Pediatr Nephrol (2002) 17:111–120

INVITED REVIEW

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Nutrition in children with preterminal chronic renal failure. Myth or important therapeutic aid?

Received: 25 January 2001 / Revised: 7 August 2001 / Accepted: 5 September 2001

Abstract Nutrition has been believed to be an important therapeutic instrument in children with chronic renal failure (i) for improving growth, and (ii) for slowing down the deterioration of renal function. The therapeutic strategies for both targets may be conflicting, at least in part, since a high calorie intake is needed for optimal growth, whereas a low protein diet, which was believed to protect renal function, places patients at risk of low calorie intake. Dietary manipulations for optimal growth are mainly effective in infants with chronic renal failure. However, growth remains suboptimal even with an energy intake above 80% of RDA. Although a low protein diet is able to slow down the rate of deterioration in renal function in rodent studies, the results of prospective clinical studies were disappointing at least for an observation period up to three years. The conclusions out of meta-analyses of these clinical studies in adults are contradictory. The progression rate was not significantly influenced by protein restriction, whereas renal replacement therapy could be postponed. However, the latter seems to be the effect of weakening uremic symptoms during the phase of end-stage renal failure. According to our present knowledge it is not justified to prescribe special diets to children early in the course of chronic renal failure, but the composition of their nutrition should follow the general concept of an optimal mixed diet.

Keywords Chronic renal failure · Children · Protein · Energy · Lipids · Diet · Progression

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Introduction

Dietary interventions have been widely used in patients with preterminal renal failure to minimize the progression of chronic renal failure (CRF). Though there is good theoretical and experimental evidence that a modification of nutrition is effective in patients even with modest renal failure, results of recent prospective clinical studies are less convincing.

The prescription of a low protein diet to reduce the progression rate of CRF may interfere with another target of nutrition in children with CRF, e.g. optimal support for growth. There is good evidence that malnutrition is one important factor for impairment of growth in CRF.

This article will review the most important dietary intervention studies and critically illuminate to what extent present dietary guidelines are evidence based.

Recommendations for macronutrient intake

The first generally accepted recommendations for nutrient intakes for all age groups had been published by the World Health Organization (WHO). While in these publications a clear differentiation was made between "recommendations", e.g. for calories, and "safe levels", e.g. for protein [1, 2], the national nutritional authorities, e.g. Food and Drug Administration (FDA) [3] (Table 1) or German Society for Nutrition (Deutsche Gesellschaft für Ernährung (DGE) [4] accepted safe levels as "recommended daily allowances" (RDA). By this, a considerable deviation from RDA to actual nationally analysed intakes became evident.

A recent nutritional survey among healthy German children demonstrated that, as in most western countries energy intakes in all age groups are 10–20% lower than RDA, with carbohydrates providing 48% of energy only, while fat with 39% and protein with 13% of energy were higher than recommended [5].

The younger the child, the higher is the need for energy and protein (kcal or g/kg per day) (Table 1). But if

Table 1 Recommendations for energy and protein intake in males given by the Committee on Dietary Allowances 1989 [3]

Age (years)	Energy (kcal/kg per day)	Protein (g/kg per day)
0–0.25	108	2.2
0.26–1.0	98	1.6
1.0–3	102	1.2
4–6	90	1.2
7–9	70	1.0
10–12	60	1.0
13–14	55	1.0
15–18	45	0.9
19–24	40	0.8
25–50	37	0.8
51+	30	0.8

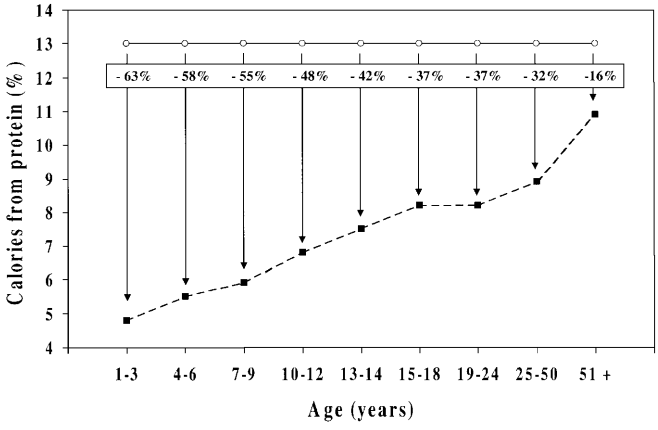

Fig. 1 Consumed protein intake of about 13% of calories in male healthy German individuals in all age groups (–○–) [5] compared to recommended dietary allowances (Table 1) [3] (--■--). (↓ % difference between consumed and recommended protein intake)

children meet their high energy needs with the same food composition as adults, e.g. with 13% of energy provided by protein, their protein intake is up to 63% higher than recommended (Fig. 1). Practical application of this concept in accordance to RDA would result in a need for totally different food composition for different age groups, i.e. different meal preparation for each family member sitting around the same table. To stick to RDA for protein in western countries, special low protein dietary products would have to be prescribed even in healthy young children.

New nutritional concepts no longer provide exact amounts in g/day for each macronutrient. They only give figures for the age-related energy requirements and recommend an optimal mix of nutrients (optimal mixed diet) [6, 7]. These recommendations even accept a protein intake up to 14% of total energy with only 50% provided from animal origin, 32% fat and 54% carbohydrates [6].

Modification of nutrition

Nutrition provides the fuel for the body to function, but, equally important nutrition plays a central role in social life and induces good feelings like satisfaction, delight and well-being. Therefore, the medical advice to modify nutrition must be based on adequate considerations. A large amount of skill and time of specialised dietitians must be invested into qualified dietary counseling [8], which provides the patient with positive information of preferable nutrients respecting the patient's eating habits, instead of providing lists of forbidden things. Otherwise, feeding problems and malnutrition will be aggravated.

All randomised studies modifying nutrition in patients with renal failure compare the outcome according to the intended intervention. Only a few studies supervised compliance and few correlated outcome to compliance [9, 10, 11]. Only in the Modification of Diet in Renal Disease (MDRD) trial patients had been asked about satisfaction with the dietary treatment. Satisfaction significantly decreased with dietary restrictions [12].

Modification of one macronutrient modifies the composition of other macronutrients and the balance of micronutrients. For instance, low protein diets based on vegetable, soy or mixed protein may provide the same amount of protein, but a totally different composition of amino acids, different amounts and compositions of lipids and different amounts of minerals, trace elements and vitamins [13, 14, 15, 16]. Low cholesterol diets may be associated with a deficit of many minerals and vitamins [17]. Therefore, a modification in the macronutient intake must be balanced and needs to be well controlled to avoid unintended effects.

Nutrition and growth

Both healthy children and children with renal disease need a high energy intake for adequate growth. Energy needs decrease from 108 kcal/kg per day in infants to 30 kcal/kg per day in adults (Table 1). However, energy and protein recommendations by RDA and WHO are 10–20% higher than actual spontaneous energy intakes in healthy infants either breast fed or bottle fed [18, 19, 20] and in well-nourished and well-growing children [5].

Adults and children with renal failure tend to develop protein-caloric malnutrition because of lack of appetite or vomiting [21, 22, 23, 24]. The spontaneous food intake varies from day to day and compliance and vomiting cannot easily be monitored. It is characteristic that most studies on nutritional intake indicate the prescribed but not the true nutrient intake. The most exact studies have been performed in infants with CRF and it has been shown that the degree of growth failure is correlated with the amount of calorie intake. However, a calorie intake above 80% of RDA did not further improve growth [25, 26, 27] (Table 1). There was no need to increase the protein intake above the amount calculated from spontaneous breast feeding in healthy infants [20].

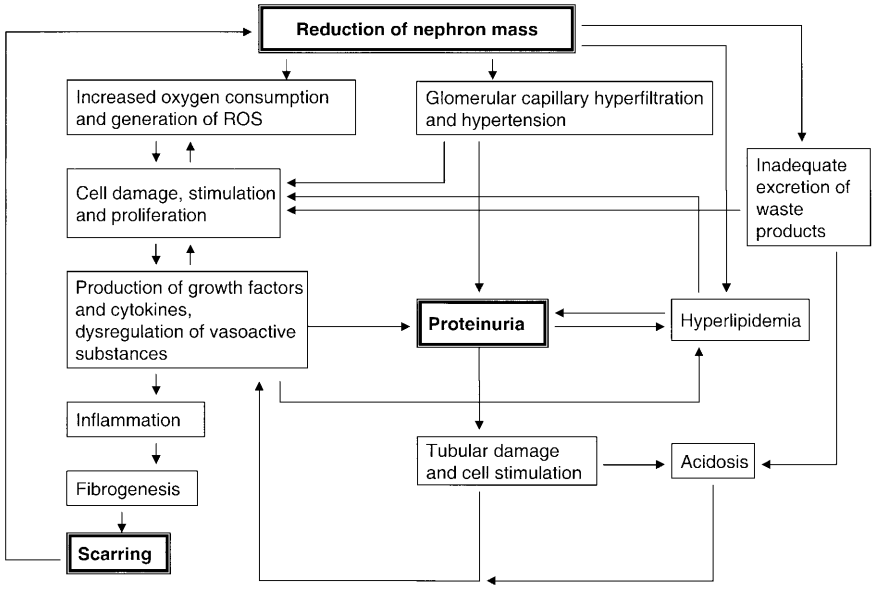

Fig. 2 Hypothetical model of events leading to progression of renal failure. *ROS* reactive oxygen species

Whereas an adequate calorie intake can prevent a fall of the growth curve of infants with CRF below the normal centiles or even induce catch-up growth, the latter effect cannot be obtained in the majority of patients with an age beyond 2 years. Furthermore, the reported success of dietary intervention is grossly dependent on the growth standards that are used for comparison. It is not justified to use old standards like those of Tanner [28] which originate from measurements performed more than 50 years ago, because the enormous secular trend for improving height and growth in Europe [29, 30, 31] and other parts of the world [32, 33] during the last century is missed and the results will look too optimistic.

Nutrition and progression of renal disease

If loss of renal function has reached a critical point, renal failure usually progresses by self-perpetuating pathways, irrespective of the primary renal disease (Fig. 2). The therapeutic aim is to halt the progression of renal disease by interruption of these pathways and elimination of possible disease modifiers such as: proteinuria [34, 35, 36, 37], glomerular hyperfiltration and hypertension [38, 39], arterial hypertension [40, 41, 42], hyperlipidemia [43, 44], acidosis [45, 46], uremic toxins [47, 48], hyperparathyroidism [49, 50] and hyperphosphatemia [51].

The complex interplay of all factors involved in the progression of renal disease makes it almost impossible to elaborate the contribution of a single player. In all human studies proteinuria and hypertension proved to be the strongest independent predictors for progression of renal failure [11, 36, 52, 53, 54]. But even these two variables are not proven to be totally independent. A lowering of blood pressure, which also resulted in reduction of proteinuria, most effectively conserved renal function in patients with initially high proteinuria [55]. Therefore, proteinuria is believed to play the key role in the progression of renal disease.

Among all therapeutic efforts, only therapy with angiotensin-II converting enzyme inhibitors and angiotensin-1 receptor antagonists has been proven to influence progression of renal failure favorably in human and/or animal studies [56, 57, 58]. These pharmaceutical agents lower systemic and intraglomerular blood pressure and reduce proteinuria. They directly interfere with inflammatory processes and fibrogenesis in the kidney [59, 60]. Additionally, recent experimental evidence points to a direct action of angiotensin-II converting enzyme inhibitors at the slit pore membrane [61]. Can similar effects also be obtained by prescribing a modified diet to children or adults with CRF?

Energy

In animal models, a low energy intake slows down the progression of renal disease [62, 63]. Vice versa, a high energy intake may result in renal failure [64] and hypertension [65]. Obese humans present with high leptin levels [66]. High leptin levels may be involved in the progression of glomerular sclerosis as noticed from animal studies [67]. Humans with morbid obesity are prone to develop focal segmental glomerulosclerosis [68]. Children [69] and adults [70] with progressive renal disease have high leptin levels which may contribute to lack of appetite [69] and progression of renal failure.

Although a sufficient energy intake is recommended for children with CRF, an exaggerated calorie intake may induce hyperlipidemia, hyperinsulinism and arterial sclerosis in the long term. Prospective studies on this subject are missing.

The utilisation of protein is correlated with the amount of energy intake even in individuals with protein and

energy intakes within the normal range [71, 72, 73]. Therefore, a low protein intake must be accompanied by high energy intake in order to avoid malnutrition.

Protein

A low protein diet postpones the development of uremic symptoms and start of dialysis in patients with advanced renal failure [74]. Lowering protein intake reduces the amount of protein degradation products that are not adequately excreted by the diseased kidney and contribute to acidosis. Acidosis is one main reason for catabolism in uremic patients [75]. This is especially seen in acute acidosis, whereas a partial metabolic adaptation is noted for chronic acidosis. Acidosis impairs growth hormone induced growth [76]. Phosphorus intake usually is reduced in parallel to protein intake. Therefore, control and prevention of hyperparathyroidism is easier in patients on a low protein intake [77]. Besides its role in renal bone disease, hyperparathyroidism and other undefined uremic toxins are suspected of augmenting insulin resistance [78] and disturbances of lipid metabolism [79, 80].

In humans and animals a high protein intake, most from animal origin, induces renal hyperfiltration [38]. In animals with reduced renal mass high protein intake provokes intraglomerular hypertension [39], loss of autoregulation of intrarenal blood pressure [81] and glomerular hypertrophy. Hyperfiltration and glomerular hypertrophy, which presumably are mediated by insulin-like-growth factor 1 (IGF-1), are in many animal models and in human diseases, e.g. diabetic nephropathy, the first visible events in the process leading to progressive renal disease [82]. However, in animal models, administration or expression of IGF-I did not result in an increased damage of renal cells [83, 84]. In children with normal renal function and normal energy intake IGF-1 diminishes with a low protein intake [85]. Considering low protein diet in children, one should keep in mind that IGF-1 is needed for body growth [86] while its unfavorable effect on renal function is not proven.

In patients with a steroid resistant nephrotic syndrome [87] and also in some patients with CRF [10] a low protein diet reduces proteinuria. At the subcellular level a low protein intake reduces the generation of reactive oxygen species (ROS) [88], the generation and activity of mediators of inflammation [89, 90], and the activation of the renin-angiotensin system [91] (Fig. 2). The activation of inflammatory pathways can add to the altered lipid metabolism and increase serum lipid levels in renal disease [92]. Recently, it has been postulated that proteinuria by virtue of the processes listed above, plus the telomere-shortening limit on hyperplasia, leads to renal cell senescence and loss of renal function [93, 94]. Telomeres are the DNA-protein complexes found at the ends of chromosomes. In the absence of the enzyme telomerase these structures shorten at each cell division. After a critical degree of shortening, cells senesce and finally die through apoptosis.

A theoretically negative aspect of a very low protein diet may be the lower L-arginine supply, which may lower the production of nitric oxide (NO), the most potent vasodilator in the body, acting against hypertension and proteinuria [95, 96]. Additionally, diets low in L-arginine may promote dyslipidemia [97]. This is in contrast to some animal models of antibody mediated glomerulonephritis, in which the favorable effect of low protein diet could be counteracted by substitution of L-arginine [98].

A more practical problem is that the minimal and maximal amount of protein intake compatible with optimal health of humans and animals not exactly known [99, 100]. In adults with renal failure, the lowering of protein intake to 0.6 g/kg per day or less (if amino acid and keto acid mixtures had been added) and in children with renal failure the lowering to the levels given by RDA (Table 1) proved to be safe for periods up to 3 years as documented in controlled studies. But, in spite of the skills of trained dietitians, lowering protein intake was accompanied by a small but significant drop in mean energy intake. In the MDRD study mean energy intake dropped from 73% of RDA at start to 63% of RDA during this study (–10%) [101] and in the European study in children from 92% to 85% of RDA (–7%) [11].

Within all positive and negative arguments for or against a low protein diet, the key question is whether such a diet can slow down the progressive loss of renal function. This question has not definitely been answered, although several well designed studies in hundreds of patients with non-diabetic renal disease, running for 2 years and longer have been performed [11, 52, 53, 102, 103 104] (Table 2).

Meta-analyses of all these studies demonstrate that a small but statistically significant reduction in the incidence of end-stage renal disease is evident for low protein diet [105, 106, 107, 108, 109]. As the progression rate was not significantly influenced by protein restriction (Table 2), this seems to reflect postponement of the start of renal replacement therapy in patients with end-stage renal failure because of amelioration of uremic symptoms by low protein intake.

Though there was no statistically significant difference in the progression rate in patients randomised for low versus normal protein intake, most studies demonstrated a higher loss of renal function in patients randomised for higher protein intakes (Table 2). It may be tempting to speculate that with a longer period of observation this difference will become significant. But, in the two studies which added 1 or 2 years of prolongation [11, 103], the difference, which was not significant after 2 years, was almost absent after 3–4 years.

In all studies (Table 2) most patients did not reach their low protein goal. Protein intakes were in the mean 30% higher than recommended. In consequence, the difference in protein intake between randomised groups was lower than planned and figured at 20–30%. In two studies only primary analyses of the results based on the intention to treat, and additional secondary analyses based on compliance have been performed [9, 10, 11]. In

Table 2 Randomised studies on the effect of a low protein diet on the progression of non-diabetic renal failure with more than 100 patients enrolled for 2 years and more

Reference	No. of patients	Age (mean, years)	Duration of observation (years)	Outcome measures		Approximate GFR at start (ml/min per 1.73 m²)	Loss of GFR (ml/min per 1.73 m²) per year		Conclusion
				Progression	Survival at a defined endpoint		Diet	Control	
[a]Rosman et al. 1984 [104]	149	48	2	1/creatinine	10% rise in serum creatinine	30	?	?	In favour of low protein diet
[a]Rosman et al. 1989 [103]	153	48	4	Endogenous creatinine clearance	50% loss of endogenous creatinine clearance	30	–2.23	–2.50	Limited effect of low protein diet
[b]Locatelli et al. 1991 [102]	456	49	2	Endogenous creatinine clearance	Doubling of serum creatinine	38	–1.8	–0.96	No effect of low protein diet
[b]D'Amico et al. 1994 [52]	128	54	2	Endogenous creatinine clearance	Halving of endogenous creatinine clearance	33	?	?	Good effect of low protein diet
[c]Klahr et al. 1994, Study A [53]	585	52	3	Iothalamate clearance	Endstage renal disease	39	–3.6	–4.0	No effect of low protein diet
[c]Klahr et al. 1994, Study B [48]	255	51	3	Iothalamate clearance	Endstage renal disease	19	–3.6	–4.4	No effect of low protein diet
[d]Wingen et al. 1997 [11]	191	10	2	Estimated creatinine clearance (Schwartz)	10 ml/min per 1.73 m² loss of creatinine clearance	37	–3.0	–3.4	No effect of low protein diet
[d]Wingen et al. 1997 [11]	112	10	3	Estimated creatinine clearance (Schwartz)	10 ml/min per 1.73 m² loss of creatinine clearance	41	–2.0	–2.0	No effect of low protein diet

[a] The same patients are reported at different times of observation. Patients in the low protein group with a GFR below 30 ml/min per 1.73 m² were instructed to lower protein intake to 0.4 g/kg per day and those with a GFR above 30 ml/min per 1.73 m² to lower protein intake to 0.6 g/kg per day. Advice was given to lower protein intake of animal origin

[b] Both studies reduced protein intake to 0.6 g/kg per day with 0.5 g of animal origin in low protein diet and to 1.0 g/kg per day with 0.6 g of animal origin in the control group

[c] In study A normal protein intake of 1.3 g/kg per day was compared to low protein intake (0.58 g/kg per day) in patients with moderate renal failure. In study B low protein intake of 0.58 g/kg per day was compared to a protein intake of 0.28 g/kg per day supplemented with 0.28 g/kg per day amino acids and keto acids in patients with advanced renal failure

[d] Protein intake was lowered to the recommendations of WHO (0.8–1.1 g/kg per day according to age) [2]. Of 191 children observed for 2 years, 112 completed an additional third year. Creatinine clearance was calculated with the formula published by Schwartz [110]

adults participating in the MDRD study [9, 10] recalculation of the data in a regression model controlled for covariates demonstrated a significantly lower loss of renal function and a reduced risk for renal death at different levels of reduction of protein intake. In patients with moderate renal failure [10] this difference became evident only when the loss of renal function was calculated from 4 months after randomisation to the end of observation. During the first 4 months the higher loss of renal function in patients with low protein intake was assumed to be induced by the loss of protein-induced hyperfiltration. In children participating in the European Study on low protein diet [11], similar calculations (data not published) did not confirm the results. In children with moderate renal failure, as defined by the MDRD study, there was no accelerated loss of renal function during the first 4 months on low protein diet and during 2 years the loss of renal function was significantly lower with higher protein intakes.

The above differences in the effect of a low protein diet in children and adults may be explained by the age of patients, totally different patterns of underlying renal diseases, higher protein intakes or different techniques to measure renal function. An additional important difference between the MDRD trial and the European Study in children is the number of patients with calculations of renal function at each point of observation. While measurement of renal function by estimating GFR from body length and serum creatinine used in the study in children [11, 110] is definitely inferior to the iothalamate clearance method used in the study in adults [53], frequent collection of creatinine data may be more complete and exact for calculations of the slope of GFR over time than infrequent iothalamate clearance data. In the trial in children available data on renal function dropped from 100% to 88% in the 2-year study and from 100% to 97% in the three-year study because of the intercurrent necessity to start renal replacement therapy. In the trial in adults the number of clearance studies available for calculation dropped from 100% to 62% at 2 years and to 24% at 3 years, while only about 215 (24%) patients dropped out for medical reasons [9, 111]. It seems questionable, if

even the best statistical methods can compensate for a heavy censoring like this.

Despite all these discussions, one should not forget that the primary analysis in both studies in adults [53] and children [11] did not document a significant benefit of a low protein diet within a period of 3 years. If there would really be a significant effect after much longer observation periods, this effect is predicted to be small and seems, at present, not to justify the prescription and the burden of a low protein diet in children over many years (starting already at an early stage of CRF). However, it seems prudent to avoid a protein excess also in view of the concomitant high phosphate intake.

Only few studies in humans with small numbers of patients address the question whether it makes any difference, if the protein supplied is from vegetable or animal origin. Few studies reveal the recommended composition of protein in the method section [52, 102]. In children it is difficult to provide the high energy requirements by strict vegetarian diets, because their energy density is low. Possibly soy protein may provide the required density of calories. Studies in animals and humans suggest that protein intake in the normal range provided by soy exerts beneficial effects on dyslipidemia and renal function [112, 113]. However, a diet based on soy protein requires a total change in nutritional habits.

Children with renal disease, as well as healthy children in western countries ingest a percentage of protein provided from animal origin of 60–70% [5, 11], while the recommendations are approximately 50%. In the European study, children with renal failure randomized to the protein-restricted group consumed 54% of protein from animal origin [11]. There was no difference in progression of renal failure in the secondary analyses based on total protein intake and percentage provided from animal origin (unpublished).

Fat

Patients with renal failure tend to have dyslipidemia, dyslipoproteinemia and high serum lipids [114, 115, 116]. In a recent study in children aged 2–18 years with glomerular filtration rates between 15 and 60 ml/min per 1.73 m^2 about 60% of patients demonstrated triglyceride, cholesterol, HDL-C, LDL-C or VLDLC levels above the 95th centile for healthy children [11]. Hyperlipidemia is correlated to the degree of renal function and proteinuria [11, 117]. Hyperparathyroidism [79, 80], high levels of cytokines [92] and low NO [97] may add to dyslipidemia.

Lipids and lipoproteins may interact with mesangial cells, vascular endothelial cells, glomerular epithelial cells and tubular epithelial cells in the kidney and induce oxidant stress [118, 119, 120, 121]. LDL oxidised by ROS, more than native LDL, exert direct toxic effects on the cell and stimulate the release of mediators of inflammation [118, 119, 122, 123]. Via binding to podocytes hyperlipidemia may disturb the glomerular filtration barrier and add to proteinuria and its negative influence on renal function (Fig. 2) [120]. Hyperlipidemia may increase angiotensin II receptor expression and thereby interfere with progressive loss of renal function [124].

In animal models with renal failure, interventions aggravating hyperlipidemia are correlated to higher rates of progression of renal failure and higher indices of renal damage [120, 123, 125, 126, 127, 128, 129, 130, 131]. In summary, lowering lipids may theoretically contribute to halt the progression of renal failure.

In humans using univariate statistical models, hyperlipidemia significantly correlated to the progression of renal disease [11, 111, 117, 132, 132, 133, 134, 135, 136]. However, in multivariate models, which include the degree of renal failure and proteinuria, the correlation of hyperlipidemia and progression was minimal or absent [11, 111, 117, 134]. Up to now it is unproven that lipids add to the progression of renal failure in humans. However, the lipid profile in patients with renal failure may add to the augmented risk of atherosclerosis in patients with renal disease.

Therapeutic options to lower serum lipids are: (i) diets low in fat, phosphate and protein or diets based on soy protein, (ii) lipid lowering drugs like statins, fibrates or probucol, and (iii) lipidapheresis. All these therapeutic options work in patients with renal failure, i.e. they lower serum lipids. Hopefully, they may reduce the risk for atherosclerosis. However, it is not yet proven in humans, if hyperlipidemia in humans is correlated to the progression of renal disease and if lowering concentrations of serum lipids halts the progression of renal disease.

Drugs such as statins, fibrates and probucol not only lower serum lipids, but specifically interfere with inflammatory processes in diseased kidneys [125, 127, 129, 130, 131, 137, 138, 139]. Therefore, the beneficial effect of these drugs on the progression of renal disease is not, or at least not only, mediated by lowering serum lipids. None of these drugs is approved for use in children. Therapy with statins or fibrates must be monitored closely, and probucol has recently been taken off the market because of severe side effects [140].

Fat has a high calorie-density of 9.3 kcal/g. Fat is an important factor for the palatability of nutrition. Therefore, a strict reduction of fat intake is not advisable for renal patients, who suffer from lack of appetite and malnutrition. But care should be taken, that the composition of fat is optimal. Fat with a good mix from animal and vegetable origin should be preferred.

Lipids derived from marine sources are enriched with omega-6 polyunsaturated fatty acids, which are metabolised to vasodilatory eicosanoids. In animal and human IgA nephropathy supplementation with fish oil significantly slowed the progression of renal failure [141, 142]. From the animal model of IgA nephropathy it may be concluded that this effect may be at least partially mediated by the alpha-tocopherol content of the preparation [141]. Vitamin E is a known antioxidant, which can block the action of ROS.

Conclusions

Given the complex roles of nutrients in the progression of CRF as outlined in this review, which practical recommendations can be given for dietary counselling of children with CRF?

In animal models, reductions of either protein or fat intake could favorably influence the complex self-perpetuating pathways of hyperfiltration and interstitial fibrosis, and slow down the progression of CRF (Fig. 2). By contrast, the results of similar dietary manipulations in humans have been disappointing so far. Reasons for this discrepancy between animal and human studies are complex and include the diversity of underlying renal diseases, genetic backgrounds, comorbid conditions and the difficulty to define, and even more, adhere to, nutritional modifications in the long term.

Based on the data accumulated so far, it cannot be excluded that strict restrictions of protein and/or fat intake may have a small beneficial effect if adhered to over extended periods of time; however, the burden would be high over many years and the psychosocial acceptability of such strict diets in children with CRF and their families must be questioned. On the other hand, one reassuring result of the European trial on low protein diet in children with CRF was that protein restriction to 0.8–1.1 g/kg daily adjusted for age and a calorie intake of at least 85% of RDA is not detrimental on longitudinal height and weight gain. Moreover, since many children tend to eat self selected diets with a very high protein intake of mainly animal origin and fat with a high content of cholesterol and saturated fat, a change in dietary habits in healthy children as well as in children with CRF seems to be worthwhile. A moderate protein intake is certainly advisable, particularly in children with advanced CRF with regard to the control of metabolic acidosis, phosphorus intake and the accumulation of toxic nitrogen waste products.

Hence, while no arguments for specific dietary interventions can be derived from the clinical trials performed so far, dietary counselling in children with CRF should aim at a calorie intake not below the recommended dietary allowances. Phosphorus and potassium intake must be reduced if necessary, and acidosis should be controlled by medication as soon as serum bicarbonate falls below 20 mmol/l. Children should follow an optimal mixed diet, composed of a large variety of foods, best depicted in the food guide pyramid [7]. The pyramid consists of a broad basis composed of bread, cereals, rice and pasta, a smaller second level comprising vegetables and fruit, and an even smaller third level composed of milk, milk products, meat, fish, beans, eggs and nuts. The small tip of the pyramid is composed of foods that should be used sparingly like sweets, oils, cream, etc. This composition follows the general concept of preventive nutrition [143].

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