

THE DIETARY MANAGEMENT OF PATIENTS WITH DIABETES AND RENAL DISEASE: CHALLENGES AND PRACTICALITIES

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SUMMARY

Diabetes Mellitus is one of the major causes of chronic kidney disease and end-stage renal disease. Diet and lifestyle modification are vital components of optimal treatment for both conditions. This paper will address appropriate and often diverse treatment for each individual, understanding that advising changes which positively impact both conditions is a major challenge for health care professionals working within either speciality. It will also highlight where overlap can be contradictory rather than complementary, and offers practical guidance to support patients in making the necessary lifestyle changes to have maximal positive impact upon both conditions and their overall health.

KEY WORDS Advice • Chronic kidney disease • Diabetes • Nutrition

INTRODUCTION

Diabetes mellitus (DM) is a complex, chronic medical condition affecting more than 346 million people worldwide (World Health Organisation, 2011). Worldwide prevalence is increasing rapidly in epidemic proportions, imposing a significant health, psychological and economic burden.

DM is one of the major causes of chronic kidney disease (CKD) and end-stage renal disease (ESRD); (USRDS; USRDS 2010; Renal Association 2011). Diet and lifestyle modification are vital components of optimal treatment for both conditions (NICE 2008; Koppell et al. 2010; Renal Association 2011).

However, appropriate treatment for each individual condition can be quite diverse, therefore advising changes which positively influence both conditions is clearly one of the major challenges for health care professionals working within either speciality, and streamlined multi-disciplinary working is essential for effective patient care.

This paper outlines some of the important elements of dietary treatment for patients with DM and CKD, highlighting where overlap can be contradictory rather than complementary, and offers practical guidance to support patients in making important changes to have maximal positive impact upon both conditions and their overall health.

BIODATA

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TYPE 1 VERSUS TYPE 2 DIABETES

It is important to distinguish between type 1 and type 2 DM, as their differing pathophysiology influences the subtle differences in dietary treatment strategies between the two. Type 1 DM is usually characterised by rapid onset pancreatic insufficiency and subsequent loss of insulin production, whereas type 2 DM is a complex metabolic disorder characterised by defects in both insulin secretion and action, and insulin resistance (SIGN 2010; NICE 2011). Insulin deficiency is progressive over time, and when coupled with insulin resistance, often leads to raised blood pressure and abnormal blood lipid levels.

Approximately 90% of those diagnosed with DM have type 2 DM (NICE 2008; WHO 2011), thought to be predominantly due to the increasing prevalence of obesity and the ageing population. Additional factors influencing risk of type 2 DM include reduced physical activity levels and family history of type 2 DM, with people of South Asian, African Caribbean,

black African and Chinese descent also having an increased risk of developing the disease (NICE 2011). Given that the majority of patients with CKD and ESRD will have type 2 DM, this paper will focus upon the dietary priorities for this patient group.

THE LINK BETWEEN DIABETES AND CKD

Diabetic nephropathy (DN) remains the most common cause of ESRD in the United Kingdom, accounting for approximately 25% of patients commencing renal replacement therapy (RRT) (UK Renal Registry 2011). The US Renal Data System (USRDS) reports that DM is present as an additional co-morbidity in over 48% of patients with CKD stages 3–5 (USRDS 2010).

Microalbuminuria is the first clinical detectable marker of DN. This often progresses to proteinuria, resulting from several renal functional changes including hyperfiltration, hyperperfusion and increased capillary permeability (Raptis & Viberti 2001; van Dijk & Berl 2004). Minimising the risk of developing the long-term complications of DM, including DN, is considered to be a cornerstone of good management (Department of Health 2001; NICE 2008). The Diabetes Control and Complications Trial (DCCT) and UK Prospective Diabetes Study (UKPDS) both demonstrated the benefit of intensive glycaemic control in delaying both the onset of microalbuminuria and subsequent progression to proteinuria and hence renal impairment (DCCT 1993; UKPDS 1998). The Steno 2 study (Gaede *et al.* 2003) highlighted the benefit of intensive multi-factorial intervention in patients with type 2 DM and microalbuminuria, with a 50% reduction in cardiovascular disease (CVD) risk and microvascular complications being achieved from intensive pharmacological therapy combined with dietary intervention and increased exercise.

Once DN is established, tight glycaemic control and optimal management of hypertension are vital to delay progression towards ESRD (NICE 2008; SIGN 2010; American Diabetes Association 2011), and diet and lifestyle modification have a pivotal role alongside medical strategies.

KEY DIETARY ASPECTS FOR DIABETES AND KIDNEY DISEASE

Lifestyle modification is a key component in the effective management of both DM and CKD (Mann *et al.* 2004; NICE 2008; SIGN 2010; Renal Association 2011). The aim of any nutritional advice is to provide the information required, in a way which allows people to make informed and appropriate choices about

the type and quantity of food they consume (Diabetes Care Advisory Committee 2003). Structured education provided by appropriate members of the diabetes multi-disciplinary team (MDT) is essential to ensure that patients acquire the necessary knowledge and skills to effectively manage disease conditions. This may consist of one to one counselling and/or group educational sessions, and care planning, which has been shown to be beneficial in diabetes management, may also be useful in maintaining a patient-centred approach (Department of Health/Diabetes UK, 2006; NICE 2008).

The following section outlines the main dietary aims for both conditions, showing not only where advice overlaps, but also where there are contradictions.

LIFESTYLE AND DISEASE PROGRESSION

Diet and lifestyle modification is essential to prevent DN and delay progression (NICE 2008), with several lifestyle factors having importance and significant influence over associated factors such as blood pressure control and obesity.

Considering that up to 50% of people with type 2 DM present with micro- or macrovascular complications at diagnosis (UKPDS 1998), earlier identification and effective management of diabetes, or even prevention, is crucial. Lifestyle is influential in preventing onset of type 2 DM (Tuomilehto *et al.* 2001; Diabetes Prevention Programme 2002). Obesity is the main contributory factor to type 2 DM (NICE 2011), and the vast majority of those diagnosed exhibiting signs of impaired glucose regulation (IGR) before developing DM (Diabetes UK 2009). Tuomilehto *et al.* (2001) showed that detailed, individualised advice to aid weight reduction, decrease total and saturated fat intake, and to undertake moderate, regular exercise, reduced the cumulative incidence of DM by 58% over a four-year period, in comparison to the control group who only received general written information regarding diet and exercise. The Diabetes Prevention Programme (2002) randomly assigned 3,234 subjects with elevated fasting and post-load plasma glucose concentrations to receive metformin, placebo or a lifestyle modification programme, which aimed to achieve 7% weight reduction and a minimum of 150 minutes of physical activity weekly. Incidence of DM was 58% less with lifestyle intervention than in the control group.

Managing type 2 DM and preventing subsequent complications is challenging, therefore it is essential that strategies such as screening high-risk individuals is implemented to enable

early identification of IGR and type 2 DM (WHO 2003; Diabetes UK 2008).

Several key factors, including lowering blood pressure, appropriate treatment of proteinuria, achieving optimal glycaemic control and smoking cessation are of significant importance in the prevention of CKD progression (NICE 2008; SIGN 2010; Renal Association 2011). Diet and lifestyle modification are important components in delaying progression of CKD, particularly given their positive influence upon both hypertension and glycaemic control (Sacks *et al.* 2001; Diabetes Care Advisory Committee, 2003; NICE 2008). However, the influence of both low protein diets and obesity upon CKD remains controversial (Klahr *et al.* 1994; Evans *et al.* 2005), and both will be subsequently discussed in more detail.

THE METABOLIC SYNDROME

The prevalence of metabolic syndrome, a common feature of vascular and metabolic diseases is increasing (Ford *et al.* 2004; Cull *et al.* 2007). It is characterised by insulin resistance, hyperglycaemia, hypertension, abdominal obesity and dyslipidaemia (WHO 1999; Balkau *et al.* 2002; Alberti *et al.* 2005) and is a commonly recognised pre-cursor for the development of type 2 DM, CVD and stroke. It may also potentially be an independent risk factor for CKD (Kurella *et al.* 2005; Tong *et al.* 2008). Obesity and lack of physical activity are important determinants of metabolic syndrome (Ford *et al.* 2004), and epidemiological data indicates that low glycaemic index (GI) diets can reduce the risk of metabolic syndrome due to the beneficial effect they exert in lowering insulin resistance.

DIABETES

All patients with CKD and/or DM should be encouraged to eat a well-balanced diet to provide all the energy, protein, vitamins, minerals and fibre that the body requires for optimal health. Incorporating foods from each of the five food groups (Figure 1) should be encouraged, in accordance with recommended food portion sizes illustrated within models used to convey healthy eating messages, such as the EatWell plate (Figure 2) in the United Kingdom (NHS Choices 2011) or the 'MyPlate' model used in the United States of America (United States Department of Agriculture 2011).

The holistic management of DM, including optimisation of glycaemic control and risk reduction for the development of CVD and microvascular complications strongly influences the goals

FOOD GROUPS	
1	Bread, cereals and potatoes
2	Fruit and vegetables
3	Meat, fish and alternatives
4	Milk and dairy foods
5	Fatty and sugary foods

Figure 1: Food groups.



Figure 2: EatWell plate (NHS choices 2011).

for dietary treatment of DM. Specific dietary guidelines for DM (Mann *et al.* 2004; NICE 2008) are summarised in Table 1 but generally follow the same principles as healthy eating for the general population.

Dietary advice should focus upon reduced consumption of carbohydrate, particularly refined carbohydrates and sugars, which often have a high GI. Particular attention should be paid to the portion sizes of carbohydrate foods, individualising recommendations to reduce the risk of both hypoglycaemia and hyperglycaemia. Carbohydrate foods have been shown to have a similar effect upon glycaemic control as high sucrose intake (Diabetes Care Advisory Committee, 2003), therefore avoiding excessive intake, particularly in type 2 DM, is beneficial. Promoting intake of lower GI foods such as fruit, vegetables and pulses, along with moderate portions of wholegrain cereals and complex carbohydrate, lean meat, fish, reduced fat dairy products and unsaturated fats are beneficial for weight control and reducing CVD risk (Diabetes Care Advisory Committee, 2003; Mann *et al.* 2004).

1	Ensure provision of individualised, culturally acceptable nutritional advice
2	Emphasise advice based on a healthy, balanced diet which applies to the general population
3	Aim to achieve healthy bodyweight (BMI 20–25 kg/m ²) and encourage weight loss (5–10% initially) in those who are overweight
4	Encourage high fibre, low GI carbohydrate foods
5	Individualise recommendations for overall carbohydrate intake, and advise limiting sucrose-containing foods to ensure avoiding excess energy intake
6	Eat plenty of fruit and vegetables
7	Include oily fish regularly within the diet
8	Reduce overall fat intake, and replace saturated fat with unsaturated fats where possible
9	Reduce salt intake to no more than 6 g per day
10	Avoid excessive alcohol consumption

Table 1: Dietary guidelines for DM (Mann *et al.* 2004; NICE 2008).

CKD STAGES 1–3

Dietary advice provided to patients with CKD stages 1–3 should focus upon the same healthy eating advice which is applicable to both the general population and those with diabetes, with particular emphasis upon reduced sodium intake, increased physical activity and weight reduction, to benefit overall health and reduces risk factors for CKD. Key aspects of appropriate advice have been included within NICE (2008) and Renal Association (2011) guidance within the United Kingdom (Table 2).

CKD STAGES 4 AND 5

As renal function deteriorates, serum levels of both potassium and phosphate often increase (Renal Association 2011), and regulation of fluid balance may also be compromised. A spontaneous reduction in overall dietary intake is often observed particularly for protein and energy intake, and is usually secondary to uraemic symptoms. The focus for dietary advice at this stage therefore shifts towards controlling levels of waste products of metabolism and fluid, as well as ensuring maintenance of optimal nutritional status. Malnutrition is common in patients with CKD and is associated with adverse outcomes, including morbidity and mortality (Locatelli *et al.* 2002). Patients commencing RRT in a poor nutritional state are shown to result in a poor outcomes (Leavey *et al.* 2001), therefore it is vital that dietary advice is patient-specific and avoids inappropriate dietary restriction.

Fouque *et al.* (2007), The Renal Association (2010b) and British Dietetic Association Renal Nutrition Group (2011) have all produced nutritional guidelines for optimal dietary manage-

1	Dietary intervention should include appropriate education, assessment and supervision and ensure the prevention of malnutrition
2	Achieving a healthy body weight should be encouraged, and patients with BMI >30 kg/m ² should receive dietary advice to assist weight loss
3	Dietary advice to restrict sodium intake to <2.4 g/day (100 mmol/day or <6 g/day of salt) should be provided
4	Advice to perform regular moderate exercise should be provided
5	If indicated, the risks and benefits of protein restriction should be discussed by an appropriately trained professional
6	Specific dietary advice to restrict potassium, phosphate, protein and/or calorie intake should be provided if indicated

Table 2: Dietary and lifestyle modification for CKD (NICE 2008; Renal Association 2011).

1	Screening for undernutrition in CKD stages 4 and 5 should take place at regular intervals, including a variety of methods such as assessing actual body weight, percentage weight loss and subjective global assessment (SGA)
2	Prevention of undernutrition in CKD stages 4 and 5 should be achieved by recommending appropriate protein and energy intake: <ul style="list-style-type: none"> • 0.75 g protein per kg ideal body weight (IBW) daily for those not on RRT, and a minimum of 1–1.2 g/kg IBW/d protein for those on RRT • 30–35 kcal/kgIBW/d for all patients
3	Established undernutrition in CKD stages 4 and 5 should be determined by appropriate clinical assessment, and treatment should include appropriate use of oral nutritional supplements or enteral or parenteral nutrition as indicated
4	Overnutrition in CKD should be determined by assessing body mass index (BMI)

Table 3: Summary of nutritional guidelines for low clearance patients and those on RRT.

ment of patients with ESRD on RRT, the key points of which are summarised in Table 3. However, it should be noted that the majority of these recommendations are based on expert opinion or lower grade evidence, highlighting the need for research in this area.

THE DIETARY TREATMENT OF PATIENTS WITH DIABETES AND RENAL DISEASE

The most appropriate dietary treatment for some patients with CKD will change over time, depending on serum biochemistry levels and current nutritional status. Furthermore, there can be some contradiction between diet for CKD and DM, thus making the management of patients with DM challenging. It is important to reassure patients that previous dietary advice was appropriate for their disease status at that time, and whilst helping them to understand changes in dietary priorities may be difficult, it is often useful to explain the health benefits of the different elements of dietary advice. It is likely that patients

developing CKD as a consequence of diabetes will have been exposed to nutrition and health information from a variety of sources. It is therefore important that these patients are routinely referred to a registered dietitian who is qualified to assess their overall diet and offer appropriate, individualised advice.

MALNUTRITION

Malnutrition is commonly seen in CKD stages 4 and 5, with reported prevalence up to 48% in those with CKD stage 4 and between 20% and 70% in patients with CKD stage 5 (Fouque *et al.* 2011). It often results from decreasing glomerular filtration rate (GFR) and increasing age, and is associated with increased morbidity and mortality (Leavey *et al.* 2001; Pifer *et al.* 2002; Dwyer *et al.* 2005). Several additional factors pre-dispose to the development of malnutrition, including symptoms associated with uraemia such as nausea and vomiting, impaired taste, anorexia and anaemia.

A meta-analysis of oral nutritional supplementation and enteral tube feeding in dialysis patients (Stratton *et al.* 2005) demonstrated that nutritional support can potentially improve clinical outcomes in malnourished patients, therefore early detection of malnutrition is vital, allowing timely nutritional intervention. Since there is no 'gold standard' method available for assessing nutritional status, routine nutritional screening and assessment should include a variety of measures to reflect nutritional status. If malnutrition is suspected, patients should be referred to a registered dietitian for nutritional assessment and support.

Patients with DM are often encouraged to reduce their carbohydrate intake to help optimise glycaemic control, or reduce fat intake to help influence their future risk of developing CVD. However, if they are fast approaching dialysis, struggling with uraemia and poor appetite the current dietetic priority is maintaining an adequate energy intake rather than preventing CVD sometime in the future. Struggling with food preparation and reduced dietary intake is further compounded by fatigue and nausea, hence the consequences of malnutrition become more of a threat to the patients' outcome than CVD. It is vital that nutritional adequacy is achieved from an early stage, and that patients are not subject to inappropriate dietary restriction.

DIETARY PROTEIN INTAKE

Optimal protein intake is required for growth, repair and maintenance of overall nutritional status. The benefit of protein

restriction in preservation of renal function remains controversial, despite being clearly implicated in the aetiology of kidney impairment through the Brenner Hypothesis (Brenner *et al.* 1982). This was an important influence in the design of the Modification of Diet in Renal Disease (MDRD) trial (Klahr *et al.* 1994), a comprehensive study evaluating the effect of protein restriction as well as blood pressure upon decline in renal function. Protein intake was only found to have a minimal effect upon the rate of decline in renal function, and only in subjects with GFR of 25–55 mL/min. However, a meta-analysis (Fouque *et al.* 2000) concluded that reduced protein intake afforded a 40% reduction in commencing RRT, and in a Cochrane Collaboration review (Fouque & Lavill 2009), results from 10 randomised studies including a total of 2,000 patients found that protein restriction followed for a minimum of one year, reduced renal death (RRT or death from any cause) by 32%. In a separate Cochrane Collaboration review of 12 studies in patients with types 1 and 2 DM, Robertson *et al.* (2009) found that lower protein intake may modestly delay progression of DN towards ESRD, although the results were not deemed to be clinically significant. Neither review could recommend optimal protein intake from their findings.

Patients with CKD stages 1–4 are not advised to follow a 'traditional' low protein intake of ≤ 0.6 g/kg IBW per day, but to aim for a protein intake of approximately 0.75–1 g/kg IBW per day. For most people this would represent a protein intake of less than 70 g per day, which represents a degree of restriction for the majority, considering that mean protein intake is between 66 and 88 g per day in the United Kingdom (National Diet and Food Survey 2010) and 70–100 g per day in the United States of America (NHANES 2008).

Patients undergoing RRT are advised to achieve a slightly higher protein intake of 1–1.2 g/kg IBW per day (Fouque *et al.* 2007; Renal Association 2010; British Dietetic Association Renal Nutrition Group 2011), in view of the increased prevalence of malnutrition in the dialysis population and to compensate protein losses on dialysis. Dietary advice regarding optimum protein intake should be given by an appropriately trained registered dietitian, as part of routine dietary assessment (NICE 2008; Renal Association 2011).

CONTROL OF WASTE PRODUCTS

Serum potassium and phosphate levels often rise as renal function deteriorates. Patients requiring advice regarding either a

low potassium or low phosphate diet (or both) must be referred to a registered dietitian to enable thorough dietary assessment upon which appropriate advice can be based (Renal Association 2011).

POTASSIUM

High potassium levels (hyperkalaemia) are a common and potentially dangerous complication in patients with CKD and declining GFR, becoming increasingly common as GFR declines below 40–60 mL/min per 1.73 m² (Hsu & Chertow 2002). Hyperkalaemia is associated with cardiac arrhythmias and death, and whilst it may be preceded by other symptoms such as muscle weakness, it often presents as cardiac arrest (Nyirenda *et al.* 2009).

Dietary potassium intake is a major determinant of serum potassium levels, and dietary potassium restriction can substantially reduce hyperkalaemia. However, non-dietary causes, including medications commonly used in CKD and DM (e.g. ACE inhibitors and angiotensin-II receptor antagonists), metabolic acidosis and diabetic ketoacidosis, should also be considered as potential causes of hyperkalaemia. Appropriate dietary advice (sometimes given in conjunction with sodium bicarbonate if metabolic acidosis is present), usually controls hyperkalaemia in CKD stages 1–4. This potentially allows ongoing optimal treatment of hypertension and proteinuria, although occasionally adjustment of medications is required. Potassium is present in many frequently consumed foods and drink, including fruit and vegetables, fruit juices, milk and meats, all of which are considered to be important elements of a normal 'healthy' diet. This can cause particular problems for those with DM, where increased consumption of fruit and vegetables is encouraged. In this instance, appropriate dietary advice regarding fruit and vegetables with a lower potassium content (Table 4), and advice regarding suitable portion sizes and cooking methods which will reduce the potassium content of potatoes and vegetables is required. Therefore, patients requiring a low potassium diet should be referred for specialist dietetic assessment and advice (Renal Association 2011). Practice varies between different renal units, but patients will generally be referred for low potassium dietary advice when serum potassium reaches 5.6–6.0 mmol/l. The specific point at which dietetic referral is made often depends upon underlying medical conditions and other treatments, particularly those which can cause hyperkalaemia such as ACE inhibitors or angiotensin-II receptor antagonists.

Fruits	Vegetables
(Portion size – 1 fruit unless otherwise stated)	(Portion size – approx. 2 tablespoons) Vegetables should be boiled where possible
Apple	
Cherries (approx. 10)	Broccoli
Clementine, Satsuma or tangerine	Cabbage
½ Grapefruit	Carrots
Nectarine	Cauliflower
1 slice fresh Pineapple	Green beans
Plum	Leeks
Raspberries (approx. 10)	Peas
Strawberries (approx. 5–6)	Runner beans
150 g any tinned fruit, drained (except prunes)	Swede
	Sweetcorn
	Turnip
	Small side salad (e.g. lettuce, cucumber, ½ small tomato)

Table 4: Lower potassium fruits and vegetables.

Consequently, patients with diabetes are more likely to require earlier intervention.

PHOSPHATE

Elevated serum phosphorus levels (hyperphosphataemia) are a common biochemical abnormality of CKD and are one of the main factors associated with Chronic Kidney Disease–Mineral Bone Disorder (CKD–MBD). Serum phosphorus levels increase with declining GFR, and hyperphosphataemia becomes increasingly common with GFR below 40 mL/min per 1.73m² (Hsu & Chertow 2002; Moranne *et al.* 2009).

It is linked to cardiovascular morbidity and mortality in CKD (Block *et al.* 2004; Dhingra *et al.* 2007; Covic *et al.* 2009), with epidemiological and observational studies demonstrating a strong association between hyperphosphataemia and mortality in dialysis patients which is thought to be mediated via vascular calcification (Covic *et al.* 2009).

Optimal management of hyperphosphataemia is multi-factorial, and therefore poses a number of significant challenges. Elevated serum phosphate levels can usually be reduced with advice to restrict dietary phosphorus intake in the first instance (Combe & Aparicio 1994) however as the amount of

phosphate found in food is closely related to its protein content, dietary advice to avoid unnecessary restriction of protein is essential (Shinaberger *et al.* 2008). Food additives and soft drinks (Sullivan *et al.* 2007; Savica *et al.* 2009) are major contributors to dietary phosphate intake which cannot be readily identified from food labelling. Many high-fibre foods are also higher in phosphate, although the extent to which this is absorbed is unclear. For patients struggling to control serum phosphate levels, reducing some high-fibre cereal foods may be beneficial, although this can appear to contradict advice for DM patients, which is to increase dietary fibre intake. Patients should therefore be referred to a specialist renal dietitian who can provide specific, individualised dietary advice (Ashurst & Dobbie 2003; Reddy *et al.* 2009). The point at which dietary intervention is initiated again varies across different renal units, but it is generally accepted that phosphate levels should be maintained between 1.1 and 1.7 mmol/l, depending upon the stage of CKD (Renal Association 2010).

Dietary restriction is not always sufficient to provide optimal phosphate control, which necessitates the use of phosphate binders (Tonelli *et al.* 2010), which work by inhibiting absorption of phosphate from food within the GI tract. Specialist renal dietitians are able to advise regarding the appropriate timing and distribution of phosphate binders with meals and snacks. Dietetic-led CKD/MBD multi-professional clinics have been shown to add value to management.

WEIGHT MANAGEMENT

Weight management is a vital component of treatment in both CKD and DM. Obesity may be an independent risk factor for the development and progression of CKD (Wang *et al.* 2008; Ting *et al.* 2009), particularly as prevalence of CKD and ESRD increases parallel to the rise in obesity (Ryu *et al.* 2008). Conversely, in patients receiving dialysis, reduced mortality risk has been observed in patients with higher BMI (Leavey *et al.* 2001; Johansen *et al.* 2004).

Few population-based epidemiological studies have examined the association between obesity and CKD (Ejerblad *et al.* 2006; Hsu *et al.* 2006), although moderate weight loss has been shown to reduce hyperfiltration and the level of metabolic stress on the kidney thereby reducing glomerular damage and proteinuria (Chagnac *et al.* 2003; Morales *et al.* 2003). Given the influence that obesity has been shown to

exert over general health, CVD risk, hypertension and glycaemic control, it seems sensible to encourage patients with CKD who are overweight to try and achieve a healthy BMI (Elsayed *et al.* 2008).

Achieving and maintaining a healthy body weight is an important goal of therapy (Anderson *et al.* 2002), particularly in type 2 DM where obesity complicates management by increasing insulin resistance and blood glucose concentrations (Klein *et al.* 2004), and results in decreased insulin sensitivity and deterioration in glycaemic control (Astrup *et al.* 2001). Weight reduction often leads to improved glycaemic control (Look AHEAD Research Study Group 2007) along with reduction in oral hypoglycaemic medication (Nathan *et al.* 2009). Even modest weight loss of under 10% body weight improves insulin sensitivity and glucose tolerance, reducing lipid levels and blood pressure, thereby reducing cardiovascular risk (Goldstein 1992; Look AHEAD Study Group 2007). The Look AHEAD randomised controlled trial (RCT) in over 2,500 patients with type 2 DM demonstrated that mean weight loss of 8.6% in the intervention group compared with 0.7% in the control group led to subsequent improvements in glycaemic control and reduction in CVD risk (Look AHEAD Research Study Group 2007).

Whilst a BMI in the range 20–25 kg/m² is desirable, this may not be achievable for all patients, particularly in those with a significantly higher BMI. With evidence suggesting that 5–10% weight loss can positively influence overall health, and specifically hypertension, glycaemic control and dyslipidaemia (Goldstein 1992; Look AHEAD Study Group 2007; Navaneethan *et al.*, 2009), this is often a realistic initial target for weight loss.

Obesity is multi-factorial, with psychological, genetic, behavioural and environmental influences (Anderson *et al.* 2001; Ting *et al.* 2009). Successful prevention and treatment therefore poses a number of challenges, and appropriate consideration of psychological and lifestyle barriers to successful weight loss is paramount. An energy deficit (overall energy intake being less than energy expenditure) is required for successful weight loss. A commonly used practical approach is to reduce energy intake from current intake by 500 kcals per day, which usually results in the loss of approximately 0.5 kg of adipose tissue per week (Eckel 2008). This can be achieved by combining a sensible structured eating programme which is

low in fat and sugar with appropriate control of portion size. Increasing physical activity alone can also modestly influence weight loss (Eckel 2008), although where possible a combination of the two should be encouraged to enhance weight loss and improve muscle mass. A steady weight loss of 0.5–1 kg (1–2 lb) per week should be encouraged, as a faster rate of weight loss leads to muscle loss rather than fat, and may also prevent future weight maintenance.

HYPERTENSION

Hypertension is one of the major determinants for progression of both CKD and DN with optimal blood pressure control being essential to help prevent macro- and microvascular complications of DM, particularly CVD and DN (NICE 2008). The target for optimal blood pressure control in patients with CKD and diabetes is 130/80 mmHg (NICE 2008; SIGN 2010; Renal Association 2011).

Several lifestyle factors, including weight loss and increased physical activity, have been shown to reduce hypertension. A meta-analysis of 25 studies indicating that blood pressure fell by 1 mmHg for each kilogram of weight loss (Neter *et al.* 2003). It is well-established that regular, moderate exercise in the general population reduces the risk of CVD (Paffenberger *et al.* 1993; Thijssen *et al.* 2010). Furthermore, aerobic physical activity has been shown to be beneficial in the non-pharmacological treatment of hypertension, and is also an integral part of a structured weight management programme, the benefits of which have also been demonstrated in renal patients (Cook *et al.* 2008).

Sodium is another major determinant of blood pressure. Western populations consume substantially higher amounts of sodium than necessary, and this is associated with a higher prevalence of hypertension and CVD (He & MacGregor 2009; Bibbins-Domingo *et al.* 2010). A meta-analysis of 28 randomised trials investigating sodium reduction on blood pressure in normotensive and hypertensive individuals showed that reducing sodium intake by 100 mmol/day significantly reduced blood pressure in hypertensive individuals (He & MacGregor 2002). The Dietary Approaches to Stop Hypertension (DASH-sodium) RCT (Sacks *et al.* 2001; Vollmer *et al.* 2001) found that reducing salt intake from 9 g per day to 6 g daily gave a significant reduction in blood pressure, with more marked effects observed in hypertensive and older subjects, and when combining salt restriction with a diet

high in fruit and vegetables and low in fat and sugar (DASH diet). A small study ($n = 14$) in the UK general population assessed whether the DASH diet could be adapted to fit UK food preferences (Harnden *et al.* 2010), and showed good compliance with the DASH-style diet, along with reduction in both systolic and diastolic blood pressure. However, some elements of the DASH-style diet, particularly increasing fruit and vegetable intake, may not be a suitable recommendation for those following low potassium diets, and further work to determine the safety of such diets in the CKD population could be warranted.

An in-depth review of 16 studies investigating the effect of sodium intake upon CKD progression (Jones-Burton *et al.* 2006) found relatively little evidence to suggest that a high sodium intake specifically influences renal outcomes, however reducing sodium intake was not found to be detrimental. A recent study from the HONEST study group (Slagman *et al.* 2011), found the combination of ACE inhibition and reduction in sodium intake from 184 to 106 mmol/day was more effective for controlling blood pressure and reducing proteinuria than dual blockade with ACE inhibitors and angiotensin-II-receptor blockers. This study also highlighted the difficulties in achieving low sodium intake, as their original target for low sodium intake was only 50 mmol/day.

The current average salt intake in the United Kingdom is approximately 9 g (approximately 3.5 g sodium) per day (Department of Health 2009), with current recommendations for the general population to reduce salt intake to 6 g (2.4 g sodium) per day. Although a greater reduction in sodium intake is thought to be more beneficial, this realistic reduction is expected to result in significant benefit for reduction in hypertension and CVD (He & MacGregor 2003, 2005). Although the effect of sodium intake upon renal outcomes remains unclear, given the positive impact of sodium reduction on blood pressure and the known impact of high blood pressure on renal function and proteinuria, it seems sensible to recommend maintaining dietary sodium intake at less than 6 g per day.

Reduction in dietary sodium intake can be practically achieved by:

- Limiting the amount of high salt and processed foods consumed (Table 5);
- Not adding salt to food at the table;
- Minimising the amount of salt used in cooking.

High salt foods to reduce	Suitable alternatives
Meat and meat products: Bacon, gammon, black pudding, tinned meat (e.g. corned beef, ham, Spam), pâté, salami, sausages, beef burgers, meat pies and most ready-made meat dishes	Plain meat (e.g. roast meats, chops and steaks) and chicken, home-made meat pies
Fish and fish products: All smoked fish, tinned fish in brine (e.g. tuna, sardines, anchovies) prawns, fish paste	Fresh fish (e.g. cod, haddock, halibut, lemon sole, plaice, salmon, trout); tinned fish in spring water or oil
Cheese: Hard cheeses (e.g. Cheddar, Cheshire, Edam); soft cheeses (e.g. brie, feta); processed cheese, e.g. cheese slices and cheese spread	Cottage cheese, cream cheese
Savoury snacks: Crisps; maize, corn and wheat snacks; salted nuts, and most other savoury snacks (e.g. Twiglets, Bombay mix, pork scratchings, salted popcorn, salted crackers)	Unsalted crisps, nuts and crackers, plain popcorn
Soup: Packet, canned and fresh soups	Homemade soups
Miscellaneous: Soy sauce, Bovril, Marmite, gravy granules, stock cubes, olives in brine	Herbs, spices, vinegar, small amounts of chutney, pickles and bottled sauces, salad cream and mayonnaise

Table 5: No added salt advice.

Stages 1 & 2	<p>Aim to minimise the risk of CKD progression and cardiovascular risk: Optimise glycaemic control (diet and medication) Weight control (appropriate reduction in energy intake and increased physical activity) Optimal blood pressure control (No added salt, weight control) Optimise lipid profile (encourage oily fish, fruit and vegetables, low saturated fat intake, increased fibre intake) Basic low potassium advice if K^+ >5.5 mmol/l</p>
Stage 3	<p>Aim to delay CKD progression: As for stages 1 & 2 Treat metabolic complications if they arise, e.g. hyperkalaemia, malnutrition, renal bone disease</p>
Stage 4	<p>Pre-dialysis advice: Emphasis on optimal nutritional intake & prevention of malnutrition No added salt Limit metabolic complications as required—low potassium, phosphate diet, fluid restriction Advice to optimise glycaemic control and/or achieve weight reduction as required</p>
Stage 5	<p>Individualised dietary advice according to biochemistry and renal replacement therapy, with emphasis on optimal nutritional intake</p>

Table 6: Suggested dietary advice across CKD stages 1–5.

Salt substitutes contain significant amounts of potassium which could be harmful for people with CKD and/or hyperkalaemia. Furthermore, they still contain substantial amounts of sodium which does not facilitate reduction in salt appetite. They should therefore not be recommended in the general population but this is of particular importance in those with CKD.

Considering that approximately 70–75% of sodium in the Western diet comes from salt in processed foods (Department of Health 2009), achieving a reduction in sodium intake in the general population is challenging. However, the Food Standards Agency in the United Kingdom (2011) has recommended a reduction in the salt content of many processed foods, to help maintain progress towards achieving the 6 g daily salt intake target.

KEY POINTS

Dietary and lifestyle advice for people with CKD and DM (Table 6) should focus on:

- Eating a varied diet;
- Achieving a healthy weight;
- Encouraging regular physical activity;
- Reducing salt intake;
- Control of metabolic complications as they arise;
- Referral to a registered dietitian working within the renal and/or diabetes MDT for specialist dietary intervention.

CONFLICT OF INTEREST

The author confirms no conflict of interest.

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