Editorial

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Metabolic Syndrome, Obesity and Irisin: State of the Art

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ABSTRACT

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Editorial

The epidemic of the twenty-first century is obesity, which is also a serious public health problem. Chronic disorders like the metabolic syndrome and its complications, cardiovascular diseases, stroke, and various types of cancer are all made more likely by being overweight [1-3]. Hence, the emphasis on adipose tissue research as the primary energy balance buffer system. Future therapeutic targets for fat cells may become apparent as a result of the function they play in metabolic disorders. Numerous peptides, in addition to «traditional» hormones, are produced by non-endocrine cells and have effects on neighboring cells or on the cells that make them (autocrine effects) [4]. These peptides' hormone-like actions necessitate contact with particular cell surface receptors [5]. Initially assumed to as «silent» or monospecialized tissues, they now appear to be regulated by endocrine, paracrine, and autocrine signals [6]. Adipose tissue, the stomach, the skin, and the muscle are all active endocrine organs that emit a variety of hormones or hormone-like compounds that are essential for controlling metabolic processes and preserving cellular energy balance. Irisin is a recently discovered polypeptide that is the chemical in concern.

It is mostly produced by muscle and fat tissue and is also known as the exercise chemokine [7]. It has also been discovered that irisin is produced by a number of tissues, including the liver, lung, tongue, ovaries, testicles, and brain cells [8]. Adipocytes were assumed to be triglyceride storage cells, and fat was formerly believed to play a passive role in the onset of obesity [9]. Adipose tissue is widely distributed throughout the body, taking up the majority of the subcutaneous space, penetrating organs and tissues, and providing mechanical and thermal protection [10]. Adipokines including leptin, adiponectin, resistin, nesfatin, and irisin, to mention a few, are also secreted by adipose tissue. Depending on the area and type of fat, these substances might just have unforeseen consequences that change the link between the metabolism of fat and overall metabolic and physiologic activity. Irisin is vital for improving metabolic health, reducing obesity, and lipogenesis. Visceral fat normally secretes it. This happens because it regulates the process by which white fat turns into brown fat [11]. Visceral obesity and subcutaneous obesity are the two main types of obesity that are identified from a clinical perspective, namely in terms of disease development [12]. Regardless of body mass index, visceral adiposity, especially ectopic fat, increases the risk of early death, but only in association with a large belly girth [13]. Subcutaneous adiposity, on the other hand, seems to be benign in terms of the frequency and seriousness of problems [14]. Metabolic advantages may result from visceral fat excision or subcutaneous fat transplantation [15].

The association between the location of body fat storage and metabolic problems has long been supported by animal models. For instance, overexpression of the enzyme 11-hydroxysteroid dehydrogenase type 1 (11-HSD-1) in the adipose tissue of transgenic mice results in metabolically unhealthy obesity with insulin resistance (IR) and changes in glucose and lipid metabolism,

whereas those overexpressing adiponectin or mitoNEET, a crucial regulator of mitochondrial function and lipid homeostasis, develop subcutaneous obesity and maintain metabolic health [16,17]. Initially, this discrepancy was attributed to differing levels of systemic inflammation linked to elevated TNF release. Similar to this, irisin has been linked to decreased pro-inflammatory cytokine production and increased anti-inflammatory cytokine secretion in adipose tissue. Exercise has long been a successful method for treating metabolic syndrome and its symptoms as well as for preventing and managing cardiometabolic risk [18]. Moreover, there are genetic variations and a variety of non-modifiable (such as gender and age) or modifiable (such as cardiorespiratory fitness, training style, and duration) factors that affect how people respond to exercise. One such component that aids in the body's reaction to exercise is skeletal muscle myokines. Since it is in charge of regulating UCP1 during the growth of beige cells, irisin stands out as the adipomyokine with the highest potential to enhance cardiometabolic health. It has been established in mice that exercise-induced irisin induces white fat to go through thermogenesis similar to that of brown fat, as indicated in the sections above [19]. As a result, numerous investigations have looked for indicators of browning development or the expression of FNDC5 in muscles, but so far with conflicting and inconclusive results [20]. For example, researchers found that muscle strength is associated with higher levels of circulating irisin and that muscle mass predicts irisin levels [21]. Therefore, one may infer that training might cause acute irisin release.

While some reported this finding, other researchers did not find such a connection [22]. Similar to this, some studies found a positive relationship between irisin release, BMI, and other anthropometric factors, while others found a negative relationship between these variables [20,23]. It has been proposed that a positive connection may act as a compensatory mechanism to make a low irisin level foretell muscle mass loss and the onset of sarcopenia [24]. Adults with excess weight have had the effect of exercise type on irisin investigated before and after an 8-week program of resistance or aerobic exercise. Anthropometric measures, maximum oxygen uptake, and muscle strength all showed significant improvements. A significant rise in circulating irisin was also observed following 8 weeks of resistance training but not after aerobic exercise, indicating that resistance training may be a useful form of exercise for people [25]. This discovery is in line with prior findings that indicated acute exercise or high-intensity exercise increased irisin levels more than low-intensity exercise did [26]. Other research found no connection between post-exercise levels of circulating irisin and participant age, training intensity, or kind of exercise; nevertheless, fitness level had a favorable effect. However, it appears that the effort may be advantageous if one maintains a healthy quantity of exercise and a normal BMI independent of the baseline level of circulating irisin [26].

Exercise does not seem to have a long-term impact on irisin levels [27]. Studies measuring irisin levels at various intervals before and after exercise, for instance, suggest that these levels rise briefly after exercise and then gradually decline [11,22]. This association between IR and cardiovascular risk suggests either an increase in the production of adipose/muscle tissue or a compensatory surge in irisin to overcome its resistance [28]. Therefore, those who have high levels of circulating irisin are more prone to get IR soon after putting on weight while on a diet [29]. However, in obese people who develop IR, calorie restriction may be harmful, which could result in T2DM. As a result, there is no discernible difference between non-diabetics and newly diagnosed ones of the same age and sex; serum irisin is elevated in non-diabetic obese people and decreased in diabetics [24].

A few of the factors that could explain these variations and discrepancies between results include physiological and experimental methods, animal versus human studies, sex, age, health status, BMI, anthropometric parameters, body composition, whether the subjects were previously trained or sedentary, the type and duration of training, the timing of irisin determination, the cardiometabolic risks, etc. Irisin has also been detected in samples that were either fresh or frozen using a variety of tests using a variety of techniques [30]. In order to identify irisin's mechanisms of action and demonstrate its significance in metabolic disorders, more research, comprising control trials, is indeed required. Considering these drawbacks, irisin continues to show interest in the prevention and treatment of obesity and metabolic syndrome, although more research is needed to support this claim.

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