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Creatine supplementation and renal function - myths and evidence - a narrative review

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Abstract

Background: Creatine is one of the most commonly used ergogenic supplements. However, many myths and misconceptions about creatine supplementation regarding renal function are still popular even among medical professionals.

Aim: The aim of this study was to summarise the current knowledge regarding the relationship between creatine supplementation and renal function, as well as to evaluate the most common myths associated with its use and to discuss creatine supplementation in patients with chronic kidney disease. Particular attention was paid to the five most common myths: that an increase in creatinine concentration always indicates kidney damage, that creatine supplementation damages the kidneys, that it causes water retention, increases the risk of kidney stones, and that it is dangerous with long-term use.

Methods and materials: A review of the literature in Polish and English was conducted using the PubMed/MEDLINE and Google Scholar databases for the period from 1998 to March 2026. A total of 189 publications were analysed, including meta-analyses, epidemiological studies and experimental studies.

Results: Current scientific evidence suggests that creatine monohydrate supplementation does not cause kidney damage in healthy individuals when administered doses remain within the recommended range. An increase in serum creatinine levels in individuals supplementing creatine does not necessarily indicate impaired kidney function, as it may result from increased creatine intake and its metabolic processes, rather than actual organ damage. Assessment of kidney function in individuals using creatine should not be based solely on serum creatinine levels; other indicators such as cystatin C, albuminuria, proteinuria or mGFR should also be considered. It has not been confirmed that creatine supplementation increases the risk of kidney stones or leads to permanent disturbances in water balance. Available studies indicate that long-term use of creatine is well tolerated and safe; however, high-quality studies covering diverse populations and long follow-up periods are still needed. Despite promising clinical indications, further studies are needed to assess the safety and efficacy of such treatment.

Conclusions: Current scientific evidence does not support the notion that creatine supplementation, when used at recommended doses, causes renal dysfunction in healthy individuals. However, careful interpretation of renal markers and further studies in clinical populations are necessary.

Keywords: creatine, creatinine, kidneys, renal function, chronic kidney disease, supplementation, safety

1. Introduction

Creatine is an endogenously synthesized compound, primarily produced in the kidneys and liver through enzymatic reactions involving the amino acids arginine, glycine, and methionine [1]. In addition to endogenous synthesis, creatine is obtained exogenously through dietary

sources such as red meat, dairy products, and seafood. In recent years, creatine supplementation has gained substantial popularity, particularly among younger populations and individuals engaged in resistance training. Beside athletes, creatine is recommended to patients suffering from myasthenia, other muscle diseases and neurological issues [2,3]. Creatine's wide use has been mostly driven by observed enhancement of athletic performance and alleged stimulation of muscle growth. While creatine supplementation alone does not directly induce muscle hypertrophy, it may facilitate muscle growth indirectly when combined with appropriate resistance training and nutritional strategies [2]. Beyond its effects on physical performance, emerging evidence suggests that creatine may exert beneficial effects on cognitive processes, including short-term memory and executive functions [4,5]. Its analogs can be used as anticancer agents during chemotherapy and there is evidence that suggests that they shield tissues from ischemic damage so may aid during organ transplants [6]. As of now creatine is the most commonly used ergogenic supplement.

Kidneys play a crucial role in keeping the state of homeostasis inside the body by filtering out waste, regulating electrolyte balance, filtering blood, as well as having endocrinological or hematopoietic functions. Creatinine, a non-enzymatic breakdown product of creatine and phosphocreatine metabolism, is produced at a relatively constant rate, largely proportional to muscle mass. Unlike creatine, creatinine is not reutilized by cells and is instead released into the bloodstream and excreted in urine [1]. Due to its relatively stable production and renal clearance, serum creatinine is widely used as a clinical biomarker for assessing kidney function [7,8].

Concerns regarding the potential nephrotoxicity of creatine supplementation originated largely from early case reports, notably the study by Pritchard and Kalra (1998) [9]. Since then, numerous studies exploring this hypothesis have been conducted. Most properly conducted studies and clinical trials seem to point to the conclusion that at right dosage creatine monohydrate does not influence renal dysfunction. However, a number of myths and misconceptions about creatine supplementation regarding renal function still remain. The goal of this review is to assess empirical evidence and refute current misconceptions about the link between creatine supplementation and renal dysfunction. We aimed to address most common myths, which include: 1. Increased serum creatinine always indicates kidney damage; 2. Creatine supplementation damages kidneys; 3. Creatine intake leads to water retention; 4. Creatine increases the risk of kidney stones; 5. Creatine is unsafe for long-term use. In addition, we discussed creatine supplementation in patients with chronic kidney disease, as well as practical implications of creatine intake.

2. Creatine metabolism and renal physiology

Creatine, also known as α -methylguanidine-acetic acid, is an endogenously produced nitrogen-containing compound. It is a crucial agent in supporting cellular energy metabolism by helping to resynthesize adenosine triphosphate (ATP) during prolonged exercise via raising intramuscular phosphocreatine concentrations. (1) Creatine is synthesized in a chain of reactions: firstly, arginine and glycine undergo a synthesis reaction catalyzed by AGAT enzyme (arginine glycine amidinotransferase), resulting in guanidinoacetate (GAA) production. GAA is then methylated by the enzyme guanidinoacetate N-methyltransferase

(GAMT) with S-adenosyl methionine (S-AMe) to form creatine. (10) The AGAT enzyme can be found in kidneys, pancreas, liver and certain brain regions; most GAA is produced in kidneys and converted to creatine in the liver. (11,12) About 95% of creatine in the human body is stored in muscle, $\frac{2}{3}$ of which as phosphocreatine (PCr) and the rest as free creatine (Cr). (1) Approximately 1-2% of creatine stored in muscle undergoes a spontaneous (non-enzymatical) and irreversible degradation to creatinine (Crn) (1,7). Conversion of creatine to creatinine is greater in individuals with larger muscle mass or higher physical activity levels. (13) Creatinine is secreted in the urine and influences estimated glomerular filtration rate (eGFR), a commonly-used determinant of renal function.

3. Myths regarding creatine and kidney damage

Kidneys play a vital role in maintaining the body's homeostasis. The question whether creatine intake affects renal function remains one of the most common concerns regarding creatine supplementation. A number of myths and misconceptions about the safety of creatine in relation to renal health is still present even among medical professionals. The most common misconception is that creatine supplementation results in kidney damage. The main source of this concern may be some early reports (9) and animal studies (14), as well as the misunderstanding of the correlation between serum creatinine levels, eGFR and mGFR (measured glomerular filtration rate).

Myth 1 – Increased serum creatinine always indicates kidney damage

Due to spontaneous creatine to creatinine conversion, the elevation of serum creatinine level is a predictable physiological response in individuals supplementing creatine. Despite increased serum creatinine being commonly associated with kidney damage, in individuals supplementing creatine relying solely on serum Crn to assess renal function can be misleading. (15) A central point in evaluation of kidney function is considered to be glomerular filtration rate (GFR), which is the amount of fluid that filters into Bowman's capsule per unit time. GFR is impossible to be directly measured and the gold-standard method to assess it remains mGFR. mGFR, i.e., measured glomerular filtration rate, is measured by continuous intravenous infusion of exogenous filtration biomarkers that are not produced by the organism and are freely filtered but neither reabsorbed nor secreted by the renal tube; such biomarkers are inulin, ^{99m}Tc -DTPA, ^{51}Cr -EDTA etc. (16) Due to its cost and invasive administration of biomarkers, the mGFR remains impractical and is generally limited to specialized facilities. In common practice, GFR is usually estimated from serum levels of endogenous biomarkers (i.e., eGFR, estimated glomerular filtration rate). (16) Biomarkers used in eGFR calculation are creatinine, cystatin C, and recently two new biomarkers: beta-trace protein (BTP) and beta-2-microglobulin (B2M) (17) Other markers indicating renal damage are proteinuria, albuminuria and increased serum urea levels. Increased serum creatinine levels have been reported in patients supplementing creatine or individuals with larger muscle mass, high-protein or meat-rich diet and lower fluid intake, without other indicators of impaired kidney function. (18) On the contrary, serum cystatin C level is not dependant on these factors, therefore, it reflects GFR - and therefore renal function- more accurately and consistently than serum creatinine level. (19) In individuals supplementing creatine elevated serum Crn levels are present; however, alternative biomarkers, such as cystatin C, remain within the normal range. (20) In summary, due to the limitations of estimated glomerular filtration rate, increased serum creatinine does not always indicate kidney damage.

Myth 2 – Creatine supplementation damages kidneys

The alleged risk of kidney damage in individuals supplementing creatine remains one of the main concerns regarding creatine intake. Origins of this misconception may be a result of a series of early studies, in which the conclusions correlating creatine to renal damage may have been drawn hastily. Renal failure in patients supplementing creatine was observed in studies conducted by Taner et al. (21), as well as Thorsteinsdottir et al. (22), however, in the former study the patient's condition was not evaluated prior to the study, whereas in the latter the patient used other types of supplements in addition to creatine, which may have contributed to kidney damage. Over the years, a number of studies, reviews and meta-analyses have reported creatine supplementation not to be harmful to renal function. In a systematic review and meta-analysis conducted by de Souza e Silva et al., it was concluded that creatine supplementation did not lead to renal function damage by analyzing 15 eligible studies including long-term ones (23). In a review of creatine supplementation studies conducted by Persky and Rawson, no increase of serum creatinine was observed in 12 studies, an increase remaining within the normal range was present in 8 studies and an increase above normal levels was observed only in 2 studies (24); it should be noted that elevated creatinine levels do not translate directly into renal damage. In a review and meta-analysis by Naeini et al., studies analyzed regarding GFR data have shown no significant changes in either eGFR or mGFR in individuals supplementing creatine. (15) Apart from increased serum creatinine or glomerular filtration rate, another possible indicator of kidney failure may be an elevated serum urea level; however, in a meta-analysis by Carvahlo et al. no changes in urea levels were observed after creatine supplementation. (25) Additionally, creatine intake did not impair kidney function in individuals with type 2 diabetes - a population prone to kidney disease - in a study conducted by Gualano et al. (26).

Based on above studies, it can be concluded that creatine supplementation is most likely safe and does not result in kidney damage.

Myth 3 - Creatine intake leads to water retention

Creatine is an osmotically active substance. Creatine is transported into muscle from plasma by a sodium-dependent creatine co-transporter (1). Since the transport involves sodium, water will also be taken up into muscle to help maintain intracellular osmolality.

The purported myth of creatine supplementation increasing total body water (TBW) is likely due to early research which showed that creatine supplementation at 20 g/day for six days was associated with water retention (27). It does appear that the most common adverse effect of creatine supplementation is water retention in the early stages (first several days) (28).

A number of exercise training studies, incorporating creatine supplementation, have shown no increases in total body water (TBW). For example, resistance-trained males who received approximately 20 g/day creatine for 7 days-and followed by 28 days 5 g creatine/day experienced no significant change in ICW (intracellular water), ECW (extracellular water), or TBW (29). Moreover, males and females ingesting creatine in a dose of 0.03 g/kg/day for six weeks experienced no significant increase in TBW (30). In contrast, when assessing TBW, ICW, and ECW content before and after 28 days of creatine supplementation in healthy males and females (n = 32), Powers et al. (31) showed that creatine supplementation was effective at

increasing muscle creatine content which was associated with an increase in body mass and TBW but did not alter ICW or ECW volumes.

In conclusion, creatine supplementation increases water retention over the short term but it does not alter total body water relative to muscle mass over longer periods of time.

Myth 4 - Creatine increases the risk of kidney stones

Kidneys play a vital role in removing creatinine from the plasma and excreting it in the urine (32). A possible increase in creatinine levels associated with CMS (creatine monohydrate supplementation) and elevated serum creatinine levels in people with kidney stones have led to speculation that creatine may cause kidney stones.

Several studies have shown that CMS in healthy individuals (25) or athletes (33) did not alter urinary creatinine levels. A meta-analysis by Silva et al. showed that an increase in serum creatinine levels caused by CMS does not indicate kidney damage (23).

In a case report published by Akbari et al., CMS was not associated with the formation of new kidney stones. To the best of the authors' knowledge, no previous study has investigated the effect of CMS on kidney stones in humans. They demonstrated that 5 g of CMS daily for two months was not associated with the recurrence of kidney stones in an athlete with a history of the condition. They suggested that their findings should be confirmed by prospective studies involving a larger number of participants under well-controlled conditions. Although individuals using creatine with a history of kidney stones are advised to monitor their health under specialist supervision, there is as yet no evidence that creatine can cause kidney stones. (34)

Myth 5 - Creatine is unsafe for long-term use

Creatine is one of the most popular supplements taken by athletes. Research shows that creatine supplementation improves exercise performance, leads to greater training adaptation, speeds up post-exercise recovery and helps prevent training-related injuries. Furthermore, a range of clinical applications for creatine supplementation has been investigated in neurodegenerative diseases (e.g. muscular dystrophy, Parkinson's disease, Huntington's disease), diabetes, osteoarthritis and ageing. Moreover, researchers have identified a number of potentially beneficial clinical applications of creatine supplementation. Studies have shown that short- and long-term supplementation (up to 30 g per day for 5 years) is safe and well tolerated by healthy individuals and across many patient populations, from infants to the elderly. It has been proven that ensuring a consistent, low intake of creatine (e.g. 3 g per day) throughout life may yield significant health benefits. (2,3)

Lobo et al. conducted a double-blind, randomised trial from November 2011 to November 2013 involving over 50 postmenopausal women with osteopenia. The aim was to demonstrate

the efficacy and safety of long-term creatine supplementation in this population. Bone health parameters, body composition and muscle function were assessed at the start of the study and after one year of intervention. It was shown that bone mineral density did not differ within or between the groups. No significant changes were observed in body weight, BMI, absolute and relative body fat, or lean body mass. No effect was demonstrated on bone health parameters, lean body mass or muscle function in older women. It was concluded that one year of low-dose creatine supplementation (1 g/day) did not cause side effects and was safe for this population. (35)

The presented studies have limitations, including small sample sizes, heterogeneous study designs, inconsistent methodologies, and a lack of dose-response studies. The lack of conclusions regarding its long-term efficacy, impact on diverse populations, optimal dosing regimens and potential benefits necessitates robust research. Further large-scale studies are required to fully understand and appreciate the efficacy of creatine supplementation over extended periods of time, however, at this moment no studies have indicated that creatine may be unsafe for long-term use.

4. Creatine supplementation in patients with CKD

Chronic Kidney Disease (CKD) is a condition marked by ongoing damage to the kidneys, leading to a gradual, permanent decline in their ability to function properly. (36) Changes in body composition in patients with CKD, such as a reduction in fat-free mass, are driven by multiple factors. This loss may be associated with increased inflammation and/or decreased physical function and capacity, as well as lower energy and protein intake. A decline in fat-free mass is an important predictor of mortality in this population, while the high prevalence of malnutrition contributes to greater morbidity, longer hospital stays, and increased risk of death. (37)

A potential risk of low-protein diets in non-dialysis CKD and high-protein, plant-focused diets in dialysis-dependent CKD is creatine deficiency. Current CKD dietary guidelines and assessments of protein-energy malnutrition do not address creatine intake or the risk of deficiency. (38)

Hemodialysis has an adverse long-term effect on fat-free mass (FFM) and muscle strength. There are studies that focus on creatine supplementation in patients with dialysis-dependent CKD. This is particularly important in these patients because 1. their ability to synthesize creatine is severely reduced due to nearly complete loss of kidney function and the associated lack of the initial enzymatic step converting arginine and glycine into creatine (38,39); 2. creatine is lost during dialysis sessions (40,41); and 3. dietary intake of creatine is often insufficient due to recommendations for primarily plant-based diets (42). Creatine deficiency has significant consequences, contributing to reduced HRQoL (Health-Related Quality of Life) and higher mortality in hemodialysis patients. Recent research shows that higher plasma creatine is linked to better muscle mass, protein status, and lower fatigue, suggesting a potential benefit of creatine supplementation in this population. (41)

The study was conducted to evaluate the impact of creatine supplementation on body composition as well as the Malnutrition Inflammation Score (MIS) in patients with CKD undergoing hemodialysis. (43) The results showed that prolonged creatine supplementation

may be associated with increase in fat-free mass (FFM) and skeletal muscle mass index (SMMI), even in the absence of exercise. This effect could be linked to increases in intracellular water content. Therefore, further research is warranted to examine the influence of creatine supplementation on hydration status, as well as to assess its effects when combined with exercise.

Another study (44) investigated the potential of intradialytic creatine supplementation in improving outcomes. Most creatine studies use oral supplementation, but in dialysis-dependent CKD patients it is impractical as it requires dissolving the creatine in large volumes of water, which may negatively affect the fluid balance. Additionally, chronic causes of creatine deficiency in these patients require long-term supplementation. Since many patients rely on dialysis for years, oral supplementation demands high compliance and does not prevent creatine losses to the dialysis fluid. In contrast to oral supplementation, intradialytic supplementation offers the possibility to supplement creatine in a controlled manner, while preventing unopposed losses of creatine to the dialysis fluid, volume overload due to the necessary ingestion of large volumes of water, and potential problems with compliance. The study (44) outlines the rationale of intradialytic creatine supplementation. Such supplementation may help maintain creatine balance, improve muscle mass, strength, and endurance, reduce fatigue and cognitive decline, support immune and vascular health, protect red blood cells, and potentially lower anemia and erythropoietin needs, ultimately enhancing quality of life and reducing complications in this population.

Despite these promising findings, the safety of creatine supplementation in patients with CKD has not yet been sufficiently established. Although creatine deficiency is biologically plausible, current evidence is limited and largely hypothetical. Further research is needed to confirm its safety and potential benefits before it can be recommended. Moreover creatine supplementation may affect the assessment of renal function by increasing serum creatinine levels, which can be misinterpreted as a decline in kidney function. (45,46) However, alternative biomarkers, such as cystatin C, often remain unchanged. (20) In addition the increase in serum creatinine with creatine supplementation may depend on its form: creatine ethyl ester, due to its higher solubility and degradation to creatinine in the gastrointestinal tract, may raise serum creatinine levels more than creatine monohydrate (47). Therefore, clinicians should consider this influence and, when necessary, rely on additional markers of renal function.

5. Practical implications

Creatine is among the most commonly used ergogenic aids in athletic populations. Supplementation protocols typically involve an initial loading phase of 0.3 g/kg/day for 5–7 days, followed by a maintenance dose of 0.03 g/kg/day, which effectively increases intramuscular phosphocreatine (PCr) levels and enhances performance during high-intensity exercise. (28,48) Notably, continuous daily supplementation without a loading phase has also been shown to elevate muscle PCr, supporting improved training adaptations and the physiological processes underlying anaerobic energy production. To date, creatine supplementation at doses up to 5 g per day over extended periods, typically six months or longer, has not been linked to any negative effects on renal function, supporting its safety in healthy individuals. (2) Although chronic creatine supplementation appears safe, strategically pausing supplementation at certain times may enhance its effectiveness. Periodized use during

high-intensity training or key competition phases allows the body to reset and may optimize performance and recovery, as suggested by recent studies. (49,50)

Creatine is primarily stored in skeletal muscle and plays a central role in cellular ATP production, supporting energy metabolism. Evidence suggests that creatine monohydrate (CrM) supplementation, even without exercise, can significantly increase total muscle creatine stores and enhance physical performance. For instance, Harris et al. (51) and Hultman et al. (27) demonstrated that a 4.5–10-day creatine-loading protocol (~20 g/day) increases total muscle creatine (phosphocreatine [PCr] and free creatine), with similar effects observed after 3 g/day for 28 days (27). Elevated intramuscular creatine may partly explain improvements in strength, anaerobic capacity, and sprint performance, even in the absence of training (52).

Forbes et al. (53), in a narrative review, concluded that CrM supplementation without exercise can improve fat-free mass, functional performance (e.g., chair rise), and reduce lower-body fatigue. Most studies used a loading phase (20 g/day for 7–10 days) or high daily doses (17–26 g/day), while those without loading generally showed no significant effects (54,35).

It has also been demonstrated that creatine supplementation has a positive effect on mental well-being. Juneja et al. conducted a literature review, concluding that creatine, traditionally used to improve physical performance, is a promising adjunct to the treatment of depression. Research indicates that creatine has a multifaceted mechanism of action, increasing brain energy metabolism, modulating neurotransmitter systems and providing neuroprotection, particularly when combined with antidepressants at a dose of 4–5 g/day for two to eight weeks. Although current research indicates significant promise, particularly among women and young people with treatment-resistant depression and those with low baseline creatine levels, creatine should be used with caution in patients with renal dysfunction and bipolar disorder. (55)

Creatine monohydrate supplementation is safe and effective for women, enhancing exercise performance, muscle strength, and recovery. The recommended dose is 3–5 g/day, with or without an initial loading phase, as consistent daily intake is equally effective. While post-exercise supplementation may support phosphocreatine replenishment, menstrual cycle phases do not appear to require adjustments in dosing. Some women may experience transient water retention, particularly during loading. Overall, personalized, consistent creatine use provides reliable benefits, though further research could refine protocols specific to women. (56)

According to the International Society of Sports Nutrition (ISSN): (2)

Creatine monohydrate is the most effective ergogenic supplement for increasing high-intensity exercise capacity and lean body mass during training. It is safe for both healthy and clinical populations, from infants to the elderly, with no evidence of harm from short- or long-term use (up to 30 g/day for 5 years).

With proper supervision, creatine monohydrate is safe for children and adolescent athletes and may serve as a safer alternative to anabolic steroids. However, supplementation is recommended only for those engaged in structured competitive training, consuming a balanced performance-oriented diet, informed about correct use, and adhering to recommended dosages.

Warnings against use in individuals under 18 are likely unnecessary, as scientific evidence supports creatine's safety in children and adolescents.

Creatine monohydrate remains the most studied and effective form for muscle uptake and performance enhancement. Adding carbohydrates, or carbohydrates and protein, may improve creatine uptake, though performance benefits are similar to creatine alone.

Rapid muscle creatine loading can be achieved with ~ 0.3 g/kg/day for 5–7 days, followed by 3–5 g/day for maintenance. Smaller daily doses (3–5 g/day) also increase stores over 3–4 weeks, though initial performance benefits are less pronounced.

Clinical populations have tolerated high doses (0.3–0.8 g/kg/day, equivalent to 21–56 g/day for a 70 kg individual) over long periods without serious adverse events.

Further research is encouraged to explore additional medical and performance benefits of creatine monohydrate and precursors such as guanidinoacetic acid.

6. Limitations

Due to this study being a review, its conclusions depend on the quality and availability of previously published research, without the possibility of controlling the variables. Furthermore, the studies analysed are characterised by heterogeneity, differing in terms of population (healthy individuals, athletes, patients with medical conditions), creatine doses and duration of supplementation. Moreover, renal function was assessed primarily based on creatinine and eGFR, which may be affected by creatine supplementation itself, constituting a significant limitation on interpretation. In many studies, factors such as diet (particularly protein intake), hydration and level of physical activity—which may influence renal parameters—were not fully controlled. The aforementioned determinants are the reason why the topic of the effect of creatine supplementation on renal function should be further investigated.

7. Conclusions

Current scientific evidence suggests that creatine monohydrate supplementation does not cause kidney damage in healthy individuals when administered doses remain within the recommended range. An increase in serum creatinine levels in individuals supplementing creatine does not necessarily indicate impaired kidney function, as it may result from increased creatine intake and its metabolic processes, rather than actual organ damage. Assessment of kidney function in individuals using creatine should not be based solely on serum creatinine levels; other indicators such as cystatin C, albuminuria, proteinuria or mGFR should also be considered. It has not been confirmed that creatine supplementation increases the risk of kidney stones or that it leads to permanent disturbances in water balance. Available studies indicate that long-term use of creatine is well tolerated and safe; however, high-quality studies covering diverse populations and long follow-up periods are still needed. In patients with chronic kidney disease, particularly those on haemodialysis, there may be a biologically plausible risk of creatine deficiency, suggesting potential benefits from supplementation;

however, current clinical evidence is insufficient to formulate definitive recommendations. In clinical populations and in individuals with pre-existing kidney disease, creatine supplementation requires caution, individual assessment and monitoring of renal parameters using appropriately selected diagnostic methods.

Disclosure

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