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## Principles of nutrition in chronic kidney disease in children - a review of the clinical trials and recommendations

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## **Abstract:**

**Introduction:** Children with chronic kidney disease (CKD) require a specialized diet to manage their condition and support healthy growth and development. A proper diet for children with CKD aims to control the intake of certain nutrients, such as protein, lipid, calcium, phosphorus, potassium, sodium, and vitamin D, while ensuring they receive adequate nutrition for growth and development.

**Aim of the study:** This article discusses the epidemiology, causes and pathomechanism of CKD in children. It also presents existing clinical trials and dietary recommendations from societies such as Kidney Disease: Improving Global Outcomes (KDIGO) 2024, Kidney Disease Outcomes Quality Initiative (KDOQI) and Pediatric Renal Nutrition Taskforce (PRNT) and identifies potential sources of selected macronutrients and micronutrients in children's diets.

**Materials and methods:** Comprehensive literature searches were performed across the main electronic databases of PubMed, Google Scholar, KDIGO2024, KDQOI and PRNT recommendations for studies published in the English language about dietary recommendations for children with CKD.

**Results:** The approach to nutrition for children with CKD is still evolving, and specific macronutrient and micronutrient ratios should be established based on the clinical condition, the child's age, and body weight. Care should be taken to ensure that the child's diet is varied, and a healthy eating style should be promoted.

**Key words:** chronic kidney disease, chronic diseases, children, macronutrients, micronutrients

## **Introduction:**

Chronic kidney disease (CKD) is a multisymptomatic disorder that can affect both adults and children. There is little data in the literature regarding the prevalence of CKD in children. According to 2007 data, the problem affects about 15-74.7 cases per million in the population for a given age [1]. According to the Kidney Disease: Improving Global Outcomes (KDIGO) 2024 guidelines, CKD is defined as abnormalities in the structure or function of the kidneys that persist for at least three months and have health consequences. The criteria apply to both adults and children over the age of 2, but CKD can also be diagnosed in a child younger than 3 months if there are severe congenital kidney and urinary tract defects. The severity of CKD is classified based on glomerular filtration rate (GFR), albuminuria and the cause of the disease. GFR classifies CKD into individual grades G1-G5, while albuminuria allows division into grades A1-A3 [Table 1]. Due to the specificity of the pediatric population and the expected long-life expectancy, KDIGO 2024 recommends increasing the cutoff to 90 ml/min per 1.73 m<sup>2</sup> for children and adolescents [2].

Risk of incidence CKD		Albuminuria categories		
		A1 <30 mg/g <3 mg/mmol	A2 30-300mg/d 3-30mg/mmol	A3 >300mg/g >30mg/mmol
G1	>90	low risk	moderately increased risk	high risk
G2	60-89	low risk	moderately increased risk	high risk
G3a	45-59	moderately increased risk	high risk	very high risk
G3b	30-44	high risk	very high risk	very high risk
G4	15-29	very high risk	very high risk	very high risk
G5	<15	very high risk	very high risk	very high risk

**Table 1.** Risk of CKD. According to KDIGO 2024 guidelines CKD is classified based on Cause, Glomerular filtration rate (GFR) category (G1–G5), and Albuminuria category (A1–A3), abbreviated as CGA. Based on KDIGO 2024 Clinical Practice Guideline for the Evaluation and Management of Chronic Kidney Disease [2].

#### **Causes and risk factors of CKD:**

The causes of CKD in the pediatric population are different than in adults. The most common cause accounting for almost 50% of CKD in children is congenital anomalies of kidney and urinary tract (CAKUT). CAKUT primarily includes disorders such as aplasia, hypo-/dysplasia of one or both kidneys, reflux nephropathy, medullary cystic disease, obstructive uropathy and vesicoureteral reflux [3]. Other much rarer causes of CKD in children include focal segmental glomerulosclerosis (FSGS), Goodpasture syndrome, IgA nephropathy, hemolytic uremic syndrome, glomerulonephritis, Wilms tumor, diabetic nephropathy, sickle cell nephropathy, oxalosis and systemic lupus erythematosus [3], [4]. However, it should be remembered that the normal number of nephrons is established around 36 weeks of fetal life, while their function is still being formed in the first years of life [2]. Consequently, target GFR values are usually established around 1-2 years of age; hence, risk factors for CKD also include prematurity, history of acute kidney injury in the neonatal period [5], or use of nephrotoxic substances in childhood [6].

#### **Mechanism of metabolic disorders and complications of CKD:**

The mechanisms underlying metabolic, lipid and electrolyte abnormalities in CKD are complex and interrelated. In CKD, progressive loss of nephron function leads to a decrease in GFR, and consequently a decrease in the kidney's ability to filter and excrete and metabolic products. Carbohydrate abnormalities in CKD take the form of hyperinsulinemia and insulin resistance and are associated with impaired renal excretion of insulin and an increase in glucagon, which acts antagonistically to insulin [7]. In CKD, there is also reduced activity of enzymes responsible for lipid metabolism, e.g., lecithin-cholesterol acyltransferase (LCAT). This manifests as dyslipidemia, which is most often associated with elevated triglyceride levels and reduced high-density lipoprotein (HDL) levels [8]. In advanced stages of CKD, chronic

inflammation develops, which will be associated with inhibition of protein synthesis and accumulation of nitrogen metabolism products [9]. The use of angiotensin-converting-enzyme inhibitors (ACEIs), angiotensin receptor blockers (ARBs) and (nonsteroidal anti-inflammatory drugs) NSAIDs and potassium-rich foods predisposes CKD patients to the development of hyperkalemia [10]. As a result of increased phosphate retention, reduced vitamin D levels and difficulties with gastrointestinal absorption, hypocalcemia is also most common in CKD [11].

The transformations described above contribute to serious complications in children with CKD such as delayed growth and maturation [12], [13], metabolic acidosis [14], renal osteodystrophy [15], cardiovascular disease [16],[17] anemia [18], and depressive disorders [19]. To avoid these complications and slow the course of the disease, it is extremely important to diagnose and treat CKD, as well as to introduce a balanced diet containing age-appropriate amounts of macronutrients and micronutrients. The main principles of protein, fat, carbohydrate, sodium, potassium, calcium, phosphorus, and vitamin D intake in children with CKD are described below.

### **Protein intake:**

Protein is essential for growth and development, so the goal is to provide enough without overloading the kidneys. Despite attempts in the past, currently, according to KDIGO 2024, it is not recommended to limit protein intake in children with CKD due to the risk of growth and maturation disorders. Protein intake targets for children with CKD G2–G5 should be at the upper limit of normal to ensure optimal growth, as children with CKD have similar energy expenditure to healthy children [2].

Two randomized controlled trials examined the effects of a low-protein diet in children with CKD. In the first one, Uauy et al. examined 24 children aged 8 months with GFR below 55ml/min/1.73m<sup>2</sup>. It was divided into two groups, the first of which received a low-protein diet for 10 months and the second - a diet with normal protein content. Protein intake in these groups was 1.4 and 2.4 g/kg per day. This study showed that the length velocity SDS from 12 to 18 months was significantly negative in the group receiving a low-protein diet, therefore special caution was recommended in the use of such a diet in children [20]. The second study included a group of 191 children aged 2-18 with chronic renal failure (CRF). Patients were further divided according to the severity of the disease and some of them were put on a diet limiting protein intake to 0.8-1.1 g/kg per day. During 3 years of using a low-protein diet, no growth disturbances were found in these children and no impact of this diet on the deterioration of renal function [21].

The exact amount of protein will depend on the stage of CKD and the child's age, size, and individual needs. A pediatric nephrologist or a registered dietitian specializing in pediatric renal nutrition should determine the child's specific protein needs. In children with CKD, emphasize high-quality protein sources that are easier on the kidneys and provide essential amino acids [22]. These include lean meats, poultry, fish, eggs, and dairy products. In some cases, plant-based protein sources may be recommended to reduce the burden on the kidneys, especially include legumes (beans, lentils, chickpeas), tofu, tempeh, and nuts/seeds [23]. However, plant-based proteins may also contain higher levels of phosphorus, so portion control and monitoring are essential [24].

### **Energy and carbohydrates intake:**

When it comes to carbohydrate intake in children with CKD, it's important to focus on providing adequate energy while managing other aspects of their diet, such as protein and phosphorus intake. Carbohydrates are a crucial source of energy, and their consumption should be monitored to prevent deficiencies or excesses. Energy demand in children with CKD should be individual and dependent on many parameters, such as chronological age, BMI, height, and physical activity [25]. The total intake of macronutrients in children with CKD should be the same as in healthy children. According to the Health Canada (federal institution of Government of Canada), children aged 1-3 years of age should require 45-65% of carbohydrates, 30-40% of fats, and 5-20% of proteins. In the group of children aged 4 to 18, the proportions are slightly different - the demand for carbohydrates does not change, fats should constitute 25-35%, and proteins 10-30%. In children under 1 year of age, carbohydrates should provide 36-56% of the energy requirement, fats 40-54%, and proteins 7-12% [25], [26].

Sources of good carbohydrates in the pediatric population, including those with CKD, can include whole grains, fruits and vegetables, legumes, dairy products, starchy vegetables, and healthy snacks. Whole grains like brown rice, whole wheat bread, whole grain pasta, quinoa, oats, and barley provide complex carbohydrates, fiber, vitamins, and minerals. Fruits and vegetables are excellent sources of carbohydrates, fiber, vitamins, and minerals. Encourage a variety of colorful fruits and vegetables to ensure a diverse nutrient intake. Milk and yogurt contain lactose, a natural sugar, which provides carbohydrates along with essential nutrients like calcium and vitamin D [27], [28]. However, in CKD, dairy intake might need to be monitored due to its phosphorus content.

According to the Pediatric Renal Nutrition Taskforce (PRNT) guidelines, if children with stage 2-5 CKD and on dialysis experience stunted growth and/or weight gain despite provision of an optimal diet, enteral tube feeding should be included. If enteral feeding is to be short-term, a nasogastric tube is the optimal solution, but due to side effects such as otitis media or sinusitis, as well as speech and swallowing disorders, it is not intended for long-term feeding [29]. According to the position of the European Society for Paediatric Gastroenterology Hepatology and Nutrition (ESPGHAN), the target method for long-term enteral feeding is gastrostomy, which can be done as percutaneous endoscopic gastrostomy (PEG), percutaneous laparoscopic-assisted gastrostomy (PLAG), percutaneous radiologically inserted gastrostomy (RIG) or open [29], [30]. Because of the increased risk of peritonitis, in children who require dialysis, it is worth remembering that, if possible, the insertion of a PEG or RIG gastrostomy should take place earlier than the implantation of a dialysis catheter [28], [31], [32], [33]. Depending on the child's clinical condition, enteral tube feeding can be used as the sole form of nutrition or as a supplementary form of nutrition after meals taken orally. The delivery of food through an enteral feeding tube can be done either during the day or exclusively at night. The latter seems preferable, as it promotes normal development by encouraging the child to try to find and eat food during the day [28]. Even if the child is not fed orally, he or she should be encouraged to develop habits that will facilitate independent food consumption in the future. In young children this can be achieved by stimulating their mouths and cheeks with a hand, pacifier, or bottle, and in older children by encouraging them to play with food [34]. Once the child begins to consume more and more by mouth, the amount of food by gavage should be gradually reduced, while carefully monitoring the rate of growth. According to data available from the population of transplanted children, it takes an average of 2-10 months to fully transition to oral feeding [34], [35].

## **Lipid intake:**

In children with CKD, managing lipid intake is crucial due to the increased risk of cardiovascular complications associated with dyslipidemia. Children with CKD often experience dyslipidemia, characterized by elevated levels of triglycerides, LDL cholesterol, and reduced levels of HDL cholesterol [36]. Dyslipidemia contributes to the progression of cardiovascular disease, which is a leading cause of morbidity and mortality in CKD patients [37], [38]. Saland et al. conducted an analysis on a group of 391 children with CKD, in which they showed that hypertriglyceridemia affects as many as 126 patients. A total of 177 children with CKD had dyslipidemia and 77 had combined dyslipidemia. It has also been shown that lower GFR values are associated with higher levels of non-high-density lipoprotein-cholesterol (non-HDL-C) fraction. These differences were particularly visible between the groups of children with GFR above 50 ml/min/1.73m<sup>2</sup> and below 30 ml/min/1.73m<sup>2</sup> [36]. Limiting saturated fat intake is essential for treating dyslipidemia. Foods high in saturated fats include fatty meats, full-fat dairy products and some oils, such as coconut and palm oil. Unsaturated fats, especially monounsaturated and polyunsaturated, are a healthier option for children with CKD. Dietary fiber should also be included in the diet of children with CKD, as its consumption is associated with a reduction in LDL and total cholesterol levels and reduced cardiovascular risk [39].

## **Sodium intake**

Managing sodium intake is crucial for children with CKD to help control blood pressure, fluid balance, and overall kidney function. Sodium plays a role in regulating fluid balance in the body. Excessive sodium intake can lead to fluid retention, exacerbating edema (swelling) and increasing the workload on the kidneys. Children with CKD may already have impaired kidney function, so monitoring and controlling sodium intake helps maintain fluid balance. High sodium intake can contribute to hypertension, which is a common complication of CKD. Controlling blood pressure is essential for slowing the progression of kidney damage and reducing the risk of cardiovascular complications [2]. Sodium requirements vary among children with CKD depending on factors such as the stage of kidney disease, presence of other medical conditions, medications, and individual tolerance levels. According to the Kidney Disease Outcomes Quality Initiative (KDOQI), it is recommended to limit sodium intake in children with CKD who have hypertension or prehypertensive conditions. According to these recommendations, children between the ages of 2-3 should consume a maximum of 1,500mg of sodium per day, children in the 4-8 age group up to 1,900mg/d of sodium, in the 9-13 age range it should be a maximum of 2,300mg/d of sodium, and above the age of 14 - max. 2,300mg/d [40]. A special group of patients with CKD are children with salt loss due to tubular disease. In such a situation, supplementation should be sought to balance the tubular loss of sodium ions [2]. In children who do not waste salt, salt intake should be in accordance with the standards described above through an appropriately balanced diet.

Processed and packaged foods are major sources of dietary sodium. Encouraging whole, fresh foods and minimizing processed food consumption can help reduce sodium intake. Using herbs, spices, and other flavorings can enhance the taste of foods without adding extra sodium. Encouraging the use of fresh herbs, garlic, lemon juice, vinegar, and other natural flavor enhancers can make low-sodium meals more appealing to children with CKD. For children accustomed to a high-sodium diet, it may be beneficial to gradually reduce sodium intake to allow taste preferences to adjust over time. This approach can help prevent resistance or

aversion to lower-sodium foods [41]. Regular monitoring of blood pressure, kidney function, and nutritional status is essential to assess the effectiveness of dietary interventions and make necessary adjustments.

### **Potassium intake**

Potassium is an essential mineral that plays a vital role in various bodily functions, including muscle contraction, nerve function, and maintaining fluid and electrolyte balance. However, in CKD, impaired kidney function can lead to difficulties in regulating potassium levels in the body [2]. Impaired kidney function reduces the ability of the kidneys to excrete excess potassium efficiently, leading to its accumulation in the bloodstream. Hyperkalemia can have serious consequences, including bradycardia, chest pain, nausea, muscle weakness. In severe cases, it can be life-threatening. Children with CKD may experience symptoms such as fatigue, weakness, palpitations, and numbness or tingling sensations [42], [43]. Depending on the severity of CKD and the child's individual potassium level, a potassium-limited diet should be considered. This involves limiting the intake of potassium-rich foods to prevent hyperkalemia.

Foods that are typically high in potassium include bananas, oranges, potatoes, tomatoes, leafy greens, dried fruits, nuts, seeds, and dairy products [44]. While these foods are nutritious, they may need to be consumed in moderation or restricted in the diet of children with CKD, particularly if they have elevated potassium levels. Certain cooking methods can help reduce the potassium content of foods. For example, cooking or rinsing vegetables in water can reduce their potassium content. Other methods such as using a microwave or pressure cooking are effective in reducing potassium levels in many foods such as meat, fruit, vegetables, and grains [45].

### **Calcium and phosphorus intake**

In CKD in children, managing calcium and phosphate intake is crucial due to the impaired kidney function affecting the regulation of these minerals in the body. Maintaining proper calcium and phosphate metabolism in CKD children is particularly important to maintain their proper growth rate and prevent complications related to loss of bone density, such as pathological fractures and osteoporosis [2], [22]. Another important side effect of abnormal calcium levels is vascular calcification and an increase in the risk of cardiovascular diseases such as left ventricular failure, hence a proper diet that considers the correct proportions of calcium and phosphorus is extremely important [46], [47], [48]. In children with CKD, managing phosphorus intake is critical because the kidneys may struggle to excrete excess phosphorus efficiently, also leading to complications such as bone disease and cardiovascular issues.

The absorption of calcium from the gastrointestinal tract depends on many factors, such as the child's age, bioavailability of calcium in the food consumed, oxalates and phytic acid, vitamin D level and diseases involving absorption disorders, e.g. inflammatory bowel disease. The main source of calcium in children's diets is dairy products, which, depending on the population studied, cover 37-70% of the demand for this element [46], [49], [50]. In the group of infants, the most important source of calcium that contains the full requirement is mother's milk or formula. Modified infant formulas have a higher calcium content than breast milk, but lower

than cow's milk or formulas intended for older children. Another source of calcium covering 14-28% of the demand for this element are cereal products, rice, pasta, and biscuits. Calcium can also be found in fish, meat, vegetables, and fruit [46], [51]. The absorption of phosphorus from the gastrointestinal tract depends primarily on the level of vitamin D. The bioavailability of phosphorus found in meat, fish, cereals, and dairy products ranges between 30 and 70%, while the bioavailability of this element in legumes is much lower and amounts to a maximum of 40% [46], [52]. Phosphate, the ionized form of phosphorus, is found abundantly in various foods, for example: milk, cheese, yogurt, meat, dairy products. Grains, legumes, vegetables are also good plant-based sources of phosphorus. [53]. Some processed foods, such as fortified cereals and instant oatmeal, are enriched with additional phosphorus and other nutrients [46].

According to the PRNT guidelines, in children with CKD stage G2-5D, the main sources of calcium and phosphorus in the diet should be identified and serum calcium, phosphorus and parathyroid hormone (PTH) should be measured, and the frequency of tests should depend on the clinical condition and age of the patient. The total calcium and phosphorus intake in the diet of children with CKD should be within age-specific norms and not exceed twice the standard deviations (SDI). There are no specific studies indicating clear standards for calcium and phosphorus intake in children with CKD. The most important source of calcium is dairy products; however, in CKD, dairy products should be taken with caution, as they are also a source of phosphorus. Unfortunately, it is not possible to translate the results of the study from the adult population to children, because in children there is continuous skeletal growth, which increases the need for calcium [22],[46]. According to KDQOI position statement, the safe upper limit of calcium intake is 200% of the reference value, and this criterion should be applied to the population of children with CKD [40]. Phosphorus retention is an unfavorable prognostic factor and is associated with CKD progression, hence the diet should be modified to avoid high serum phosphorus concentrations [2]. Unfortunately, strong dietary phosphorus restriction is difficult, as it would involve reducing other essential nutrients - especially protein. However, according to PRNT guidelines, it is worth monitoring serum phosphorus concentrations, as supplementation may be necessary for some dialysis patients or those with renal phosphorus loss [46].

### **Vitamin D metabolism and intake**

The primary source of vitamin D is through the synthesis in the skin when it is exposed to ultraviolet B (UVB) radiation from sunlight. Specifically, 7-dehydrocholesterol in the skin is converted into previtamin D<sub>3</sub>, which then undergoes a thermal isomerization to form vitamin D<sub>3</sub> (cholecalciferol). Vitamin D can also be obtained from certain foods, such as fatty fish, egg yolks, and fortified foods. Both dietary vitamin D and vitamin D synthesized in the skin are absorbed in the small intestine. They are incorporated into chylomicrons, which are then transported to the liver. In the liver, vitamin D undergoes hydroxylation by the enzyme 25-hydroxylase to form 25-hydroxyvitamin D [25(OH)D], also known as calcidiol. This is the major circulating form of vitamin D and is used as a marker to assess vitamin D status. 25(OH)D is further metabolized in the kidneys by the enzyme 1-alpha-hydroxylase to form the biologically active form of vitamin D, which is 1,25-dihydroxyvitamin D [1,25(OH)<sub>2</sub>D], also known as calcitriol. Calcitriol plays a crucial role in regulating calcium and phosphate metabolism, bone health, and immune function. The production of calcitriol in the kidneys is tightly regulated by various factors, including parathyroid hormone (PTH), calcium levels, and phosphate levels in the blood. Vitamin D can also be found in the form of ergocalciferol (vitamin D<sub>2</sub>). This form of vitamin D is derived from plant sources and is commonly used in

supplements. It undergoes conversion in the liver to form calcidiol, which is then further metabolized in the kidneys to its active form, calcitriol. Overall, the metabolism of vitamin D is essential for maintaining calcium and phosphate homeostasis, bone health, and various other physiological functions in the body [54].

Supplementing vitamin D in children with CKD is crucial because impaired kidney function can lead to decreased activation of vitamin D, resulting in low levels of active vitamin D in the body [55], [56]. This deficiency can lead to various complications such as bone disorders, impaired growth, and weakened immune function. The table below [Table 2] presents Polish guidelines for vitamin D supplementation in the pediatric population and in groups at risk of vitamin D deficiency.

Age	Recommended dose of vitamin D IU/24h
newborns born $\leq$ 32. week of pregnancy	800
newborns born between 33 and 36 weeks of pregnancy	400
0-6 months	400
7-12 months	400-600
1-10 years old	600-1000
11-18 years old	800-2000
obese children and adolescents 1-10 years old	1200-2000
obese children and adolescents 11-18 years old	1600-4000

**Table 2.** Polish guidelines for vitamin D supplementation in the pediatric population and in groups at risk of vitamin D deficiency. Based on [https://www.mp.pl/pytania/pediatrica/table/025\\_2033](https://www.mp.pl/pytania/pediatrica/table/025_2033)

There have also been a few clinical trials demonstrating the effects of supplementation with both the active and native form of vitamin D in children with CKD. Schroff et al. conducted a randomized clinical trial in a group of children with CKD, during which 24 patients were given vitamin D<sub>2</sub> orally for 52 weeks. During this time, a control sample of 47 children with CKD received a placebo. Through this study, it was shown that the group receiving vitamin D<sub>2</sub> developed secondary hyperparathyroidism much later, but optimal 25(OH)D levels were much more difficult to maintain in stage 3-4 patients than in stage 2. There are also many observational prospective studies showing the benefits of native vitamin D supplementation in CKD patients [57]. Kari et al. showed that administration of a single intramuscular dose of 300 000 IU of vitamin D<sub>3</sub> in children in stage 2-5 resulted in a transient increase in 25(OH)D levels and reduced PTH levels with preserved calcium, phosphorus, and alkaline phosphatase (ALP) levels [58]. In contrast, oral D<sub>3</sub> supplementation 2 doses of 2000 IU per day for 26 weeks in stage 2-5 children resulted in normalization of 25(OH)D levels in only 11% of patients and no effect of D<sub>2</sub> supplementation on calcium, phosphorus, PTH and ALP levels was observed [59]. Hari et al. modified vitamin D dosage and administered vitamin D<sub>3</sub> orally for 3 days daily at 600 000 IU. After such supplementation, a decrease in serum 25(OH)D and PTH levels with unchanged calcium and phosphorus values was observed at week 6 [60]. Nadeem et al. used oral vitamin D<sub>3</sub> supplementation of 1000 IU or 4000 IU for 6 months in children with stage 3-5 CKD. The use of 4000 IU daily was shown to achieve 25(OH)D<sub>3</sub> values of  $\geq$ 30 ng/dL at

months 3 and 6 in a significantly higher percentage of children than in the 1000 IU supplement group, without leading to toxicity or hypercalcemia [61].

There have also been several reports regarding the use of an active form of vitamin D. Hahn et al. compared a series of randomized clinical trials using calcitriol in children with stage 2-5D CKD [62]. Salusky et al. demonstrated that intraperitoneal administration of calcitriol three times a week lowered PTH levels more than using it orally at the same time, but the form of administration did not affect growth and bone histopathology results [63]. Ardissino et al. compared oral administration of calcitriol daily at 10ng/kg or twice weekly at 35ng/kg for 8 weeks. They found no differences in growth rate, PTH levels or incidence of hypercalcemia. Serum total and ionized calcium levels were higher in children treated with intraperitoneally administered calcitriol, while serum phosphorus and alkaline phosphatase levels were higher in those given oral calcitriol [64]. Jones et al. also administered calcitriol intraperitoneally and orally but did so alternately for 3 months at a dose of 0.01-0.02 mg/kg/day. This mode of calcitriol administration did not induce any changes in plasma calcium, phosphorus and PTH levels [65]. Four clinical trials comparing the effects of active forms of vitamin D such as paricalcitol, calcitriol and 1- $\alpha$ -hydroxyvitamin D in children with CKD showed that all the above-mentioned preparations contribute to a decrease in plasma PTH, and one study showed an increased risk of hypercalcemia after intravenous administration of calcitriol. No differences were observed in the growth rate of children with CKD using different forms of active vitamin D [56].

## **Discussion**

Our knowledge regarding the pathogenesis and complications of CKD is constantly evolving and changing. The pediatric population is special in this regard, as the incidence and causes of CKD are quite different in this group than in adults. There are also far fewer clinical trials testing the optimal principles of nutrition in children with CKD than in adults. The present study focuses primarily on the proper supply of energy, protein, fat, sodium, potassium, calcium, phosphorus, and vitamin D in children with CKD. Despite concerns about excessive production of toxic products of protein metabolism, current guidelines agree that in children and adolescents with CKD, its dietary intake should not be restricted due to growth and maturation [2], [22]. Energy and lipid requirements in children with CKD also remain at a similar level as in healthy children, but the higher incidence of dyslipidemia in children with CKD should be considered and the intake of fats should be directed toward unsaturated ones [39]. Electrolyte abnormalities such as hyperkalemia and hypernatremia are particularly dangerous during CKD, so monitoring blood pressure and plasma levels of these ions is recommended if large amounts of sodium- and potassium-rich foods are consumed [2].

The proper supply of calcium and phosphorus is also a major challenge for the developing body. The amount supplied with food of these two elements should be adequate to ensure proper skeletal development on the one hand, and on the other to prevent complications associated with hypercalcemia such as vascular calcification and increased risk of cardiovascular incidents. Vitamin D is required for proper absorption of calcium and phosphorus from the gastrointestinal tract. During CKD, the hydroxylation of vitamin D in the kidneys is impaired, which impairs the formation of its active metabolite, hence several clinical trials have been conducted in which vitamin D was supplemented in both its active and native forms. The results of the trials varied depending on the dose used, route and time of administration, but most showed elevated plasma 25(OH)D levels and reduced plasma PTH levels, with varying concentrations of calcium and

phosphorus. An interesting new line of research in children with CKD is zinc supplementation. Escobedo-Monge et al. supplemented zinc at 15mg/day or 30mg/day for one year in patients with CKD under 18 years of age. In both groups, zinc supplementation had a normalizing effect on BMI, hypoalbuminemia, and CRP [66]. To counteract chronic inflammation in CKD, Hamedi-Kalajahi et al. used L-carnitine supplementation at a dose of 50mg/kg for 10 weeks in dialyzed children. In this group, L-carnitine supplementation resulted in a significant reduction in plasma IL-6 levels, fasting blood sugar, an increase in free carnitine and an improvement in clinical status as assessed by the Pediatrics Quality of Life scale [67]. The clinical trials described above underscore the interdisciplinary approach to the problem of nutrition in CKD patients and point to further research directions.

## **Conclusion**

Children with CKD require a specialized diet to manage their condition and promote healthy growth and development. In most cases, the intake ranges of many macronutrients and micronutrients are like those in healthy children, but the final decision should be made primarily depending on the child's general condition, age, body weight and stage of the disease. In older children, it is also worth talking to them about their dietary preferences to create a balanced diet that the child will follow. In summary, an appropriate diet for children with CKD includes careful control of food intake, especially of protein, fat, sodium, potassium, calcium, phosphorus, and vitamin D. Individualized nutritional plans developed by dietitians and proper cooperation with doctors are essential to ensure optimal nutrition while monitoring progress. CKD and minimizing complications.

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Authors do not report any disclosures.

## **Authors contribution**

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The authors declare no conflict of interest.

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