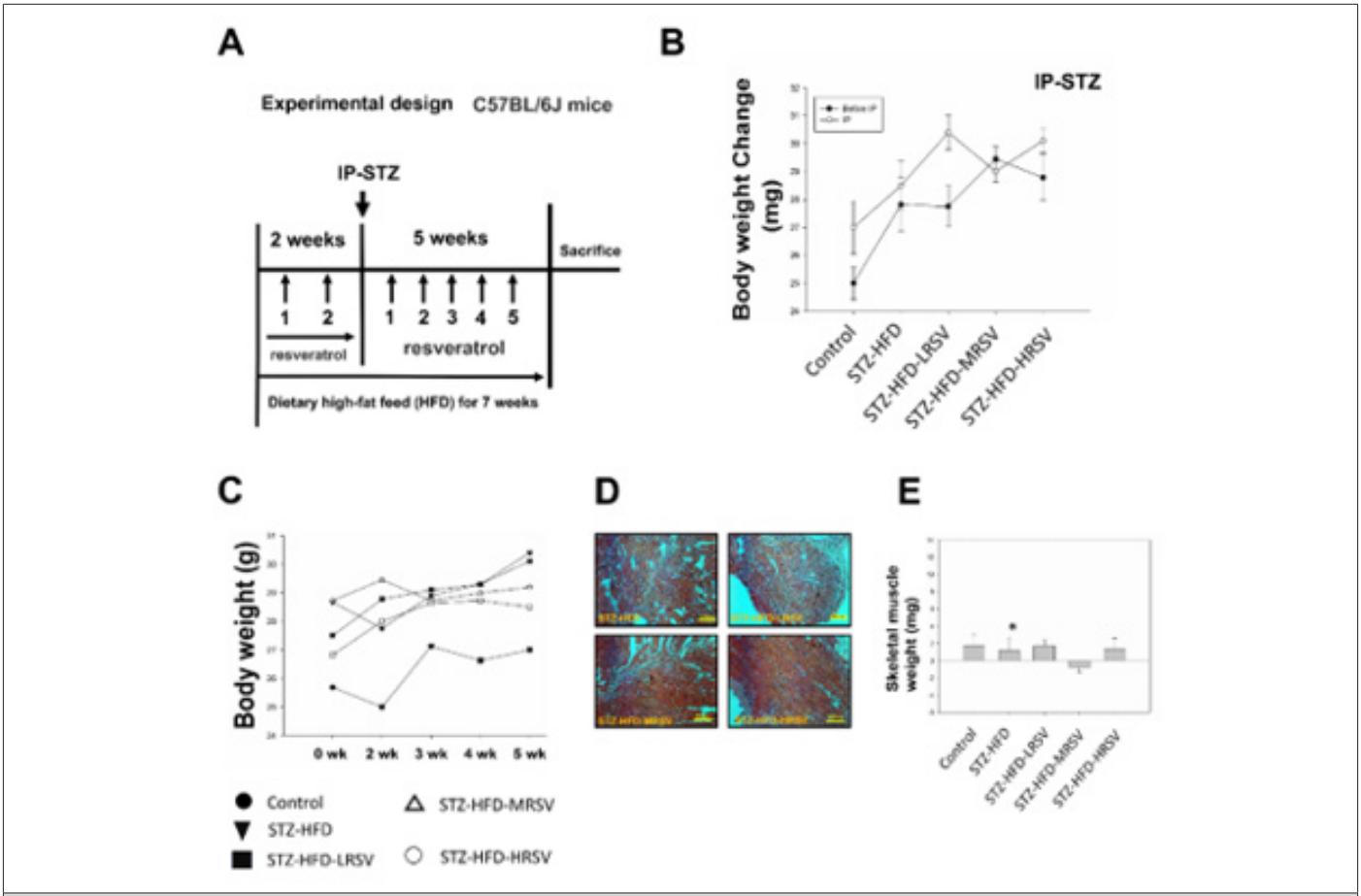

Figure 1: The effects of resveratrol intake on STZ-HFD-Induced sarcopenic obesity C57BL/6J mice body weight change.
a) Study design.
b) The changes in body weight were measured in mice fed HFD with STZ injected. Resveratrol intake reversed body weight after IP-STZ was observed by middle-dose resveratrol. STZ: streptozotocin, HFD: high-fat diet, L-RSV: Low-dose resveratrol, M-RSV: Middle-dose resveratrol, HRSV: High-dose resveratrol.
c) The body weight changes after a 5-week HFD feed and STZ injected with low-, middle-, and high-dose resveratrol. STZ: streptozotocin, HFD: high-fat diet, L-RSV: Low-dose resveratrol, M-RSV: Middle-dose resveratrol, HRSV: High-dose resveratrol.
d) Histological of skeletal muscle tissue by H&E staining.
e) The skeletal muscle weight changed after a 5-week HFD feed and STZ injected with low-, middle-, and high-dose resveratrol. STZ: streptozotocin, HFD: high-fat diet, L-RSV: Low-dose resveratrol, M-RSV: Middle-dose resveratrol, HRSV: High-dose resveratrol.

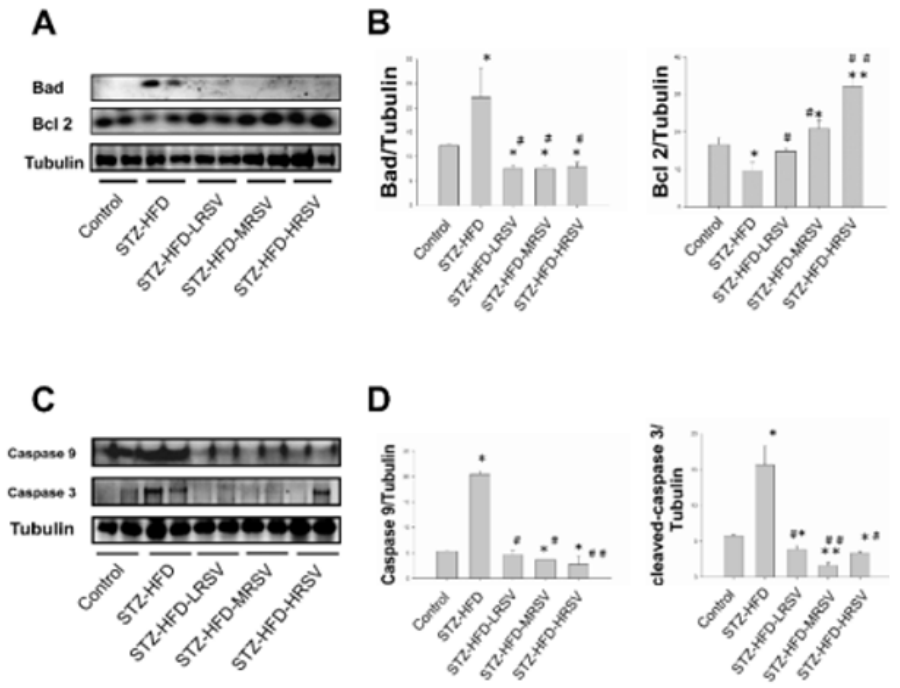

Figure 2: The effect of oxidative stress proteins on resveratrol from diabetes-induced sarcopenic obesity using western blot. The levels of protein expression related to oxidative stress in the C57BL/6J mice fed with HFD and STZ-injected showed the protein expression levels of Bcl-2, Bad, caspase-9, and caspase-3 in sarcopenic obesity.

a) Protein expression levels of Bad and Bcl-2 in the STZ-HFD-LRSV, STZ-HFD-MRSV, and STZ-HFD-HRSV groups.

b) Data are presented as means ± SEM. n=8. *p<0.05, **p<0.001 vs. the control group. #p<0.05, ##p<0.01 vs. the STZ-HFD group. STZ: streptozotocin, HFD: high-fat diet, L-RSV: Low-dose resveratrol, M-RSV: Middle-dose resveratrol, HRSV: High-dose resveratrol.

c) High-fat diet (HFD) with STZ injection in sarcopenic obesity protein expression levels of caspase-3 and caspase-9 by western blotting analysis.

d) Data are presented as means ± SEM. n=8. *p<0.05, **p<0.001 vs. the control group. #p<0.05, ##p<0.01 vs. the STZ-HFD group. STZ: streptozotocin, HFD: high-fat diet, L-RSV: Low-dose resveratrol, M-RSV: Middle-dose resveratrol, HRSV: High-dose resveratrol.

Resveratrol has anti-inflammatory properties in sarcopenia obesity in T2DM C57BL/6J mice skeletal muscle

The FoxO transcription factor integrates signal is a protective role in DNA repair for genomic stability in longevity pathways. The Foxo longevity-related pathways play a crucial role in aging and longevity in STZ-HFD-fed mice model skeletal muscle. Resveratrol intake may improve insulin signaling in skeletal muscle cells via Foxo transcription factor integrates signals. Our results showed Foxo1a/Foxo3a protein expression was caused significantly lower by STZ-HFD by western blotting analysis (Figure 3A). Resveratrol supplementation reversed sarcopenic obesity by Foxo1a/Foxo3a. Resveratrol was higher Foxo 1a expression by STZ-HFD-HRSV ($p<0.05$). Furthermore, resveratrol alleviated Foxo 3a protein expression levels by STZ-HFD-LRSV and STZ-HFD-MRSV ($p<0.05$) (Figure 3B). Foxo1a was lower in low-dose resveratrol intake but higher in high-dose resveratrol STZ-HFD-HRSV group. Resveratrol reverted all muscle during aging. It is well known that TNF-alpha has been shown to impair type 2 DM-induced sarcopenic obesity and is associated with inflammation in skeletal muscle tissue. TNF-alpha and IL-6 in type 2 DM are associated with obesity. IL-6 has been reported to cause skeletal muscle inflammation. Resveratrol was inhibiting the STZ-HFD-induced sarcopenic obesity skeletal muscle inflammation through TNF-alpha and IL-6. Sarcopenia obesity is correlated with increased inflammation markers, TNF-alpha and IL-6. Sarcopenic obesity in type 2 diabetes has increased body weight. STZ has beneficial effects on endothelial dysfunction to induce DM. DM is evidenced to alleviate TNF-α-induced muscle inflammation. Our results found inflammatory markers, TNF-α and IL-6, have elevated by STZ-HFD. Resveratrol has inhibited TNF-α and IL-6 protein expression by western blotting (Figure 3C). STZ-HFD in type 2 DM is associated with obesity. Resveratrol was observed to inhibit IL-6 by STZ-HFD-LRSV ($p<0.05$) and STZ-HFD-MRSV ($p<0.05$) (Figure 3D). PPAR-α is involved in fatty acid oxidation in skeletal muscle and PPAR-Ɣ is involved in lipid storage and insulin sensitivity. Results showed PPAR-α has decreased in the STZ-HFD group, but PPAR-Ɣ has been promoted in the STZ-HFD group (Figures 4 A and 4B). Resveratrol intake has a direct effect on fatty acid oxidation and insulin resistance resulting in PPAR-α and PPAR-Ɣ increases by STZ-HFD-LRSV ($p<0.05$), STZ-HFD-MRSV ($p<0.05$), and STZ-HFD-HRSV ($p<0.05$) (Figure 4B). However, obese and T2DM individuals have greater amounts of IMAT and IRS. IMAT is associated with fat deposition and insulin resistance (IRS) proteins. Our results showed that resveratrol supplementation improvement of insulin resistance occurred through the inhibition of IMAT and IRS by STZ-HFD-LRSV ($p<0.05$), STZ-HFD-MRSV ($p<0.05$), and STZ-HFD-HRSV ($p<0.05$) (Figure 4C and 4D). Thus, resveratrol acts directly or indirectly affect DM-induced sarcopenic obesity mice skeletal muscle health.

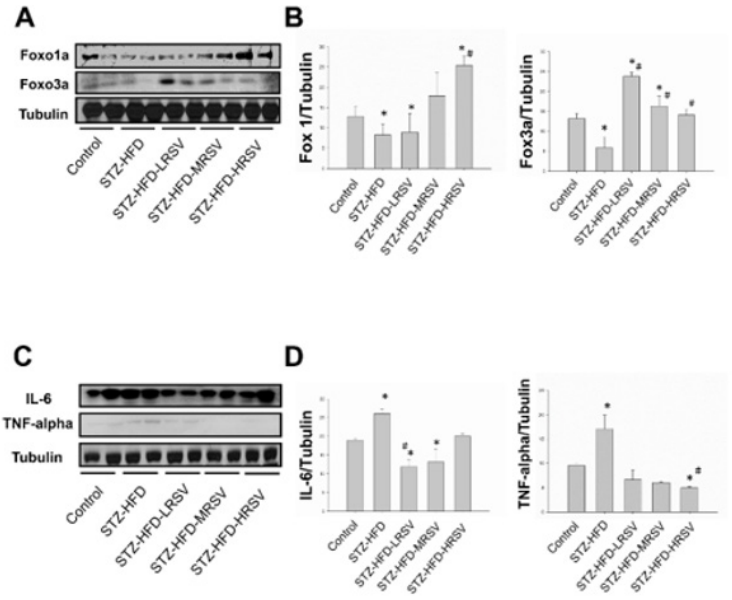

Figure3: The levels of inflammation proteins related to DM-induced sarcopenic obesity including Foxo1a, Foxo3a, IL-6, and TNF-alpha by western blotting analysis. The effects of resveratrol intake suppressed inflammation on STZ-HFD-induced sarcopenic obesity by STZ-HFD-LRSV, STZ-HFD-MRSV, and STZ-HFD-HRSV.

a) Protein expression of Foxo1a and Foxo3a in STZ-HFD C57BL/6J mice by STZ-HFD-LRSV, STZ-HFD-MRSV, and STZ-HFD-HRSV by western blot.

b) All data are presented as means ± SEM. n=8. *p<0.05, **p<0.001 vs. the control group. #p<0.05, ##p<0.01 vs. the STZ-HFD group. STZ: streptozotocin, HFD: high-fat diet, L-RSV: Low-dose resveratrol, M-RSV: Middle-dose resveratrol, HRSV: High-dose resveratrol.

c) Inflammation protein expression of IL-6 and TNF-α in STZ-HFD C57BL/6J mice by STZ-HFD-LRSV, STZ-HFD-MRSV, and STZ-HFD-HRSV using western blot.

d) All data are presented as means ± SEM. n=8. *p<0.05, **p<0.001 vs. the control group. #p<0.05, ##p<0.01 vs. the STZ-HFD group. STZ: streptozotocin, HFD: high-fat diet, L-RSV: Low-dose resveratrol, M-RSV: Middle-dose resveratrol, HRSV: High-dose resveratrol.

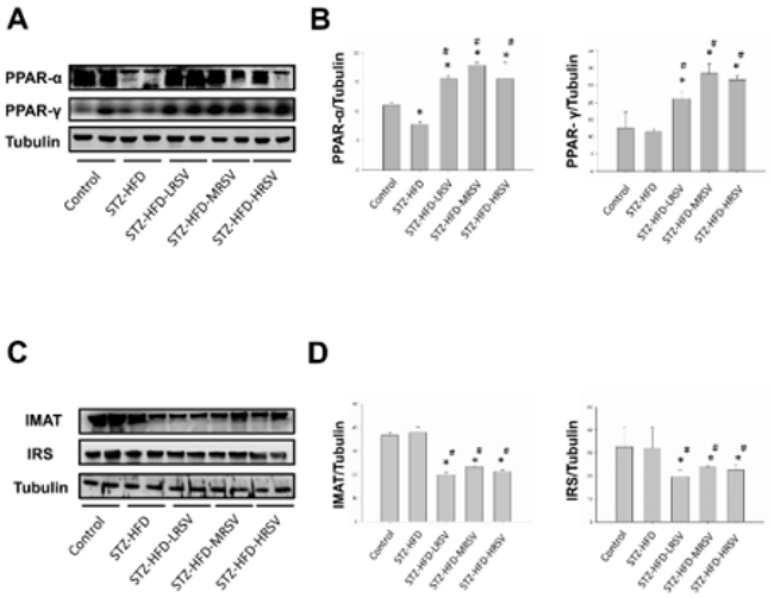

Figure 4: Resveratrol decreased the protein expression in the skeletal muscle fat deposition related to insulin resistance of C57BL/6J mice with diabetes.

a) Resveratrol increases the protein expression of PPAR-α and PPAR-γ using western blotting.

b) Data are presented as means ± SEM. n=8. *p<0.05, **p<0.001 vs. the control group. #p<0.05, ##p<0.01 vs. the STZ-HFD group. STZ: streptozotocin, HFD: high-fat diet, L-RSV: Low-dose resveratrol, M-RSV: Middle-dose resveratrol, HRSV: High-dose resveratrol.

c) Resveratrol decreases the protein expression of IMAT and IRS in the skeletal muscle tissue of C57BL/6J mice with diabetes using western blotting.

d) Data are presented as means ± SEM. n=8. *p<0.05, **p<0.001 vs. the control group. #p<0.05, ##p<0.01 vs. the STZ-HFD group. STZ: streptozotocin, HFD: high-fat diet, L-RSV: Low-dose resveratrol, M-RSV: Middle-dose resveratrol, HRSV: High-dose resveratrol.

Discussion

Sarcopenia is due to type-2 diabetes (T2DM) induced. Sarcopenic obesity is the sarcopenia and obesity combination [10]. Thus, sarcopenic obesity has a synergistic effect on individual effects of sarcopenia and obesity [11]. Thus, we use a high-fat diet and streptozotocin (STZ) to establish DM sarcopenic obesity C57BL/6J mice model. Skeletal muscle weight decrease is linked to sarcopenic obesity to an increased risk of diabetes mellitus (DM). The body weights of the control and STZ-HFD groups were lower initially but higher in the later weeks of high-fed diet (HFD) and resveratrol intake (Figure 1). However, both sarcopenia and obesity are highly associated with DM [12]. Sarcopenic obesity leads to higher mortality rates than either sarcopenia or obesity alone. Sarcopenic obesity is a condition referred to as insulin resistance induced-T2DM. In this study, we want to determine in C57BL/6J mice fed HFD and injected STZ with diabetes with or without sarcopenia. Sarcopenic obesity and obese sarcopenic remain controversial. Sarcopenia with obesity, obesity with sarcopenia, and T2DM-induced sarcopenic obesity in mice or humans are difficult to determine. The results showed that resveratrol reversed sarcopenic obesity-induced low-level inflammation in muscle tissue, resulting in an atrophic state (Figure 1D). STZ-HFD-induced low-level inflammation resulted in skeletal muscle loss (Figure 1E). Bad, Bcl-2, Caspase-3, and Caspase-9 are the associated factors of sarcopenic obesity including aging. Our data regarding sarcopenic obesity with T2DM are scarce. Resveratrol supplementation triggers an abnormal rise in Bad, Bcl-2, Caspase-3, and Caspase-9 that leads to blindness in STZ-HFD-LRSV, STZ-HFD-MRSV, and STZ-HFD-HRSV groups (Figure 2). These results suggest that resveratrol could alleviate skeletal muscle insulin resistance by modulating oxidative stress. Sarcopenic obesity is a pro-inflammatory phenotype and oxidative stress, which can lead to sarcopenia and obesity complications [13]. Thus, incubation of skeletal muscle mass with resveratrol will reverse the insulin-resistant state induced by DM. Sarcopenic obesity-induced low-grade inflammation combined with metabolic aberration by prolonged TNF-α and IL-6 [14]. Resveratrol intake is referred to lower initial IL-6 and TNF-α protein expression levels but high Foxo1a and Foxo3a protein expression levels (Figure 3). However, skeletal muscle is the pathogenesis of insulin resistance mediated by IRS [15, 16]. Our results observed body weights were lower initially but higher in the later stage of aging. Resveratrol improvement of PPAR-α and PPAR-ϒ increases occurred through inhibition of IMAT and IRS in mice skeletal muscle (Figure 4). IMAT is fat deposition, IRS is insulin resistance, and PPAR-α and PPAR- ϒ are fatty oxidation [17]. Indeed, resveratrol concentrations within skeletal muscle increase across the lifespan. IMAT and IRS are associated with poor metabolic and muscle health [18, 19]. Therefore, resveratrol has the potential for activating regulators' anti-oxidative stress and anti-inflammatory proteins to increase suppressed insulin resistance to prevent type 2 diabetes mellitus-induced sarcopenic obesity in older age (Figure 5).

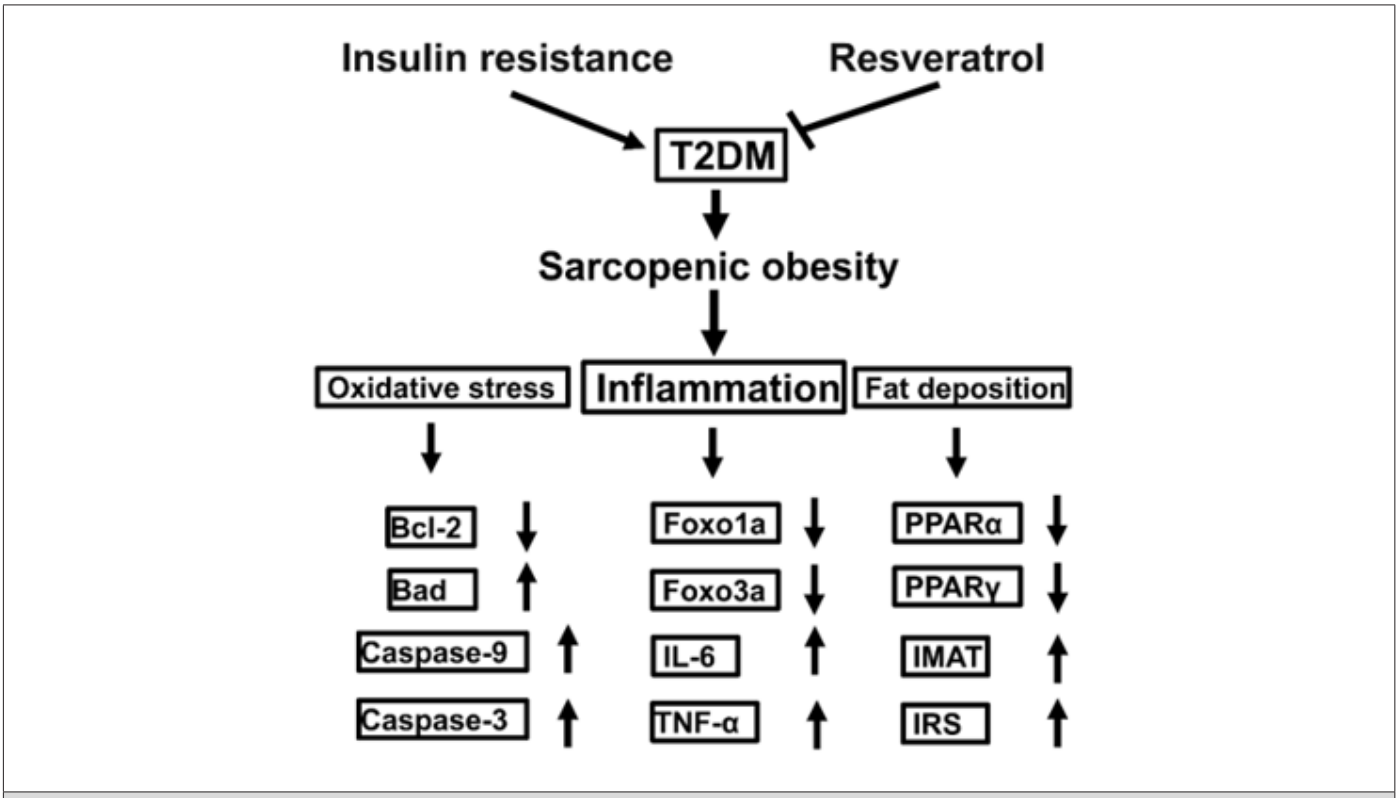

Figure 5: Resveratrol has been developed for pharmacological interest to prevent insulin resistance-induced type 2 diabetes mellitus (T2DM) on sarcopenic obesity diseases-related oxidative stress (Bcl-2, Bad, Caspase-3, Caspase-9), inflammation (Foxo1a, Foxo3a, IL-6, and TNF-α), and fat deposition (PPAR-α, PPAR-γ, IMAT, IRS) in skeletal muscle tissue. Sarcopenic obesity leads to proteins of Bad, Caspase-3, Caspase-9, IL-6, TNF-α, IMAT, and IRS increases and Bcl-2, Foxo1a, Foxo3a, PPAR-α, and PPAR-γ decreases.

Conclusion

Resveratrol supplementary intake is non-toxic and easily absorbed by animals and humans. Therefore, resveratrol may be a therapeutic option for a type-1 diabetes mellitus protective effect in sarcopenia obesity. Therefore, T2DM-induced sarcopenic obesity skeletal muscle insulin resistance was reverted by resveratrol in T2DM C57BL/6J mice skeletal muscle (Figure 6). Resveratrol intake has the potential protection from anti-obesity and obesity-dependent anti-sarcopenic effects in sarcopenic obesity.

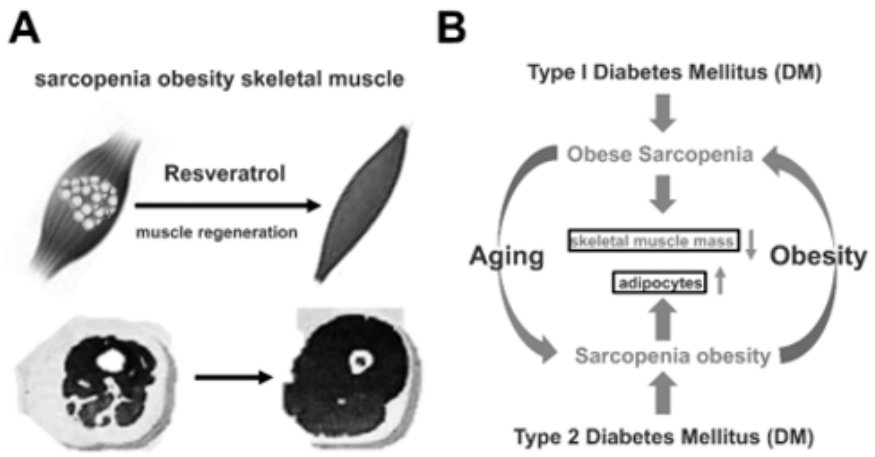

Figure 6: Evidence to support the view that sarcopenic obesity.
a) After feeding for four weeks of resveratrol intake, sarcopenic obesity mice's skeletal muscle mass increased and skeletal muscle regeneration increased.
b) Increased adipocytes and skeletal muscle loss weight are also common sarcopenic obesity and obese sarcopenia transfer. T1DM and T2DM are induced in two different ways of sarcopenia and obesity with progressive diabetes. Obesity is media condition aging and older age has accumulated with excess body fat weight.

Acknowledgments

N/A.

References

1. Liu Z, Huang S, Yuan X, Wang Y, Liu Y, et al. (2023) The role of vitamin D deficiency in the development of paediatric diseases. Ann Med 55: 127-135.
2. Molinari M, (2020) Sarcopenia, obesity, and postoperative outcomes after hepatectomy for hepatocellular carcinoma. Hepatobiliary Surg Nutr 9: 198-201.
3. Mohamed AA, Risse K, Stock J, Heinzel A, Mottaghy FM, et al. (2022) Body Composition as a Predictor of the Survival in Anal Cancer. Cancers (Basel) 18;14(18): 4521.
4. Bahat G. (2022) Sarcopenic obesity: a hot yet under considered evolving concept. Eur Geriatr Med 13: 1023-1024.
5. Kim HI, Ahn SH, Kim Y, Lee JE, Choi E, et al. (2022) Effects of sarcopenia and sarcopenic obesity on joint pain and degenerative osteoarthritis in postmenopausal women. Sci Rep 12(1): 13543.
6. Tanabe H, Hirai H, Saito H, Tanaka K, Masuzaki H, et al. (2022) Detecting Sarcopenia Risk by Diabetes Clustering: A Japanese Prospective Cohort Study. J Clin Endocrinol Metab 107: 2729-2736.
7. Argyropoulou D, Geladas ND, Nomikos T, Paschalis V (2022) Exercise and Nutrition Strategies for Combating Sarcopenia and Type 2 Diabetes Mellitus in Older Adults. J Funct Morphol Kinesiol 7(2): 48.
8. Gao S, Yu L, Yi G, Li T, Chen Z, et al. (2022) Exercise Intervention as a Therapy in Patients with Diabetes Mellitus and Sarcopenia: A Meta-Analysis. Diabetes Ther 13: 1311-1325.
9. Kumar R, García-Compeán D, Maji T (2022) Hepatogenous diabetes: Knowledge, evidence, and skepticism. World J Hepatol 14: 1291-1306.
10. Patrícia da Silva Souza A, Lopes de Souza S, Rodrigues Alves Dos Santos ME, Lucĺecia da Silva M, Almeida Barros WM (2022) Letter to editor: Comment on "sarcopenic obesity is associated with cognitive impairment in community-dwelling older adults: The bunkyo health study" clinical nutrition 2022. Clin Nutr 41(9): 2054-2055.
11. Zembura M, Matusik P (2022) Sarcopenic Obesity in Children and Adolescents: A Systematic Review. Front Endocrinol (Lausanne) 13: 914740.
12. Chen SH, Liang YJ (2022) The Role of Lifestyle Intervention, in Addition to Drugs, for Diabetic Kidney Disease with Sarcopenic Obesity. Life (Basel) 12: 380.
13. Kumar R, García Compeán D, Maji T (2022) Hepatogenous diabetes: Knowledge, evidence, and skepticism. World J Hepatol 14: 1291-1306.
14. Merchant RA, Soong JTY, Morley JE (2022) Gender Differences in Body Composition in Pre-Frail Older Adults with Diabetes Mellitus. Front Endocrinol (Lausanne) 13: 795594.
15. Yoo MC, Won CW, Soh Y (2022) Association of high body mass index, waist circumference, and body fat percentage with sarcopenia in older women. BMC Geriatr 22: 937.
16. Konishi K, Nakagawa H, Asaoka T, Kasamatsu Y, Goto T, et al. (2022) Sarcopenia among people living with HIV and the effect of antiretroviral therapy on body composition. Medicine (Baltimore) 101: e31349.
17. Villani A (2022) Lifestyle Strategies for the Management of Obesity in Older Adults: From Controversies to Alternative Interventions. Healthcare (Basel) 10: 2107.
18. Kim HI, Ahn SH, Kim Y, Lee JE, Choi E, et al. (2022) Effects of sarcopenia and sarcopenic obesity on joint pain and degenerative osteoarthritis in postmenopausal women. Sci Rep 12: 13543.
19. Guan L, Li T, Wang X, Yu K, Xiao R, et al. (2022) Predictive Roles of Basal Metabolic Rate and Body Water Distribution in Sarcopenia and Sarcopenic Obesity: The link to Carbohydrates. Nutrients 14: 3911.