



The Role of Insulin-Like Growth Factor 1 And Irisin in Muscle Dystrophy: Effect of Age and Comparison Among Patients

Zeena Firas Haider ¹, Shaima Razaq Ibraheem ²

^{1,2} University of Baghdad , Biotechnology Department , College of Sciences.

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Abstract

Background: Muscle dystrophy and sarcopenia involve progressive muscle wasting and functional decline. Insulin-like growth factor 1 (IGF-1) and irisin are key myokines regulating muscle growth and metabolism. Changes in their levels may reflect disease and aging effects. To evaluate the role of IGF-1 and irisin in muscle dystrophy. **Methods:** A case-control study was conducted in Baghdad hospitals (Dec 2024–Feb 2025) involving 55 patients with muscle dystrophy and 35 healthy controls. Serum IGF-1 and irisin were quantified utilizing ELISA, and data were analyzed with SPSS v26 ($p \leq 0.05$). **Results:** Patients had significantly lower IGF-1 (75.18 ± 3.16 vs. 112.14 ± 8.75 ng/ml, $p = 0.0001$) and irisin (5.19 ± 0.22 vs. 9.92 ± 0.78 ng/ml, $p = 0.0001$) levels. Irisin declined with age, especially >70 years ($p = 0.0481$). No correlation was found between IGF-1 and irisin. **Conclusion:** This study suggests a potential role of IGF-1 and irisin in muscle dystrophy progression

Keywords: Muscle dystrophy, IGF-1, ELISA, irisin.

¹ University of Baghdad , Biotechnology Department , College of Sciences.
zena.ferras1806a@sc.uobaghdad.edu.iq

² University of Baghdad , Biotechnology Department , College of Sciences.
Shaima.ibraheem@sc.uobaghdad.edu.iq

Introduction

Sarcopenia, or age-related muscle loss, affects 5-10% of older adults. It first shows clinical signs in the sixth or seventh decade of life, but the underlying deterioration begins decades earlier (Gustafsson & Ulfhake, 2024). Compared to younger adults, older people display significant changes in muscle morphology. While these morphological changes are accompanied by functional loss, the decrease in lower-body strength with age is more noticeable than in the upper body (Sarto *et al.*, 2023). Irisin is one of these myokines involved in the paracrine and/or endocrine regulation of various tissues and organs, including the liver, fat, and brain, as well as the autocrine HCs of metabolism within the muscle (Iglesias, 2025). Irisin can enhance resistance to oxidative stress-induced muscle atrophy, endurance, and muscular strength, which is attributed to irisin's effects on the biogenesis of mitochondria, autophagy, and dynamics in skeletal muscles of the skeleton. Its multifunctional properties also include promoting the metabolism of bones, where it stimulates osteoblast differentiation and proliferation, potentially contributing to a treatment for osteoporosis and other musculoskeletal conditions (Zhao, Wu, Zhu, Liu, & Xu, 2024). The liver and skeletal muscle produce IGF1, which has endocrine and paracrine effects on muscle fibers. It appears that this growth factor is crucial for activating a specific signaling pathway in muscle fibers, resulting in muscle protein synthesis (MPS). This signaling cascade is initiated by the local production and release of IGF1 during muscle contraction, which then binds to its receptor on the sarcolemma (Erskine & Degens, 2019). IGF-1 and irisin have been researched individually in connection to neuromuscular diseases, but joint research in relation to muscle dystrophy is minimal. The current literature provides inconsistent data regarding IGF-1 and irisin levels in relation to disease types and ages, and an interaction between them has yet to be determined. The relationship between serum levels of IGF-1 and irisin in relation to muscular dystrophy patients and normal participants, alterations in irisin levels in different ages, and a connection between IGF-1 and irisin levels will also be considered in this study.

Materials and Methods

Subject

Peripheral blood samples were obtained from patients with muscle diseases at Al-Wasiti Hospital, Saad Al-Watary Hospital, and the City of Medicine in Baghdad. During the period from December 2024 until February 2025. The Iraqi Ministry of Health approved the protocol of work. A total of 90 blood samples were collected. 55 samples from patients diagnosed with muscle disease (aged 27-77 years), and 35 samples from healthy individuals (aged 20-73), serving as a control group, were included in this work. For all participants, body mass index (BMI) and skeletal muscle mass (SMM) were quantified utilizing an InBody device (InBody Co., Ltd/South Korea), and anthropometric indexes were recorded. The patients were diagnosed with muscle dystrophy by specialized neurologists. They were so diagnosed through clinical observation, case history, physical and neurological exams, as well as laboratory tests (where available). Only patients with confirmed dystrophy and stable health status at the time of sampling were selected.

Inclusion and exclusion criteria

Inclusion criteria, included adults aged ≥ 18 years with confirmed muscle dystrophy but not treated in the previous months with agents impacting muscle metabolism or endocrine biomarkers, while exclusion criteria, included inflammatory diseases, cancers, serious conditions in the liver or kidneys, other diseases in the endocrine system than muscle dystrophy, recent major surgeries, or agents impacting IGF-1/irisin levels such as systemic corticosteroids or anabolic agents. Healthy subjects should not have had neuromuscular or metabolic diseases or chronic systemic conditions, with inclusion based on age if feasible to control for variables impacting the biomarkers in question.

Blood Collection and Preparation

3ml of peripheral venous blood were drawn from every contributor utilizing sterile disposable syringe utilizing a sterile disposable syringe. For 30 minutes at room temperature, the samples were permitted to clot. Then, at 3000 rpm for five minutes, they were put in a centrifuge in order to disperse the sera. The obtained serum was transferred into a clean Eppendorf tube and stored at a low temperature in a refrigerator until laboratory analysis

Immunological study

Based on Biotin double antibody sandwich technology (Shanghai YL Biont, China), serum levels of Irisin and IGF1 were quantified utilizing enzyme-linked immunosorbent assay (ELISA).

Statistical Analysis

The analyses of the results used the software "SPSS version 26." The data was checked for normality before carrying out any tests. The analysis for the two independent groups (muscle dystrophy patients vs. control participants) was conducted using independent samples t-tests for the mean serum IGF-1 and irisin. Post hoc tests: LS-D and Duncan's test were conducted to identify intergroup differences that followed overall group comparisons for various age groups. Chi-square test and Pearson correlation test were used for distribution and correlation analysis. All tests were made to correspond to variable types and the design of the study; test statistics, p-values, and significance were standardized. Significance was set to $p \leq 0.05$, and $p \leq 0.01$ was highly significant (SPSS, 2019).

Results

The results in Table 1 show that significant decline in serum IGF-1 levels in the patient group (75.18 ± 3.16 ng/ml) compared to the HCs group (112.14 ± 8.75 ng/ml) with p-value 0.0001), highlighting its potential role in illness-related muscle and bone alterations.

Table 1. Comparison between the variation patients and control groups in IGF-1

Group	Means $\pm$ SE of IGF-1 (ng/ml)
Patients	75.18 ± 3.16
Control	112.14 ± 8.75
T-test	15.927 **
P-value	0.0001

** ($P \leq 0.01$).

The results in Table 2 demonstrate that significant decline in serum irisin levels in the patient group (5.19 ± 0.22 ng/ml) compared to the HCs group (9.92 ± 0.78 ng/ml) with p-value (0.0001), indicating a potential role of this myokine in illness-related muscle and metabolic alterations.

Table 2. Comparison between the variation patients and control groups in Irisin

Group	Means $\pm$ SE of Irisin (ng/ml)
Patients	5.19 ± 0.22
HCS	9.92 ± 0.78
T-test	1.357 **

** ($P \leq 0.01$).

The results in Table 3 indicate that the correlation between IGF-1 and irisin levels was weak and not statistically significant in both groups. In patients, the correlation coefficient was negative ($r = -0.11$, $P = 0.397$), while in the HCs group it was slightly positive ($P = 0.499$; $r = 0.12$), implying that the regulation of these biomarkers may occur independently in the studied population.

Table 3. Estimate of the Correlation coefficient among varied parameters in patients and HCs groups

Parameters		Patients		HCs	
		r.	P-value	r.	P-value
IGF1	Irisin	-0.11 NS	0.397	0.12 NS	0.499

* ($P \leq 0.05$), NS: Non-significant.

Discussion

The results of the current study were in line, indicating a reduced level of serum IGF-1 in patients in comparison to healthy individuals, thereby confirming the hypothesis that the pathophysiology in muscle dystrophy is a result of the deficiency of IGF-1. Being an anabolic factor, the reduced level of IGF-1 in the circulation may be due to an impairment in the signaling pathway in the target tissues. Differences in the level of regulation of IGF-1 in various diseases have been noted. Higher levels of IGF-1 were identified in patients suffering from autism spectrum disorder (Robinson-Agramonte *et al.*, 2022), whereas in older individuals, the prevalence of sarcopenia decreased with an increase in the levels of IGF-1 in men, but not in women, in the context of age-related sarcopenia (Jiang *et al.*, 2022). However, these results, together with the U-shaped relationship between IGF-1 concentration and mortality rates (Rahmani *et al.*, 2022), suggest that the role of IGF-1 is condition- and dose-dependent, contrary to the general idea that IGF-1 has either negative or positive roles. Exercise studies also prove the biological significance of IGF-1 modulation, in which resistance or endurance training, as well as exogenous administration of IGF-1, suppressed muscle atrophy through the activation of the IGF-1/PI3K/Akt pathway (de Alcantara Borba *et al.*, 2020; Feng *et al.*, 2022; Sakai *et al.*, 2021), which could be therapeutically useful in individuals with low levels of IGF-1. In addition, irisin expression was found to be lower in patients and correlated with muscle dysfunction, thus providing support for the hypothesis that



muscle dysfunction is a result of myokine irregularities. Lower irisin expression was also found to result in lower muscle strength and quality in Charcot-Marie-Tooth disease, and sarcopenia, malnutrition, and high rates of mortality in liver cirrhosis patients (Colaianne *et al.*, 2022). In contrast, higher irisin expression was found in certain disease conditions, likely owing to compensatory reactions, as in the case of Becker muscular dystrophy and metabolic liver disease (Baek *et al.*, 2022) and Sheptulina *et al.* (2024). The endocrine and nutritional regulation on irisin expression is also supported by a recent observation that vitamin D administration results in the stimulation of FNDC5 expression and subsequent irisin expression (Sanesi *et al.*, 2023). The finding of the age-related decline of irisin, especially in persons over the age of 70 years, supports the hypothesis that the dysregulation of myokines by the aging process itself is mediated by the reduced muscle mass and activity of the affected individuals. This finding fits the metabolic dysfunction of the elderly with sarcopenia and the findings of coordinated disruption of the IGF-1 and irisin signaling cascades in metabolic diseases (Merawati *et al.*, 2023). It must be highlighted that the lack of a significant relationship between IGF-1 and irisin in the present study contradicts the hypothesis that these biomarkers are interdependently controlled but rather supports the alternative hypothesis that these are controlled by two self-contained processes. This finding fits the findings in patients with acromegaly in which no direct relationship exists between irisin levels and the levels of the GH/IGF-1 axis despite the severe disturbance of the hormonal levels (Mizera *et al.*, 2021; Sendur *et al.*, 2022), suggesting that in the disrupted endocrine states of GH or IGF-1, the regulatory control of irisin levels differs from the findings in the elderly with sarcopenia.

Conclusions

Serum IGF-1 and irisin were found to be lower in patients with muscular dystrophy than in healthy controls, associating the two markers with muscular impairment in disease. Irisin was found to be reduced in older patients, indicating signs of aging. Lack of correlation between IGF-1 and irisin indicates that they are potentially regulated in an autonomous manner. In terms of applications, both IGF-1 and irisin can act as markers indicating muscle status and exercise or training response. Designed training, particularly resistance training and endurance training, can affect these markers and promote functional improvement in affected patients. Future studies should investigate post-exercise changes, nutrition, and training responses related to predictive properties regarding performance, strength, and functional status in muscle dystrophy patients.

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