Nutritional Care for Adults with Chronic Kidney Disease

A Guide to Clinical Practice

Editor
Kalliopi-Anna Poulia
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Acknowledgements
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Pathophysiology of the Kidney
1.1 Kidney’s Structure

The components of the kidney are: the nephrons, the collecting ducts, and a unique microvasculature. The multi-papillary human kidney contains approximately one million nephrons, a number already established during prenatal development. New nephrons cannot be developed after birth, and that is the reason why a lost nephron cannot be replaced.

1.2 Physiology

The main function of the kidneys is maintaining a stable milieu intérieur by the selective retention or elimination of water, electrolytes, and other solutes. This is achieved in three steps. First, circulating blood is filtered from the glomerulus, so that the ultrafiltrate of plasma in Bowman’s space is formed. Secondly, a selective reabsorption (from tubular fluid to blood) across the cells’ lining the renal tubule happens. Finally, selective secretion from peritubular capillary blood to tubular fluid takes place.

The total rate at which fluid is filtered into all nephrons is called the Glomerular Filtration Rate (GFR). The typical value of GFR is 120-130 ml/min per 1.73m² surface area, but the normal range differs considerably (according to sex, age, body size,

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1 consisting of a renal corpuscle (glomerulus), which after being connected to a complicated and twisted tubule, finally drains into a collecting duct.

2 formation in the renal cortex of the joining of several nephrons.
activity, diet regime, drug treatment, etc). GFR is approximately 8% higher in young men than in women and declines with age; the mean rate of decline is approximately 0.75 ml/min per year after 40 years of age. The renal clearance of any substance not metabolized by the kidneys is the volume of plasma required to provide that amount of the substance excreted in the urine per unit time. Given a typical GFR, roughly 180 liters of mostly protein-free plasma is filtered daily, which leads to a need for massive reabsorption by the nephron as a whole. A major function of the kidneys is to regulate the blood volume; this is achieved through the regulation of effective circulating volume\textsuperscript{3}, which is largely controlled by the body’s sodium content.

A decline in the number of nephrons can lead to reduction in GFR, as also a decline in the single-nephron GFR from physiologic or hemodynamic alterations can happen. If the latter increases (as in increased glomerular capillary pressure or glomerular hypertrophy), this can compensate for a decrease in nephron number; therefore GFR may not reflect the loss of nephrons and this may result in causing substantial kidney damage before GFR actually decreases.

### 1.3 Pathophysiology of the kidney

#### 1.3.1 Acute Kidney Injury

Acute kidney injury -AKI\textsuperscript{4} is a clinical syndrome denoted by a sufficient decline in GFR to decrease the elimination of nitrogenous waste products\textsuperscript{5}, and other uremic toxins. A proposed definition for AKI, is “a decline in kidney function during 48hs as demonstrated by a) an increase in serum creatinine of >0.3 mg/dl, b) an increase in serum creatinine of

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\textsuperscript{3} An inmeasurable, conceptual volume that reflects the degree of fullness of the vasculature.

\textsuperscript{4} Has previously been referred to as acute renal failure-ARF.

\textsuperscript{5} Urea and creatinine
Nutritional Care for Adults with Chronic Kidney Disease

>50%, or the development of oliguria. Staging criteria, which are of prognostic value, has also been based on the magnitude of the rise in serum creatinine and changes in the volume of urine output during 1 week.

The development of AKI can be due to a wide range of causes; differential diagnosis must be considered in a systematic way in order to avoid missing multiple factors that may be contributing to the condition. The traditional paradigm divides AKI into prerenal, renal, and postrenal causes. Prerenal AKI may be due to hypovolemia or a decreased effective arterial volume. Postrenal obstructive renal failure is usually diagnosed by urinary tract dilation on renal ultrasound or computed tomography scanning. Intrinsic renal causes should be taken under consideration under the different anatomic components of the kidney (vascular supply, glomerular, tubular, and interstitial disease).

Wise approaches for early detection of AKI include identification and monitoring. The clinical manifestations of AKI (which can also be asymptomatic) depend on the severity and duration of the kidney dysfunction and are accompanied by metabolic disturbances (i.e. metabolic acidosis, hyperkalemia, abnormal body fluid balance and effects on other organ systems). AKI is more common in hospitalized patients; AKI episodes can lead to chronic kidney disease (CKD), accelerate the progression to end-stage renal disease, and contribute to higher long-term mortality risk.

1.3.2 Chronic Kidney Disease

Chronic kidney disease-CKD- can be defined as kidney damage or a GFR <60 ml/min per 1.73 m² for 3 months or more irrespective of the cause. The classification of CKD

6 Asymptomatic AKI can result in significant delay in the diagnosis and will most likely be found in routine biochemical screening (as an increase in blood urea nitrogen (BUN) and creatinine concentrations) of hospitalized patients.
based on GFR as proposed by the Kidney Disease Outcomes Quality Initiative (KDOQI) guidelines and modified by NICE in 2008, can been seen in Table 1.1

<table>
<thead>
<tr>
<th>CKD stage</th>
<th>Definition</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>Normal or increased GFR; some evidence of kidney damage reflected by microalbuminemia, proteinuria, and hematuria, as well as radiologic or histologic changes</td>
</tr>
<tr>
<td>2</td>
<td>Mild decrease in GFR (89-60 ml/min per 1.73 m²) with some evidence of kidney damage reflected by microalbuminuria, proteinuria and hematuria as well as radiologic or histologic changes</td>
</tr>
<tr>
<td>3</td>
<td>GFR 59-30 ml/min per 1.73 m²</td>
</tr>
<tr>
<td>3A</td>
<td>GFR 59-45 ml/min per 1.73 m²</td>
</tr>
<tr>
<td>3B</td>
<td>GFR 44-30 ml/min per 1.73 m²</td>
</tr>
<tr>
<td>4</td>
<td>GFR 29-15 ml/min per 1.73 m²</td>
</tr>
<tr>
<td>5</td>
<td>GFR &lt;15 ml/min per 1.73 m²; when renal replacement therapy in the form of dialysis or transplantation has to be considered to sustain life.</td>
</tr>
</tbody>
</table>

The suffix p to be added to the stage in proteinuric patients (proteinuria <0.5 g/24h)

Since early to moderate CKD is usually asymptomatic, the actual incidence and prevalence of CKD within a community is difficult to be defined. Different study groups suggest a prevalence of CKD around 10%, albuminuria (mostly microalbuminuria) round 7%, and a GFR below 60 ml/min per 1.73 m² around 3%. Risk factors for CKD include susceptibility (predisposing to CKD), initiation (directly triggering KD), and progression (leading to worsening of KD) factors. Identification
of susceptibility and initiation factors is useful in order to define those at high risk for development of CKD, whereas the identification of progression factors helps foresee those at higher risk for a possible loss of kidney function.

CKD progression is influenced by a variety of modifiable and non-modifiable progression risk factors (PRF), as can been seen in Table 1.2.

Since the majority of CKD patients die of non-renal causes (mostly CVD), early diagnosis of CKD is of high importance and can delay its progression and in that way also delays relevant CVD complications.

Table 1.2: Modifiable and non-modifiable risk factors for the progression of Chronic Kidney Disease

<table>
<thead>
<tr>
<th>Non-modifiable PRF</th>
<th>Modifiable PRF</th>
<th>Additional PRF</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age</td>
<td>Hypertension</td>
<td>Alcohol and recreational drugs</td>
</tr>
<tr>
<td>Gender</td>
<td>Proteinuria</td>
<td>Analgesics and NSAIDs</td>
</tr>
<tr>
<td>Race</td>
<td>Albuminuria, Chronic Kidney Disease, and Cardiovascular Disease</td>
<td>Lead and heavy metals exposure</td>
</tr>
<tr>
<td>Loss of Renal Mass</td>
<td>Renin-Angiotensin System</td>
<td></td>
</tr>
<tr>
<td>Genetics</td>
<td>Glycemia</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Obesity</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Lipids</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Smoking</td>
<td></td>
</tr>
</tbody>
</table>
### Table 1.3. Clinical manifestations of CKD

<table>
<thead>
<tr>
<th>Hypertension</th>
<th>Immunity</th>
</tr>
</thead>
<tbody>
<tr>
<td>Dyslipidemia</td>
<td>Psychological manifestations</td>
</tr>
<tr>
<td>Anemia</td>
<td>Other complications</td>
</tr>
<tr>
<td>Bone and mineral metabolism</td>
<td>Bleeding diathesis</td>
</tr>
<tr>
<td>Malnutrition</td>
<td>Neurologic problems</td>
</tr>
<tr>
<td>Sodium and water retention</td>
<td>Dermatologic manifestations</td>
</tr>
<tr>
<td>Potassium</td>
<td>Uremic pericarditis</td>
</tr>
<tr>
<td>Endocrine abnormalities</td>
<td></td>
</tr>
</tbody>
</table>

#### 1.4 Diabetic nephropathy

Diabetic nephropathy-DN, which is the leading cause of end-stage renal disease (ESRD) in western countries, can develop in both type 1 and type 2 diabetes mellitus (DM-1 and DM-2 respectively), but also in other forms of DM. Only approximately 1 of every 3 patients with DM-1 or DM-2 will ultimately develop DN, which shows that the risk varies in different racial and ethnic groups and is therefore co-determined by polygenetic factors.

Detection of proteinuria is the main diagnostic sign for DN, whereas the majority of patients also have hypertension and retinopathy. The main diagnostic procedures completed on a patient with suspected DN should include:

- Measurement of urinary microalbumin or albumin or protein
- Measurement of serum creatinine concentration and estimation of GFR
- Measurement of Blood Pressure (BP)
- Ophthalmologic examination
Change of renal function, followed by an increase in GFR are among the first to be found in DM. This is paralleled by an increase in renal size and then the development of albuminuria follows. DM-patients with persistent microalbuminuria\(^7\) demonstrate a marked increase in risk of developing DN (which is heralded by the development of proteinuria\(^8\)).

The presence of DN greatly increases\(^9\) mortality in both DM-1 and DM-2. Glycemic control and blood pressure control are the main general measures for prevention of DN. Since DM is associated with an increased risk of CVD morbidity and mortality, treatment of dyslipidemia together with diet and lifestyle modifications (such as physical activity, weight reduction and smoking cessation), can significantly lead to a decreased CVD risk.

### 1.5 Nephrotic syndrome

Nephrotic Syndrome-NS\(^{10}\) is characterized by the presence of the following symptoms:

- Proteinuria >3,5g/24h/1,73m\(^2\) body surface (in normal GFR)
- Lipiduria
- Hypoalbuminemia
- Peripheral or general oedema
- Hyperlipidemia

Nephrotic syndrome demonstrates metabolic effects that can influence the patient’s general health. For most patients, it is

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\(^7\) Albumin excretion rates between 0 and 30 mg/day are called normoalbuminuria, and between 30 and 300 mg/day, microalbuminuria.

\(^8\) albuminuria >300 mg/day

\(^9\) 20- to 200-fold in patients with proteinuria.

\(^{10}\) The relative frequency of the different glomerular diseases leading to NS development is variable to age. Additionally, patients may be nephrotic having preserved renal function; on the other hand in many patients progressive renal failure will become superimposed.
a chronic condition, since only some episodes of NS are self-limited, and a few respond completely to specific treatment (e.g., corticosteroids in MCD). Some patients, even with proteinuria >3.5 g/24 h, have a normal serum albumin concentration, and therefore do not demonstrate full NS. This might be the result of the varied response to protein metabolism by which an increase in albumin synthesis in response to heavy proteinuria (that could even normalize serum albumin), can be sustained by only some patients.

Hypoalbuminemia,\textsuperscript{11} is mostly a consequence of urinary loss. Heavy proteinuria will lead to negative nitrogen balance (usually measured by serum albumin). Increasing protein intake does not improve albumin metabolism, since the hemodynamic response to an increased intake is a rise in glomerular pressure, producing enhanced urine protein losses. On the other hand, a low-protein diet might reduce proteinuria, but also leads to a reduction in the synthesis rate of albumin; the latter may deteriorate the negative nitrogen balance.

Hypercoaguability is also common in patients with NS. Multiple proteins participating at the coagulation cascade, demonstrate altered levels in NS,\textsuperscript{12} Thromboembolic events increase markedly if the serum albumin concentration reaches <2g/dl. Important complications are renal vein thrombosis and pulmonary embolism.

Hyperlipidemia is regarded as an integral feature of NS, since it is frequently present in patients with heavy proteinuria. Serum cholesterol concentration >500 mg/dl is not at all uncommon, while serum triglyceride levels are highly variable.

\textsuperscript{11} White bands in the nails (as in Muehrcke’s bands) are a characteristic clinical sign.

\textsuperscript{12} The net effect is a hypercoagulable state.
Due to their dyslipidemia, patients with NS carry almost a 5-fold increased risk of coronary death.

Lipiduria is manifested by the presence of refractile accumulations of lipids in cellular debris and casts and mostly appears to be a consequence of the proteinuria and not of the plasma lipid abnormalities.

Among the different mechanisms accounted for the lipid abnormalities in NS (which might rapidly produce characteristic clinical signs, such as xanthelasmas), are the increased hepatic synthesis of LDL, VLDL and LP(a), the defective peripheral lipoprotein lipase activity, and the urinary losses of HDL.
References


Nutritional screening and assessment for the chronic kidney disease patient
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Learning outcomes

- Knowledge of How to Assess a Patient’s Nutritional Needs
- Knowledge of Different Methods of Assessment
- Knowledge of Nutritional Assessment Scoring Systems

Dietary intervention is of high importance for patients with chronic kidney disease (CKD), both for disease outcome and for prevention and treatment of malnutrition (over- and under-nutrition), which is quite common in renal patients. Therefore, a detailed nutritional assessment is of paramount importance in providing optimal care to individuals with CKD of all disease stages. Classically, three major lines of inquiry, i.e. dietary intake, biochemical measures, and body composition, are used to assess the protein-energy nutritional status; a fourth category of nutritional assessment, composite indices, that include a combination of assessment measures within these categories, are also utilized and will be reviewed in this chapter.

2.1 Dietary intake assessment

During the nutritional assessment of patients with CKD, dieticians should focus on the quality and quantity of dietary intake, in order to assess the adequacy of energy and nutrient intakes, as well as to rule out the excess intake of nutrients that may harm renal function (e.g. protein, sodium, phosphorus and potassium). With regard to dietary assessment, the most commonly used methods include dietary recalls over short periods of time (e.g. 24 hrs), food records with or without supplementary dietary interviews conducted over short periods.
Nutritional screening and assessment for the chronic kidney disease patient

of time (e.g. 3 to 7 days), and long term (weeks to months) food histories in the form of food frequency questionnaires (FFQs)\textsuperscript{1,2}. Additionally, in hemodialysis (HD) patients (particularly those with little or no residual renal function) the extent of the rise in serum urea between two consecutive HD treatments allows protein intake to be estimated\textsuperscript{3}. In particular, most maintenance HD patients cannot excrete a significant amount of urinary nitrogen. Hence, the rate of increase in serum urea nitrogen between two consecutive HD sessions reflects dietary nitrogen intake, provided that the individual is not in substantially negative or positive nitrogen balance. This indirect but conveniently available measure of protein intake is referred to as the urea kinetic and is based on protein equivalent of total nitrogen appearance (PNA) or protein catabolic rate (PCR), which is usually normalized (n) for the patient’s body weight or an estimate of the volume of distribution of urea; hence the term, nPNA or nPCR\textsuperscript{4}. Modifications of this method can be used in non-dialysis CKD patients and chronic peritoneal dialysis patients as well. Strengths and limitations of the aforementioned methods have been recently reviewed and are presented in Table 2.1\textsuperscript{2}. Practically, no method is ideal and the combination of more than one is usually recommended. Apart from information collected through the above mentioned methods, dieticians should interview the patient and review medical records to assess common factors that are usually related to protein-energy malnutrition in CKD (Table 2.2)\textsuperscript{5}.

2.2 Anthropometric and body composition assessment

2.2.1 Body weight and Body mass Index

Anthropometric methods are practical, cost-effective techniques that describe body size and can identify levels of fatness and leanness\textsuperscript{6}. Stature and weight provide a general description of body size and mass. Weight is also a measure of total energy stores, leanness, and obesity; however it is affected by hydration status and fluid retention. Percentage of weight
change is a useful marker of adequate energy intake, however in patients with fluid retention this is not applicable. On the contrary, dry body weight changes in HD patients can provide useful information for nutritional assessment. Most patients with high body weight tend to have high amounts of body fat. Weight combined with stature squared in the body mass index (BMI) is a descriptive index of leanness or obesity. There are extensive national reference data available for BMI, and BMI values greater than 25 kg/m² are associated with increased morbidity and mortality in the general population.

Table 2.1. Strengths and limitations of dietary assessment methods for CKD patients².

<table>
<thead>
<tr>
<th></th>
<th>Strengths</th>
<th>Limitations</th>
</tr>
</thead>
<tbody>
<tr>
<td>24-hrs recall</td>
<td>• Convenience</td>
<td>• Reliance on patient’s memory</td>
</tr>
<tr>
<td></td>
<td>• Rapidity</td>
<td>• Lack of ability to represent a longer period esp. in dialysis patients whose food intake pattern on dialysis and non-dialysis days may be significantly different</td>
</tr>
<tr>
<td></td>
<td>• No need to provide or prepare records</td>
<td>• Reliance on patient’s cooperation and communication ability</td>
</tr>
<tr>
<td></td>
<td>• Possibility to be performed over the phone</td>
<td>• Reliance on interviewer’s skills, comprehensiveness and prompts</td>
</tr>
<tr>
<td></td>
<td>• Ability to evaluate the validity of other assessment tools</td>
<td>• Reliance on interviewer’s familiarity with background food culture</td>
</tr>
<tr>
<td>Diet records and diaries</td>
<td>• Expecting real time recording on the ingested food and the extended</td>
<td>• Reliance on compliance of the patients with instructions</td>
</tr>
<tr>
<td></td>
<td>period of time beyond 24 hrs.</td>
<td>• Possibility of missing or inaccurate recordings of food items</td>
</tr>
<tr>
<td></td>
<td>• Ability to evaluate the validity of other assessment tools</td>
<td>• Lack of capturing seasonal or other cycling variations in dietary pattern.</td>
</tr>
</tbody>
</table>
### Nutritional screening and assessment for the chronic kidney disease patient

<table>
<thead>
<tr>
<th>Strengths</th>
<th>Limitations</th>
</tr>
</thead>
<tbody>
<tr>
<td>• Convenience, especially if self-administered for use in large populations</td>
<td>• Under or even over estimation of nutrient intake at individual level</td>
</tr>
<tr>
<td>• Large temporal catchment (months to years) hence less sensitivity to seasonal variations</td>
<td>• Lack of accuracy to use to assess the amounts or adequacy of dietary intakes of individuals or small groups of people</td>
</tr>
<tr>
<td>• Relatively high reliability in ranking subjects across each food item</td>
<td>• Inadequate coverage to include all available food items</td>
</tr>
<tr>
<td>• Feasibility and low cost for large scale epidemiologic studies</td>
<td>• Inclusion of diverse varieties of a given food under one single food item question, and hence, failure to capture significant differences among different food subtypes</td>
</tr>
</tbody>
</table>

- **Food screeners and FFQ**

- **Urea dynamic calculated protein intake (nPCR or nPNA)**

  • No need for direct dietary assessment

  • Lack of ability to assess other nutrients intakes other than protein

  • Variations due to non-dietary factors related to urea generation such as in catabolic or anabolic states

  • Inaccurate or misleading if residual renal function is not clear.

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*FFQ: food frequency questionnaire; nPNA: normalized protein equivalent of total nitrogen appearance; nPCR: normalized protein catabolic rate.*

In CKD, BMI is known to predict the clinical outcome of disease. BMI is dependent on muscle and fat mass and total body water content, however weight changes over a period of time can still be of clinical value and more so in the case of unplanned weight loss over a short period of time. When assessing BMI it should be remembered that a higher percentage of muscle mass is seen in young people, athletes and body builders and a higher percentage of fat mass in less mobile and elderly patients. Obesity is not uncommon among dialysis patients; however, sarcopenia (loss of muscle mass) can cause a
normal-weight person to become “obese” because of a high percentage of body fat, whereas a greater BMI may also reflect higher amounts of muscle mass and therefore better disease outcomes. Several studies have shown that a BMI of 23 kg/m² and higher reduces the risk of morbidity and mortality in HD patients.1

Table 2.2. Conditions related to protein-energy malnutrition as a cause of wasting syndrome in CKD patients.

<table>
<thead>
<tr>
<th>Inadequate nutrient intake</th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td>• Anorexia</td>
<td></td>
</tr>
<tr>
<td>o Due to uraemic toxicity, altered taste sensation</td>
<td></td>
</tr>
<tr>
<td>o Due to impaired gastric emptying</td>
<td></td>
</tr>
<tr>
<td>o Due to inflammation with or without comorbid conditionsa</td>
<td></td>
</tr>
<tr>
<td>o Due to emotional and/or psychological disorders</td>
<td></td>
</tr>
<tr>
<td>• Dietary restrictions</td>
<td></td>
</tr>
<tr>
<td>o Prescribed restrictions: low-potassium, low-phosphate regimens</td>
<td></td>
</tr>
<tr>
<td>o Social constraints: poverty, inadequate dietary support</td>
<td></td>
</tr>
<tr>
<td>o Physical incapacity: inability to acquire or prepare food or to eat</td>
<td></td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Nutrient losses during dialysis</th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td>• Loss through haemodialysis membrane into haemodialysate</td>
<td></td>
</tr>
<tr>
<td>• Loss into peritoneal dialysate</td>
<td></td>
</tr>
<tr>
<td>• Hyercatabolism due to comorbid illnesses</td>
<td></td>
</tr>
<tr>
<td>o Cardiovascular diseasesa</td>
<td></td>
</tr>
<tr>
<td>o Diabetic complications</td>
<td></td>
</tr>
<tr>
<td>o Infection and/or sepsisa</td>
<td></td>
</tr>
<tr>
<td>o Other comorbid conditionsa</td>
<td></td>
</tr>
<tr>
<td>• Hyercatabolism associated with dialysis treatment</td>
<td></td>
</tr>
<tr>
<td>o Negative protein balance</td>
<td></td>
</tr>
<tr>
<td>o Negative energy balance</td>
<td></td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Endocrine disorders of uraemia</th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td>• Resistance to insulin</td>
<td></td>
</tr>
</tbody>
</table>

| Acidaemia with metabolic acidosis |   |

| Concurrent nutrient loss with frequent blood losses |   |

* These factors may also be associated with inflammation
2.2.2 Skinfolds

The triceps and subscapular skinfolds measure subcutaneous fat thickness on the limbs and trunk, and abdominal circumference is an index of internal adipose tissue. Mid-arm circumference (MAC) and triceps skinfold thickness are combined to calculate mid-arm muscle circumference (MAMC) and area (MAMA). MAMC and MAMA are related to levels of protein stores or are used as a marker for fat-free mass (FFM) in CKD patients. Calf circumference is an indirect measure of muscle mass.

For patients on HD, all the above anthropometric variables should be measured immediately after a dialysis session and on the non-fistula arm. The main limitation of anthropometric methods is that the standards used for these measurements are mainly derived from cohorts of healthy individuals. Because of these restricted databases and the lack of correction for age, hydration status, and physical activity, the use of these standards to identify protein or fat depletion is problematic. However, recently referenced anthropometric data have been created, taking into account patients’ age, gender, race, presence of diabetes, and length of time on dialysis therapy. Although this method was based on a selected sample of patients participating in the HEMO study (with dry body weight <85 kg), the anthropometric standards derived from this study seem to be more appropriate for CKD patients than the ones based on healthy populations.

2.2.3 Bioelectrical Impedence Analysis and Dual X-ray Absorptiometry

The assessment of body composition, or of somatic protein stores, involves the measurement of different body compartments (water, fat, bone, muscle, and visceral organs). The most widely used body composition methods in clinical practice are bioelectrical impedance analysis (BIA) and
Dual X-ray absorptiometry (DXA). BIA estimates body-fluid volumes by measuring the resistance to a high frequency, low amplitude alternating electric current (50 kHz at 500–800 mA). BIA has been mostly used for fluid management but has been increasingly used for nutritional assessment. Advantages of BIA include safety, general acceptance by the patients, ease of use, and minimal inter-operator variability. Disadvantages include inaccurate estimations of fat and fat free mass in situations of variable fluid status. For pre-dialysis patients, BIA should be performed with caution, whereas for accurate measurements of FFM in HD patients, BIA should be conducted when the patient is under stable fluid status, without major shifts among different body pools. For this reason it has been suggested that BIA can be appropriately performed at anytime during the 2 hours after a HD session. DEXA is a 3-compartment model initially designed for assessing bone density and later adapted to quantify soft-tissues (fat and fat free mass). It has the advantage of measuring not only whole-body but also segmental body composition (head, trunk, arms, and legs). Hydration status of the patient must be considered for each evaluation, although studies performing DEXA before and after dialysis suggest that there are minimal effects of body-water changes on fat mass and bone measurements. Main advantages of DEXA include the minor discomfort to the patient (lying supine for 6–10 minutes) and minimal radiation exposure, which allows safe serial measurements. It also has excellent precision (1.5% variability or less), suitable for all ages and a reasonably wide range in body sizes, quickness to perform, and the sensitivity for detecting small changes in body composition. The main disadvantages of DEXA include the high cost of the equipment and the need for a specialized operator, which limits its application in every day clinical practice.
2.2.4 Measurements of muscle function

Just as measuring body composition offers a qualitative aspect of nutritional status, muscle function represents a dynamic indicator of muscle mass. Measurement of muscle function as an indicator of functional as well as nutritional status has therefore gained considerable attention in the past years. Hand-grip strength (HGS) is a validated and the most feasible bedside method of voluntary muscle strength evaluation and has been described as a useful tool in assessing muscle function because it is a noninvasive, rapid, objective, and inexpensive procedure. This technique has been related to mortality and complications in the elderly, as well as in surgical and stroke patients and patients with gastrointestinal or pulmonary diseases\textsuperscript{11}. Recently, a systematic review of the literature on the use of HGS as a parameter for nutritional assessment and a prognostic marker in patients on dialysis was published\textsuperscript{12} and concluded that patients on HD presented a high prevalence of muscle function loss assessed by HGS. Early detection of muscle function loss, even in the presence of overweight, may allow the implementation of appropriate therapeutic measures. Moreover, HGS may be used as a reliable nutritional marker during HD because it is not influenced by dialysis variables.

2.3 Biochemical markers

Biochemical parameters have been widely used to assess and monitor nutritional status in several different clinical conditions, including CKD. The size of the visceral protein pool, which is expected to be lower in depleted states, can be estimated by the measurement of circulating proteins in the blood, such as serum albumin, serum prealbumin, serum transferrin, and other less available markers such as serum retinol-binding protein and insulin-like growth factor-I. A commonly used laboratory marker to estimate the somatic protein pool is serum creatinine. Additionally, biochemical markers such as blood urea nitrogen (BUN) and serum cholesterol can be useful
Nutritional Care for Adults with Chronic Kidney Disease

in the interpretation of dietary intake. However, the analysis of these biochemical markers in the CKD population should be performed with caution because they can be modified by conditions inherent to the kidney disease itself and in some instances not be related to the nutritional status\textsuperscript{7}. According to KDOQI Guidelines for CKD Care\textsuperscript{13} serum albumin $<4$ g/dl, prealbumin $<30$mg/dl, predialysis creatinine $<10$ mg/dl and cholesterol $<150$ mg/dl are clinically useful markers indicating high possibility of protein-energy under-nutrition in CKD patients, whereas the International Society of Renal Nutrition and Metabolism recently included serum albumin $<3.8$ g/dl as one of three biochemical diagnostic criteria for protein–energy wasting (among transthyretin and cholesterol levels)\textsuperscript{14}. However, since albumin levels also reflect several non-nutritional factors which are frequently present in CKD patients, including inflammation and infection, urinary and dialysate losses as well as hydration status, serum albumin validity as a clinically useful measure of protein energy under-nutrition has been recently questioned by Friedman and Fadem\textsuperscript{15}. They support that a plethora of corroborative clinical evidence in the general population and in patients with CKD demonstrates that serum albumin is an insensitive indicator of malnutrition, except possibly the very rare circumstance of kwashiorkor-like states. In the same article authors suggest a clinical algorithm for interpreting serum albumin, which is presented in Figure 2.1. On the other hand, a non-classical biochemical marker for which increasing data emerges supporting its use in CKD nutritional assessment, is total lymphocyte count that has been associated with mortality and hospitalization especially in HD patients\textsuperscript{16-18}. 
2.4 Nutritional assessment scoring systems

Subjective Global Assessment (SGA) is a clinical method for evaluating nutritional status at a broader perspective, including history, symptoms, and physical parameters\(^1\). SGA is useful to identify malnourished patients at increased risk for medical complications and who will presumably benefit from nutrition therapy. The basis of this assessment is to determine whether nutrient assimilation has been restricted because of decreased...
food intake, maldigestion, or malabsorption, whether any effects of malnutrition on organ function and body composition have occurred, and whether the patient’s disease process influences nutrient requirements. The history used in the SGA focuses on 5 areas: body weight, dietary intake, gastrointestinal symptoms, functional capacity, and disease state. In addition to the medical history, there is also a physical examination, which is noted as normal, mild, moderate, or severe alterations and evaluates loss of subcutaneous fat, muscle wasting, and the presence of edema, based on subjective evaluation of the examiner. The findings of the history and physical examination are used to categorize patients as being well nourished (A), moderately (or suspected of being) malnourished (B) or severely malnourished (C). Based on SGA, other tools have been also applied in CKD patients’ nutritional assessment and are presented in Table 2.3. SGA, along with modified versions, has shown clinical and/or predictive validity in CKD for both dialysis and non-dialysis populations. When considering which tool is best to apply, it is important to consider the purpose of the nutritional assessment. Tools displaying diagnostic evidence indicate the ability to distinguish between other known measures of nutrition status, by clinical and/or criterion validity. Prognostic evidence refers to the ability of the tool to predict negative outcomes, such as morbidity and mortality, achieved via predictive validity. In general, Subjective Global Assessment (A vs. B and C) is a valid method for assessing the presence of malnutrition for diagnostic and prognostic purposes. Continuous scores may improve the clinician’s ability to assess small changes in nutrition status, as an ideal nutritional marker should reliably measure change in nutrition status, not just predict clinically important outcomes. In combination, these tools can provide a comprehensive assessment for both clinical and research settings.
Table 3. Nutritional assessment scoring systems used in chronic kidney disease patients

<table>
<thead>
<tr>
<th>Tool</th>
<th>Method</th>
<th>Modification from SGA</th>
<th>Advantages</th>
<th>Disadvantages</th>
</tr>
</thead>
<tbody>
<tr>
<td>SGA</td>
<td>Rating A, B, C</td>
<td></td>
<td>Appears to have the best diagnostic evidence to adequately detect the presence of abnormal nutrition status and prognostic evidence to predict poor outcome.</td>
<td>Agreements between the ratings of the SGA categories appear not to be very sensitive.</td>
</tr>
<tr>
<td>Retrospective mSGA</td>
<td>Rating A, B, C</td>
<td>Retrospective ‘self-rating’ on A, B, C scale</td>
<td>Conducted as a survey (self-reported)</td>
<td>Relies on self-report and caregiver’s physical assessment</td>
</tr>
<tr>
<td>4-point SGA</td>
<td>Rating 1–4</td>
<td>Expands the ‘B’ category to two. Ratings &gt;2 represent malnutrition</td>
<td>May delineate poor nutrition status</td>
<td>Similar issues to original, difficult to note changes over time</td>
</tr>
<tr>
<td><strong>Nutritional Care for Adults with Chronic Kidney Disease</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>---</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th><strong>Tool</strong></th>
<th><strong>Method</strong></th>
<th><strong>Modification from SGA</strong></th>
<th><strong>Advantages</strong></th>
<th><strong>Disadvantages</strong></th>
</tr>
</thead>
<tbody>
<tr>
<td>7-point SGA</td>
<td>Rating 7 – 1</td>
<td>Expands the three categories of the original SGA, to seven on a Likert-type scale</td>
<td>May delineate levels of nutrition status</td>
<td>May increase inter-observer variation</td>
</tr>
<tr>
<td>Dialysis Malnutrition Score (DMS)</td>
<td>Scored 7–35</td>
<td>Scores seven components of the SGA as 1 (normal) to 5 (very severe)</td>
<td>Scores so less subjectivity</td>
<td>Allocation of scores not based on evidence</td>
</tr>
<tr>
<td>Malnutrition Inflammation Score (MIS)</td>
<td>Scored 0–30</td>
<td>Ten components, the DMS with BMI, serum albumin and total-iron binding capacity, scored according to severity 0 (normal) to 3 (very severe)</td>
<td>Includes objective categories—less reliance on subjectivity</td>
<td>Requires biochemistry (albumin and iron studies), and weight/height measures for BMI</td>
</tr>
<tr>
<td>Patient-Generated Subjective Global Assessment (PG-SGA)</td>
<td>Scored 0–50 and A, B, C</td>
<td>Provides a numerical score, dependent on the impact of each SGA component on nutrition status</td>
<td>Patient completes medical history, scored so less subjective</td>
<td>May require more patient input</td>
</tr>
</tbody>
</table>

**Expands the three categories of the original SGA, to seven on a Likert-type scale**

**May delineate levels of nutrition status**

**Scores seven components of the SGA as 1 (normal) to 5 (very severe)**

**Scores so less subjectivity**

**Ten components, the DMS with BMI, serum albumin and total-iron binding capacity, scored according to severity 0 (normal) to 3 (very severe)**

**Includes objective categories—less reliance on subjectivity**

**Requires biochemistry (albumin and iron studies), and weight/height measures for BMI**

**Provides a numerical score, dependent on the impact of each SGA component on nutrition status**

**Patient completes medical history, scored so less subjective**

**May require more patient input**
Nutritional screening and assessment for the chronic kidney disease patient

References


Nutritional screening and assessment for the chronic kidney disease patient
Nutrition & Hypertension
3.1 Introduction

Hypertension is defined by the World Health Organisation and International Society of Hypertension (WHO/ISH) as persistent blood pressure (BP) readings ≥140 mmHg for systolic and ≥90 mmHg for diastolic\(^1\). The WHO/ISH recommended BP targets for patients at high cardiovascular risk; those with hypertension, chronic kidney disease, smoking, high cholesterol, obesity and physical inactivity. The recommendations are for a BP<130 mmHg for systolic and <80 mmHg for diastolic, which are based on clinical trial evidence and epidemiological data.

Hypertension is usually divided into two categories: primary (or essential) and secondary. Primary or essential hypertension is of unknown cause and responsible for at least 90% of cases\(^2\). Secondary hypertension develops due to an underlying primary disease, such as kidney disease.

3.2 Prevalence

Hypertension is an important public-health challenge worldwide\(^3\). The estimated total number of adults with hypertension in 2000 was 972 million; 333 million in economically developed countries and 639 million in economically developing countries\(^4\). This prevalence is predicted to increase by 2025 to about 60% when a total of 1.56 billion people globally may be affected\(^4\). Worldwide, hypertension is estimated to cause 7.1 million premature deaths and 4.5% of the total disease burden\(^5\). Additionally, secondary hypertension is a common feature of CKD patients. The NEOERICA study showed a crude odds ratio of 3.45 (95% CI 3.23 – 3.57) for hypertension in patients with GFR <60 ml/min/1.73 m\(^2\)\(^6\). This prevalence increases as renal function decreases, thus, in patients with GFR <30 ml/min/1.73 m\(^2\) the prevalence is high at 87.8%\(^7\). Furthermore, uncontrolled hypertension in renal disease has
been associated with an increased risk of rapid progressive renal failure\textsuperscript{8,9}. The Multiple Risk Factor Intervention Trial (MRFIT) identified hypertension as a significant risk factor for the progression of CKD\textsuperscript{10}. These results were corroborated by an Italian study showing that patients with raised blood pressure exhibited an accelerated loss of renal function over a 30-month follow-up period, compared to those with lower BP\textsuperscript{11}. Recently, a prospective Japanese 17-year follow-up study of 98,759 subjects showed that hypertension (before and after adjustment for age and body mass index) was an independent risk factor for CKD progression\textsuperscript{12}. A large USA prospective cohort study of 23,534 men and women supported these findings, demonstrating a strong graded increased risk for CKD progression with the increments in systolic blood pressure\textsuperscript{8}.

### 3.3 Hypertension management

Hypertension treatment is a high priority; this is acknowledged by health expert committees that developed guidelines and recommendations to control high blood pressure.

The USA Joint National Committee (JNC) on Prevention, Detection and Treatment of High Blood Pressure\textsuperscript{13} published recommendations for the targets and treatment of high blood pressure. The recommended treatment is lifestyle modification and medication (Table 3.1). Lifestyle modification recommendations are evidence-based and include adoption of a healthy diet, exercise and maintenance of an optimal weight (Figure 3.1).
Table 3.1 Adapted lifestyle modifications recommendations by Joint National Committee on prevention, detection and treatment of high blood pressure on the management of hypertension

<table>
<thead>
<tr>
<th>Recommendations</th>
<th>Actions required</th>
</tr>
</thead>
<tbody>
<tr>
<td>Weight reduction</td>
<td>Maintain normal body weight (body mass index: 18.5–24.9 kg/m²)</td>
</tr>
<tr>
<td>DASH eating plan</td>
<td>Consume a diet rich in fruits, vegetables, and low-fat dairy products with a reduced content of saturated and total fat</td>
</tr>
<tr>
<td>Reduction in salt (sodium) intake</td>
<td>Reduce dietary sodium intake to no more than 100 mmol/day (6 g salt)</td>
</tr>
<tr>
<td>Physical activity</td>
<td>Engage in regular aerobic physical activity such as brisk walking (at least 30 min per day, most days of the week)</td>
</tr>
<tr>
<td>Moderation of daily alcohol consumption</td>
<td>Limit consumption to no more than 2 drinks (e.g., 24 oz beer, 10 oz wine, or 3 oz 80-proof whiskey) per day in most men, and to no more than 1 drink per day in women and lighter weight persons</td>
</tr>
</tbody>
</table>

Source: Joint National Committee on Prevention, Detection and Treatment of High Blood Pressure.¹³

Figure 3.1 Treatment effect of lifestyle modification on Systolic Blood Pressure

<table>
<thead>
<tr>
<th>Lifestyle Modification to Manage Hypertension*</th>
<th>Approximate Systolic BP Reduction Range</th>
</tr>
</thead>
<tbody>
<tr>
<td>Weight</td>
<td>Maintain normal body weight (BMI: 18.5-24.9) 5-20 mmHg/ 10-kg weight loss</td>
</tr>
<tr>
<td>DASH diet</td>
<td>Consume diet rich in fruits, vegetables, lower – fat milk products 8-14 mmHg</td>
</tr>
<tr>
<td>Dietary Sodium</td>
<td>Reduce sodium intake to no more than 2400 mg sodium or 6000 mg salt (sodium chloride) 2-8 mmHg</td>
</tr>
<tr>
<td>Physical activity</td>
<td>Engage in regular aerobic physical activity at least 30 minutes/ day most days of the week 4-9 mmHg</td>
</tr>
<tr>
<td>Alcohol</td>
<td>Limit consumption to ≤2 drinks/ day for men and ≤1 drink/ day for women 2-4 mmHg</td>
</tr>
</tbody>
</table>

Abbreviations: BMI: Body Mass Index, BP: Blood pressure, DASH: Dietary Approaches to Stop Hypertension

*For overall cardiovascular risk reduction, stop smoking. The effects of implementing these modifications are dose and time dependent and could be higher for some individuals.

Adopted from Chobanian el al⁶

Source: Joint National Committee on Prevention, Detection and Treatment of High Blood Pressure.¹³
The European Society of Hypertension and the European Society Cardiology (ESH/ESC)\textsuperscript{14} have combined forces and produced guidelines for the management of arterial hypertension (Table 3.2).

In the UK, the British Hypertension Society guidelines\textsuperscript{15} recommend lifestyle measures for up to six months.

Raised BP is commonly seen as part of the ageing process in the Western countries and has also been associated with a high dietary salt intake.

\textit{Table 3.2. ESH/ESC guidelines for the management of hypertension}

<table>
<thead>
<tr>
<th>Blood Pressure</th>
<th>Other risk factors OD or disease</th>
<th>No other risk factors</th>
<th>1-2 risk factors</th>
<th>≥3 risk factors</th>
<th>Diabetes</th>
<th>Established CV or renal disease</th>
</tr>
</thead>
<tbody>
<tr>
<td>Normal</td>
<td>SBP 120-129 or DBP 80-84</td>
<td>No BP interventions</td>
<td>Lifestyle changes</td>
<td>Lifestyle changes</td>
<td>Lifestyle changes</td>
<td>Lifestyle changes + drug treatment</td>
</tr>
<tr>
<td>High normal</td>
<td>SBP 130-139 or DBP 85-89</td>
<td>Lifestyle changes</td>
<td>Lifestyle changes</td>
<td>Lifestyle changes + drug treatment</td>
<td>Drug treatment</td>
<td>Drug treatment</td>
</tr>
<tr>
<td>Grade 1 HT</td>
<td>SBP 140-159 or DBP 90-99</td>
<td>Lifestyle changes for several months then drug treatment if BP uncontrolled</td>
<td>Lifestyle changes for several months then drug treatment if BP uncontrolled</td>
<td>Lifestyle changes</td>
<td>Immediate drug treatment</td>
<td>Immediate drug treatment</td>
</tr>
<tr>
<td>Grade 1 HT</td>
<td>SBP 160-179 or DBP 100-109</td>
<td>Lifestyle changes for several months then drug treatment if BP uncontrolled</td>
<td>Lifestyle changes for several months then drug treatment if BP uncontrolled</td>
<td>Lifestyle changes + Immediate drug treatment</td>
<td>Drug treatment</td>
<td>Drug treatment</td>
</tr>
<tr>
<td>Grade 1 HT</td>
<td>SBP ≥180 or DBP ≥110</td>
<td>Lifestyle changes</td>
<td>Lifestyle changes</td>
<td>Lifestyle changes</td>
<td>Drug treatment</td>
<td>Drug treatment</td>
</tr>
<tr>
<td>Grade 1 HT</td>
<td>SBP ≥180 or DBP ≥110</td>
<td>Lifestyle changes</td>
<td>Lifestyle changes</td>
<td>Lifestyle changes</td>
<td>Immediate drug treatment</td>
<td>Immediate drug treatment</td>
</tr>
</tbody>
</table>

\textit{Source: ESH/ESC hypertension guidelines.}\textsuperscript{14}
Table 3.3 Lifestyle change recommendations by the BHS, 2004 guidelines

<table>
<thead>
<tr>
<th>Changes</th>
<th>Lifestyle recommendations</th>
</tr>
</thead>
<tbody>
<tr>
<td>Weight reduction</td>
<td>Maintain normal weight for adults (body mass index 20-25 kg/m²)</td>
</tr>
<tr>
<td>Salt intake</td>
<td>Reduce salt to &lt; 100 mmol/day (&lt; 6g NaCl or &lt; 2.4 g sodium/day)</td>
</tr>
<tr>
<td>Alcohol</td>
<td>Limit alcohol consumption to ≤ 3 units/day for men and ≤ 2 units/day for women</td>
</tr>
<tr>
<td>Physical activity</td>
<td>Engage in regular aerobic physical exercise (brisk walking rather than weightlifting) for ≥ 30 minutes per day, ideally on most of days of the week but at least on three days of the week</td>
</tr>
<tr>
<td>Potassium</td>
<td>Consume at least five portions/day of fresh fruits and vegetables</td>
</tr>
</tbody>
</table>

Source: British Hypertension Society guidelines.

3.3.1 Dietary salt

Salt has always been important historically. Table salt comprises 40% sodium and 60% chloride, where sodium is the key agent for flavouring and preservation. Salt is lost from the body through the urine and sweat. Twenty four hours urine collection is the most accurate method to determine dietary salt intake, assuming the body is in balance for this mineral, as in the steady state oral intake must equal losses in the urine and sweat. Thus, urinary sodium excretion (mmol/24h) can be converted to provide an estimate of dietary sodium intake (g/day), as 1g salt contains 1mmol sodium.

The association between salt and health relates specifically to the sodium component. Nevertheless, the term salt is still often used, because the major dietary source of sodium is salt, sodium chloride. The adult dietary salt survey showed that approximately 90% of the sodium in our diet comes from sodium chloride. The other 10% comes from other forms of sodium in our diet, mainly used as additives in food processing.
Therefore, recommendations for a reduction in sodium intake should translate directly into a reduction in dietary salt.

### 3.3.2.1 Evidence for the link between salt and hypertension

Epidemiological and intervention studies provide very strong evidence of a causal link between high salt intake and high BP\textsuperscript{17-20}. Epidemiological studies have shown that salt intake has a key role in determining population blood pressure incremental change with age\textsuperscript{21,22}. A classic study in a primitive society was conducted over three decades ago with the Yanomamo Indians, an unacculturated tribe living isolated in the rain forest of Northern Brazil, who do not use salt in their diet\textsuperscript{23}. Control subjects were members of the expedition who ate a ‘normal’ diet with table salt ad libitum. Urinary 24hrs sodium (Na) excretion mean was 1mEq compared to 104 mEq for the control group, whilst potassium (K) excretion mean was 152 mEq compared to 38 mEq for the control group, giving the lowest Na:K ratio ever observed; 0.066 in the Yanomamo tribe. Blood pressure measurements in the Yanomamo Indians, in contrast to the usual finding in Western societies, did not rise with age and even seemed to decline slightly.

Epidemiological studies by Law et al.\textsuperscript{18,24,25} provide further evidence for a causal relationship between high dietary salt intake and the development of essential hypertension. In their first study, these authors analyzed the published epidemiological data from observational studies and trials to examine the relationship between blood pressure and sodium intake among populations\textsuperscript{24}. They showed that in undeveloped communities with very low sodium intake blood pressure did not rise with age and was on average 110/72 mmHg. Thus, a difference in sodium intake of 100 mmol/24hr was associated with an average difference in systolic blood pressure that ranged from 5 mmHg at age 15-19 years to 10 mmHg at age 60-69. In their second study, these authors analyzed observational data within populations to determine whether the
association between blood pressure and salt intake observed between different populations were consistent with those within populations (Law, Frost et al. 1991b). Collectively, the studies demonstrated a highly significant (p<0.001) association between blood pressure and sodium intake. In their final study, they carried out a meta-analysis of data from trials of salt reduction18. The data demonstrated that salt reduction lowers BP not only in individuals with high blood pressure, but also in individuals with normal BP, although the extent of reduction depends on the initial BP. Additionally, it suggests that simply by not adding salt in cooking or at the table and avoiding salty foods an average daily sodium reduction of 50mmol (3g salt) can be achieved. This reduction led to a decrease of an average 5 mmHg in BP of individuals at 50 years of age and to a drop of an average 7mmHg in individuals with hypertension. A further reduction of 100 mmol, achieved by avoiding many common processed foods, can lower systolic BP by an average of 14 mmHg in those with high blood pressure26.

The INTERSALT study, an epidemiological comparative study, was the first large international study to provide robust evidence for a causal relationship between salt and high blood pressure17. The INTERSALT study applied standardized methods across 52 canters worldwide in 32 countries with a total of 10,079 subjects. In their analysis they found a significant linear relationship between salt excretion and blood pressure. They also found that four isolated populations; two Brazilian Indian (Yanomamo and Xingu), one Papua New Guinean and one Kenyan, had the lowest urinary sodium excretion (0.2, 5.8, 26.8 and 51.3 mmol/24hr respectively) and had decreases or minor increases of systolic blood pressure with age (-0.079, 0.052, -0.14, and 0.20 mmHg/year, respectively) with an average systolic 95-110 and diastolic 61-68 mmHg.

The findings from epidemiological studies are confirmed by dose-response studies27,28. Dose-response study provides the most persuasive evidence about the effects of salt restriction
on BP management, but few have been performed to date. The DASH-Sodium trial is the largest reported dose-response study, testing the effects of three different sodium intakes separately in two distinct diets: the DASH (Dietary Approach to Stop Hypertension) diet and the control diet (typical USA diet) (Sacks, Svetkey et al. 2001). The sodium levels were high (150mmol/day), intermediate (100mmol/day) and low (50mmol/day). Mean urinary sodium excretions were respectively; 142, 107 and 65mmol/day during the high, intermediate and low sodium periods. The main key findings were; BP can be reduced both in the normal USA diet and the DASH diet by sodium reduction of 40mmol/day, and the DASH diet combined with low sodium intakes had greatest effect, lowering systolic BP by 11.5 and 7.1 mmHg in hypertensive and normotensive individuals respectively. MacGregor et al smaller study of three sodium intakes, one of the first dose-response studies, corroborates this progressive BP fall as salt intake is reduced28. The study showed that from a BP of 163/100 mmHg with a 200 mmol dietary sodium intake, BP fell to 155/95 mmHg and 147/91 mmHg when sodium intake was halved to 100mmol and 50mmol/day respectively.

A Cochrane meta-analysis was conducted to assess the effect of moderate salt reduction on blood pressure of hypertensive and normotensive individuals and investigate whether there is a dose-dependent response to salt reduction29. The study included 31 randomized trials conducted over 4 or more weeks; 20 trials in hypertensive individuals (n=802) and 11 trials in normotensive individuals (n=2220). The dose-response curve analysis showed that a reduction of 100 mmol/day (6g/day) in salt intake predicts a fall in BP of 7.2/3.8 mmHg and 3.6/1.7 mmHg for hypertensive and normotensive individuals respectively.

The DASH diet success led to its adaptation to suit the CKD population that could not cope with the very high levels of potassium due to reduced renal function (Table 3.4).
Furthermore, these robust hypertension trials have formed the basis of various national and international guidelines and recommendations on BP control in the CKD population (Table 3.5).

Table 3.4 Nutritional content of the DASH diet adapted for renal failure patients

<table>
<thead>
<tr>
<th>Nutrient</th>
<th>Stage of CKD</th>
</tr>
</thead>
<tbody>
<tr>
<td>Sodium (g/d)*</td>
<td>&lt;2.4</td>
</tr>
<tr>
<td>Total Fat (% of calories)</td>
<td>&lt;30</td>
</tr>
<tr>
<td>Saturated fat (%calories)</td>
<td>&lt;10</td>
</tr>
<tr>
<td>Cholesterol (mg/g)</td>
<td>&lt;200</td>
</tr>
<tr>
<td>Carbohydrate (%calories)**</td>
<td>50-60</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Nutrient</th>
<th>Stages 1-2</th>
<th>Stages 3-4</th>
</tr>
</thead>
<tbody>
<tr>
<td>Protein (g/kg, % of calories)</td>
<td>1.4 (~18)</td>
<td>0.6-0.8 (~10)</td>
</tr>
<tr>
<td>Phosphorus (g/d)</td>
<td>1.7</td>
<td>0.8-1.0</td>
</tr>
<tr>
<td>Potassium (g/d)</td>
<td>&gt;4</td>
<td>2-4</td>
</tr>
</tbody>
</table>

*Not recommended for patients with salt wasting
**Adjust so total calories from protein, fat and carbohydrate is 100%

http://www.kidney.org/professionals/KDOQI/guidelines_ckd/p4_class_g1.htm

Table 3.5 Treatment targets for hypertension management in CKD

<table>
<thead>
<tr>
<th>Strategies</th>
<th>Recommendations</th>
</tr>
</thead>
<tbody>
<tr>
<td>Diet</td>
<td>DASH diet for CKD stages 1-2, modified sodium, protein, phosphorus and potassium diet for CKD 3-4</td>
</tr>
<tr>
<td>Others</td>
<td>Weight control, exercise, moderation of alcohol intake, smoking cessation</td>
</tr>
<tr>
<td>Initiation</td>
<td>For diabetic or nondiabetic kidney diseases and spot urine total protein to creatinine ratio ≥2200mg/g, or blood pressure ≥130/80mmHg</td>
</tr>
<tr>
<td>Selection of “preferred agents”</td>
<td>ACE inhibitors and ARBs for diabetic kidney disease and nondiabetic kidney disease with spot urine total protein-to-creatinine ratio ≥200mg/g</td>
</tr>
<tr>
<td>Dose of “preferred agents”</td>
<td>Moderate to high doses, as used in controlled trials</td>
</tr>
<tr>
<td>Anti-hypertensive agents</td>
<td>Diuretics first for diabetic and nondiabetic kidney disease. Others as necessary to reduce CVD risk and reach target blood pressure</td>
</tr>
<tr>
<td>Additional agents</td>
<td></td>
</tr>
<tr>
<td>Therapeutic targets</td>
<td>Target level</td>
</tr>
<tr>
<td>Blood pressure</td>
<td>&lt;130/80mmHg (possibly lower if spot urine total protein-to-creatinine ratio &gt;500-1000 mg/g)</td>
</tr>
<tr>
<td>Consider urine protein excretion</td>
<td>Spot urine total protein-to-creatinine ratio &lt;500-1000 mg/g</td>
</tr>
<tr>
<td>Other CVD risk factors</td>
<td>As appropriate for CVD risk reduction (lipid lowering therapy, management of diabetes, etc)</td>
</tr>
</tbody>
</table>

http://www.kidney.org/professionals/KDOQI/guidelines_ckd/p4_class_g1.htm
3.3.2 Other dietary and lifestyle approaches to treat hypertension

3.3.2.1 Increased potassium intake

There is a large body of evidence showing an association, albeit at times inconsistent, between high potassium intake and reduced BP. Supporting this association, three meta-analyses of clinical trials documented a significant inverse relationship between potassium intake and BP control in hypertensive individuals. The first meta-analysis by Cappuccio & MacGregor\(^{30}\) showed that a median potassium increment of 63 mmol/24hr resulted in a BP drop of 6/3 mmHg. This was followed by Whelton et al.\(^{31}\) meta-analysis where a median potassium increment of 50 mmol/24hr was associated with a reduction in blood pressure of 3/2 mmHg. The most recent meta-analysis carried out by Geleijnse et al.\(^{32}\) showed a BP reduction of 2.4/1.6 mmHg from a median increase of 44 mmol/24hr in potassium intake. In contrast, a recent Cochrane review concluded that potassium supplementation had no statistical significant effect on blood pressure\(^{33}\). The reviewers argued that the evidence was not conclusive in view of heterogeneity between trials, the small number of participants and only two high quality trials of short duration and follow-up. Despite not reaching statistical significance, the studies in the review showed that the BP lowering effects of potassium were greater in hypertensive individuals and more pronounced in those eating a high dietary sodium intake. The beneficial effects of potassium were also shown in the DASH trial, where participants increased fruit and vegetable consumption making a total of 120mmol/day of potassium intake\(^{27}\). In addition, this study demonstrated that the effects of potassium on BP depended on the levels of salt intake; increased dietary potassium has a greater effect in the context of a higher salt intake. It should be noted that 120mmol/day of potassium is a very high level for people in industrialized nations that has a high consumption of processed foods thereby ingesting 30 to
70mmol of potassium per day and as much as 100 to 400mmol of sodium per day\textsuperscript{34}.

### 3.3.2.2 Weight loss

Numerous clinical studies have examined the relationship between weight reduction and decrease in blood pressure. A recent meta-analysis of 25 studies concluded that for every 1kg weight loss there is an approximate 1 mmHg drop in blood pressure\textsuperscript{35}. This report also stated that blood pressure reduction was accomplished even without individuals attaining a healthy weight status. The Trial of Hypertension Prevention (TOHP) supports these studies, demonstrating that a 2kg weight loss resulted in a BP drop of 3.7/2.7 mmHg\textsuperscript{36}. It is clear in the literature that weight loss, regardless how it is obtained, results in clinically significant reductions in blood pressure and has the attractiveness of its multipurpose healthy benefits\textsuperscript{37}.

### 3.3.2.3 Exercise

Physical inactivity has been shown to be a risk for high blood pressure and for cardiovascular disease\textsuperscript{36}. Conversely, increased physical activity has been associated to reductions in blood pressure. This was demonstrated by Murphy et al.\textsuperscript{38} meta-analysis of 1128 participants on a walking program for a mean length of 34.9 weeks; BP reduced by 0.8\% and 2\% for systolic and diastolic respectively. In addition, a reduction in body weight and BMI of 1.4\% and 1.1\% respectively was also observed in this meta-analysis. Similar results have been demonstrated in other meta-analysis\textsuperscript{39,40}. The meta-analysis conducted by Whelton et al. assessed the effect of aerobic exercise on BP in 54 clinical trials which involved 2419 participants\textsuperscript{40}. The results showed a reduction of 3.8/2.6 mmHg in BP in hypertensive and normotensive participants that were either overweight or normal weight. In this meta-analysis the overall weight change was (-0.42Kg) neither
statistically nor clinically significant. This clearly demonstrates that the effects of aerobic exercise on blood pressure control may be independent of change in body weight. Additionally, aerobic exercise had a slightly greater effect on hypertensive (-4.94/-3.73 mmHg) than normotensive (-4.04/-2.33 mmHg) participants. The effect was higher in high intensity aerobic activity (cycling) when compared to walking; -5.58 vs -2.59 mmHg systolic blood pressure respectively for each type of activity. The positive effect of walking has been illustrated by Kelley et al. (Kelley, Kelley et al. 2001) meta-analysis of 16 studies using walking as the only intervention. The results showed a reduction of 2% for both systolic and diastolic resting blood pressure; initial resting BP was good at a mean of 128/80 mmHg. Once again, no statistically significant changes were observed in body weight, body mass index or percentage of body fat. Given these results and the other health benefits accrued from increased physical activity, it is reasonable to suggest that all adults should be encouraged to be physically active.
References


Nutritional Management of the Nephrotic Syndrome
4.1 Definition and Aetiology

Glomerular diseases are among the most common renal pathologies leading frequently to end-stage renal disease. Clinical disease can be divided into five different groups, the features of which are determined by the underlying pathophysiology. One of these five clinical syndromes is the nephrotic syndrome.

Nephrotic syndrome is defined by the association of a proteinuria higher than 3.5 g/24 hours, hypoalbuminaemia, oedema and dyslipidaemia. The prevalence of this syndrome is high, mainly due to its frequency in diabetic patients. The aetiological causes of nephrotic syndrome are however miscellaneous, ranging from primary renal diseases to systemic illnesses with various histopathological presentations (Tables 4.1 and 4.2). The renal disease revealed by nephrotic syndrome must be precisely characterised, since although many therapeutic strategies are common to all nephrotic patients, specific treatments exist and must be evaluated in each case. Independent of the underlying disease, urinary protein loss may lead to several complications, such as oedema, dyslipidaemia, hypertension, anaemia, secondary hyperparathyroidism and hypovitaminosis D, due either to the toxicity of proteinuria on the kidney, or to plasma depletion of specific proteins.
4.2 Treatments

In secondary causes of nephrotic syndrome, specific treatment varies depending on the causative illness, e.g. in diabetes, metabolic control is the mainstay of treatment. Among primary renal diseases, an underlying immunological disorder is suspected. Immunosuppressive or immunomodulatory drugs are therefore the mainstay of treatment, therapeutic strategies depending on the clinical and histological features. However, irrespective of these specific treatments, nonspecific protective therapies must be implemented in patients with nephrotic syndrome.

Table 4.1: Primary glomerular diseases associated with nephrotic syndrome.

<table>
<thead>
<tr>
<th>Primary glomerular diseases (frequent; rare)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Membranous glomerulopathy</td>
</tr>
<tr>
<td>Focal and segmental glomerulosclerosis</td>
</tr>
<tr>
<td>Minimal change disease (MCD) glomerulopathy</td>
</tr>
<tr>
<td>IgA nephropathy</td>
</tr>
<tr>
<td>Membranoproliferative glomerulonephritis</td>
</tr>
<tr>
<td>C1q glomerulopathy</td>
</tr>
<tr>
<td>Fibrillar glomerulopathy</td>
</tr>
<tr>
<td>Congenital podocyte anomaly</td>
</tr>
</tbody>
</table>

Antihypertensive treatment, NSAIDs, and dietary factors are therefore either complementary to a specific treatment or the sole therapy available to lower proteinuria. In contrast to specific treatment, complete remission of proteinuria cannot be expected with these treatments. A reasonable goal is to aim for a 50% reduction in proteinuria, or, even better, for a proteinuria below 1g/day, which seems to be a critical threshold for long term renal survival\(^3,4\).
4.2.1 Dietary factors

A proper **nephrotic syndrome dietary plan** is an essential component of the treatment that the patient should implement. The purpose of nutritional care in nephrotic syndrome is to minimize edema and other manifestations of the syndrome, to replace nutrients lost in the urine and to reduce risks of further progression of renal disease and also atherosclerosis. As a result, the diet is modified primarily in protein and sodium. However, additional modifications in fat cholesterol, calcium and calorie intake may also be indicated.5

<table>
<thead>
<tr>
<th>Medications (non exhaustive)</th>
<th>Allergens, immunisations Pollens, seric illness, vaccines, bee sting</th>
</tr>
</thead>
<tbody>
<tr>
<td>NSAIDs, pamidronate, rifampicin, IFN alpha, gold, lithium, interferon alpha</td>
<td></td>
</tr>
<tr>
<td>Infections</td>
<td>Systemic illnesses (most frequent) Systemic lupus erythematosus (SLE), Rheumatoid polyarthritis, Schonlein-Henoch purpura MGUS, amyloidosis</td>
</tr>
<tr>
<td>Bacterial: Endocarditis, syphilis, tuberculosis, mycoplasma infections</td>
<td></td>
</tr>
<tr>
<td>Viral: HIV, HBV, HCV, EBV, CMV, VZV</td>
<td></td>
</tr>
<tr>
<td>Protozoal: Toxoplasmosis, malaria</td>
<td></td>
</tr>
<tr>
<td>Helminthic: Schisostomiasis, trypanosomiasis, filariasis</td>
<td></td>
</tr>
<tr>
<td>Neoplasia</td>
<td>Metabolic diseases and heredofamilial (non exhaustive) Type I and II diabetes Hypothyroidism Alport syndrome Graves disease Fabry disease</td>
</tr>
<tr>
<td>Solid tumours</td>
<td></td>
</tr>
<tr>
<td>Haemo- or lymphopathies</td>
<td></td>
</tr>
<tr>
<td>Multiple myeloma</td>
<td></td>
</tr>
<tr>
<td>GVHD post marrow transplantation</td>
<td></td>
</tr>
<tr>
<td>Miscellaneous (examples)</td>
<td></td>
</tr>
<tr>
<td>Pregnancy-associated</td>
<td></td>
</tr>
<tr>
<td>Chronic allograft failure</td>
<td></td>
</tr>
<tr>
<td>Nephronic reduction</td>
<td></td>
</tr>
<tr>
<td>Renal artery stenosis</td>
<td></td>
</tr>
<tr>
<td>Obesity</td>
<td></td>
</tr>
<tr>
<td>Heart failure (right/left)</td>
<td></td>
</tr>
<tr>
<td>and pericarditis</td>
<td></td>
</tr>
</tbody>
</table>
4.2.1.1 Protein

Protein intake has been a subject of debate in nephrotic syndrome. Determining the optimal level of protein needed to maintain protein reserves without promoting renal disease progression and protein losses in patients with nephrotic syndrome is difficult. Various studies have demonstrated that a high protein diet (to correct for the urinary losses) was ineffective in correcting hypoalbuminaemia. Moreover, the increased protein intake tends to further increase proteinuria and glomerular hyperfiltration, and is therefore probably deleterious. Conversely, low protein diets (<0.8 g/kg/d) have a slight anti-proteinuric effect which might be valuable.

Vegetable proteins appear to be more beneficial in reducing proteinuria when compared to animal proteins. However, due to muscle wasting being a major problem in nephrotic patients, and a low protein intake diet increasing the risk of malnutrition, it is not recommended. The maintenance of appetite and overall nutrition may be difficult in the early stages of treatment and modification of protein intake may worsen these difficulties. In practice, a range of 0.8-1.0 g protein per kg dry body weight or weight adjusted for obesity, depending on the glomerular filtration rate (GFR) and nutritional status is recommended, with a preference for vegetable and fish proteins. The use of intravenous albumin preparations would seem unjustified on both medical and financial grounds except in the presence of serious haemodynamic complications (including acute renal failure) and/or the presence of severe resistant oedema. In these latter cases, short-term use of IV albumin may prove to be a useful supplement to sequential diuretic administration and sodium restriction.

4.2.1.2 Sodium and fluid

Controlled sodium intake in conjunction with pharmacological therapy is recommended to control oedema and hypertension.
Depending on the presence and level of oedema and hypertension, sodium intake can reasonably be limited to 2 - 3 g/day, in order to not only minimise oedema and hypertension, but also potentiate the effect of angiotensin – converting enzyme (ACE) inhibitors and angiotensin type 1 receptor antagonists (ARBs). Again, as with perturbations in dietary protein, extreme reduction in salt can lead to reduced palatability and worsening of anorexia.

Fluid intake is generally unrestricted. It is recommended reversing the oedema slowly, with a target weight loss of 0.5-1 kg a day, because aggressive diuresis can cause electrolyte disturbances, acute renal failure, and thromboembolism as a result of haemoconcentration. Therefore, daily weights are needed to monitor and assess patient fluid status.

### 4.2.1.3 Fat

Dyslipidaemia is common in patients with nephrotic syndrome. Dyslipidaemia may be marked, with an increase in total cholesterol, LDL, triglycerides and lipoprotein (a). Dyslipidaemia in nephrotic syndrome contributes to the increased cardiovascular mortality in these patients, and may also be involved in renal disease progression. Screening and treatment of dyslipidaemia are therefore of critical importance. The pathogenesis of dyslipidaemia in nephrotic patients is not yet totally understood. Decreased plasma oncotic pressure stimulates hepatic synthesis of different proteins and may contribute to the increase in LDL. Acquired HDL metabolism abnormalities resulting in increased triglycerides and decreased HDL synthesis have also been observed.

Hyperlipidaemia appears to be proportional to the degree of proteinuria and hypoalbuminemia. Short-term hyperlipidaemia is not considered to be particularly harmful. Thus, immediate treatment of nephrotic hyperlipidaemia in patients who are undergoing treatment of the underlying renal disease with
expected remission is not imperative, since proteinuria reduction is the best treatment for dyslipidaemia correction. However, for patients for whom remission is not likely, dietary modifications that minimize proteinuria and dyslipidaemia are recommended.

Specific diet modifications are consistent with the Therapeutic Lifestyle Changes (TLC) diet, which include limiting dietary cholesterol to less than 200 mg/d and restricting total fat to 25-35% of total calories with less than 7% of the day’s total calories coming from saturated fat and with a polyunsaturated – saturated ratio of at least 1:1. There is evidence that fish oil lowers serum triglycerides and cholesterol in mice with nephritic syndrome. In high – risk patients with IgA nephropathy, early and prolonged treatment with fish oil supplements (1.9g EPA and 1.4g DHA) has been shown to slow disease progression. In case of non-responsiveness to diet, pharmacologic intervention may be used in combination with diet to maximize control of serum lipid levels.

4.2.1.4 Energy
Calorie intake should be sufficient to achieve and maintain a desirable dry body weight. Adequate calorie intake is essential to preserve protein reserves. Complex carbohydrates should supply the majority of calories, since protein intake and fat intake will be limited. Simple carbohydrates should be avoided, since most of the patients with NS, receive corticosteroids. If a patient is obese, supervised gradual weight reduction is recommended to assist in controlling blood pressure and dyslipidaemia.

4.2.1.5 Vitamins and minerals
Renal failure is associated with secondary hyperparathyroidism and hypovitaminosis D. In nephrotic patients this may be further complicated by urinary loss of albumin and globulins
that are vitamin D transporters. Monitoring of serum calcium and phosphates as well as vitamin D and PTH measurement is warranted in these patients. Steroid treatments of nephrotic syndrome also provides a significant risk for early development of osteoporosis. Depending on the clinical context and associated medications, mostly corticosteroids, a dual absorption densitometry and/or bisphosphonate therapy may be indicated\textsuperscript{10}. If possible, supplementation with calcium and vitamin D is recommended in all patients in order to preserve bone density and to facilitate calcium absorption.

Anaemia of chronic renal disease is related to inadequate erythropoietin production by the kidneys (endocrine failure). In nephrotic patients this can be aggravated by urinary losses of erythropoietin, transferrin and iron. The association of nephrotic syndrome with anaemia in the absence or renal dysfunction is still a subject of debate\textsuperscript{11}. In cases of anaemia a standard evaluation should be performed (erythrocytes count, B12, folate, iron status). In the absence of renal dysfunction and with an unexplained regenerative anaemia, erythropoietin measurement is useful. If the erythropoietin level is low, administration of erythropoiesis synthesis agents (ESA) is indicated, targeting a haemoglobin level of 11–12 g/l. Iron should be supplemented only when there is a proven deficiency, since as mentioned above anaemia in NS patients is more commonly due to losses of erythropoietin rather than iron deficiency\textsuperscript{4}.

Increased potassium and magnesium losses due to secondary hyperaldosteronism, potassium – wasting diuretics and steroids may require replacement. Therefore, potassium level should be monitored and dietary potassium adjusted as needed. Generally, though, there is no modification for potassium\textsuperscript{12}.

In conclusion, evaluation and treatment of complications are crucial in order to minimise mortality and morbidity associated with nephrotic syndrome. A nutritionist consultation is recommended to every nephrotic patient\textsuperscript{4}.
References


Nutritional Management of Early Stage Chronic Kidney Disease
Nutritional Care for Adults with Chronic Kidney Disease

Introduction

In the United Kingdom alone it is estimated that 10% of the adult population have early CKD. This increases with age to approximately 20% over 65 years and greater than 30% over 80 years. Although nutrition plays an important role during all stages of CKD, optimising nutritional status early in the CKD trajectory has the potential to reduce the risk of progression to end-stage renal disease (CKD requiring dialysis). The focus of this chapter is to explore the nutritional management of early CKD. The opening sections of this chapter will examine the aims of nutritional strategies early in CKD. The core components of nutritional assessment will then be addressed, prior to outlining nutritional interventions. Within the context of this chapter, early CKD refers to CKD Stages 1 to 3.

The aims of nutritional strategies in early CKD

In accordance with Cohen et al. (2007), the aims of nutritional therapy in early CKD are two-fold encompassing:

- Preventing progression of CKD.
- Reducing the risk factors for cardiovascular disease.

Learning outcomes

- Describe the aims of nutritional management in early chronic kidney disease (CKD)
- Outline the key principles of nutritional assessment for the patient with early stage CKD
- Discuss the core nutritional interventions which may be utilised to halt the progression of CKD
Hypertension, obesity, dyslipidaemia, and sub-optimal glucose control in diabetes mellitus comprise risk factors for CKD progression. As each of these risk factors may be readily modified by nutritional interventions, dietetic strategies should be implemented at the earliest opportunity.

1. **Hypertension**

Hypertension is both a cause and consequence of renal disease and is present in 50% to 75% of patients with early CKD. Moreover, high blood pressure per se may be independently associated with the development of type 2 diabetes mellitus, which constitutes the single most important cause of CKD worldwide. The nutritional management of hypertension in patients with early CKD focuses on the achievement and maintenance of a desirable body weight in combination with dietary sodium restriction.

2. **Obesity**

Obesity (body mass index [BMI] >30 kg/m²) constitutes a risk factor for progression to CKD in patients with pre-existing renal disease or reduced renal mass. Furthermore, patients with obesity are at increased risk for developing obesity-related glomerulopathy, where on renal biopsy the glomeruli are enlarged. Dietary approaches to the management of obesity in the early CKD patient population are based on achieving a BMI <25 kg/m² through encouraging a balanced diet and physical activity.

3. **Dyslipidaemia**

Dyslipidaemia is a common complication of progressive kidney disease, which is characterised by high triglyceride and low high-density lipoprotein (HDL) cholesterol levels, accumulation of remnant particles, a predominance of small dense low-density lipoprotein (LDL) particles, and increased levels of lipoprotein A. Further to accelerating
the development of systemic atherosclerosis, dyslipidaemia may also predict glomerular filtration rate decline\textsuperscript{10}. The aims of dietary interventions in the management of dyslipidaemia in early CKD are to lower LDL cholesterol, reduce trans-fatty acids to <1%, and limit total fat consumption to approximately 25%-35% of calories\textsuperscript{11}.

4. Glucose control in diabetes mellitus

Diabetes mellitus is the lead cause of CKD worldwide, and diabetic nephropathy is responsible for most deaths attributed to microvascular complications\textsuperscript{4}. Nephropathy is also considered to be a major risk factor for macrovascular complications and particularly for coronary heart disease\textsuperscript{4}. Poor glycaemic control in patients with early CKD accelerates the progression of nephropathy. Subsequently, dietetic management aims to optimise glycaemic control by maintaining an HbA1C <7.0\textsuperscript{12}.

Prior to the planning of and implementation of nutritional strategies, the patient’s nutritional status needs to be comprehensively assessed. Nutritional assessment provides the basis on which to formulate individualised plans of care, tailored to meet the specific requirements of the patient. The core components of nutritional assessment will now be addressed.

Assessment of nutritional status

The assessment of nutritional status does not lend itself to one simple test, and an optimal panel of measures for screening nutritional status is a requisite\textsuperscript{2}. Table 1 outlines the core components of nutritional assessment.
<table>
<thead>
<tr>
<th>Components</th>
<th>Assessments</th>
</tr>
</thead>
</table>
| Assessment of nutritional intake | • Diet history e.g. a three-, seven-, 14-, or 30-day history of nutritional intake  
• Food diaries                       |

  - Assist/increase the accuracy of dietary recall  
  - Items to include:  
    ▪ Foods consumed  
    ▪ Amount consumed  
    ▪ Method of food preparation e.g. grilled, baked, fried, boiled  
    ▪ Times of meals e.g. breakfast, lunch, dinner, supper, snacks  
    ▪ Portion sizes  
    ▪ Speed with which food was consumed  
    ▪ The body’s reaction to the foods e.g. nausea, bloating, indigestion |
| Anthropometric measurements       | • Body weight (kg)  
• Height (cm)  
• BMI (kg/m²)  
  - A practical method for assessing adiposity/classifying obesity  
  - BMI categories:  
    ▪ Underweight <18.5  
    ▪ Ideal weight = 18.5-24.9  
    ▪ Overweight = 25.0-29.9  
    ▪ Obese ≥30  
• Weight change  
  - Current weight compared to weight three, six, or twelve months previously  
  - Percentage of weight change i.e.  
    *([IW-CW]/IW) x 100 = % weight change  
• Skinfold thickness  
  - An effective measure of adiposity  
• Mid-upper-arm muscle circumference (MAMC)  
  - A reliable measure of muscle mass  
  - +MAC (cm) - [3.14 x TSF (cm)] = MAMC (cm) |
# Nutritional Care for Adults with Chronic Kidney Disease

**Components Assessments**

<table>
<thead>
<tr>
<th>Body composition</th>
<th>Assessments</th>
</tr>
</thead>
</table>
| • Neutron activation analysis  
  – A technique used to assess body protein content |
| • Ultrasonography |
| • Bioelectrical impedance analysis  
  – A method used to estimate total body water and fat-free body mass |
| • Dual-energy x-ray absorptiometry  
  – A means of measuring bone mineral density |

<table>
<thead>
<tr>
<th>Biochemical determinations</th>
<th>Assessments</th>
</tr>
</thead>
<tbody>
<tr>
<td>• Serum electrolytes e.g. potassium, sodium, calcium</td>
<td></td>
</tr>
<tr>
<td>• Serum proteins e.g. albumin, C-reactive protein</td>
<td></td>
</tr>
<tr>
<td>• Serum cholesterol, HDL, LDL, triglycerides</td>
<td></td>
</tr>
<tr>
<td>• Serum glucose, HbA1c</td>
<td></td>
</tr>
</tbody>
</table>
| • Creatinine index i.e. creatinine synthesis rate  
  – Facilitates the assessment of dietary skeletal muscle protein intake and skeletal muscle mass |

<table>
<thead>
<tr>
<th>Subjective global assessment (SGA)</th>
<th>Assessments</th>
</tr>
</thead>
</table>
| • A comprehensive nutritional classification system which incorporates:  
  – Medical history to establish:  
    ▪ Weight change  
    ▪ Dietary intake  
    ▪ Gastrointestinal symptoms  
    ▪ Functional impairment  
  – Physical examination to identify:  
    ▪ Loss of subcutaneous fat  
    ▪ Muscle wasting  
    ▪ Oedema |
| • Classifies patients as:  
  – Well-nourished  
  – Mildly malnourished or suspected of malnutrition  
  – Severely malnourished |
Nutritional Management of Early Stage Chronic Kidney Disease

**Components Assessments**

<table>
<thead>
<tr>
<th>Components</th>
<th>Assessments</th>
</tr>
</thead>
<tbody>
<tr>
<td>Functional tests</td>
<td>• Hand grip strength</td>
</tr>
<tr>
<td></td>
<td>− An indicator of muscle function</td>
</tr>
<tr>
<td></td>
<td>− Impaired muscle strength is a well-known phenomenon occurring in disease-related malnutrition</td>
</tr>
</tbody>
</table>

* OW=Original weight; CW=Current weight; + MAC=Midarm circumference; TSF=Triceps skinfold

References: Bircher and Doherty (2007); Anderson (2011); Norman et al. (2011).  

Comprehensive nutritional assessment, as outlined in Table 1, functions to identify patient-specific nutritional deficits. Moreover, the outcomes of nutritional evaluation, when interpreted within the context of pre-existing risk factors (i.e. hypertension, obesity, dyslipidaemia, diabetes mellitus), facilitate the selection of appropriate nutritional interventions which, in turn, have the potential to halt CKD progression.

**Nutritional interventions in early CKD**

Nutritional therapy is a fundamental component of patient care in early CKD. Whilst the overall focus of nutritional counselling is to promote a healthy and well-balanced diet, specific interventions exist to target the key CKD progression risk factors.

The nutritional management of hypertension

Cohen et al. (2007) propose that the dietary management of hypertension in patients with early CKD should focus primarily on weight reduction (discussed in the nutritional management of obesity section) and reducing sodium intake. Nevertheless, findings from the Dietary Approaches to Stop Hypertension (DASH) study indicate that a diet rich in fruits, vegetables, and low-fat dairy foods and with reduced saturated and total fat may also substantially lower blood pressure.
Sodium retention occurs with CKD, and associated extracellular volume expansion plays a principal role in the development of hypertension\(^8\). Subsequently, a reduction in sodium intake has the potential to reduce systolic and diastolic blood pressure. The DASH-sodium study\(^{15}\) demonstrated that a daily salt reduction to <100mmols sodium was associated with significant blood pressure lowering. Therefore, nutritional education, which illustrates the principles and benefits of sodium restriction, forms the basis of patient management. Key components of educational strategies encompass:

- Encouraging fresh, unprocessed food choices
- Promoting foods containing 5%-10% of the recommended sodium intake per serving (115-230mg)
- Discouraging pre-packaged and processed foods e.g. regular breads, cereals, smoked or cured meats, cheeses, and canned products
- Providing advice on reducing the amount of salt added to foods during the cooking process
- Recommend alternative methods for flavouring foods e.g. adding spices

The nutritional management of obesity

Among the documented benefits of losing weight is lower blood pressure, reduction in proteinuria, and improved insulin sensitivity\(^8\). Therapeutic strategies for the management of obesity in patients with early CKD comprise weight reduction through healthy eating combined with increased regular physical activity. Table 2 depicts the therapeutic strategies recommended for the non-pharmacological management of obesity.
Table 2: Therapeutic strategies for the management of obesity in patients with early CKD

<table>
<thead>
<tr>
<th>Component</th>
<th>Recommendation</th>
</tr>
</thead>
<tbody>
<tr>
<td>Weight reduction to</td>
<td>Calorie-restricted, balanced diet</td>
</tr>
<tr>
<td>achieve a BMI &lt;25 kg/m²</td>
<td></td>
</tr>
<tr>
<td>Exercise and physical</td>
<td>Moderate intensity for 30 minutes per day, most</td>
</tr>
<tr>
<td>activity</td>
<td>days of the week</td>
</tr>
<tr>
<td>Moderation of alcohol</td>
<td>≤2 alcoholic beverages per day (males)</td>
</tr>
<tr>
<td>intake</td>
<td>≤1 alcoholic beverage per day (females)</td>
</tr>
<tr>
<td>Smoking cessation</td>
<td>Counselling, nicotine supplementation</td>
</tr>
</tbody>
</table>


The nutritional management of dyslipidaemia

In the majority of cases, even in patients with CKD, dyslipidaemia is at least partly secondary to lifestyle factors such as diet, physical inactivity, cigarette smoking, obesity, and excessive alcohol usage16. Consequently, therapeutic lifestyle changes constitute important components in the management of dyslipidaemia and the prevention of cardiovascular disease16.

In addition to the lifestyle interventions depicted in Table 2, nutritional strategies target fat intake, particularly in relation to the type of fat consumed. With reference to Chan et al. (2011)16, the aims of nutritional interventions in dyslipidaemia management are to:

- Lower LDL cholesterol by replacing saturated fat (e.g. whole milk) with unsaturated fat (e.g. soya milk)
- Reduce trans-fatty acids to <1% e.g. by eliminating fried foods from the diet (French fries, chicken nuggets)
- Limit total fat consumption to about 25%-35% of calories
- Increase the consumption of soluble or viscous fibres (e.g. legumes and apples, respectively)

**Nutritional management in diabetes mellitus**

As with hypertension, obesity, and dyslipidaemia, lifestyle modification is integral to the management of diabetes mellitus in early CKD. Nonetheless, the management of nutrition in a patient with both diabetes mellitus and CKD is complex because intake of protein, potassium, sodium, and phosphorus, among other nutrients, must be considered cautiously\(^12\).

The optimisation of glycaemic control, as evidenced by HbA1c <7.0\(^{12}\), is the focus of nutritional interventions in diabetes mellitus management. The following interventions, if timely implemented, have the potential to delay the progression of diabetic nephropathy:

1. *Weight loss* to achieve a goal BMI <25 kg/m\(^2\) (achieved by implementing a low-calorie, carbohydrate-restricted, or fat-restricted diet)\(^12\)
2. *Protein restriction* to 0.8 g/kg body weight per day\(^12\)
3. *Lipid management* to target a LDL cholesterol of <100 mg/dL (<2.59 mmol/L)\(^3\)

**Nutritional counselling**

Given the well-known difficulties associated with lifestyle modification, patients wishing to adopt lifestyle changes are likely to need healthcare provider support. Karanja et al. (2004)\(^17\) advocate the ‘five A’s’ counselling framework as a method by which to enhance the success of lifestyle modification interventions. Table 3 summarises the ‘five A’s’ counselling framework.
Table 3: The ‘five A’s’ counselling framework

<table>
<thead>
<tr>
<th>The five A’s of counselling</th>
<th>Components</th>
</tr>
</thead>
</table>
| **Assess**                  | • Assess the patient’s eating behaviour and readiness to change  
                               • Identify other factors that might interfere with attempts to make lifestyle changes |
| **Advise**                  | • Provide clear, specific advice tailored to the patient  
                               • Emphasise the value of making the proposed lifestyle adjustments e.g. weight loss |
| **Agree**                   | • Ensure that the patient agrees on the most appropriate target behaviour to focus on, and the best method to achieve this behaviour i.e. the patient selects a dietary goal (e.g. increase fruit and vegetable intake) and creates an action plan for achieving this goal (e.g. have a piece of fruit for breakfast) |
| **Assist**                  | • Assist the patient as he or she attempts to make the required lifestyle changes e.g. provide patients with the skills necessary to overcome behavioural change  
                               • Provide nutritional education e.g. information regarding serving sizes and tips on how to shop for and prepare food |
| **Arrange**                 | • Schedule follow-up contacts to provide ongoing support and advice  
                               • Adjust lifestyle goals as necessary  
                               • Refer for intensive counselling if indicated |

Summary

This chapter has explored the nutritional management of early CKD, and, in doing so, has highlighted the importance of nutritional assessment. Nutritional deficits, as identified by detailed patient assessment, provide the foundations on which to plan, implement, and evaluate patient-focused nutritional interventions. Nutritional counselling, within the context of the ‘five A’s’ framework, has the potential to maximise nutritional outcomes in the early CKD patient population.
References


Nutritional needs for CKD Stages 4-5
Learning outcomes

- Knowledge as to why CKD patients need medical nutrition therapy
- Knowledge of different dietary deficiencies in CKD 4-5 and how to regulate them

Chronic kidney disease (CKD) is a condition characterized by a gradual decline of renal function over time. It is classified into five stages based on the reduction of renal function measured with glomerular filtration rate (GFR) and/or the presence of renal damage, that is albuminuria.

Major causes of CKD include: age, diabetes, hypertension, glomerulonephritis, cystic kidney diseases and tubulointerstitial diseases. Cardiovascular disease (CVD) complications are frequent in these patients and are much more frequent than end-stage renal disease (CKD). Reduction in renal function due to ageing is a non-modifiable cause, whereas management of CVD risk factors, valid for the general population, should be applied for CKD patients, especially in the early stages. In this context, together with medical therapy, nutritional therapy is essential as it includes: weight control, blood pressure maintenance, a satisfactory glycaemia level and a normal lipid profile while it promotes both smoking cessation and increased physical activity.

When patients progress to CKD, medical nutrition therapy (MNT) is essential for:

- Promoting a balanced and healthy diet, in accordance with the guidelines for the general population and
individual preference, within the limitations imposed by the disease;

- Maintaining blood urea nitrogen (BUN), electrolytes and fluids under control;
- Preventing metabolic complications such as metabolic acidosis, hyperparathyroidism and insulin resistance;
- Ensuring the maintenance / achievement of a satisfactory nutritional status.

### 6.1 Dietary energy intake in CKD Stages 4-5

One of the most common complications of chronic renal failure (CRF) is the development of protein energy malnutrition (PEM) that can originate even in stage IV of CKD, because of a spontaneous reduction in food intake\(^2\). In dialysis, the main cause of under-nutrition is a low energy intake often due to an overly restrictive dietary regimen. The role of psychosocial factors such as depression, solitude and inability to prepare meals should also be considered. Moreover, a low energy intake stimulates protein catabolism for energy production and can be increased by chronic inflammation and resistance to anabolic factors\(^3\). At the same time, overweight and obesity are becoming progressively more common in renal patients and many observational studies have shown that obesity might be associated with better outcomes in dialysis patients\(^4\).

To avoid malnutrition - in excess or defect - it is necessary to accurately assess patients’ nutritional needs. In order to obtain this, a dietary history needs to be compiled by an expert renal dietician, who is able to identify discrepancies between prescribed and actual dietary intake. Energy requirements of patients with CKD are similar to healthy population\(^5\) and can be estimated using a formula provided by guidelines on nutritional care of renal patients.

A summary of the available guidelines is presented in Table 6.1. For patients in CKD Stages 4-5, an intake of 35 kcal/kg
ideal body weight (IBW) or 30/kcal/kg IBW for those patients with a low physical activity or an age older than 60 years. When clinical or biological indices of malnutrition are found, it is suggested that energy intake be increased to 40 kcal/kg by frequent dietetic counseling and/or oral supplement.

Table 6.1 Recommended Suggested energy intake for CKD patients.\textsuperscript{24} ADA 2002,\textsuperscript{6,23}

<table>
<thead>
<tr>
<th></th>
<th>Predialysis</th>
<th>Haemodialysis</th>
<th>Peritoneal dialysis</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>DOQI</strong></td>
<td>35 kcal/kg BW/day</td>
<td>35 kcal/kg BW/day</td>
<td>35 kcal/kg BW/day</td>
</tr>
<tr>
<td></td>
<td>30-35 kcal/kg BW/day if age ≥ 60 years</td>
<td>30-35 kcal/kg BW/day if age ≥ 60 years</td>
<td>30-35 kcal/kg BW/day if age ≥ 60 years</td>
</tr>
<tr>
<td><strong>EBPG</strong></td>
<td>-----------------------------------------</td>
<td>30-40 kcal/kg IBW/day</td>
<td>35 kcal/kg /day</td>
</tr>
<tr>
<td></td>
<td>Adjusted to age, gender and physical activity level</td>
<td>Adjusted to age, including both diet and peritoneal absorption</td>
<td></td>
</tr>
<tr>
<td><strong>ADA</strong></td>
<td>Individualized; to maintain reasonable weight; or ≥ 35 kcal/kg IBW</td>
<td>Individualized; or use 30-35 kcal/kg IBW or adjusted weight</td>
<td>Individualized; or use 25-35 kcal/kg IBW or adjusted weight</td>
</tr>
<tr>
<td><strong>EDTNA/ERCA</strong></td>
<td>35 kcal/kg IBW/day</td>
<td>35 kcal/kg IBW/day</td>
<td>35 kcal/kg IBW/day</td>
</tr>
<tr>
<td></td>
<td>30 kcal/kg IBW/day in the elderly and patients with reduced activity</td>
<td>30 kcal/kg IBW/day in the elderly and patients with reduced activity</td>
<td>30 kcal/kg IBW/day in the elderly and patients with reduced activity</td>
</tr>
</tbody>
</table>

Abbreviations: DOQI = Disease Outcomes Quality Initiative; ADA = American Dietetic Association; EBPG = European Best Practice Guidelines; EDTNA/ERCA = European Dialysis and Transplant Nurses Association/European Renal Care Association; IBW = ideal body weight.
For peritoneal dialysis (PD) patients, the energy obtained from the absorption of glucose must be considered, as 60% to 75% of glucose, during a 6-hour peritoneal dwell, reaches the general circulation resulting in a glucose absorption of 100-200 g glucose/24 h.\(^7\).

### 6.2 Dietary Protein intake in CKD Stage 4 -5

Prior to the advent of dialysis, protein restriction was essential for prolonging survival of CKD patients, permitting symptom control. In fact, limiting dietary protein is associated with decreased waste products and uremic toxins, blood urea nitrogen levels and metabolic acidosis, improvement in all uremia symptoms such as anorexia, nausea, vomiting, weakness and fatigue.

With dialysis therapy, much less importance has been given to nutritional treatment, but recent studies have shown that lowering protein intake in patients with CKD reduces the occurrence of renal death by 32%, compared with higher or unrestricted protein intake\(^8\). The optimal level of protein restriction has not been determined, but a significant decrease in normal intake has been reported to produce favorable effects in terms of progression rate reduction. Indeed, a diet low in protein is related with reduced urinary urea nitrogen output and a decrease in kidney workload, having a potential effect to protect kidneys.

Moreover, a linear relationship between reduction in dietary protein intake and a decrease in proteinuria has been reported as an additive antiproteinuric effect to ACE inhibition (Ruilopec\(^9,10\). As proteinuria is the most important and independent factor affecting the progression of CKD, trying to lower it is a key aim of the therapy\(^11\). In addition, a low protein diet:

- Reduces phosphate load, leading to both a better control of metabolic bone disorders and a reduction in secondary hyperparathyroidism
Nutritional Care for Adults with Chronic Kidney Disease

- Improves anemia control
- Ameliorates insulin resistance
- Reduces metabolic acidosis and controls dyslipidemia

The Kidney Disease Outcomes Quality Initiative (KDOQI) recommends a protein intake of 0.6 g/kg day; if the patient cannot tolerate such a diet or is unable to maintain adequate daily energy intake (DEI), an intake of 0.75 g/kg/day may be prescribed. The European Dialysis Transplantation Nurse Association/Renal Care Association (EDTNA/ERCA) and the American Dietetic Association (ADA) recommend a protein intake between 0.6 and 1.0 g/kg IBW day. Moreover, EDTNA/ERCA states that it is necessary to ensure that patients who are advised to reduce their intake to below 0.8/kg IBW/day are provided with adequate follow-up from trained renal dieticians due to their increased risk of malnutrition. (Table 6.2). It should be added that at least 50% of the prescribed protein intake should be of high biological value.

Another therapeutic option is very low protein diets (VLPD), which could be used in selected patients, including elderly patients who are highly motivated to delay the start of dialysis. This dietary plan provides a protein intake of 0.3g/kg IBW and a supplementation with ketoanalogues of essential amino acids (EAA) to reduce urea accumulation by allowing endogenous nitrogen to be reused for new protein synthesis while preventing EAA deficiency. Bellizzi et al reported that in patients treated for 6 months with VLPD, blood pressure control improved significantly, thereby concluding that VLPD has an antihypertensive effect, which was attributed to the reduction of salt intake, ketoanalogues supplementation and type of proteins.

An interesting conclusion of the INTERMAP Study was that a diet high in vegetable products is recommended as part of healthy lifestyle for prevention of high blood pressure and related diseases. Previously, Cupisti et al suggested that
the adoption of a vegetable based low protein diet, alternated with a conventional low protein diet, can improve patient compliance\textsuperscript{17}.

6.2.1 Dietary Protein intake in Haemodialysis

Haemodialysis treatment induces a loss of nutrients including glucose, amino acids, vitamins and trace elements. The procedure itself is responsible for protein catabolism. Moreover, research data indicate that a spontaneous decrease in calories and protein intake may occur, possibly leading to PEM\textsuperscript{18}.

Many observations have lead to recommend a safe level of protein intake of 1.2 g/kg day to ensure a neutral or positive nitrogen balance. More recently, it has been stated that, with adequate caloric intake, even 1.1 g/kg of protein is sufficient for nitrogen balance in stable hemodialysis patients who are absent of co-morbidities or catabolic events. At least 50% of the protein should be of high biological value to improve the efficiency of amino acid utilization. (Table 6.2)

6.2.2 Dietary Protein intake in Peritoneal dialysis

Protein needs during peritoneal dialysis are higher due to the protein losses through the peritoneal membrane (3-15 g / day of protein)\textsuperscript{19}. Many guidelines indicate 1-1.2 g / kg as the ideal intake for a neutral or positive nitrogen balance. This value is set to meet the 97.5% of the population, however, some patients have a stable nutritional status with lower intake and being so, recent guidelines indicate an intake > 1g/kg as acceptable if the patient does not exhibit any clinical signs of decline in nutritional status. During episodes of peritonitis, protein intake should be increased for the catabolism associated with inflammation. (Table 6.2)
### Table 6.2 Suggested protein intake for CKD patients. Adopted from: 24 ADA 2002; 6,23

<table>
<thead>
<tr>
<th>DOQI</th>
<th>Predialysis</th>
<th>Haemodialysis</th>
<th>Peritoneal dialysis</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>0.6 g/kg BW/day</td>
<td>1.2 g/kg BW/day</td>
<td>1.2-1.3 g/kg BW/day</td>
</tr>
<tr>
<td></td>
<td>0.75 if low DEI</td>
<td>≥ 50% HBV</td>
<td>≥ 50% HBV</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>EBPG</th>
<th></th>
<th></th>
<th>At least 1.0 g/kg IBW/day</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>At least 1.1 g/kg IBW/day</td>
<td>Balanced intake of high quality animal protein and vegetable protein source</td>
<td>At least 1.0 g/kg IBW/day</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>ADA</th>
<th></th>
<th></th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Based on CrCl, GRF or protein losses: 0.6-1 g/kg IBW; 50% IBW</td>
<td>1.1-1.4 g/kg IBW</td>
<td>1.2-1.5 g/kg IBW</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>EDTNA/ERCA</th>
<th></th>
<th></th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>0.6-1 g/kg IBW &gt; 50% IBW</td>
<td>1.1-1.2 g/kg IBW/day</td>
<td>1.0-1.2 g/kg IBW/day</td>
</tr>
<tr>
<td></td>
<td>≥ 50% HBV</td>
<td>≥ 50% HBV</td>
<td>≥ 50% HBV</td>
</tr>
</tbody>
</table>

**Abbreviations:** DOQI = Disease Outcomes Quality Initiative; ADA = American Dietetic Association; EBPG = European Best Practice Guidelines; EDTNA/ERCA = European Dialysis and Transplant Nurses Association/European Renal Care Association; IBW = ideal body weight; HBV = high biological value; DEI = Dietary energy intake.

### 6.3 Phosphorous intake in CKD Stages 4-5

Phosphorous intake should be restricted in all stages of CKD to control hyperphosphatemia as this is implicated in
hyperparathyroidism, mineral bone disease and cardiovascular disease.

During CKD 4, a reduction in protein intake allows for a good control of phosphorous. In CKD, the need for an adequate protein intake leads to a high phosphate load while a standard hemodialysis session removes only 500-700 mg of phosphate. In this context, dialysis adequacy and schedule - both prolonged or daily dialyses - are able to influence phosphorous removal.

Foods with high protein content provide 12-16 mg of phosphate per gram of protein and dairy products have the highest ratio. Thus, the selection of food with favorable ratios of phosphate per gram of protein and the prescription of appropriate phosphate binders - selection, dose and time of assumption - are important, in order to allow for an adequate protein intake. The need for phosphate binders is associated with problems of adherence and side effects such as gastrointestinal symptoms.

Recent studies have reported that phosphorous containing additives are commonly found in processed foods, including meats, dairy products, baked goods and beverages. Over the last few decades, the consumption of these processed foods has increased markedly20 and can duplicate the normal amount of dietary phosphorous so patients should be advised to avoid food that have added phosphorous salts.

EDTNA/ERCA suggests intakes of 600-1000 mg/day in conservative management and 1000-1400 mg/day in replacement therapy, while ADA indicates an individualized intake in conservative management and <17/mg/kg in replacement therapy (Table 6.3).
Table 6.3 Suggested phosphorus intake for CKD patients. Adopted from \cite{25}; ADA 2002; \cite{6,23}

<table>
<thead>
<tr>
<th></th>
<th>Predialysis</th>
<th>Haemodialysis</th>
<th>Peritoneal dialysis</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>DOQI</strong></td>
<td>Restricted to 800-1000 mg/day if serum phosphorus level &gt; 5.5 mg/dl</td>
<td>Restricted to 800-1000 mg/day if serum phosphorus level &gt; 5.5 mg/dl</td>
<td>----</td>
</tr>
<tr>
<td><strong>EBPG</strong></td>
<td>-----------------</td>
<td>800-1000 mg/day</td>
<td>----</td>
</tr>
<tr>
<td><strong>ADA</strong></td>
<td>Individualised: 8-12 mg/kg/IBW</td>
<td>≤ 17 mg/kg IBW</td>
<td>≤ 17 mg/kg IBW</td>
</tr>
<tr>
<td><strong>EDTNA/ERCA</strong></td>
<td>600-1000 mg/day</td>
<td>1000-1400 mg/day</td>
<td>1000-1400 mg/day</td>
</tr>
</tbody>
</table>

Abbreviations: DOQI = Disease Outcomes Quality Initiative; ADA = American Dietetic Association; EBPG = European Best Practice Guidelines; EDTNA/ERCA = European Dialysis and Transplant Nurses Association/European Renal Care Association; IBW = ideal body weight.

6.4 Potassium intake in CKD Stages 4-5

Potassium control is important to prevent episodes of hyperkalemia that can lead to cardiac arrhythmia. From a multidisciplinary point of view, it is essential to consider non-dietetic factors that can influence blood potassium.

In healthy subjects 90% of dietary potassium is excreted by the kidneys, whereas in CKD patients, potassium intestinal excretion is increased as a compensatory mechanism. For this reason, constipation has to be avoided, while diarrhea may be responsible for hypokalaemia, because of potassium loss. Many drugs may induce hyperkalaemia including steroids, ACE-inhibitors and potassium-sparing diuretics; moreover, acidosis and hyperglycaemia may cause a shift of potassium from intracellular to extracellular fluid\cite{21}.

In CKD, the maintenance of acceptable potassium levels is preserved by dietary intake and dialysis adequacy. Generally,
as the serum level increases, dietary potassium intake needs to be monitored. Restriction should be a short-term solution because, if protracted, it may negatively affect nutritional adequacy and quality of life.

Finally, patients need to be advised to both avoid the use of salt substitutes as part of a low sodium diet and attentively read food labels for the presence of potassium salt or the phrase “enhanced with a natural solution of…”.

Being so, examined guidelines suggest a potassium intake of 2000 mg/day both in conservative and replacement therapy (EDTNA/ERCA) or adjusted in relation to serum level (ADA) (Table 6.4)

Table 6.4 Suggested potassium intake for CKD patients. Adopted from: 24 ADA 2002; 6,23

<table>
<thead>
<tr>
<th></th>
<th>Predialysis</th>
<th>Haemodialysis</th>
<th>Peritoneal dialysis</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>DOQI</strong></td>
<td>35 kcal/kg BW/day</td>
<td>35 kcal/kg BW/day</td>
<td>35 kcal/kg BW/day</td>
</tr>
<tr>
<td></td>
<td>30-35 kcal/kg BW/day if age ≥ 60 years</td>
<td>30-35 kcal/kg BW/day if age ≥ 60 years</td>
<td>30-35 kcal/kg BW/day if age ≥ 60 years</td>
</tr>
<tr>
<td><strong>EBPG</strong></td>
<td>&lt; 1 mEq/kg IBW/day or 50-70 mmoL/day</td>
<td>&lt; 1 mEq/kg IBW/day or 50-70 mmoL/day</td>
<td>&lt; 1 mEq/kg IBW/day or 50-70 mmoL/day</td>
</tr>
<tr>
<td><strong>ADA</strong></td>
<td>Individualized per lab values</td>
<td>Approximately 40 mg/kg IBW</td>
<td>Restricted by lab values</td>
</tr>
<tr>
<td><strong>EDTNA/ERCA</strong></td>
<td>2000-2500 mg/day</td>
<td>2000-2500 mg/day</td>
<td>2000-2500 mg/day</td>
</tr>
</tbody>
</table>

Abbreviations: DOQI = Disease Outcomes Quality Initiative; ADA = American Dietetic Association; EBPG = European Best Practice Guidelines; EDTNA/ERCA = European Dialysis and Transplant Nurses Association/European Renal Care Association; IBW = ideal body weight.
6.5 Sodium and liquid intake in CKD Stages 4-5

In healthy individuals, the kidneys maintain blood osmolarity and regulate body fluid volume through the excretion of sodium and water. During CKD, the kidneys lose this ability and dialysis becomes the principal way of maintaining fluid balance.

Sodium restriction is indicated for all stages of CKD as it helps to control extracellular volume and, at the same time, is important for blood pressure control.

In hemodialysis, sodium control is essential for preventing an excessive interdialytic weight gain (IDWG) in anuric and oliguric patients, given that it reduces thirst and improves compliance with fluid restriction. It should be noted that the rapid removal of a large amount of fluids during a session of dialysis may contribute to episodes of hypotension, cramps, arrhythmias and angina.

Examined guidelines (Table 6.5) recommend a fluid intake of 500-1000 cc in addition to daily urine output for HD patients in order to achieve an IDWG of 2-2.5 kg or 4% of dry body weight. All foods that is liquid form at room temperature (18-20°) should be considered as fluid, except oils.
Table 6.5 Suggested liquid intake for CKD patients. Adopted from: 24 ADA 2002; 6,23

<table>
<thead>
<tr>
<th></th>
<th>Predialysis</th>
<th>Haemodialysis</th>
<th>Peritoneal dialysis</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>DOQI</strong></td>
<td>-----</td>
<td>-----</td>
<td></td>
</tr>
<tr>
<td><strong>EBPG</strong></td>
<td>------------</td>
<td>Intradialytic weight gain</td>
<td>&lt; 4-4.5% of DBW</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>-----</td>
</tr>
<tr>
<td><strong>ADA</strong></td>
<td>To maintain appropriate hydration status</td>
<td>500-750 ml + UO</td>
<td>Maintain fluid balance</td>
</tr>
<tr>
<td></td>
<td></td>
<td>1000 ml/ day if anuric</td>
<td></td>
</tr>
<tr>
<td><strong>EDTNA/ERCA</strong></td>
<td>reduced fluid intake if oedematous or medically indicated</td>
<td>500 mL + daily urine output – includes only foods that are liquid at room temperature and those with high fluid content</td>
<td>800 mL + daily urine output – includes only foods that are liquid at room temperature and those with high fluid content</td>
</tr>
</tbody>
</table>

*Abbreviations: DOQI = Disease Outcomes Quality Initiative; ADA = American Dietetic Association; EBPG = European Best Practice Guidelines; EDTNA/ERCA = European Dialysis and Transplant Nurses Association/European Renal Care Association; IBW = ideal body weight; DBW = dry body weight; UO = Urine output*

Fluid balance is easier in CAPD patients as they generally have renal residual functions; for these patients examined guidelines recommend 500 cc + daily urine output + ultrafiltrate.
Finally, examined guidelines recommend a low daily sodium intake for both conservative and replacement therapies (Table 6.6).

### 6.6 Vitamins in CKD Stages 4-5

Routine supplements of vitamins involved in iron and calcium metabolism is common in CKD patients; other vitamins should be prescribed only when necessary\(^\text{23}\). Dialysis can remove some water-soluble vitamins; this loss is partly neutralized by the loss of renal function, which decreases their catabolism. Moreover, dietary restriction aimed at controlling potassium and sodium intake may contribute to clinically relevant losses of some vitamins.

<table>
<thead>
<tr>
<th>Table 6</th>
<th>Suggested sodium intake for CKD patients. Adapted from: NKF 2000 (DOQI); ADA 2002; Fouque et al 2007 (EPBG); EDTNA/ERCA 2002;</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Predialysis</td>
</tr>
<tr>
<td><strong>DOQI</strong></td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
</tr>
<tr>
<td><strong>EBPG</strong></td>
<td>------------</td>
</tr>
<tr>
<td><strong>ADA</strong></td>
<td>1-3 g/day</td>
</tr>
<tr>
<td><strong>EDTNA/ERCA</strong></td>
<td>1800-2500 mg/day</td>
</tr>
</tbody>
</table>

Abbreviations: DOQI = Disease Outcomes Quality Initiative; ADA = American Dietetic Association; EBPG = European Best Practice Guidelines; EDTNA/ERCA = European Dialysis and Transplant Nurses Association/European Renal Care Association; IBW = ideal body weight; HBV = high biological value.
References


Managing Symptoms
Learning outcomes

- To gain knowledge and understanding of the importance of maintaining a good nutritional status after a kidney transplantation.
- To examine the needs in the post-transplant recovery state, in the short term and in the long term (maintenance) phase.
- To see the common problems and complications a transplanted patient have to face in the short term and in the long term.

7.1 Introduction

Kidney transplantation begun in the 1950s as a mode of therapy for patients with end stage kidney disease. Kidney transplants are categorized according to the donor source as:

- Cadaveric, when the donor is a cadaver
- Living – related, when the donor is a blood relative
- Living donor, when he is a non related person.

Maintaining a good nutritional status is one of the main goals for these patients. After transplantation their needs are altered significantly in the early post transplant phase and also in the maintenance state and patients should receive relative nutritional guidelines from a renal dietician for each state.

7.2 Post transplant recovery phase

As malnutrition increases the risk of post transplant complications, i.e. impaired wound healing, hospital infections
and extended rehabilitation time, sufficient provision of nutrients is essential in the early post transplant state as nutritional needs are increased in this state\textsuperscript{1}.

After surgery, patients generally begin with clear liquid diet when bowel sounds are audible, usually within 24 hours, and they can advance in solid foods relatively quick, according to the patients’ tolerance. Dietary and liquid recommendations are strictly individualized, according to the function of the graft. In patients with delayed graft function, restrictions regarding fluids and electrolytes intake similar to the pre transplant state may be imposed\textsuperscript{2}.

### 7.2.1 Nutrient needs

#### 7.2.1.1 Energy

Caloric needs in that phase are elevated due to the metabolic stress of the transplantation and the high dose of corticosteroids. Caloric needs ideally should be measured with indirect calorimetry. In general, 30 – 35 Kcal/ kg of dry body weight could cover the needs of the majority of the patients\textsuperscript{1}.

#### 7.2.1.2 Protein

Immediately postoperatively nitrogen losses are increased. Protein requirements for acute post transplanted patients with functioning grafts are estimated at 1.3 – 2.0 g/kg of dry body weight. Restrictions in protein intake should be avoided in that state as they could worsen the negative nitrogen balance caused mainly by the stress of the operation and the high dosage of corticosteroids\textsuperscript{3}. In case of acute tubular necrosis and delayed graft function after transplantation, patients may need haemodialysis, a procedure that also affects negatively their nitrogen balance\textsuperscript{3}. 
7.2.1.3 Carbohydrates and fat
Glucose intolerance is common early post operatively, due to the high dose of corticosteroid therapy and immunosuppressive therapy. Nonetheless, dietary restrictions are not supported and optimizing diabetic therapy is preferred. Non protein calories should provide up to 70% of the total energy intake, putting emphasis on complex carbohydrates and monounsaturated fat intake².

7.2.1.4 Fluid and electrolyte intake
In the early post – transplant state high volume of fluids are necessary in order to stimulate graft function and urine output. Patients should be weighed daily and fluid intake and output should be closely monitored to avoid dehydration. Dietary recommendations should also be based on blood pressure monitoring and electrolyte status.

7.3 Maintenance – Long term phase
In the long term – maintenance state the main goals are:

- The achievement of a desirable body weight, within the normal values of Body Mass Index and the maintenance of a healthy lifestyle, regarding nutrition and physical activity.
- The control of blood sugar and blood lipid levels and achieve normal blood pressure levels.
- The maintenance of a normal bone density.
- The minimization of medications’ side effects.

7.3.1 Nutrient needs
7.3.1.1 Energy
Obesity is one of the most frequent complications following a transplantation and has a negative impact on graft survival⁴.
The cause of weight gain is multifactorial and includes the alleviation, the appetite stimulation due to corticosteroids, the lack of physical exercise and the non compliance to dietary recommendations\textsuperscript{5}. To avoid excessive weight gain caloric needs should be individually estimated, in order to maintain to achieve and/or maintain body weight within the normal range for Body Mass Index (BMI), i.e. 18.5 – 25 kg/m\textsuperscript{2}. Aerobic exercise should be encouraged, according to functional capacity of each patient\textsuperscript{2}.

7.3.1.2 Protein

Protein intake recommendations of long term kidney graft recipients do not differ from the healthy population, ranging from 0.8 – 1.0 g/kg of body weight, provided that they have a normal graft function. These needs should be covered with high biological value, low fat protein choices, as lean meat, poultry, fish, low fat dairy products and soya, in order to limit saturated fat intake\textsuperscript{2}.

7.3.1.3 Carbohydrates

Hyperglycaemia is very common in transplanted patients\textsuperscript{6}. As diabetes is one of the main cause of kidney disease, pre existing diabetes mellitus is common in patients undergoing renal transplantation and it’s control worsens under the effect of immunosuppressive therapy, corticosteroids and insulin resistance due to the elevated body weight often observed as mentioned earlier. Total carbohydrate intake should provide 45-50\% of the total energy intake, and for better blood glucose control patients are encouraged to limit simple sugars intake and to give emphasis to high dietary fibre intake (i.e. 25- 30 g of fibre per day) in order to achieve a better blood glucose and triglyceride control\textsuperscript{2}.
7.3.1.4 Fat

Hyperlipidaemia is also common in transplanted patients. Lipid abnormalities include hypercholesterolaemia, hypertriglyceridaemia, elevated Low Density Lipoprotein cholesterol (LDL-C) levels\(^7\). Hyperlipidaemias pose patients to higher risk for developing atherosclerosis and other vascular diseases\(^8\). Total fat intake should provide approximately 30% of total energy intake, with emphasis on the limitation of saturated fat and sufficient provision on mono and polyunsaturated fat. Mediterranean diet could also be recommended as it has be shown to be beneficial in controlling blood lipids and metabolic syndrome in transplanted patients\(^9,10\). Cholesterol intake should also be limited to 300 mg/day, while \(\omega\)-\(3\) fatty acids have been proven effective in reducing triglyceride levels\(^2\).

7.3.1.5 Fluid and electrolytes

Fluid in general are not restricted in the long term. Sufficient hydration is essential in hot climates in order to optimize graft function. In case that the graft loses its functionality fluid intake should follow guidelines for CKD. Sodium should be restricted to 2,000 mg per day to avoid fluid retention due to corticosteroid therapy and to help blood pressure control. Potassium rarely needs to be restricted and the aim is that blood potassium should be within normal levels\(^2\).

7.3.1.6 Vitamins and minerals

Serum calcium, phosphorus and magnesium should be monitored in long term transplanted patients. Since these patients are in chronic corticosteroid treatment which impairs calcium absorption from the gastrointestinal tract, increases bone absorption and suppresses osteoblast function, sufficient calcium and vitamin D intake are essential in optimize bone density\(^11\). Weight-bear exercise, in cases that proteinuria is absent, is also recommended. Moreover phosphorus may
Nutritional Needs in Kidney Transplantation

need to be supplemented as hypophosphataemia is common due to parathyroidectomy performed in many CKD patients prior to transplantation\(^\text{12}\).

**Table 7.1 Nutrient recommendations for Kidney transplanted patients**

<table>
<thead>
<tr>
<th>Nutrient</th>
<th>Immediately post transplant</th>
<th>Long-term transplant</th>
</tr>
</thead>
<tbody>
<tr>
<td>Energy</td>
<td>30 -35 kcal/ kg dry body weight</td>
<td>1.2-1.3 x REE</td>
</tr>
<tr>
<td>Protein</td>
<td>1.2 – 2 g/kg dry body weight</td>
<td>0.8 – 1 g/kg</td>
</tr>
<tr>
<td>Carbohydrates and Fat</td>
<td>50-70% of non protein calories</td>
<td>CHO: 45-50% of total energy intake</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Lipids: Limit saturated fat and cholesterol. Increase monounsaturated and polyunsaturated fat intake</td>
</tr>
<tr>
<td>Sodium</td>
<td>2,000 mg/ day</td>
<td>2,000 – 4,000 mg/day according to Blood pressure control</td>
</tr>
<tr>
<td>Potassium</td>
<td>Maintain normal serum levels</td>
<td>Maintain normal serum levels</td>
</tr>
<tr>
<td>Phosphorus</td>
<td>Individualized</td>
<td>Individualized</td>
</tr>
<tr>
<td>Calcium</td>
<td>Individualized</td>
<td>Often needs to be supplemented (800 – 1500 mg/d)</td>
</tr>
<tr>
<td>Fluids</td>
<td>Individualized, according to graft functionality</td>
<td>Usually unrestricted</td>
</tr>
</tbody>
</table>
References


Nutritional Management of Diabetic Nephropathy
Learning outcomes

- To gain knowledge and understanding of the basic principles of the nutritional management of diabetic nephropathy.

Alternative names for diabetic nephropathy

- Kimmelstiel-Wilson disease; Diabetic glomerulosclerosis; Nephropathy - diabetic

8.1 Introduction

The kidneys via the glomeruli filter circulating blood and help remove waste from the circulation. Diabetes mellitus (DM) over a long period of time may result in changes in the glomeruli that can damage the kidneys. This may be the result of either type 1 or type 2 DM. Diabetic nephropathy (DN) is defined as the type of kidney disease that occurs in individuals with diabetes. Diabetic nephropathy is the most common cause of end-stage renal disease and is associated with increased cardiovascular morbidity\(^1\). It is estimated that approximately 20-40% of patients with diabetes will develop diabetic nephropathy\(^1\).

Usually there are no symptoms as the kidney damage slowly develops and damage can begin 5-10 years before symptoms start. Signs and symptoms of diabetic nephropathy include poor appetite, feeling tired most of the time, general ill feeling, headache, nausea, vomiting, swelling of the legs, etc.
Diabetic nephropathy has two distinct stages: microalbuminuria and macroalbuminuria\(^2\) as shown in the Table 8.1:

<table>
<thead>
<tr>
<th>Category</th>
<th>Spot collection (mcg/mg creatinine)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Normal</td>
<td>&lt;30</td>
</tr>
<tr>
<td>Microalbuminuria</td>
<td>30 to 299</td>
</tr>
<tr>
<td>Macroalbuminuria</td>
<td>≥300</td>
</tr>
</tbody>
</table>

### 8.2 Aims of treatment of diabetic nephropathy

- Achieve blood glucose levels within or as close as possible to the normal range
- Slow down the progression of the disease/reduce albuminuria
- Achieve a lipid profile that reduces the risk of cardiovascular disease
- Achieve normal blood pressure levels
- Prevent and/or slow the progression of the complications of diabetes
- To meet nutritional needs of individuals according to their gender, age, preferences, etc.

The most **important modifiable risk factors** for diabetic nephropathy are the following\(^1\):

- Hyperglycemia
- Hypertension
- Glomerular filtration rate
- Smoking
Nutritional Care for Adults with Chronic Kidney Disease

- Dyslipidemia
- Proteinuria levels
- Nutrition
- Exercise

Other non-modifiable risk factors include family history as not everyone with diabetes will develop diabetic kidney disease; as well as ethnicity as patients of African-American, Hispanic and American Indian Origin are more likely to develop diabetic nephropathy.

8.3 Nutritional management of diabetic nephropathy

In the nutritional management of diabetic nephropathy glycaemic control is the main focus for diabetes management. Tight glycaemic control has been shown to delay the development as well as the development of albuminuria\(^1\).

According to the American Diabetes Association (2012) the glycaemic and blood lipid recommendations for nonpregnant adults with diabetes are shown in Table 8.2 and Table 8.3:

### Table 8.2: Glycaemic recommendations for nonpregnant adults with diabetes\(^1\)

<table>
<thead>
<tr>
<th>Glycaemic recommendations for nonpregnant adults with diabetes</th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td>HbA1C</td>
<td>&lt;7.0%</td>
</tr>
<tr>
<td>Pre-prandial capillary plasma glucose</td>
<td>70-130mg/dL (3.9-7.2 mmol/L)</td>
</tr>
<tr>
<td>Peak postprandial capillary plasma glucose</td>
<td>&lt;180mg/dL (&lt;10.0mmol/L)</td>
</tr>
</tbody>
</table>

*Prandial = meal*
Nutritional Management of Diabetic Nephropathy

Table 8.3: Lipid recommendations for individuals with diabetes (American Diabetes Association, 2012).

<table>
<thead>
<tr>
<th>Lipid recommendations for individuals with diabetes</th>
</tr>
</thead>
<tbody>
<tr>
<td>Total cholesterol</td>
</tr>
<tr>
<td>Low-density lipoprotein cholesterol</td>
</tr>
<tr>
<td>High-Density lipoprotein cholesterol</td>
</tr>
<tr>
<td></td>
</tr>
<tr>
<td>Triglycerides</td>
</tr>
</tbody>
</table>

Goals need to be individualised for every patient depending on several factors such as: duration of diabetes, age and life expectancy, comorbid conditions, known CVD and other DM complications, etc (American Diabetes Association, 2012).

Research evaluating the effectiveness of nutrition in diabetes care indicates reduction in HbA1C by 1% in type I and 1-2% in type II diabetes\(^3,4\). As excessive body weight contributes to insulin resistance\(^5\) it is important that overweight or obese individuals reduce and consequently maintain a weight within the normal body mass index (BMI).

8.3.1.1 Weight management

Weight loss is associated with decreased proteinuria and microalbuminuria, however the long-term effect of weight loss on CKD progression is unknown\(^6\). In addition, obesity is an independent risk factor for the onset as well as rate of progression of chronic kidney disease\(^7\).

The National Kidney Foundation recommends a target BMI within the normal range. Weight management programs should include lifestyle measures such as dietary restriction and physical activity, anti-obesity medications if needed along
with appropriate counseling. Bariatric surgery may be considered appropriate based on individual benefits versus risks, for patients with type 2 DM with a BMI>35kg/m².

Although long-term weight loss and maintenance is difficult, short-term studies have demonstrated that 5% weight loss is linked to glycemic control, improved lipid profile and blood pressure. High protein diets are not recommended as a method for weight loss for patients with diabetic nephropathy (See section 8.3.1.3 Protein).

### 8.3.1.2 Carbohydrates

Carbohydrate (CHO) intake should be evenly distributed throughout the day, on a day-to-day basis, as it has been shown to result in improved glycemic control in individuals receiving either nutrition therapy or pharmacotherapy. For those with type 1 DM insulin therapy should be given according to one’s diet and physical activity whereas in those with type 2 DM glucose monitoring will be used to determine if adjustments in meal content and frequency or medications need to be made. In addition:

- CHO counting, exchanges or experience-based estimation can be used in order to monitor carbohydrate intake, as the strategy towards glycemic control.
- Glycaemic index and load in addition to the above might provide a modest additional benefit.
- Sucrose can be substituted with isocaloric amounts of starch but excess energy intake should be avoided.
- There is no evidence to suggest a beneficial percentage of macronutrients for diabetes therefore healthy eating is recommended. The same applies to dietary fibre however it is recommended that individuals with DM consume a minimum of 7-13g of soluble fibre per day as it reduces total cholesterol by 2-3% and LDL cholesterol up to 7%.
• Non-nutritive sweeteners are safe if consumed within the recommended amounts and do not affect glycaemic responses.

• Management of hypoglycaemia: Patients should be advised on safety guidelines to prevent hypoglycaemia which include the following: insulin dose/CHO intake adjustment, use of CHO containing snacks during exercise and blood pressure monitoring. CHOs should be ingested if glucose levels are \(<(5.5 \text{ mmol/l}).\)

8.3.1.3 Protein

Patients with normal renal function and diabetes should consume no more than 20% of their energy intake from protein\(^{11}\). For patients with diabetic nephropathy although there is no definitive evidence to suggest protein restriction (protein intake 0.8-1.0g/kg/day) a recent systematic review\(^{12}\) suggests a 6 month trial of protein restriction in all individuals, with continuation only in those who responded well as there might be individual benefit. This might be particularly considered for patients with progressive nephropathy despite optimal glycemic, blood pressure control as well as blood pressure medications\(^{13}\).

8.3.1.4 Micronutrients

There is no clear evidence of benefit from supplementation with micronutrients such as vitamin E, vitamin C, carotene and chromium in people with no micronutrient deficiencies. In addition there is concern about their long-term safety. A daily multivitamin, however, may be beneficial in older adults with reduced energy intake.
8.3.1.5 Alcohol

Alcohol is safe when consumed within the daily intake, i.e. one drink per day for women or two drinks per day for men. In order to reduce the risk of hypoglycemia during the night when using insulin or insulin secretagogues, alcohol should be consumed with food.

8.3.2 Prevention of Cardiovascular disease

Albuminuria apart from being the first manifestation of nephropathy it also is a marker of increased cardiovascular morbidity and mortality for patients with DM. Therefore the following are important in order to minimize the risk of developing cardiovascular disease:

- Glycemic control
- Balanced diets that are high in fruits, vegetables, whole grains and nuts without excess energy intake.
- Two or more servings of oily fish (containing omega-3 fatty acids) per week.
- Blood pressure control (see below).
- Limit saturated fat to <7% of total energy intake.
- Reduce to a minimum the consumption of trans-fat.
- Limit dietary cholesterol to <200mg/day.

8.3.3 Blood pressure

The UK Prospective Diabetes Study provided strong evidence that improved blood pressure can reduce the risk of macroalbuminuria by 25% in patients with type 2 diabetes whereas in a study by Wrone et al. microalbuminuria was associated with diabetes and hypertension in the highest quintile\textsuperscript{14}. 
The Dietary Approaches to Stop Hypertension (DASH) eating pattern should be incorporated into the hypertensive patients’ diet pattern. The DASH eating pattern is high in potassium, calcium, magnesium and fiber, low in sodium (between 1,500-2,400mg/day)\textsuperscript{15}, and should be used with caution in presence of CKD. In addition it is important to achieve and maintain a weight within the normal range as well as engage in regular physical activity and limit alcohol consumption.

Lifestyle modifications such as those discussed above lead to significant reductions in blood pressure among patients with hypertension\textsuperscript{16-19} however many patients with hypertension will eventually require medications to control their blood pressure. On the other hand some patients already taking one blood pressure medication might discontinue it after intensive nutrition therapy\textsuperscript{20}.

If angiotensin converting enzyme inhibitors or angiotensin receptor blockers are used for the treatment of blood pressure then potassium levels need to be monitored in order to avoid hyperkalaemia\textsuperscript{21}.

\textbf{8.3.4 Exercise and physical activity}

Exercise and physical activity are also important as they have a modest effect on weight loss and maintenance, but also improve sensitivity to insulin independent of weight loss and lower blood pressure\textsuperscript{11}. Although exercise will not improve glycemic control in type I diabetics, it will offer the same benefits as in the general public such as a reduction in the risk of CVD and a sense of well-being\textsuperscript{22}.

\textbf{8.4 Summary}

Diabetic nephropathy is a major diabetes complication, which can lead to increased morbidity and mortality among patients with DM. Early detection and appropriate nutritional
management along with medical treatment when necessary help to delay its onset as well as slow down its progression. When kidney damage progresses this might lead to the need for dialysis or a kidney transplant.
References


Nutritional Needs of Special Populations with Chronic Kidney Disease
9.1 Nutritional management of Chronic Kidney Disease in pregnancy

Women with CKD generally do not become pregnant as frequently as those with normal kidney function. Women with CKD undergoing dialysis have a marked decrease in conception, and fertility usually returns for women with well-functioning kidney transplants, but immunosuppressive drug therapy, especially cyclosporine, has been known to result in the birth of babies who are small for gestational age. These drugs have not been associated with an increase in congenital anomalies, but all groups of women with CKD are at risk for premature births.
Pregnant women with CKD are at increased risk of hypertension, proteinuria, pre-eclampsia, premature labour, urinary tract infections and thrombosis. The most serious problem for women with CKD who conceive is the potential for rapid loss of renal function during pregnancy. This occurs irrespective of the cause of the underlying renal disease, and the risk of rapid progression appears to increase dramatically once a critical degree of baseline renal insufficiency is present.

A woman with CKD who becomes pregnant presents many challenges to the health care team, as substantive research on the specialised medical and nutritional needs for this unique patient population is lacking. Adequate nutrition, including vitamin and mineral therapy, is particularly important during pregnancy to promote a successful outcome. Also the amount of dialysis treatment must be increased during pregnancy to more closely assimilate the function of normal kidneys during fetal development, and women undergoing dialysis must realize the time commitment involved. Pregnancy in the face of kidney disease poses a new dimension to medical nutrition therapy. Individualisation for specific nutrient needs is paramount, whether the patient has renal insufficiency, requires haemodialysis or peritoneal dialysis, or has a renal transplant.

The dietician plays a crucial role in guiding and educating the patient to meet the increased protein, energy, and vitamin and mineral needs of pregnancy protecting the kidney function that affects serum levels of electrolytes, calcium and phosphorous, the volume status and particular the vitamin needs. Weight gain also has to be monitored carefully due to the greater potential for fluid retention with CKD. Women in the early stages of CKD with confirmed pregnancy have to meet dietician as soon as possible and then monthly for follow up assessment, education and plan of an individualised nutrition program. Women undergoing dialysis should be followed up, counseling and educating weekly after the pregnancy is
confirmed for assessment of nutrient intake and laboratory values.

9.1.2 Energy and protein needs

The energy requirements during all stages of CKD and after kidney transplantation meet the requirements for the pregnant women without CKD. Generally, 35 kcal/kg of ideal body weight (IBW) or adjusted IBW per day are prescribed in the first semester and for the second and third semester added to this value at least 300 kcal/d\(^2\). However, definitive information regarding energy requirements for pregnant women with CKD does not seem to exist in the literature.

An important role is selecting the correct energy consumption in pregnant women (with or without CKD) relative to the body weight at the beginning of pregnancy. BMI of women in the first trimester of pregnancy is associated with the risk of adverse pregnancy outcome\(^3\). Caloric intake requirements increase as pregnancy progresses, and healthy maternal weight gain in a woman with a normal BMI should range from 12-17 kilos. Dry Body Weight of pregnant women on dialysis can be difficult to ascertain. During the first trimester, expected weight gain is minimal, while during the second and third trimesters, the dry weight can be expected to increase by up to 0.5 kg/week.

For women with CKD Stages 1-3, protein intake of 0.8 g/kg/d plus 10 g/d added for the pregnancy seems to be reasonable, although specific guidelines are lacking\(^2\). For CKD Stage 4 and end stage without dialysis to introduced a lower protein intake about 0.6 g/kg IBW plus 10 g/day\(^2\). Protein requirements for pregnant women undergoing haemodialysis HD and peritoneal dialysis (PD) are generally 1.2 to 1.3 g/kg IBW plus at least 10 g/day\(^2\). For the pregnant dialysis patients these needs are easier to meet because in this case, the diet can be frequently liberalized for sodium, potassium, and phosphorous content due to increased solute removal with more intensive dialysis.
9.1.3.1 Low-protein Diet

Low-protein diets are considered an important tool in management of CKD patients and are considered to slow CKD progression in selected patients. However, there is little available data on the risk/benefits of a low protein diet in pregnant CKD patients. In one report 12 pregnancies in 11 patients did show that the vegetarian supplemented low-protein diet was a safe option for pregnant CKD patients with good maternal and fetus outcomes. The median week of delivery was 32 weeks, only one patient had doubling of serum creatinine, and no mothers required dialysis4.

9.1.3.2 Vitamins and minerals

Vitamin and mineral requirements for pregnant adults in the earlier stages of CKD or after kidney transplantation are almost the same as for the general healthy population during pregnancy. Pre-natal vitamins are usually prescribed to meet these needs, including at least 11mg of zinc, 27 mg of iron, 800 μg of vitamin A per day, but excess should be avoided.

Pregnant women undergoing dialysis treatment are frequently prescribed a double dose of a daily multivitamin and prescribed 5 mg of folic acid or renal vitamins with greater than 1 mg of folic acid because water-soluble vitamins and minerals can be removed by intensified dialysis. The good clearance associated with intensified dialysis allows for an unrestricted diet.

Dialysate potassium concentration is typically increased to 3.0 mEq/l, and phosphate is supplemented by the addition of sodium phosphate (fle enema) to the dialysate. Supplemental calcium, when necessary, is given in addition to what is provided in a prenatal vitamin during the early stages of CKD and after transplantation. At least 1000 mg/d is recommended from diet and supplements to meet the increased requirements for skeletal development in the
Nutritional Care for Adults with Chronic Kidney Disease

fetus. Calcium intake requirements for women undergoing dialysis increase as pregnancy progresses. Calcium intake must include an additional 30 g/day primarily during the third semester for fetal skeletal development. For this reason, the dialysate calcium concentration often needs to be increased to 3.25 mEq/l (1.75 mmol) or higher to ensure adequate supplementation.

Vitamin D analogs have been given intravenously during hemodialysis to pregnant women who need suppression of parathyroid glands. Dialysate calcium must be adjusted so that PTH is kept within Kidney Disease Outcomes and Quality Initiative guidelines and bone derived alkaline phosphatase stays in normal levels. For this reason serum phosphorous may be lower than expected as a result of its incorporation into the fetal skeleton. This should be addressed by further increase in dialysate phosphate as necessary to normalized pre- and post- dialysis values.

Anaemia usually occurs during pregnancy; iron is generally provided in a prenatal vitamin preparation for women with CKD Stages 1 to 3 and after kidney transplantation. For those women undergoing haemodialysis, intravenous iron has been safely given during the treatment to work in conjunction with epoetin alfa (erythropoeitin stimulating agents). Women who are receiving peritoneal dialysis may be prescribed oral forms of iron, as well as intravenous iron. The values provided should be based on achieved the goals for haemoglobin status, transferrin saturation and ferritin utilised in the general dialysis population. In case anaemia worsens usually it is necessary to increase the requirements for iron and erythropoietin, particularly for women undergoing haemodialysis. Thus, erythropoietin requirements can be expected to at least double and typical iron requirements exceed the usual 30 mg/day recommended for healthy pregnant women.
9.1.3.3 Fluids and sodium intake

Careful clinical assessments of fluid status remains the best mechanism to determine appropriate ultrafiltration goals and guide blood pressure treatment, and is critical to assist with the diagnosis of pre-eclampsia. In the absence of urine output revealing proteinuria, the diagnosis of pre-eclampsia relies on the assessment of worsening blood pressure.

Sodium allowances for pregnant patients with early stages of CKD, while undergoing dialysis or after kidney transplantation depend upon fluid retention and hypertension. Women undergoing haemodialysis, when dialysed more frequently, can generally tolerate higher sodium intakes without excessive interdialytic fluid weight gains.

9.2 Kidney disease in Patients with HIV infection and AIDS

As patients infected with human immunodeficiency virus (HIV) live longer while receiving antiretroviral therapy, CKD has emerged as a significant cause of morbidity and mortality. Black race, older age, hypertension, diabetes, low CD4+ cell count, and high viral load remain important risk factors for CKD in this population. Primary renal disease is also associated with HIV infection. In 1984 a specific form of HIV associated primary renal disease was first described. This is known as HIV associated nephropathy (HIVAN) and is characterized by proteinuria, often of sudden onset, with rapidly progressive renal dysfunction, resulting in end stage renal disease (CKD requiring dialysis) over a few months.

9.2.1 Nutrition guidelines for patients with kidney disease and HIV/AIDS

Nutritional advice and monitoring are a crucial part of the management of patients with CKD and HIV infection. Clinical
guidelines for the nutrition care of the patient with HIV and kidney disease have not been formally established. For the time being the best nutritional intervention for the HIV/AIDS patient with CKD is individualised assessment, integrating the specific nutrition challenges of HIV/AIDS with the appropriate guidelines for patients with CKD and for those receiving replacement therapy.

Nutritional challenges specific to the patients with HIV can be treated by first recognising the factors contributing to the development of nutrition problems and then categorising and identifying the problems. The dietician may need to schedule a plan for nutrition screening to develop an individualized intervention. The Nutritional and Cardio metabolic events with HIV infection are illustrated in Figure 1.

*Figure 1. Nutritional and cardiometabolic events with HIV infection*
Wasting syndrome

Severe malnutrition and weight loss especially loss of lean tissue and delayed weight gain has been associated with an increase risk of morbidity in this patient population. Patients with kidney disease and HIV with active weight loss should be evaluated for gastrointestinal disease, malignancy, opportunistic infections, hypogonadism and adrenal insufficiency.

The main factors contributing to wasting syndrome are:

- Reduced appetite and or taste alterations as a side effects of the medical therapy
- Fever
- Sore mouth due to oral and oesophagical infections caused by *Candida*
- Diarrhoea caused by bacterial infections by *Salmonella*, *Mycobacterium avium intercellular*, CMV or parasitic infections like *Giardia*, *C. parvum*, and *E. Bieneusi*
- Nausea/vomiting as a side effect of medications

It is quite difficult to differentiate underlying causes of wasting in the HIV/AIDS patient who also has kidney disease. Chronic uraemia is often characterized by wasting of muscle and fat mass, which has been defined as protein-energy wasting (PEW), and is responsible increasing morbidity and mortality, mostly from cardiovascular events. Extensive research in this field has been indicating that the pathogenesis of PEW in kidney disease is complex and multifactorial. Complexity involves underlying metabolic alterations, including inflammation, oxidative stress, and insulin resistance. In addition, patient heterogeneity is increasing with large numbers of obese individuals as a result of the ongoing obesity epidemics. Several issues are involved in discussion and contribute to metabolic derangements, including adipose tissue, the gut, and the central nervous system, with novel mediators including the gastric hormone ghrelin.
Renal osteodystrophy

CKD is associated with renal osteodystrophy. In HIV infected patients the prevalence of bone problems especially osteopenia and osteoporosis is increasing\textsuperscript{11}. Although the underlying mechanisms triggering bone loss in HIV-infected patients are not completely defined, traditional risk factors, HIV infection itself, HIV-associated fat redistribution, antiretroviral therapy for HIV infection, and increased production of proinflammatory cytokines (such as TNF-α and IL-6) may influence osteoclast activation and bone resorption\textsuperscript{12}. HIV-infected patients have been also been reported to have low baseline and maximal secretion of parathyroid hormone and 1,25-dihydroxyvitamin D3 levels\textsuperscript{12}. Although data on bone disease in patients with CKD and HIV infection is not available in the literature, it is known that HIV-infected patients with CKD develop complications of altered calcium and phosphate metabolism similar to HIV-negative patients\textsuperscript{12}. Medical nutrition intervention currently recommended includes daily: 1500 mg calcium, 400 to 1000 IU Vitamin D, and moderate physical activity.

Lipodystrophy

Treatment with antiretroviral agents - protease inhibitors in particular - has uncovered the syndrome of lipodystrophy. The term lipodystrophy is used to describe a diverse group of disorders characterized by body composition and metabolic alterations. Changes in body composition include lipoatrophy, which is complete or partial loss of adipose tissue compartments, and lipohypertrophy, which is a pathological accumulation of adipose tissue in distinct body compartments. Lipoatrophy and lipohypertrophy may or may not coexist. Metabolic abnormalities include insulin resistance and dyslipidemia. Hepatic steatosis may also be present. The severity of these co-morbidities usually correlates with the degree of adipose tissue loss\textsuperscript{13}. 
The cause of the syndrome seems to be multifactorial; however, its exact mechanisms have yet to be clarified. Accelerated risk for cardiovascular disease is likely to be a major concern in these patients in the future. The available treatment options range from switching the different antiretroviral drugs and lifestyle modifications to the use of pharmacologic agents to treat patients with dyslipidemia, impaired glucose tolerance and/or diabetes, and changes in body composition\textsuperscript{14}.

Nutrition recommendations for patients with HIV/AIDS lipodystrophy include nutritional changes (namely low saturated fat intake, and increased intake of dietary fibre and low glycaemic index carbohydrates) and regular physical exercise\textsuperscript{15}.

### 9.3 Nutrition management of chronic kidney disease in the elderly

The National Health and Nutrition Examination Survey (NHANES) suggest that chronic kidney disease (CKD) prevalence is higher in the elderly\textsuperscript{16}. Furthermore, data from population-based prospective cohort studies, such as the Community Health Study (CHS) add from clinical populations such as Kaiser Permanente, support that risk of cardiovascular disease morbidity and mortality associated with decreased kidney function is dramatically increased in the elderly\textsuperscript{17}.

The cause of CKD is often not readily apparent in many elderly patients. Epidemiological evidence suggests that vascular disease may be the predominant etiology for CKD in this population. Many CVD risk factors, including diabetes, hypertension, and obesity, are prevalent in patients with CKD and are associated with albuminuria and decreased GFR\textsuperscript{18}.

Current guidelines available for the management of CKD are intended to be applied to adult patients in general and guidelines specific to older adults with CKD do not exist. However, most nephrologists would agree that caring for
older patients with CKD presents unique challenges that arise because of the substantial differences between older and younger patients with this disease.

9.3.1 Malnutrition in the elderly with CKD

Affects almost one-third of the dialysis patients have mild to moderate malnutrition, while 6-8% have severe malnutrition, which is associated with increased morbidity and mortality rates and numerous pre-existing factors directly correlated with, or existing prior to, renal replacement therapy. Moderate to severe malnutrition, accounting for 10-30% of the cases, however, is an independent risk factor for death among the elderly. Many of these patients also suffer from cardiovascular diseases and other co-morbidities, which, independent of any underlying uraemia and/or dialysis, impacts negatively on both their quality of life and clinical status. Moreover, their condition is often further exacerbated by dialysis itself, with its acute (e.g., hypotension and sensorial alterations) and chronic complications, including an exacerbation of malnutrition and systemic vascular disease.

Malnutrition in the third age is a multifactorial problem, consisting of physiological, social and economic parameters, often referred as the “nine d’s”, namely poor dentition, dysgeusia, dysphagia, diarrhoea, depression, disease, dementia, dysfunction and drugs, factors which often coexist, resulting in compromised nutritional intake and subsequent poor nutritional status. Malnutrition can occur secondary not only to insufficient and/or unbalanced nutritional intake or uraemia, but it may also depend on the patient’s level of tolerance to dialysis and on the dialysis modality. Despite the improvements made to dialysis techniques, the nutritional condition of elderly patients on dialysis for chronic renal failure remains a cause for concern.
9.3.2 Aims of nutritional therapy

- To promote a balanced and healthy dietary plan, covering the special needs of the elderly patients, without a negative effect on the quality of life of the patient.
- To preserve good nutritional status and prevent malnutrition and sarcopenia.
- To control biochemical parameters related to CKD and retain them between acceptable range.
- To optimize fluid control.

9.3.3 Energy intake

The sufficient energy intake is vital in older renal patients in order to prevent malnutrition, to maintain Body Mass Index (BMI) within the normal range and to achieve a nitrogen balance. According to the recent recommendations the provision of 35 kcal/kg can cover the needs of the majority or pre – and dialysis renal patients. In patients over 60 years it is recommended that lower energy intake, i.e. 30 kcal/kg/ day could sufficiently cover their needs, due to the reduced energy expenditure of the above mentioned population, especially when they lead a sedentary way of life.

Elderly patients are more susceptible to undernutrition comparing to younger adults due to physiological, psychological and socioeconomic reasons. Anorexia is more common in older patients, which is closely related to age associated alterations in taste and smell, loss of dentition, depression and cognitive decline due to ageing. In End Stage Renal Disease (CKD) anorexia and malnutrition affect over 50% of the patients over the age 65 years, and are probably even more frequent in diabetics. For the prevention of energy – protein malnutrition in clinically stable chronic haemodialysis patients, energy intake should be 30-40 kcal/day, adjusted for age, gender, physical activity levels.
For patients undergoing peritoneal dialysis, calories absorbed from the dialysate should be included in the assessment of the nutritional needs of the patients. 60 -70% of the dextrose of the dialysate is absorbed during its dwell in the peritoneal cavity\textsuperscript{26}. Despite the caloric intake by the dextrose in the dialysate, malnutrition is common in PD patients, due to the feeling of fullness that the dialysate causes the patient, the loss of appetite and/or the increased energy needs in case of peritonitis. Therefore, the energy intake for elderly PD patients should be 30 kcal/kg/day, including the calories for the dialysate\textsuperscript{22}.

9.3.4 Protein intake

Close control of protein intake is vital for patients with Chronic Kidney Disease (CKD), Stages 4 – 5. According to a secondary analysis or the results of Modification of Diet in Renal Disease (MDRD) study, a favourable effect of the tight control of blood pressure and modification of protein intake (i.e. 0.6 g/kg/day) in pre-dialysis patients with Glomerular Filtration Rate (GFR)< 25 ml/min was revealed\textsuperscript{27}. A recent Cochrane review supported the benefit of the reduction in protein intake in preserving renal function and delaying the progression of renal disease. [28]. Nevertheless, the evidence about the need of exact level of protein intake reduction remains inconclusive\textsuperscript{28,29}.

Considering both the absence of studies in older adults and the potential hazards of a long term restrictive in protein diet, it is suggested that the recommendations for older adults should be analogous to the ones for the younger ones. According to the NKF/ KDOQI recommendations protein should be limited to 0.6 g/kg/day for patients with GFR<30 ml/min, while in EDTNA/ERCA guidelines protein intake for CKD patients can range from 0.6 – 1.0 g/kg/day. Emphasis on the biological value of the proteins should be given, as more than 50% of the protein intake should be of high biological value (i.e. protein from meat, poultry, dairy products and soya)\textsuperscript{22,30}. Protein
intake should be higher than 1.0g/kg/day in patients with overt malnutrition and/or older than 80 years. In these patients the risk of malnutrition is probably higher than any benefit from the protein restriction\textsuperscript{31}.

In patients with CKD recommendations are similar to the ones for younger adult patients. Protein intake is more liberal due to the protein and amino-acid losses during the dialysis. These losses can be as high as 10-12 g/session of haemodialysis\textsuperscript{32} and should be replaced for the achievement of a positive nitrogen balance in these patients. Therefore, dietary intake in stable haemodialysis patients should be between 1.0 and 1.2 g/kg/day, with emphasis on proteins of high biological value\textsuperscript{22}.

In patients undergoing peritoneal dialysis (PD) protein loses in the dialysate can vary greatly, ranging from 4-12 g/day. In cases of peritonitis these losses can be raised up to 70%, resulting in a negative nitrogen balance. Therefore, protein intake of 1.0g/kg/day is considered be the minimum for stable, non-catabolic patients. Higher levels of protein intake are recommended for patients with peritonitis or catabolic stress, when it is suggested an intake of 1.5 g/kg/day\textsuperscript{33}.

### 9.3.5 Potassium

Hyperkalaemia is common in patients with Stage 4 to 5 CKD, raising their risk of sudden cardiac death. Renal insufficiency, constipation, metabolic acidosis, lean body mass catabolism and insufficient dialysis are some of the causes of hyperkalaemia in these patients\textsuperscript{34}. Moreover, medical therapies such as angiotensin – converting enzyme inhibitors (ACEI), angiotensine receptive blockers (ARB), beta-blockers, potassium sparing diuretics, non steroidal anti-inflammatory drugs, corticosteroids and cyclosporine use can contribute to hyperkalaemia\textsuperscript{35}.

Recommendations regarding potassium intake do not differ in older adults from the younger In patients with Stage 4
renal disease, serum potassium should be monitored and potassium intake restrictions should be followed in case of abnormal laboratory values\textsuperscript{22}. In patients with Stage 5 renal disease, potassium intake should be limited from 2,000 to 3,000 mg/day (8-17 mg/kg/day). In haemodialysis patients with pre-dialysis serum potassium > 6 mmol/l, a daily intake of potassium of 1950 – 2730 mg (50-70 mmol) or 1 mmol/kg IBW is recommended\textsuperscript{22,25}.

Since normal bowel function can also contribute to normal serum potassium values, older people often suffer for hyperkalaemia due to constipation. Constipation is a common problem in older age and since large intestine increases stool potassium content in order to compensate the renal insufficiency, high blood potassium levels are rather common. Therefore, the prevention of constipation can help in achieving normal serum potassium levels\textsuperscript{35}. And since restriction in potassium intake include restriction in whole grain foods, fruits and vegetables, dietary fibre supplements may be a solution to the prevention of constipation.

\textbf{9.3.6 Sodium and Fluids}

Sodium restriction is essential in elderly patients with CKD. Keeping in mind that older people often suffer from heart failure apart from CKD, sodium intake should be rather limited on a daily basis\textsuperscript{31}. In CKD, as urine output decreases, sodium filtration decreases as well. In Stage 4 and 5 CKD patients’ sodium intake should be limited to 2000 – 2300 mg/day (80 – 100 mmol/l)\textsuperscript{22,25}. Sodium restriction can also contribute to better fluid intake control through lowering the thirst sensation\textsuperscript{36}.

Fluid intake in Stage 4 CKD should be restricted only if edema or fluid retention is experienced. In patients undergoing haemodialysis, hypertension is strongly connected to excess fluid intake between hemodialysis sessions. Fluid control should be monitored by the interdialytic weight gain, which
should be approximately less than 1 kg per day, or 4 – 4.5% of dry body weight (oedema free body weight) to avoid fluid overload.\textsuperscript{22,25}. To achieve this fluid intake should be matched to the volume removed during the treatment and should be limited to 1000 ml plus the remnant urine output/day\textsuperscript{22}. In older adults the reduction of the sensation of thirst may facilitate the restriction in fluids. Lower fluid intake though may increase the risk of constipation and subsequent hyperkalaemia. In PD patients the fluid equilibrium is controlled by ultrafiltrate (i.e. the fluid removed by the dialysate) and dietary fluid restriction is easier to be achieved.

\textbf{9.3.7 Phosphorus and calcium}

Hyperphosphataemia is a common problem for patients with late stages of CKD. Phosphorus retention is directly connected to the development of secondary hyperparathyroidism\textsuperscript{37}. Serum phosphate should be kept within the range of 2.7–4.6 mg/dl (0.87 – 1.49 mmol/L) in patients with GFR between 15 and 59 ml/min (Stage 3 and 4 patients). Dietary phosphorus intake should be limited in the case of abnormal laboratory values of serum phosphate. In patients with early stages of CKD, who follow a low protein diet, the restriction in phosphorus is easy to be achieved, since they are advised to limit their intake of the main dietary sources of this mineral (i.e. dairy products, meat and animal protein)\textsuperscript{22}.

Disturbed balance of phosphorus and calcium can increase the soft tissue and vascular calcification, leading to higher cardiovascular disease prevalence and mortality in patients with Stage 3 to 5 CKD\textsuperscript{38}. Therefore, the balance between phosphate and calcium is crucial. More specifically, the calcium phosphate product (corrected calcium x phosphate) should be maintained < 55 mg\textsuperscript{2}/dl\textsuperscript{2} in these patients\textsuperscript{22}.

In HD and PD patients the restriction of dietary phosphate is more complex, and patients should be advised from a renal
dietician, in order to limit their phosphate without limiting their protein intake. According to the K/DOQI and the European guidelines, phosphate intake should be limited to 800 – 1000 mg/ day. High protein foods, with lower phosphate content should be recommended, in combination with non-dietary strategies, i.e. phosphate binders. Calcium intake should not exceed 2000 mg, including calcium obtained from calcium-based phosphate binders, for the management of renal bone disease and metabolism\textsuperscript{22,30}. 
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References


Nutritional Care for Adults with Chronic Kidney Disease


Malnutrition in the CKD Patient
10.1 Malnutrition-definition

Malnutrition is a general term indicating a state of nutrition in which a deficiency or excess (or imbalance) of energy, protein, and other nutrients causes measurable adverse effects on tissue/ body form (body shape, size and composition) and function, and clinical outcome\(^1\). The first and most important type is protein-energy malnutrition (PEM) or protein-energy wasting (PEW), which is defined as a lack of sufficient energy or protein to meet the body`s metabolic demands\(^2\). The causes for malnutrition are:

a. inadequate dietary intake of protein,
b. intake of poor quality dietary protein,
c. increased demands due to disease,
d. increased nutrient losses\(^3\).

10.2 Epidemiology and pathophysiology

Overnutrition is commonly encountered in general population and a major risk factor for developing metabolic syndrome and cardiovascular disease (CVD), with subsequent increase in risk of mortality\(^4\). However, in chronic kidney disease (CKD), and especially in the maintenance dialysis patients, the so-called uremic malnutrition (also known as PEW) is one of the strongest predictors of mortality\(^2,4\). Also, the prevalence of PEM increases progressively along with the loss of residual renal function. CKD Stages 3 and 5 are associated with spontaneous reduction of the mean protein intake from 1.0 gr/kg body weight/day to about 0.5 g/kg body weight/day accompanied by a reduction in energy intake\(^3\).

Various factors contribute to malnutrition in CKD patients and they are presented in Table 10.1. Alterations in protein...
metabolism caused by uraemia and gastrointestinal tract function can result in poor nutritional profile, which in turn increases the risks of CVD and infections\(^2\). But the increase of the CVD prevalence in CKD patients may be attributed to other, non-traditional risk factors (other than old age, lifestyle, smoking, hypertension, dyslipidaemia, diabetes, left ventricular hypertrophy, heart failure) which per se may promote endothelial dysfunction and/or atherogenesis.

The phenomenon of the “reverse epidemiology” in dialysis patients is thought as an example of the relevance of non-traditional risk factors\(^2\). The paradox is that in dialysis patients the effect of overweight or obesity leads to an improved survival with decreased cardiovascular risk and with all-cause mortality. During the course of CKD, the mechanisms responsible for malnutrition and the increase in cardiovascular risk in CKD are the loss of kidney excretory and metabolic functions proceed together with the activation of pathways of endothelial damage, inflammation, acidosis, alterations in insulin signalling and anorexia which are likely to orchestrate net protein catabolism and the protein-energy wasting (PEW) syndrome\(^2,3\).

**Table 10.1. Factors contributing to wasting in CKD patients (3, 5)**

<table>
<thead>
<tr>
<th>Anorexia due to:</th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td>Nausea, emesis, medications</td>
<td></td>
</tr>
<tr>
<td>Uraemia/uremic stage of metabolism</td>
<td></td>
</tr>
<tr>
<td>Under - dialysis</td>
<td></td>
</tr>
<tr>
<td>Accumulation of uremic toxins not completely removed dialysis</td>
<td></td>
</tr>
</tbody>
</table>

**Inflammation**

<table>
<thead>
<tr>
<th>Contributing anorexia</th>
<th>Inducing catabolism</th>
<th>Due to co morbidities</th>
</tr>
</thead>
<tbody>
<tr>
<td>Related to the dialysis procedure (such as impure dialysate, backfiltration)</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>
### 10.3 Diagnosis of malnutrition

A comprehensive evaluation of the nutritional status of a patient can best be judged by a combination of clinical parameters, laboratory findings and certain technical examinations.

<table>
<thead>
<tr>
<th>Metabolic Acidosis</th>
</tr>
</thead>
<tbody>
<tr>
<td>Endocrine Disorders</td>
</tr>
<tr>
<td>Insulin resistance</td>
</tr>
<tr>
<td>hyperparathyroidism</td>
</tr>
<tr>
<td>Impaired response to IGF-1</td>
</tr>
<tr>
<td>Comorbidity</td>
</tr>
<tr>
<td>Infections</td>
</tr>
<tr>
<td>Diabetes mellitus (diabetic gastroparesis)</td>
</tr>
<tr>
<td>CVD</td>
</tr>
<tr>
<td>Dental problems</td>
</tr>
<tr>
<td>Dialysis-Related</td>
</tr>
<tr>
<td>Inadequate doses</td>
</tr>
<tr>
<td>Bio-incompatible membranes</td>
</tr>
<tr>
<td>Loss of amino acids and protein loss</td>
</tr>
<tr>
<td>Reuse with bleach, nausea, hypotension</td>
</tr>
<tr>
<td>Psychosocial</td>
</tr>
<tr>
<td>Depression</td>
</tr>
<tr>
<td>Low physical activity</td>
</tr>
<tr>
<td>Loneliness</td>
</tr>
<tr>
<td>Poverty</td>
</tr>
<tr>
<td>Inadequate Dietary Recommendations</td>
</tr>
<tr>
<td>Immobility and Reduced Ability to Purchase Food</td>
</tr>
<tr>
<td>Mechanical Compression of Stomach and Intestine in Polycystic Kidney Disease</td>
</tr>
</tbody>
</table>
10.3.1 Clinical parameters

10.3.1.1 Clinical symptoms

Continuous weight loss, reductions in subcutaneous fat and in muscle mass in specific locations and in more advanced stages of malnutrition vomiting, diarrhea and ascites.

10.3.1.2 Body weight

In order to interpret body weight, it is necessary to calculate oedema-free normal body weight (men: height (cm)-100; women:[height (cm)-100]-10%).

Unintentional loss of dry weight over time: 5% over 3 months or 10% over 6 months is a diagnostic tool for PEW.4,5

10.3.1.3 Body mass index (BMI)

BMI <23kg/m² is a diagnostic tool for PEW

10.3.1.4 Anthropometry

Reduced mid-arm muscle circumference (<10th percentile) is a diagnostic tool for PEW.4,5

10.3.1.5 Nutritional Scoring systems

Malnutrition -inflammation score ≥5, Subjective Global Assessment and its modifications in the malnourished range and other scoring tools in the malnourished range are diagnostic tools for PEW.4,5

10.3.1.6 Assessment of dietary protein and energy intake

Dietary protein intake <1 gr/kg per day for dialysis patients, or <0.5 gr/kg per day for patients with nondialysis-dependent
Nutritional Care for Adults with Chronic Kidney Disease

CKD and dietary energy intake <25 kcal/kg per day for at least 2 months are diagnostic tools for PEW\textsuperscript{4,5}.

10.3.1.7 Assessment of appetite
Relative anorexia which means subjectively reported poor appetite is a diagnostic tool for PEW\textsuperscript{4,5}.

10.3.2 Laboratory parameters
Laboratory parameters allow determination of the visceral protein levels and a longitudinal assessment of nutritional status

10.3.2.1 Total protein (T1/2: 6 weeks)
It is a long-term parameter of nutritional status and mainly represents serum albumin levels\textsuperscript{3}.

10.3.2.2 Serum albumin
Nutrient intake is not the single cause of hypoalbuminemia in CKD patients (Table 10.2). Serum albumin serves as an indicator of nutritional status, as a negative acute phase reactant and also as an indicator of chronic inflammation. Diagnostic criteria are serum albumin levels <40g/l for hemodialysis patients, or 38g/l for peritoneal dialysis patients and non-dialysis CKD patients\textsuperscript{2,4-6}.

Table 10.2. Factors that may affect serum albumin levels in CKD patients (4, 5)

<table>
<thead>
<tr>
<th>Inflammation</th>
</tr>
</thead>
<tbody>
<tr>
<td>Poor energy and protein intake</td>
</tr>
<tr>
<td>Catabolic and anabolic processes</td>
</tr>
</tbody>
</table>
Malnutrition in the CKD Patient

<table>
<thead>
<tr>
<th>Age</th>
<th>Co-morbidity (CVD, diabetes mellitus)</th>
<th>External protein losses (urine, dialysate)</th>
<th>Fluid overload</th>
</tr>
</thead>
</table>

10.3.2.3 Prealbumin (T1/2 : 2-3 days)

Serum prealbumin levels <300 mg/l (for maintenance dialysis patients)$^{4,5}$

10.3.2.4 Transferrin (T1/2 : 8 days)

Serum transferrin levels <1.85 μmol/l$^{4,5}$.

10.3.2.5 Serum cholesterol

Serum total cholesterol levels <2.59mmol/l$^{4,5}$.

10.3.2.6 Serum phosphate

Low levels of serum phosphate in dialysis patients without receiving phosphate-binding agents indicates poor dietary protein intake$^3$.

10.3.2.7 Bicarbonate

Pre-dialysis bicarbonate levels should be ≥22mmol/l$^3$.

10.3.2.8 C-reactive protein (CRP)

It is not an indicator of nutritional status but can be helpful in interpreting levels of other visceral proteins$^3$. 
10.3.2.9 Serum creatinine

Low serum creatinine concentration (adjusted for renal status), or low calculated creatinine appearance is a diagnostic tool for PEW\textsuperscript{4,5}.

10.3.2.10 Pre-dialysis blood urea nitrogen (BUN) concentration

A repeatedly low pre-dialysis BUN may be used as an indicator for poor dietary protein intake\textsuperscript{3}.

10.3.2.11 Normalized protein equivalent-nitrogen-appearance (nPNA)

It is not a sensitive indicator of realistic protein intake, but it is helpful for monitoring the nutritional status during treatment of malnutrition\textsuperscript{3,5}.

10.3.3 Technical methods

10.3.3.1 Dual energy X-ray

Although the best method available for analysis of body composition, DEXA is rather expensive and it cannot be used for routine clinical use. Total body fat percentage <10\% and reduced lean body mass >5\% over 3 months or >10\% over 6 months are diagnostic tools for PEW\textsuperscript{2,4,5}.

10.3.3.2 Bioimpedance

It measures the body water content and body cell mass (BCM), but its value is controversial\textsuperscript{3,5}.

10.3.3.3 Hand grip muscle strength

It is important for routine clinical use as a measurement of muscle strength and nutritional status\textsuperscript{3}.
10.4 Dietary recommendations for the malnourished patient with CKD Stages 3-5

10.4.1 CKD Stage 3 (glomerular filtration rate (GFR) 60-30 ml/min per 1.73 m²)
For malnourished patients with CKD Stage 3 the recommendations for protein and energy intake are 0.6-0.8 g/Kg body weight/day and at least 35 kcal/Kg body weight respectively. According to a meta-analysis, reducing protein intake appears to slow progression to kidney failure³⁷. In nephrotic syndrome the dietary protein supplementation to compensate for renal protein loss is not recommended. Generally, the dietary protein restriction bears the risk for development of malnutrition. It is also important than any dietary intervention be followed by dietary counselling.

10.4.2 CKD Stage 4-5 (GFR< 30 ml/min per 1.73 m²)
For patients with advanced CKD the recommendations for protein and energy intake are 0.6-0.8 g/Kg body weight/day and 30-35 kcal/Kg body weight respectively³⁷. Because patients with uncontrolled dietary intake usually report a decline in spontaneous protein and energy intake which is particularly notable below a GFR of about 25 ml/min together with a progressive decline in anthropometric values, and biochemical markers of nutritional status, dietary protein restriction should be discontinued if malnutrition is diagnosed.

10.4.3 Maintenance dialysis Treatment
Patients on maintenance dialysis have increased dietary protein requirements due to catabolic effects of the dialysis procedure itself and to the loss of amino acids and proteins through the dialysate³⁸⁹. As the condition becomes chronic, the patients also have increased basal energy expenditure and energy requirements. For these patients the recommendations
for protein and energy intake are greater or equal to 1.0-1.2 g/Kg body weight/day (based upon oedema-free normal weight) and 30-35 kcal/Kg body weight respectively\textsuperscript{3,7}. Furthermore, any peritoneal protein loss in peritoneal dialysis patients should be compensated for by increased dietary protein intake. Energy intake should correspond to the activity level of the patient. It has also been shown that the consumption of an energy- and protein-rich meal during the hemodialysis session decreased whole-body protein breakdown and increased protein synthesis and oxidation abolishing the negative effect of dialysis on whole protein balance\textsuperscript{3,9,10}.

10.5 Treatment of malnutrition in dialysis patients

Although there is a small number of studies on treatment of malnutrition in dialysis patients, some of them support the theory that the increases in protein and energy intake can lead to increases in body weight and serum albumin concentration\textsuperscript{3,9,10}. It is very important that before any dietary intervention, the target oedema-free standard weight of the patient should be determined with the use of NHANES tables or the BMI cut-offs. The patient should be recommended to maintain a dietary protein intake of at least 1.2g/kg target weight/day\textsuperscript{3,7}. The energy intake should be adjusted to physical activity levels between 30 and 35 and maximally 40-45 kcal/kg target weight/day. Fat intake should not exceed the 30% of the total caloric intake\textsuperscript{3,7}.

Although PEW might be the result of non-nutritional conditions, dietary interventions such as enteral feeding with high-protein meals or supplements might improve nutritional status and outcomes\textsuperscript{4}. If the patient is severely malnourished, then tube feeding or the placement of a percutaneous gastrostomy tube may be considered.

In the fields of diagnosis and therapy of malnutrition (Figure 10.1.), patients with advanced CKD (Stages 4 and 5 without...
Malnutrition in the CKD Patient

dialysis) should be examined at least once a year for the presence of malnutrition\(^3\). With the initiation of dialysis, the nutritional status of the patient should be determined within 4-6 weeks and in regular intervals every 6-12 months following the initial examination. Screening involve a number of parameters (Figure 10.1), but if malnutrition is suspected, additional diagnostic procedures should be introduced.

Based on the results of the further assessment of the nutritional status, a decision on the need for dietary intervention should be made. If there is no evidence for malnutrition, additional dietary supplements are unnecessary and recommendations for protein and energy intake should involve the oedema-free normal body weight\(^7\).

In case of established malnutrition, a trained renal dietician should initiate the dietary counselling of the patient\(^3,7\). Possible causes of malnutrition (Table 10.1) should be treated and the administered dialysis doses (Kt/V) should be adapted to the patient’s target weight. The dialytic adequacy is very important for the prevention and the treatment of malnutrition. Daily haemodialysis or six haemodialysis sessions per week improves appetite and food intake\(^8-10\). The decreased appetite and food intake is usually caused by decreased dose of medications (e.g. phosphate binders), strict dietetic prescriptions and higher levels of uremic toxins.

In case of mild to moderate malnutrition, there is a need for increased dietary protein and energy intake, according to the pre-defined oedema-free target weight\(^3\). The use of specific dietary supplements may not be necessary, but the increased protein intake may cause the development or worsening of hyperphosphatemia which is treated with phosphate binders. In the fields of severe malnutrition, a sole increase in spontaneous dietary energy intake usually is not sufficient. For these patients, oral administration of dietary products formulated especially for dialysis patients (high protein and energy density with low potassium and phosphorus content)
Nutritional Care for Adults with Chronic Kidney Disease

is required\textsuperscript{3,8-10}. Intradialytic administration of oral supplements may improve patient compliance and motivation. If the above strategies fail, then insertion of a gastric tube or a percutaneous gastrostomy tube (PEG) should be proposed. Intraperitoneal or intradialytic parenteral nutrition should be considered for patients with PEW whenever enteral interventions are not possible or ineffective\textsuperscript{3, 7}.

After the initiation of the therapy, the monitoring should consist of regularly analyzed dietary records, nutrition-related laboratory parameters and nPNA and assessment of nutritional status\textsuperscript{3}. The nutritional status should be assessed regularly at 6-8 weeks intervals. Nutrition therapy must be intensified if malnutrition persists or worsens. Also in cases of moderate malnutrition and resistance to dietary interventions, the use of gastric of PEG tube should be considered early enough to prevent further deterioration\textsuperscript{7}.

Ideally, in dialysis units, patients and nursing staff should be aware of the importance of adequate patient nutrition and regularly participate in courses related to nutrition. Dietary counselling should be established for every new dialysis patient from the trained renal dietician with the help of the ‘nutrition specialist’ who should be designated and trained from amongst the nursing staff. The existence of cooperation with other dialysis units is of great importance, in order to facilitate access to and exchange of nutrition-specific information. Finally, nephrologists should be aware of current scientific advances in the fields of nutrition related to CKD patients.
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**Fig. 10.1. Flow chart for diagnosis and treatment of malnutrition in CKD patients.**

**Screening of nutritional status**
- Dietary history
- Interdialytic weight gain
- Long-term weight change
- Body-Mass-Index (BMI)
- Serum albumin, cholesterol, phosphate
- Pre-dialysis BUN

**Dietary recommendations for eunourished patients**
- Protein: 1.0-1.2 g/kg b.w./day
- Energy: 35-35 kcal/kg b.w./day

**Extended diagnostic steps**
- Subjective global assessment
- Prealbumin, Transferrin
- Bicarbonate
- CRP
- nPNA

**Therapy initiation**
- Dietary counseling
- Dietary diaries
- Search for causes (Table 1)
- Definition of target weight (t.w.)
- Adjustment of dialysis dose

**Therapy monitoring:**
- Dietary counseling
- Dietary diaries
- Assessment of nutritional status
- Adjustment of dialysis dose

- Increase spontaneous nutrient intake to:
  - Protein: 1.2 gr./kg t.w./day
  - Energy: 35 kcal/kg t.w./day without oral supplements

- Increase enteral nutrient intake to:
  - Protein: 1.2-1.4 gr./kg t.w./day
  - Energy: 35 kcal/kg t.w./day using dialysis-adapted dietary supplements either oral or via gastric or PEG tube

**Improvement of nutritional status**

**Established MN**

- No evidence for MN

**Some evidence for MN**

- No evidence for MN

**Mild to moderate MN MN**

- Severe MN

- Improvement of nutritional status

- No improvement or exacerbation of MN
References


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