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Irisin and kidney disease; new concepts

Parivash Nouri[1], Hamid Nasri[2*]

Chronic renal failure is known by reduced glomerular filtration rate and/or albuminuria, is currently thought as a main public health problem due to its increasing prevalence globally and its independent relationship with cardiovascular mortality and generally by its all-cause mortality and also by its progression to end-stage kidney failure. Individuals with chronic renal failure have distorted energy expenditure (1-3). Disturbed energy expenditure in chronic renal failure leads to protein-energy waste and resultant greater mortality (2). The exact mechanisms responsible for deregulation of energy expenditure in chronic renal failure patients are not fully understood. Renal insufficiency results in various perturbations in cellular metabolism, containing impaired glucose metabolism, metabolic acidosis, micro-inflammation and altered cellular protein turnover (2-4). Numerous evidences demonstrate that skeletal muscle can express and release substances like as cytokines or other various peptides able of modulating metabolic processes. Hence, skeletal muscle can act like a paracrine, autocrine, or even an endocrine organ, acting in a hormone-like model and therefore exerting specific endocrine influences on other systems (3-5). Consequently, these cytokines are categorized as 'myokines,' which work as hormones both locally within the muscle and by targeting distant systems (6-8) Irisin has been recognized newly as an exercise-induced hormone secreted through skeletal muscle. By cleavage and shedding of the membrane fraction of fibronectin type III domain containing 5 (FNDC5), this myokine can release in the circulation through the response to activation of peroxisome proliferator-activated receptor-γ coactivator-1α (PGC-1α). This novel peptidic-myokine acts on cells of white adipose tissue, to promote the acquisition of a brown adipocyte phenotype prone to energy expenditure (7-9).

Irisin rises the total energy expenditure, reduces body weight, mitigates diet-induced insulin resistance and extends life expectancy. Therefore irisin can reduce obesity and insulin resistance. This mechanism explains the importance of exercise with regard to the over-expression of myokines. Additionally, it has been recently detected that irisin is not only a myokine but also acts as an adipokine too (6-8). It has also been found that circulating irisin concentration are lower in type 2 diabetes mellitus compared to nondiabetic individuals (6,10). Importantly recent studies have shown the lower level of irisn in patients with chronic renal failure (7,10). Likewise it was detected that, irisin protect diet and ageing-induced obesity and diabetes in experimental studies (6,9). Lower concentrations of irisin are independently associated with lower HDL-C levels. These findings propose that irisin may be involved in the regulation of HDL-C levels (7). In fact, exercise is found to decrease inflammation and oxidative stress and improve muscular strength and the quality of life in chronic renal failure patients. However, further investigations are necessary to determine whether irisin have influences similar to those induced by exercise in patients with chronic renal failure (7-10).

Implication for health policy/practice/research/medical education

Disturbed energy expenditure in chronic renal failure leads to protein-energy waste and resultant greater mortality. The exact mechanisms responsible for deregulation of energy expenditure in chronic renal failure patients are not fully understood. Recent studies have shown the lower level of irisn in patients with chronic renal failure. Whether irisin have influences similar to those induced by exercise in patients with chronic renal failure, needs further investigation.

Keywords: Chronic renal failure, Irisin, End-stage kidney failure, Myokines

Authors' contribution

Both authors contributed equally to the writing of this paper.

Conflicts of interest

The authors declared no competing interests.

Ethical considerations

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[1]Nickan Research Institute, Isfahan, Iran. [2]Department of Nephrology, Isfahan University of Medical Sciences, Isfahan, Iran.
***Corresponding author:** Prof. Hamid Nasri, Email: hamidnasri@med.mui.ac.ir

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