Kidney failure (KF) is characterized by the loss of the ability of the kidneys to filter metabolic waste, salts and blood fluids. The main causes of this disease are systemic arterial hypertension and diabetes mellitus [1,2]. However, other causes are described, such as the use of nephrotoxic drugs, prolonged dehydration and infection [1]. IR can be classified as acute (fast and reversible) and chronic (gradual, slow and without reversibility) [1,2]. KF represents a serious public health problem in Brazil. It is estimated that 134,000 chronic kidney patients require replacement therapy (hemodialysis) [3]. These numbers grow every year, in 2018 alone 42,546 new people were diagnosed who needed this therapy [3].

Blood creatinine and urea levels are the main biomarkers used to determine the diagnosis of chronic KF [4]. Creatinine is the metabolic product resulting from the degradation of muscle phosphocreatine, used for the production of adenosine triphosphate (ATP). As it cannot be reused by the body, creatinine is easily filtered by the kidneys and is not reabsorbed at any stage of urine formation. Therefore, it becomes the main marker of renal function [5].

Through creatinine, it is possible to determine the glomerular filtration rate (GFR), which consists of the capacity for the clearance of metabolic residues (urea and creatinine) and salts (sodium, potassium, chlorine) by nephrons [1]. To do this, it is necessary to compare serum and urinary creatinine levels after 24 hours [4]. GFR can also be estimated using Cockcroft-Gault (CG) formulas [6] and the Modification of Diet in Renal Disease (MDRD) [7]. This measure is used to stratify the stage of chronic kidney disease, considering as normal a GFR ≥ 90 mL/min and those with hemodialysis needing those with GFR <15 mL/min [1,4].
Urea is a metabolic residue synthesized by the liver failure, resulting from the breakdown of proteins ingested through the diet [8]. In the face of liver, kidney failure or excessive protein consumption, serum urea levels may be increased causing symptoms such as nausea, vomiting, shortness of breath, drowsiness and headache. Urea has low molecular weight, facilitating the glomerular filtration process, with 50% being reabsorbed in the proximal contorted tubule. At low flows as in dehydration situations, about 70% of urea is reabsorbed by the kidneys [5].

In addition to renal function, serum urea levels can determine the individual’s protein status in a short period of time [9]. Therefore, it is necessary to draw attention to the doses of food supplementation in protein compounds such as Whey Protein (milk protein). These supplements are used to gain muscle mass and strength, in addition to helping with nutrition and wound healing. However, when the individual’s renal function is not properly dosed or analyzed, it can cause severe complications [10].

Muscle mass and the type of physical activity performed also contribute to the serum levels of creatinine and urea. A study carried out in rural workers who performed activities in the field with approximately 8h daily demonstrated that the time of execution of the physical activity influences the doses of creatinine and plasma urea, being higher in the individuals who collected the samples at the end of the day, after work [11]. Another study carried out in trained individuals showed an increase in creatinine and urea levels after intense exercise, this is because during its execution there was an increase in creatine metabolism and a reduction in urinary excretion, also increasing serum urea levels [12].

In this edition of the Revista Brasileira de Fisiologia do Exercício, Vieira et al. [13], published a clinical trial with male Rattus Norvegicus Wistar Albino, supplemented with whey protein and compared to the control. The rats were allocated into 6 groups in which: two groups were supplemented with 2 g kg d⁻¹ (with and without exercise) two with 4 g kg d⁻¹ (with and without exercise), one group was the control training and the another only control. The rats were submitted to neuromuscular exercise for 12 weeks with a frequency of 3x a week. The load determination was done through the maximum loaded weight test (PMC) and the prescription was 4 climbs on the ladder per training session with increasing intensity of 50%, 75%, 90% and 100% of the PMC.

The rats that performed exercises associated with protein supplementation had higher excretion of creatinine in the urine when compared to the control groups. When performing a serum analysis, no statistical difference was identified between the groups [13]. During intense exercise, there is an increase in systemic blood pressure, resulting in an increase in glomerular pressure [12]. These physiological adaptations increase blood filtration processes, thus eliminating a greater concentration of metabolic waste from the blood.

It is possible to observe that the groups that did physical exercise associated with protein supplementation also presented higher levels of urea concentration.
With the increase in hydrostatic pressure in the renal capillary due to intense physical effort, the flow of the tubular fluid becomes faster, thus reducing the reabsorption of urea in the contorted proximal, distal tubule and collecting duct. Thus, it is justified to increase the concentrations of this residue in the urine [14].

Studies like this are necessary to identify the protein supplementation thresholds, in addition to guiding professionals who work directly with physical exercise regarding the correct prescription. Too many doses of supplementation and exercise are harmful to the body, requiring more time to eliminate metabolic excesses and organic regeneration [5,14]. In addition, it is necessary to pool the information that constitutes individuals. Larger doses of supplementation require adaptation of the type and intensity of exercise and the opposite relationship also holds. That said, the study by Vieira et al. [13] draws attention to this theme and encourages the production of new studies with similar proposals.

References