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WISP-1 in Obesity and Metabolic Dysfunction: The Modulatory Role of Exercise Training-A Narrative Review

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A B S T R A C T

Obesity is a major global health concern strongly associated with metabolic disorders, chronic low-grade inflammation, insulin resistance, and an increased risk of cardiovascular disease. Among the emerging biomarkers implicated in obesity-related metabolic dysfunction, Wnt1-inducible signaling pathway protein-1 (WISP-1), a matricellular protein belonging to the CCN (Cyr61, C: Cyr61(Cysteine-rich angiogenic inducer 61/ C: CTGF (Connective Tissue Growth Factor), N: NOV (Nephroblastoma Overexpressed gene) family, has attracted increasing attention. WISP-1 is involved in tissue remodeling, extracellular matrix regulation, inflammatory signaling, and metabolic processes, and accumulating evidence suggests that elevated circulating levels are associated with visceral adiposity, insulin resistance, and systemic inflammation. Physical activity is widely recognized as a cornerstone intervention for improving metabolic health and reducing obesity-related complications. Exercise training modulates adipose tissue function, inflammatory pathways, insulin signaling, and extracellular matrix remodeling mechanisms that may be linked to alterations in WISP-1 expression. However, the extent to which exercise directly regulates WISP-1 remains incompletely understood, and existing findings are not entirely consistent across populations and intervention types. This narrative review synthesizes current evidence regarding the relationship between physical activity and WISP-1 levels in individuals with obesity. Available studies suggest that aerobic training, resistance exercise, and high-intensity interval training (HIIT) may reduce circulating WISP-1 concentrations, often in parallel with improvements in insulin sensitivity, body composition, and inflammatory markers. Nevertheless, the number of interventional studies remains limited, sample sizes are generally small, and methodological heterogeneity complicates definitive interpretation. Furthermore, much of the mechanistic understanding linking WISP-1 to insulin resistance derives from experimental or associative human data, highlighting the need for cautious interpretation of causality. Variations in exercise modality, intensity, duration, and participant characteristics may influence WISP-1 responses, underscoring the complexity of its regulation. Overall, while emerging evidence supports a potential link between exercise-induced metabolic improvements and modulation of WISP-1, well-designed longitudinal and mechanistic studies are required to clarify its physiological role and determine whether WISP-1 can serve as a reliable biomarker or therapeutic target in obesity related metabolic dysfunction.

Keywords: *WISP-1, physical activity, obesity, inflammation, extracellular matrix, metabolic health*

1. Introduction

Obesity is a global health challenge associated with numerous comorbidities, including insulin resistance,

chronic inflammation, type 2 diabetes, and cardiovascular diseases (1-3). The prevalence of obesity has increased dramatically in recent decades, prompting intensified efforts to better understand the molecular and physiological mechanisms underlying its pathophysiology and to identify effective therapeutic strategies (4-6). Among these mechanisms, adipose tissue derived factors, known as adipokines, have garnered significant attention due to their regulatory role in metabolic homeostasis and inflammatory processes (6, 7). WNT1-inducible signaling pathway protein 1 (WISP-1), a member of the CCN family of matricellular proteins, has recently emerged as a molecule of interest in obesity research. WISP-1 is expressed in multiple tissues, including adipose tissue, and has been implicated in extracellular matrix remodeling, inflammatory signaling, and metabolic regulation (8). Accumulating evidence indicates that circulating WISP-1 levels are positively associated with markers of adiposity, insulin resistance, and chronic low grade inflammation (9). However, much of the current evidence is observational or derived from experimental models, and definitive mechanistic conclusions in humans remain limited.

Exercise training is widely recognized as a cornerstone intervention for managing obesity and improving metabolic health. Regular physical activity contributes to enhanced insulin sensitivity, improved body composition, and reduced systemic inflammation (10). Exercise has also been shown to influence adipokine secretion profiles, suggesting a potential interaction between physical activity and WISP-1 regulation (11). Nevertheless, the extent to which exercise directly modulates WISP-1 expression, and whether such changes mediate improvements in metabolic outcomes, remains incompletely understood. Recent studies have explored the effects of aerobic training, resistance exercise, and high-intensity interval training (HIIT) on circulating WISP-1 levels in individuals with obesity and related metabolic disorders. Although several interventions report reductions in serum WISP-1 concentrations following structured exercise programs, findings are not entirely consistent, and study designs often involve small sample sizes and short intervention durations (11-13). Furthermore, variability in participant characteristics including; age, sex, metabolic status, and comorbid conditions; may influence reported outcomes. Importantly, while reductions in WISP-1 are frequently observed alongside improvements in insulin sensitivity and inflammatory markers, the available data primarily support associative relationships rather than established causality (13). Therefore, careful interpretation of these findings is warranted.

Given the emerging but still evolving evidence, this narrative review aims to synthesize and critically evaluate current findings regarding the relationship between exercise training and WISP-1 levels in individuals with obesity. In addition to summarizing existing studies, this review highlights methodological limitations, identifies gaps in mechanistic understanding, and discusses the clinical relevance of WISP-1 modulation in the context of exercise based interventions.

2. WISP-1 and Obesity

WISP-1, or WNT1-inducible signaling pathway protein 1, is a matricellular protein that plays a key role in the regulation of cellular processes such as proliferation, migration, differentiation, and survival (8). Part of the CCN protein family, WISP-1 is expressed in various tissues, including adipose tissue, where it functions as an adipokine. Accumulating evidence suggests that WISP-1 levels may be elevated in individuals with obesity, particularly in visceral adipose tissue, and may be associated with systemic inflammation and metabolic dysregulation (9, 14).

The role of WISP-1 in metabolic disorders has attracted increasing attention, particularly its potential involvement in obesity-related complications. Mechanistically, WISP-1 is proposed to interact with key signaling pathways, including WNT/β-catenin, which may influence adipogenesis, inflammation, and extracellular matrix remodeling (14). However, it is important to note that most evidence is associative or derived from experimental models, and definitive mechanistic conclusions in humans remain limited. Elevated WISP-1 levels have been observed alongside increased inflammatory markers, such as tumor necrosis factor-α (TNF-α) and interleukin-6 (IL-6) (15), which are central to obesity induced chronic inflammation. These cytokines have been implicated in the impairment of insulin signaling and metabolic regulation, suggesting that WISP-1 may contribute, alongside other

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factors, to a pro-inflammatory and metabolically dysregulated state in obesity (7).

Recent studies have highlighted the potential of WISP-1 as a biomarker for obesity-related complications. For example, elevated serum WISP-1 levels have been reported in individuals with type 2 diabetes, cardiovascular disease, and certain cancers, although the predictive utility and causative role remain under investigation (16). In the context of exercise, WISP-1 has emerged as a potentially modifiable factor. Interventional studies suggest that exercise training may reduce circulating WISP-1 levels in obese individuals, often alongside improvements in insulin sensitivity, body composition, and inflammatory markers (11, 12). However, the extent to which these changes are direct or mediated by broader metabolic adaptations is not fully clear.

Despite these advances, many questions remain regarding the regulation of WISP-1 in obesity and its precise role in mediating the effects of exercise. Heterogeneity in study populations, exercise modalities, intensity, and duration, as well as small sample sizes, limit the ability to draw definitive conclusions. Understanding the mechanisms underlying WISP-1 modulation by exercise may provide important insights, but further well designed studies are needed to establish its causal role and potential as a therapeutic target.

3. WISP-1 and insulin resistance

WISP-1 is a protein from the CCN family that regulates various cellular processes, including inflammation, growth, and fibrosis. Evidence from observational and experimental studies indicates that WISP-1 may be associated with insulin resistance and metabolic disturbances, although causality in humans has not been firmly established (9, 17, 18). Several potential mechanisms have been proposed: WISP-1 may stimulate inflammatory processes in adipose tissue and muscle, partly through increases in TNF-α and IL-6, which can interfere with insulin signaling (14). WISP-1 is suggested to influence key insulin signaling pathways, including PI3K/Akt and MAPK, which are central to glucose and lipid metabolism (9, 14, 19). Overactivation of these pathways in experimental models has been associated with decreased insulin sensitivity, but human evidence remains largely associative. WISP-1 acts as a fibrotic factor, promoting extracellular matrix production and tissue remodeling in adipose tissue, muscle, and liver (20, 21). This may indirectly impair insulin responsiveness, but the extent to which WISP-1 independently drives fibrosis versus reflecting systemic metabolic stress is unclear (9, 19)

Furthermore, WISP-1 is linked to fat metabolism and distribution, particularly in visceral fat and the liver, which are key contributors to insulin resistance in obesity and type 2 diabetes (22, 23). In individuals with metabolic disorders, elevated WISP-1 is frequently observed alongside inflammatory and fibrotic changes, suggesting a multifaceted association with metabolic dysregulation, although direct causal pathways remain to be conclusively demonstrated (23, 24).

Exercise interventions have been reported to modulate WISP-1 levels, with aerobic, resistance, and high-intensity interval training often resulting in reductions in circulating concentrations, concomitant with improvements in insulin sensitivity and inflammatory markers. Nevertheless, differences in study design, population characteristics, intervention type, and duration limit the generalizability of these findings, and causality cannot be inferred with certainty (23).

Overall, WISP-1 appears to be a multifactorial mediator associated with insulin resistance and obesity related metabolic disturbances. While exercise may influence WISP-1, current evidence primarily supports associative relationships, emphasizing the need for longitudinal and mechanistic studies to clarify its physiological and therapeutic relevance.

4. Effects of exercise training on WISP-1 levels

Emerging evidence suggests that physical activity may influence circulating WISP-1 levels in individuals with obesity and related metabolic conditions. Aerobic exercise, resistance training, and high-intensity interval training (HIIT) have all been studied, with many interventions reporting reductions in serum WISP-1, often accompanied by improvements in insulin sensitivity, body composition, and inflammatory markers (12, 13).

For example, circuit resistance training in individuals with type 2 diabetes and obesity was associated with decreased WISP-1 and WISP-2 levels, along with

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reductions in TNF-α and improvements in insulin resistance (12). Similarly, aerobic training in obese men with type 2 diabetes appeared to reduce serum WISP-1 concentrations and associated inflammatory markers, although sample sizes were limited and intervention durations relatively short (25). HIIT and moderate intensity continuous training (MICT) in obese individuals have been reported to lower WISP-1, with some studies suggesting a more pronounced effect with HIIT, but heterogeneity in population characteristics and exercise protocols complicates interpretation (13).

In overweight and obese girls, both interval aerobic and continuous aerobic training were associated with reductions in serum WISP-1 and TNF-α, although the magnitude of effect differed slightly by training modality (26). Resistance training in obese men also appeared to reduce WISP-1 and WISP-2 levels, alongside improvements in body composition and insulin sensitivity (27). Generally, these findings suggest that WISP-1 may be responsive to physical activity, but the extent and consistency of these effects remain uncertain.

It is important to note several limitations in the current literature. Many studies are limited by small sample sizes, short intervention durations (typically 8-12 weeks), heterogeneous populations, and variations in exercise modality, intensity, and adherence**.** Additionally, while reductions in WISP-1 frequently co-occur with improvements in metabolic markers, causality cannot be inferred, and it remains unclear whether changes in WISP-1 mediate or merely reflect broader metabolic adaptations.

Despite these limitations, the observed associations provide preliminary support for the potential of WISP-1 as a modifiable factor in exercise interventions. Future research should aim to include larger, well controlled trials with standardized exercise protocols, longitudinal follow up, and mechanistic assessments to determine whether exercise induced modulation of WISP-1 contributes directly to improvements in metabolic health. In summary, while exercise training appears to influence WISP-1, current evidence predominantly supports associative relationships, highlighting the need for further investigation to clarify mechanistic links and potential clinical implications.

Table 1

Effect of exercise training on WISP-1 levels

Study	Population	Sample Size	Study Design	Exercise Intervention	Duration	Main Findings	Limitations
Tayebi et al. (11)	Individuals with type 2 diabetes and obesity	28	Randomized controlled trial	Circuit resistance training	8 weeks	Decreased serum WISP-1 and WISP-2; improved insulin resistance; reduced TNF-α	Small sample, short duration, limited generalizability
Kharghani et al. (25)	Obese men with type 2 diabetes	24	RCT	Aerobic training (moderate intensity)	8 weeks	Reduced serum WISP-1 and TNF-α; improved HOMA-IR	Small sample, men only, short-term follow-up
Gholami et al. (13)	Obese men	30	RCT	HIIT vs. MICT	10 weeks	Both interventions decreased WISP-1; HIIT showed slightly greater reduction	Small sample, limited population diversity
Bahreini et al. (26)	Overweight/obese girls	32	RCT	Interval aerobic vs. continuous aerobic	8 weeks	Both reduced WISP-1 and TNF-α; interval training slightly more effective	Small sample, girls only, short duration
Kermani et al. (27)	Obese men	26	RCT	Resistance training	8 weeks	Reduced WISP-1 and WISP-2; improved insulin sensitivity and	Small sample, men only, no long-term follow-up

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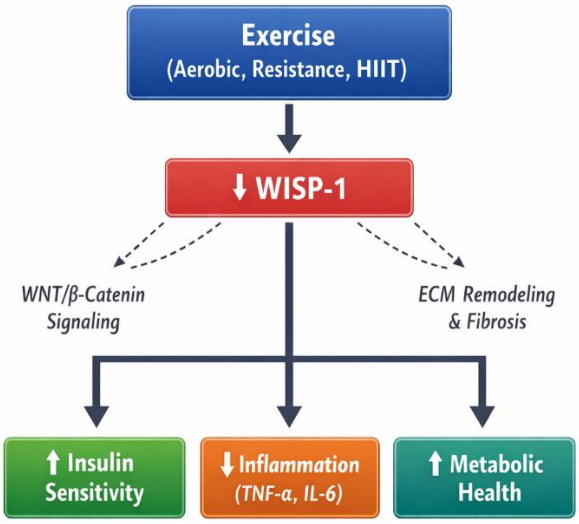
body composition

Figure 1

Potential effects of exercise on WISP-1 and metabolic health in obesity



Solid arrows indicate observed associations in human intervention studies, including reductions in WISP-1 after aerobic, resistance, or HIIT exercise, along with improvements in insulin sensitivity, inflammation, and overall metabolic health. These associations do not confirm direct causality. Dashed arrows represent proposed mechanistic pathways that remain under investigation, including modulation of WNT/β-catenin signaling, extracellular matrix (ECM) remodeling, and adipose tissue adaptations.

Abbreviations: WISP-1, Wnt1-inducible signaling pathway protein-1; HIIT, high-intensity interval training; ECM, extracellular matrix; TNF-α, tumor necrosis factor-alpha; IL-6, interleukin-6.

5. Mechanisms of WISP-1 reduction by exercise training

Exercise training reduces WISP-1 levels through a variety of interrelated mechanisms that impact metabolic and inflammatory pathways. These mechanisms include:

1. **Reduction in Adipose Tissue Mass**: Exercise promotes weight loss and reduces visceral and subcutaneous adipose tissue, leading to a decrease in adipokine secretion, including WISP-1. Adipose tissue is a primary site of WISP-1 expression, and its reduction directly correlates with lowered WISP-1 levels (13, 25, 27).
2. **Improved Insulin Sensitivity**: Physical activity enhances insulin sensitivity by increasing glucose uptake in skeletal muscles and reducing insulin resistance. Lower insulin resistance is associated with decreased WISP-1 levels, as WISP-1 is implicated in pathways that exacerbate insulin resistance (25, 27).
3. **Anti-inflammatory Effects**: Regular exercise reduces chronic low-grade inflammation, characterized by lower levels of pro-inflammatory cytokines such as TNF-α and IL-6. Since WISP-1 expression is positively regulated by inflammation, its levels decrease as the inflammatory milieu improves (25).
4. **Modulation of the WNT/β-catenin Pathway**: Exercise affects the WNT/β-catenin signaling pathway, which is involved in adipogenesis and

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WISP-1 regulation. Modulating this pathway reduces WISP-1 expression and its downstream effects on adipocyte differentiation and inflammation (13).

5. **Improvement in Body Composition**: Exercise-induced improvements in lean body mass and reductions in fat mass contribute to the modulation of adipokine profiles, including WISP-1. Enhanced muscle mass increases the secretion of anti-inflammatory myokines, which may further suppress WISP-1 expression (10).
6. **Exercise Intensity and Type**: Different exercise modalities, such as high-intensity interval training (HIIT), moderate-intensity continuous training (MICT), and resistance training, have been shown to variably influence WISP-1 levels. HIIT, in particular, may have a more pronounced effect due to its impact on metabolic rate and inflammatory cytokine profiles (13).

These mechanisms highlight the multifaceted effects of exercise training on WISP-1 levels and underscore the importance of physical activity as a non-pharmacological intervention for improving metabolic health in obese individuals.

6. Clinical Implications

Understanding the impact of exercise on WISP-1 levels has significant clinical implications:

1. **Non-Pharmacological Management:** Exercise serves as a cost-effective and accessible intervention for reducing WISP-1 levels and improving metabolic health.
2. **Biomarker Potential:** Monitoring WISP-1 levels could provide insights into the efficacy of exercise interventions and the progression of metabolic disorders.
3. **Tailored Exercise Programs:** Personalized exercise regimens targeting WISP-1 regulation could enhance therapeutic outcomes for obese individuals.

Few studies have directly examined the relationship between exercise and WISP-1 levels. Further research is needed to validate these findings in larger and more diverse populations. The molecular pathways linking exercise to WISP-1 modulation remain unclear and warrant deeper investigation. Studies exploring the sustained impact of exercise on WISP-1 levels and its clinical significance are necessary.

7. Conclusion

Exercise training appears to be a promising strategy for modulating circulating WISP-1 levels and potentially improving obesity related inflammation and metabolic dysfunction. Evidence from human studies suggests that aerobic, resistance, and HIIT may reduce WISP-1 concentrations, often in parallel with improvements in insulin sensitivity, body composition, and inflammatory markers such as TNF-α and IL-6. However, the current literature is limited by small sample sizes, short intervention durations, heterogeneous populations, and variations in exercise modality, intensity, and adherence. As a result, causal relationships between exercise-induced changes in WISP-1 and improvements in metabolic health remain to be fully established. Future research should focus on well-controlled, longitudinal studies with larger and more diverse populations, standardized exercise protocols, and mechanistic investigations to clarify whether changes in WISP-1 contribute directly to metabolic improvements. Such studies could inform the development of optimized exercise interventions targeting WISP-1 and provide insight into its potential role as a biomarker or therapeutic target in obesity and related metabolic disorders.

Authors' Contributions

F. D. & A. A. were equally involved in writing, editing, and methodology, and checked and approved the final draft of the manuscript.

Declaration

Artificial intelligence tools were not used for data analysis or interpretation. AI assistance was limited to language editing and formatting during manuscript preparation.

Transparency Statement

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The data that support the findings of this study are available from the corresponding author upon reasonable request.

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Declaration of Interest

The authors report no conflict of interest.

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Ethics Considerations

No ethical approval was required for this study because it is a narrative review of published studies and did not involve animal experimentation or human participants.

References

1. Organization WH. Obese and overweight. 2024.
2. Tayebi SM, Ghanbari-Niaki A, Saeidi A, Hackney AC. Exercise training, neuregulin 4 and obesity. Annals of applied sport science. 2017;5(2):1. [PMID: 30899900] [PMCID: PMC6424364] [DOI]
3. Saeidi A, Tayebi SM, Khosravi A, Malekian F, Khodamoradi A, Sellami M, et al. Effects of exercise training on type 2-diabetes: the role of Meteorin-like protein. Health promotion perspectives. 2019;9(2):89. [PMID: 31249794] [PMCID: PMC6588808] [DOI]
4. Saeidi A, Nouri-Habashi A, Razi O, Ataeinosrat A, Rahmani H, Mollabashi SS, et al. Astaxanthin supplemented with high-intensity functional training decreases adipokines levels and Cardiovascular Risk factors in men with obesity. Nutrients. 2023;15(2):286. [PMID: 36678157] [PMCID: PMC9866205] [DOI]
5. Saeidi A, Jabbour G, Ahmadian M, Abbassi-Daloii A, Malekian F, Hackney AC, et al. Independent and combined effects of antioxidant supplementation and circuit resistance training on selected adipokines in postmenopausal women. Frontiers in physiology. 2019;10:484. [PMID: 31105587] [PMCID: PMC6499001] [DOI]
6. Delfan M, Amadeh Juybari R, Gorgani-Firuzjaee S, Høiriis Nielsen J, Delfan N, Laher I, et al. High-intensity interval training improves cardiac function by miR-206 dependent HSP60 induction in diabetic rats. Frontiers in Cardiovascular Medicine. 2022;9:927956. [PMID: 35845054] [PMCID: PMC9277013] [DOI]
7. Ouchi N, Parker JL, Lugus JJ, Walsh K. Adipokines in inflammation and metabolic disease. Nature reviews immunology. 2011;11(2):85-97. [PMID: 21252989] [PMCID: PMC3518031] [DOI]
8. Jun J-I, Lau LF. Taking aim at the extracellular matrix: CCN proteins as emerging therapeutic targets. Nature reviews Drug discovery. 2011;10(12):945-63. [PMID: 22129992] [PMCID: PMC3663145] [DOI]
9. Mirr M, Owecki M. An update to the WISP-1/CCN4 role in obesity, insulin resistance and diabetes. Medicina. 2021;57(2):100. [PMID: 33498604] [PMCID: PMC7911315] [DOI]
10. Pedersen BK, Saltin B. Exercise as medicine–evidence for prescribing exercise as therapy in 26 different chronic diseases. Scandinavian journal of medicine & science in sports. 2015;25:1-72. [PMID: 26606383] [PMCID: PMC12189298] [DOI]
11. Tayebi SM, Saeidi A, Shahghasi R, Golmohammadi M. The eight-week circuit resistance training decreased the serum levels of WISP-1 and WISP-2 in individuals with type 2 diabetes. Annals of Applied Sport Science. 2023;11(4):0-. [PMCID: PMC12694805] [DOI]
12. Chang JS, Kim TH, Kong ID. Exercise intervention lowers aberrant serum WISP-1 levels with insulin resistance in breast cancer survivors: A randomized controlled trial. Scientific reports. 2020;10(1):10898. [PMID: 32616883] [PMCID: PMC7331642] [DOI]
13. Gholami M. Changes of WNT1-inducible signaling pathway protein-1 (WISP-1), ICAM-1 and VCAM-1 in type 2 diabetic women following 12-week high intensity interval training and moderate intensity continuous training. Daneshvar Medicine. 2023;31(3):68-80.
14. Barchetta I, Cimini FA, Capoccia D, De Gioannis R, Porzia A, Mainiero F, et al. WISP1 is a marker of systemic and adipose tissue inflammation in dysmetabolic subjects with or without type 2 diabetes. Journal of the Endocrine Society. 2017;1(6):660-70. [PMID: 29264519] [PMCID: PMC5686652] [DOI]
15. Klimontov VV, Bulumbaeva DM, Fazullina ON, Lykov AP, Bgatova NP, Orlov NB, et al. Circulating Wnt1-inducible signaling pathway protein-1 (WISP-1/CCN4) is a novel biomarker of adiposity in subjects with type 2 diabetes. Journal of Cell Communication and Signaling. 2020;14:101-9. [PMID: 31782053] [PMCID: PMC7176766] [DOI]
16. Habib NS, EL-Hefnawy MH, El-Mesallamy HO. Assessment of circulating Wnt1-inducible signaling pathway protein 1 (WISP1) in obesity and type 2 diabetes mellitus patients. Archives of Pharmaceutical Sciences Ain Shams University. 2018;2(1):47-53. [DOI]
17. Saeidi A, Hackney AC, Tayebi SM, Ahmadian M, Zouhal H. Diabetes, insulin resistance, fetuin-B and exercise training. Annals of applied sport science. 2019;7(2):1-2. [PMID: 31321377] [PMCID: PMC6639044] [DOI]
18. Saeidi A, Seifi-Ski-Shahr F, Soltani M, Daraei A, Shirvani H, Laher I, et al. Resistance training, gremlin 1 and macrophage migration inhibitory factor in obese men: a randomised trial. Archives of Physiology and Biochemistry. 2023;129(3):640-8. [PMID: 33370549] [DOI]
19. Maiese K. WISP1: Clinical insights for a proliferative and restorative member of the CCN family. Current Neurovascular Research. 2014;11(4):378-89. [PMID: 25219658] [PMCID: PMC4205162] [DOI]
20. González D, Campos G, Pütter L, Friebel A, Holland CH, Holländer L, et al. Role of WISP1 in Stellate Cell Migration and Liver Fibrosis. Cells. 2024;13(19):1629. [PMID: 39404393] [PMCID: PMC11475959] [DOI]
21. Christopoulou M-E, Aletras AJ, Papakonstantinou E, Stolz D, Skandalis SS. WISP1 and macrophage migration inhibitory factor in respiratory inflammation: Novel insights and therapeutic potentials for asthma and copd. International Journal

Health Nexus
E-ISSN: 2981-2569

of Molecular Sciences. 2024;25(18):10049. [PMID: 39337534] [PMCID: PMC11432718] [DOI]
22. Hörbelt T. The role of the novel adipokines WISP1 and sFRP4 in obesity and type 2 diabetes and their impact on insulin action and energy metabolism in muscle and liver: Dissertation, Düsseldorf, Heinrich-Heine-Universität, 2018; 2018.
23. Grünberg J. WISP-2-A Novel Adipokine Related to Obesity and Insulin resistance2015.
24. Singh K, Oladipupo SS. An overview of CCN4 (WISP1) role in human diseases. Journal of Translational Medicine. 2024;22(1):601. [PMID: 38937782] [PMCID: PMC11212430] [DOI]
25. Kharghani A, Rezaeian N, Yaghoubi A. Effect of Aerobic Training on Serum Levels of WISP1 and TNF-α and Insulin Resistance in Obese Men with Type 2 Diabetes. Journal of Applied Exercise Physiology. 2022;18(35):153-64.
26. Bahreini A, Fathi R. Comparing the Effect of Eight Weeks of Interval and Continuous Aerobic Training on Serum Levels of WISP1 and TNF-Î±in Overweight/0bese Girls. Journal of Sports and Biomotor Sciences. 2017;9(18):1-12.
27. Kermani S, Abbassi Daloii A, Abdi A, Saeidi A. The effect of resistance training on hormones secreted from the adipose tissue (WISP-1 and WISP-2) in obese men. Ebnesina. 2022;24(3):15-24.

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E-ISSN: 2981-2569