CHAPTER 12

NUTRITIONAL MANAGEMENT OF CHRONIC DISEASES

Renée Blaauw, Martani J. Lombard, Nelia Steyn, and Petro Wolmarans

Outline

• Diabetes mellitus
  • Classification of different types of diabetes
  • Diagnosis
  • Dietary management
  • Treatment with medications
  • Complications
• Cardiovascular disease (stroke and ischaemic heart disease)
  • Risk factors
  • Abnormalities of blood lipids
  • Dietary management
  • Hypertension
  • Dietary and lifestyle recommendations for people at increased risk
• Role of diet in the treatment of cancer

Objectives

At the completion of this chapter you should be able to:

• Demonstrate a good understanding of the classification of diabetes mellitus, cardiovascular disease, and lifestyle-related cancers
• Describe the best dietary treatment for diabetes
• Describe the best dietary treatment for the management of risk factors related to cardiovascular disease (stroke and ischaemic heart disease)
• Describe appropriate dietary recommendations for patients with cancer
• Plan a regime for dietary management of nutrition-related chronic diseases
Abbreviations

CDL  chronic diseases of lifestyle
CVD  cardiovascular diseases
DM  diabetes mellitus
HDL-C  high-density lipoprotein cholesterol
IHD  ischaemic heart disease
LDL-C  low-density lipoprotein cholesterol
MUFA  monounsaturated fatty acids
OGTT  oral glucose tolerance test
PUFA  polyunsaturated fatty acids
SFA  saturated fatty acids
TC  total cholesterol
TE  total energy
TG  triglyceride

1. INTRODUCTION

According to the World Health Organization (WHO), the overnourished population of the world is now 1.2 billion people (WHO, 2008); this is roughly the same as the undernourished population. Globally, there are more than one billion adults who are overweight or obese. The increase in obesity is associated with escalating prevalences of the metabolic syndrome, type 2 diabetes mellitus (DM), and cardiovascular diseases (CVD) in developing countries (Misra & Khurana, 2008). This is especially true for underprivileged people residing in urban areas (Misra et al., 2001). The main reasons believed to be responsible for this trend are increasing urbanization, reduced physical activity, and the nutrition transition. Indeed, programmes aimed at undernourished children may exacerbate the problem, particularly since perinatal undernutrition and early stunting have been closely associated with the adult onset of type 2 DM and the metabolic syndrome (Misra & Khurana, 2008).

These diseases are known as chronic diseases of lifestyle (CDL), which is the term used in this book. Another commonly used term is non-communicable diseases.

The growth of CDL in low- and middle-income countries has become an epidemic in terms of prevalence and cost to treat. The good news, however, is that lifestyle modification can delay or prevent the majority of CDL, including obesity, type 2 DM, CVD, and several major types of cancer. For that reason, lifestyle modification should be introduced for the prevention of these diseases. In particular, there is a great need for large-scale community intervention programmes that focus on physical activity and a healthier diet (Misra & Khurana, 2008).

This is all explained in detail in this chapter and Chapter 13. The major focus of this chapter is the management of DM (both type 1 and type 2), CVD, and several major types of cancer. In the next chapter we examine the role of diet and other lifestyle factors in the prevention of cancer, CVD, type 2 DM, and obesity.

2. DIABETES MELLITUS

2.1 Introduction

Diabetes mellitus (DM) is considered to be the most common endocrine disorder around the world (Bastaki, 2005). In 2010, among adults aged 20 to 79 years, the prevalence in sub-Saharan Africa was in the range of 2.2% to 4.7%; in Asia, 3.2% to 11.6%; in the Middle-East crescent, 3.0% to 16.8%; and in Europe and North America, 3.6% to 10.3%. On average, the world DM prevalence is currently 6.4%; it is expected to increase to 7.7% by 2030 (see Table 12.1). The largest proportional increase from 2010 to 2030 is expected for the African region, followed closely by the Eastern Mediterranean and Middle-East (EMME) region (Shaw et al.,
Overall, the increase in the number of adults with DM will be much greater in developing countries than in developed countries.

**Table 12.1:** Current and projected prevalence of diabetes among adults, by world region, 2010 and 2030

<table>
<thead>
<tr>
<th>Region</th>
<th>Total adult population (000s)</th>
<th>No. of adults with diabetes (000s)</th>
<th>Diabetes prevalence (%)</th>
<th>Total adult population (000s)</th>
<th>No. of adults with diabetes (000s)</th>
<th>Diabetes prevalence (%)</th>
<th>Increase in the no. of adults with diabetes (%)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Africa</td>
<td>379</td>
<td>12.1</td>
<td>3.8</td>
<td>653</td>
<td>23.9</td>
<td>4.7</td>
<td>98.1</td>
</tr>
<tr>
<td>EMME*</td>
<td>344</td>
<td>26.6</td>
<td>9.3</td>
<td>533</td>
<td>51.7</td>
<td>10.8</td>
<td>93.9</td>
</tr>
<tr>
<td>Europe</td>
<td>646</td>
<td>55.4</td>
<td>6.9</td>
<td>659</td>
<td>66.5</td>
<td>8.1</td>
<td>20.0</td>
</tr>
<tr>
<td>North America</td>
<td>320</td>
<td>37.4</td>
<td>10.2</td>
<td>390</td>
<td>53.2</td>
<td>12.1</td>
<td>42.4</td>
</tr>
<tr>
<td>South and Central America</td>
<td>287</td>
<td>18.0</td>
<td>6.6</td>
<td>382</td>
<td>29.6</td>
<td>7.8</td>
<td>65.1</td>
</tr>
<tr>
<td>South Asia</td>
<td>838</td>
<td>58.7</td>
<td>7.6</td>
<td>1200</td>
<td>101.0</td>
<td>9.1</td>
<td>72.1</td>
</tr>
<tr>
<td>Western Pacific</td>
<td>1531</td>
<td>76.7</td>
<td>4.7</td>
<td>1772</td>
<td>112.8</td>
<td>5.7</td>
<td>47.0</td>
</tr>
<tr>
<td>World</td>
<td>4345</td>
<td>284.8</td>
<td>6.4</td>
<td>5589</td>
<td>438.7</td>
<td>7.7</td>
<td>54.1</td>
</tr>
</tbody>
</table>

* EMME = Eastern Mediterranean and Middle-East region

Source: Shaw et al., 2010.

The major contributing factors responsible for these trends are believed to be urbanization, sedentary lifestyles, poor diet, obesity, and increased longevity (Shaw et al., 2010). The role of diet and other aspects of lifestyle in the causation and prevention of DM is examined in Chapter 13.

### 2.2 Definition

DM is a collection of various metabolic disorders. In particular, there is an increased blood glucose concentration (hyperglycaemia) as a result of either decreased amounts of insulin, defective insulin action, or both.

### 2.3 Classification

#### 2.3.1 Type 1 diabetes

Type 1 diabetes is an autoimmune reaction in which the pancreas islet beta-cells are completely destroyed. As a result, daily insulin injections are required. The disease typically appears during childhood or early adulthood. Although patients with type 1 DM are mostly thin or underweight, obesity may occur in rare cases (ADA, 2009a; Daneman, 2006; Bastaki, 2005).
2.3.2 **Type 2 diabetes**
Type 2 DM, accounting for about 90% of those with diabetes, is associated with insulin resistance and decreased insulin production (ADA, 2009a, 2009b). It is mostly lifestyle related, with a gradual onset. Type 2 diabetic patients are typically older, overweight or obese, and inactive. Diet, weight loss, and physical activity are imperative for blood glucose control. Oral glucose-lowering agents or insulin can be used for further control (ADA, 2009a; Bastaki, 2005).

2.3.3 **Insulin resistance and glucose intolerance**
Insulin resistance is also known as prediabetes. People with this condition have raised glucose concentrations, but not as high as those in people formally diagnosed with type 2 DM (ADA, 2009a). People with insulin resistance will mostly have fasting plasma glucose (FPG) concentrations between 5.6 and 7.0 mmol/l (100–126 mg/dl) and 2-hour post-prandial values during the oral glucose tolerance test (OGTT) between 7.8 and 11.1 mmol/l (140–200 mg/dl) (ADA, 2009a).

2.3.4 **Gestational diabetes**
Gestational diabetes is defined as the presence of increased blood glucose concentrations during pregnancy. Dietary changes and/or insulin treatment is crucial, depending on blood glucose concentrations. Blood glucose concentrations sometimes stay elevated after delivery (ADA, 2009a). In the years following the pregnancy there is a high risk of developing DM (Franz, 2012). It is therefore important that women with this condition maintain a reasonable body weight and healthy lifestyle (Bastaki, 2005).

2.4 **Diagnosis**
The criteria used for the diagnosis of DM by the American Diabetic Association (ADA) and the Society of Endocrinology and Metabolic Disorders in South Africa (SEMDSA) are as follows (ADA, 2009a, 2009b; Levitt et al., 2009; Cerielo, 2008):

1. Symptoms of DM + random plasma glucose >11.1 mmol/l (200 mg/dl)
2. Fasting plasma glucose >7.0 mmol/l (126 mg/dl)
3. 2-hour plasma glucose >11.1 mmol/l (200 mg/dl) during OGTT
4. Hypoglycaemia; plasma glucose <4 mmol/l (72 mg/dl)

Symptoms are similar in all types of DM but differ in intensity and speed of onset. Symptoms indicating hyperglycaemia include polyurea (excessive urination), polydipsia (excessive thirst), polyphagia (excessive eating), unexplained weight loss, sweating, fatigue, blurred vision, and increased infections. Symptoms indicating hypoglycaemia (low blood glucose) can be very similar and thus confusing to the patient. They include anxiety, sweating, shaking, blurred vision, fatigue, and sleepiness. Depending on the presence of complications, other specific symptoms can also be experienced (Bastaki, 2005).

2.5 **Dietary Management**
Chronic exposure to hyperglycaemia leads to secondary complications including blindness, kidney failure, limb amputations, and damage to the heart and blood vessels (ADA, 2009b; Bastaki, 2005). DM requires continuing medical and nutritional care and effective patient education to manage acute complications (hyperglycaemia) and to reduce the development of long-term complications (ADA, 2008).

The primary treatment goal is control of blood glucose in order to keep the concentrations within the normal range. Food and medication that reduce the rise in post-prandial (after-meal) blood glucose concentrations are important to such treatment. The blood glucose response following a meal is dependent on the rate of digestion and absorption of glucose from the food and its clearance from the blood circulation. Insulin is normally secreted by the endocrine system to keep blood glucose within its normal range, but in individuals with DM, defects in insulin management hamper effective glucose control (ADA, 2008).
In type 1 DM, insulin therapy is the most important treatment modality, whereas in type 2 DM, the key aspects of treatment are diet, oral medication, and lifestyle modifications (Franz, 2012; Bastaki, 2005). Insulin therapy can also be employed in type 2 patients if blood glucose concentrations cannot be successfully controlled by diet and oral medication.

2.5.1 Diet

Diet remains the cornerstone of the management of DM. Intensive therapy, consisting of a combination of medication, diet, and physical activity, effectively delays the onset of DM, manages existing DM, and slows the development and progression of complications in patients with insulin-dependent DM (ADA, 2008). It is important to emphasize that the diabetic diet is not a special diet in the true sense of the word, but rather a balanced eating pattern that should be followed by the whole family. The goals of nutrition intervention in diabetic patients are summarized in Table 12.2.

Table 12.2: Goals of nutrition intervention in diabetic patients

- Establish and maintain euglycaemia (normal glucose concentrations)
- Promote a reasonable body weight
- Improve blood lipid concentrations in order to reduce the risk for CVD
- Achieve and maintain blood pressure levels within a normal range
- Prevent, or delay the onset of complications
- Treat, complications
- Maintain normal growth and development in children and adolescents
- Provide adequate nutrition for pregnant and lactating women
- Encourage healthy eating habits
- Encourage physical activity


Successful medical nutrition therapy has been proven through clinical trials to result in decreased glycosylated haemoglobin (HbA1c) and reduced low-density lipoprotein cholesterol (LDL-C) concentrations (ADA, 2008). It is recommended that the HbA1c level should be <7%, which indicates long-term blood glucose control (Levitt et al., 2009; ADA, 2009b; Nathan, 2009; Cerielo, 2008).

Various dietary recommendations for the management of DM have been proposed by many associations around the world. The following table provides a summary of the essence of the recommendations, though different countries vary somewhat in some details (see Table 12.3).

Energy. Achieving weight loss and maintaining a reasonable body weight should be the first goal of the treatment plan. Long-term weight loss is achieved with programmes that emphasize lifestyle changes, including education, individualized counselling, reduced dietary energy and fat intake, regular physical activity, and frequent patient contact (ADA, 2005, 2008; Franz, 2012; Mann, 2006). Strategies to achieve weight loss are examined in Chapter 13.

Carbohydrate and Fat. Various contributions of these macronutrients to total energy (TE) are proposed, ranging from the conventional high carbohydrate (45% to 65% of TE), low fat (<30% of TE) to a combined carbohydrate and monounsaturated fat (MUFA) intake of 60% to 70% of TE. The various approaches are dependent on the individual patient response to carbohydrate intake and blood glucose and lipid control (ADA, 2005, 2008; Franz, 2012; Mann, 2006).

Irrespective of the percentage contribution of carbohydrates, the aspect that all agree upon is the importance of a diet rich in complex carbohydrate and fibre. The benefits of a high fibre intake for blood glucose and lipid control has been proven, and one should aim for an intake of 14 g/1000 kcal or 20 to 40 g/day. Emphasis should be placed on including sources of soluble fibre – e.g., fruit, oats, dried beans, and legumes. Soluble fibre is also known as viscous fibre.
Table 12.3: Dietary recommendations for individuals with diabetes

<table>
<thead>
<tr>
<th>Nutrient</th>
<th>Prescription</th>
</tr>
</thead>
<tbody>
<tr>
<td>Energy</td>
<td>Individualized</td>
</tr>
<tr>
<td></td>
<td>Goal is to achieve weight loss and maintain a reasonable body weight</td>
</tr>
<tr>
<td>Carbohydrates</td>
<td>Make up 45% to 65% of TE</td>
</tr>
<tr>
<td></td>
<td>Carbohydrate and MUFA are 60% to 70% of TE</td>
</tr>
<tr>
<td></td>
<td>must be &gt;130 g carbohydrate per day</td>
</tr>
<tr>
<td></td>
<td>Fibre is 20 to 40 g/day or 14 to 25 g/1000 kcal/day</td>
</tr>
<tr>
<td></td>
<td>Approximately 50% of fibre should be soluble</td>
</tr>
<tr>
<td></td>
<td>Low glycaemic index (GI) foods are encouraged</td>
</tr>
<tr>
<td></td>
<td>Sucrose and other sugars are &lt;10% of TE, with sucrose &lt;50 g/day</td>
</tr>
<tr>
<td>Proteins</td>
<td>Make up 10% to 20% of TE</td>
</tr>
<tr>
<td></td>
<td>Total 0.8 g/kg in the case of nephropathy (kidney disease)</td>
</tr>
<tr>
<td>Fats</td>
<td>Make up 20% to 35% of TE</td>
</tr>
<tr>
<td></td>
<td>Saturated fat and trans fats are 7% to 10% of TE</td>
</tr>
<tr>
<td></td>
<td>PUFA are 10% of TE</td>
</tr>
<tr>
<td></td>
<td>MUFA are 10% to 20% of TE</td>
</tr>
<tr>
<td></td>
<td>Limit cholesterol to 200 to 300 mg/day</td>
</tr>
<tr>
<td></td>
<td>Emphasis is on omega-3 fatty acids</td>
</tr>
<tr>
<td></td>
<td>Allow 2 g/day sterols and stanols</td>
</tr>
<tr>
<td>Vitamins and minerals</td>
<td>There is no evidence that warrants supplementation if a balanced diet is followed; an individualized approach may, however, be beneficial</td>
</tr>
<tr>
<td>Salt</td>
<td>Maximum 3000 mg/day salt or 2300 mg/day if hypertensive</td>
</tr>
</tbody>
</table>

Notes: MUFA = monounsaturated fatty acids; PUFA = polyunsaturated fatty acids; TE = total energy.

The relationship between the digestibility of carbohydrate foods and their glycaemic response is indicated by the glycaemic index (GI). Low-GI diets have produced significant improvements in blood glucose control and lipid metabolism. Hence foods with a low GI are recommended for people with DM (ADA, 2008; Mann, 2006; Wheeler & Pi-Sunyer, 2008; Ceriello, 2008).

Protein. Dietary protein does not affect blood glucose concentrations directly but does increase insulin responses. Protein intake should be 10% to 20% of TE. Children and adolescents need about 12% to 20% of TE as protein, or 0.9 to 2.2 g/kg body weight. During pregnancy an additional 10 g protein should be consumed per day, and during lactation an additional 15 g of protein per day during the first 6 months and 12 g/day thereafter. Half of protein intake should be of high biologic value, that is, containing all nine essential amino acids (ADA, 2008; Mann, 2006).

Micronutrients. Although certain micronutrients may affect glucose and insulin metabolism, no convincing evidence exists to date documenting the role of micronutrients in the development of DM (ADA, 2008). It is also likely that the response of patients to micronutrient supplements is determined by a patient’s micronutrient status, such that only people with micronutrient deficiencies will respond to supplements favourably. Therefore, routine supplementation of micronutrients and antioxidants in people with DM is not
recommended (ADA, 2008; Mann, 2006; Bastaki, 2005). However, in people following an energy-restricted diet, including pregnant or lactating women, strict vegetarians, or the elderly, a multivitamin supplement may be needed (ADA, 2008). The supplement should not exceed nutrient intakes in excess of the Dietary Reference Intake (DRI). Eating a balanced diet and obtaining all the nutrients through natural foods should always be the first priority (ADA, 2008; Mann, 2006).

2.5.2 Meal distribution
It is important that every diabetic patient maintains a balance between his or her food intake and the type or amount of medication at all times, in order to maintain euglycaemia.

Meals should be eaten at more or less the same time each day. The total energy, and specifically carbohydrate distribution, must be taken into account when determining actual meal size and the meal distribution for the day. Thus in order for a patient with DM to prevent blood glucose fluctuations, dietary manipulation of meal size and the timing thereof is necessary; this must coincide with the peak action of the various medications prescribed (ADA, 2008). There is therefore no standard dietary prescription regarding food distribution other than the three basic meals that need to be consumed each day. The size of the meals and the intake of snacks must be individualized according to the medication that the patient receives.

2.5.3 Sweeteners and sucrose
Several alternative sweeteners are available for use by individuals with DM. They are categorized as nutritive (energy containing) and non-nutritive (non–energy-containing) (see Table 12.4). They are all considered safe provided intake falls within the intake levels established by the Food and Drug Administration (FDA) (ADA, 2008; Wheeler & Pi-Sunyer, 2008).

<table>
<thead>
<tr>
<th>Nutritive</th>
<th>Non-nutritive (approved by the FDA)</th>
</tr>
</thead>
<tbody>
<tr>
<td>- Fructose</td>
<td>- Saccharin</td>
</tr>
<tr>
<td>- Sugar alcohols, e.g., sorbitol, xylitol, maltitol, erythritol, isomalt, lactitol, mannitol, tagatose, and hydrogenated starch hydrolysates</td>
<td>- Aspartame</td>
</tr>
<tr>
<td></td>
<td>- Acesulfame potassium</td>
</tr>
<tr>
<td></td>
<td>- Neotame</td>
</tr>
<tr>
<td></td>
<td>- Sucralose</td>
</tr>
</tbody>
</table>

Sources: ADA, 2008; Wheeler & Pi-Sunyer, 2008.

The use of nutritive sweeteners by diabetic patients should be evaluated on the basis of the individual’s blood glucose and lipid control, as well as in the overall context of the diet. Moderation in the consumption of nutritive sweeteners would appear to be prudent as they may be involved in the aetiology of diabetic complications (ADA, 2008; Bastaki, 2005).

If a patient with DM wishes to use sweeteners, a combination of sweeteners should be ingested so as to minimize any possible risks. A modest intake of sucrose and other refined sugars is allowed, contingent on metabolic control and body weight. If sucrose is included in the diet, it should form part of a high-carbohydrate, high-fibre, low-fat diet and should not exceed 50 grams per day or be more than 10% of TE. Randomized controlled trials have found that glycaemic and lipid control are not adversely affected by such amounts of sugar (ADA, 2008; Wheeler & Pi-Sunyer, 2008).

2.5.4 Alcohol
People with DM need to be aware of the physiological effects of alcoholic beverages. The consumption of alcohol can result in hypoglycaemia. Because the absorption of alcohol is delayed by food, it should always be ingested together with a meal, not on an empty stomach (ADA, 2008). Alcohol intake should not exceed...
one drink per day for women or two drinks for men (Bastaki, 2005). Alcohol consumption is contra-indicated in DM patients with hypertriglyceridaemia, uncontrolled hypertension, peripheral neuropathy (disease of peripheral nerves), or those on medication that interacts with alcohol (ADA, 2008; Bastaki, 2005). Alcohol is discussed in more detail in Chapter 13.

2.5.5 Diet during pregnancy
It is of the utmost importance for a mother and baby’s health that the mother’s blood glucose is kept between 4 and 6 mmol/l. This can be achieved with either diet therapy alone or with insulin therapy (basal bolus), with or without snacks. The mother should, if possible, test her blood sugar levels 6 times per day (before meals and 2 hours after meals) and provide the results to the dietitian/doctor. Decisions such as whether to include snacks should be based on these readings and the type of medication used (ADA, 2008).

2.6 Physical Activity
One of the most important goals in the treatment of DM today is to give the patient a chance to live as normal a life as possible. This includes being physically active and able to participate in different sports and leisure-time activities. Regular physical activity has several benefits for health; people with DM share these benefits as well. Such benefits include better glycaemic control, the prevention or delay of cardiovascular complications, weight maintenance, and psychological benefits (Bastaki, 2005). Physical activity is therefore an important component in the treatment of patients with DM (ADA, 2008).

Regular activity of 150 minutes/week or 30 to 45 minutes/day, 3 to 5 days per week initially, is recommended (ADA, 2005, 2008, 2009b; Mann, 2006). Daily exercise should be gradually increased in duration and frequency to 60 to 75 minutes of moderate-intensity activity (e.g., walking) or 35 minutes of vigorous activity (e.g., jogging) (ADA, 2005, 2009b).

2.7 Medications
2.7.1 Oral medications
The aim of treating type 2 DM with an oral medication is to decrease peripheral insulin resistance and to increase pancreatic insulin secretion. The main objective of these drugs is to correct the underlying disorder, namely either decreased secretion of insulin by the pancreas or impaired insulin function (i.e., peripheral insulin resistance). The drugs are prescribed in combination with diet and lifestyle modifications (Bastaki, 2005). The oral hypoglycaemic agents can be divided into insulin secretagogues (sulphonylureas) and insulin sensitizers (biguanides, thiazolidinediones) (Bastaki, 2005). Table 12.5 indicates the mechanisms of action and other differences of the oral agents (Bastaki, 2005; Levitt et al., 2009; Nathan et al., 2009).

2.7.2 Insulin
Patients requiring insulin therapy have an absolute insulin deficiency and therefore need exogenous insulin injections to mimic physiological insulin delivery. A slow basal insulin secretion is necessary to control hepatic glucose production, while bolus, post-prandial secretions are needed to control peripheral glucose uptake (Bastaki, 2005; Daneman, 2006; Levitt et al., 2009). Because the duration of action and more specifically the peak action of the different insulin preparations differ, meal distributions will vary according to each treatment. Table 12.6 indicates the different insulin therapies available and their nutritional effects (Daneman, 2006; Bastaki, 2005).

Individuals using fixed daily insulin dosages need a consistent intake of carbohydrates in terms of time and amount, whereas individuals using rapid-acting insulin by injections or insulin pumps can adjust their meal and snack insulin amount based on the carbohydrate content of the meal or snack (ADA, 2008).
Table 12.5: Oral hypoglycaemic medications used in the treatment of type 2 diabetes

<table>
<thead>
<tr>
<th>Classification</th>
<th>Side effects</th>
<th>Contraindications</th>
<th>Nutritional effects</th>
<th>Function</th>
</tr>
</thead>
<tbody>
<tr>
<td>Insulin sensitizers</td>
<td>Gastro-intestinal tract side effects, including heartburn, reflux, diarrhoea, or constipation (one or more of these may occur)</td>
<td>Pregnancy and lactation</td>
<td>Does not increase insulin levels; only 3 meals per day are needed (unless otherwise decided)</td>
<td>Improves insulin resistance</td>
</tr>
<tr>
<td>Biguanides, Metformin</td>
<td></td>
<td>Renal dysfunction</td>
<td></td>
<td></td>
</tr>
<tr>
<td>(taken with or directly after meals)</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Pioglitazone, Rosiglitazone</td>
<td>Weight gain</td>
<td>Childhood</td>
<td>Does not increase insulin levels; only 3 meals per day are needed (unless otherwise decided)</td>
<td>Improves insulin resistance</td>
</tr>
<tr>
<td>(taken with or directly after meals)</td>
<td>Oedema</td>
<td>Hypersensitivity to pioglitazones Impaired liver function Cardiac failure Pregnancy and lactation Type 1 DM Ketoacidosis</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Insulin secretogogues</td>
<td>Hypoglycaemia might occur</td>
<td>Hypersensitivity to gliclazide Renal dysfunction Liver impairment Type 1 DM Ketoacidosis Pregnancy and lactation Childhood</td>
<td>Three meals are needed; some patients with hypoglycaemic symptoms might need a snack 2 hours after taking the tablet</td>
<td>Stimulates the beta-cells of the pancreas to produce insulin</td>
</tr>
<tr>
<td>Sulphonylureas, Gliclazide, Glibenclamide, Glimepiride (taken 30 minutes before meals)</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

Table 12.6: Insulin therapies and their nutritional effects

<table>
<thead>
<tr>
<th>Classification</th>
<th>Product names and manufacturers</th>
<th>Administration</th>
<th>Nutritional effects</th>
</tr>
</thead>
<tbody>
<tr>
<td>Rapid insulin (ultra-short working)</td>
<td>Novorapid (Novo Nordisk), Humalog (Lilly), Apidra (Sanofi-Aventis)</td>
<td>Directly before a meal</td>
<td>Duration is about 3 hours and peak after one hour, snacks in-between are therefore unnecessary and might lead to increased blood sugar (unless patient experiences hypoglycaemia).</td>
</tr>
<tr>
<td>Short-acting insulin</td>
<td>Humalin R (Lilly), Actrapid (Novo Nordisk)</td>
<td>30 minutes before meals</td>
<td>Duration is about 3–6 hours and peak time is after 1–3 hours, snacks 2 hours after administration are essential.</td>
</tr>
<tr>
<td>Intermediate-acting</td>
<td>Protophane (Novo Nordisk), Humalin N (Lilly)</td>
<td>At bedtime</td>
<td>Duration is about 18–20 hours and peak time occurs after about 90 minutes after administration. A single snack 2 hours after administration is important.</td>
</tr>
</tbody>
</table>
### 2.8 Acute Complications

Both hypoglycaemia and diabetic ketoacidosis are extremely dangerous and should be treated immediately. Frequent self-monitoring of glucose concentrations is vital in preventing these complications, especially for diabetic patients on insulin treatment. Diabetic persons should therefore be encouraged to obtain a glucometer (from pharmacies or government institutions). The diabetic person and their family and friends should also be trained in these acute situations regarding prevention and treatment.

#### 2.8.1 Hypoglycaemia

Hypoglycaemia occurs when blood glucose concentrations drop below 4 mmol/l (72 mg/dl). Those with frequent hypoglycaemia might lose the ability to recognize the symptoms. Hypoglycaemia is more frequent with insulin therapy and to some degree sulphonylureas. It can occur when too much insulin is injected, when not enough or no food is consumed with insulin, during exercise, or when excessive alcohol is consumed (ADA, 2009b; Daneman, 2006; Bastaki, 2005).

If hypoglycaemia is suspected, it must be tested for immediately by measuring blood glucose. If hypoglycaemia is confirmed, a fast-energy release carbohydrate (e.g., sugar, honey, or syrup) as well as a slow-energy release carbohydrate (such as whole-wheat biscuit or a sandwich) must be consumed immediately. Blood glucose concentrations should increase within 10–15 minutes. This should be followed up with a meal to prevent the recurrence of hypoglycaemia (ADA, 2008, 2009b). Fatty foods and chocolate must be avoided under these circumstances because the fat will delay the absorption of glucose. An unconscious person should not be given something to eat or drink because this might cause choking. Sticky glucose, such as syrup, must be smeared on the inside of the cheek, where glucose is rapidly absorbed (ADA, 2008, 2009b; Bastaki, 2005).

#### 2.8.2 Diabetic ketoacidosis

Diabetic ketoacidosis (DKA) is a severe and acute complication caused by a lack of insulin or a failure to follow dietary restrictions. The main symptoms are similar to hyperglycaemia (polyuria, polydipsia, weakness, weight loss). Nausea and vomiting can also occur. The combination of hyperglycaemia, ketonuria, and metabolic acidosis is highly indicative of DKA (Van Zyl, 2008).

DKA can leave the person comatose. The situation demands immediate reaction and treatment, which consist of fluid replacement, insulin treatment, potassium and phosphorus corrections, and administration of biphosphonates. Blood glucose concentrations and urine must be tested hourly (Van Zyl, 2008).

### 2.9 Long-term Complications

The major long-term complications of DM can be divided into macro- and microvascular complications (see Table 12.7).
Table 12.7: Classification of complications in diabetes

<table>
<thead>
<tr>
<th>Classification</th>
<th>Description</th>
</tr>
</thead>
<tbody>
<tr>
<td>Macrovascular complications</td>
<td>Ischaemic heart disease</td>
</tr>
<tr>
<td></td>
<td>Stroke</td>
</tr>
<tr>
<td></td>
<td>Peripheral vascular disease</td>
</tr>
<tr>
<td>Microvascular complications</td>
<td>Retinopathy</td>
</tr>
<tr>
<td></td>
<td>Nephropathy</td>
</tr>
<tr>
<td></td>
<td>Neuropathy</td>
</tr>
</tbody>
</table>

The most important risk factor for the development of the complications of DM is, apart from poor glycaemic control, the duration of the disease. Since the latter cannot be controlled, it is important to focus treatment on control of glycaemic levels. Even though the relationship between glycaemic control and the incidence of complications has been much debated, the latest emerging evidence shows that the incidence of complications can be reduced by up to 70% with good glycaemic control (Franz, 2012).

2.9.1 Macrovascular complications
Cardiovascular disease (CVD) accounts for approximately 80% of total mortality resulting from DM (Daneman, 2006; ADA, 2009b). The majority of this mortality is the consequence of ischaemic heart disease (IHD), and the remaining results from stroke as well as peripheral vascular disease. Atherosclerosis is the dominant underlying pathological process. Dyslipidaemia is common amongst these patients and is characterized by elevated concentrations of triglyceride (TG), very low-density lipoprotein cholesterol (VLDL-C), and reduced high-density lipoprotein cholesterol (HDL-C). The elevated TG concentration is closely related to the atherogenic changes in the level of low-density lipoprotein cholesterol (LDL-C) particles that increase the risk of IHD. The level of HDL-C is inversely related to IHD and is thought to have a protective effect twice that of the atherogenic effect of LDL-C (Ceriolo, 2008).

The management of dyslipidaemic patients involves blood glucose control together with the treatment of lipid abnormalities, hypertension, smoking, and obesity. Regular exercise is an important component of the treatment regimen. Dietary strategies include energy restriction to attain weight loss and an adaptation of the fat content of the diet according to the specific lipid abnormality present. The restriction of total fat to <30% of the TE, with saturated fat (including trans fatty acids) <10%, PUFA 10%, and MUFA 10% to 20%, should be the first approach. If, after a 6-month period, a significant improvement of the dyslipidaemia has not been achieved, or if there is pre-existing CVD, the total fat content should be further reduced to 20% to 25% of the TE, with saturated fat <7% (ADA, 2005, 2009b). This decrease in the fat content of the diet will mean an increase in the total carbohydrate content.

2.9.2 Microvascular complications
Retinopathy: Retinopathy (disease of the retina resulting in vision impairment or loss) occurs in all types of DM, with a prevalence of more than 90% after 20 years. The development of this condition is dependent primarily on the duration of the disease (Daneman, 2006; ADA, 2009b; Ceriolo, 2008).

Nephropathy: Diabetic nephropathy (damage to or disease of the kidney) is the most common cause of renal failure in the developed world (Daneman, 2006). Nephropathy occurs in 35% to 45% of type 1 DM patients and in less than 20% of type 2 patients. The most important risk factors for the development of this condition are duration of DM and HbA1c levels, ratio of LDL-C to HDL-C, and waist-to-hip ratio (i.e., abdominal obesity) (Daneman, 2006; ADA, 2009b). Concurrent CVD in the majority of these patients makes nephropathy the complication with the highest mortality.

In the presence of diabetic nephropathy, dietary protein should be restricted to 10% of TE or 0.8 g per kg body weight (Mann, 2006; ADA, 2008).
**Neuropathy.** The most important risk factors for the development of neuropathy (nerve damage) are the duration of DM and poor blood glucose control (Shakil et al., 2008). The risk is increased by the presence of hypertension, hypercholesterolaemia, and obesity. More than 70% of diabetic patients suffer from one or more gastro-intestinal symptoms, which may be related to autonomic neuropathy. This condition affects gastric and intestinal motility. Table 12.8 lists the most common symptoms associated with autonomic neuropathy.

**Table 12.8:** Gastric and intestinal symptoms of autonomic neuropathy

- Dysphagia
- Heartburn
- Anorexia, nausea, vomiting
- Gastric distension / bloating
- Delayed gastric emptying (gastroparesis diabeticorum)
- Abdominal pain
- Constipation alternating with diarrhoea

*Sources: ADA, 2009b; Shakil et al., 2008.*

The symptoms indicative of gastric retention are collectively known as gastroparesis, and their treatment focuses on the relief of the individual symptoms (Shakil et al., 2008). A diet consisting of 6 small meals per day, in conjunction with intensive therapy to improve glycaemic control, may be helpful. Drugs that regulate intestinal motility are very effective. Difficulty in maintaining euglycaemia and the presence of alternating hyper- and hypoglycaemia are typically seen in patients with delayed gastric emptying, because the latter affects the absorption of nutrients in an unpredictable way.

Chronic diarrhoea, commonly occurring at night, occurs in up to 85% of patients who have had poor blood glucose control for 2 to 5 years. Treatment consists of glycaemic control as well as antidiarrhoeal agents, and antibiotics in the case of proven bacterial overgrowth (Shakil et al., 2008).

Constipation, which often occurs intermittently with diarrhoea, can be treated with high-fibre foods. In the presence of gastroparesis, however, soluble fibre can be counter-productive because it will further impair gastric emptying. Insoluble fibre, administered post-pylorically, is thought to be beneficial (Shakil et al., 2008).

3. **CARDIOVASCULAR DISEASE**

3.1 **Introduction**

Cardiovascular disease (CVD) encompasses a range of diseases, the dominant ones being stroke (cerebrovascular disease) and ischaemic heart disease (IHD; also known as coronary heart disease).

It is estimated that of the 58 million annual deaths globally, 30% can be attributed to CVD (WHO, 2007). This has been the leading cause of death for several decades. Also, about 85% of these deaths occur in low- and middle-income countries (see Table 12.9) (WHO, 2002, 2003). Each year an estimated 32 million heart attacks and strokes occur around the world (Mathers et al., 2002). Research indicates that in sub-Saharan Africa and the Caribbean, fatalities from stroke are much higher than in developed countries (Walker et al., 2000), mainly because of limited health-care facilities, untreated risk factors, and an increase in the incidence of atherosclerotic diseases.

The major focus of this section is the management of risk factors for CVD, especially IHD. In particular, we look at abnormalities of blood lipid concentrations (dyslipidaemia) and of hypertension. In Chapter 13, we look more generally at the prevention of CVD.
Table 12.9: Global cardiovascular disease mortality, for 2001 (in thousands)

<table>
<thead>
<tr>
<th></th>
<th>African region</th>
<th>American region</th>
<th>European region</th>
<th>South-East Asia region</th>
<th>Western Pacific region</th>
<th>Eastern Mediterranean region</th>
</tr>
</thead>
<tbody>
<tr>
<td>Stroke</td>
<td>307</td>
<td>454</td>
<td>1480</td>
<td>1070</td>
<td>1926</td>
<td>218</td>
</tr>
<tr>
<td>Ischaemic heart disease (IHD)</td>
<td>333</td>
<td>967</td>
<td>2423</td>
<td>1972</td>
<td>963</td>
<td>523</td>
</tr>
<tr>
<td>Hypertensive heart disease</td>
<td>54</td>
<td>131</td>
<td>175</td>
<td>138</td>
<td>285</td>
<td>91</td>
</tr>
<tr>
<td>All CVDs</td>
<td>985</td>
<td>1979</td>
<td>5042</td>
<td>3797</td>
<td>3745</td>
<td>1037</td>
</tr>
</tbody>
</table>


3.2 Assessment of Risk of IHD and Diagnosis of Lipid Abnormalities

Most of the burden of IHD is caused by one or more cardiovascular risk factors, especially hypertension (high blood pressure), smoking, dyslipidaemia, lack of exercise, and abdominal obesity (large waist circumference) (WHO, 2002, 2003). The presence of DM is an especially important risk factor for IHD. Each of these risk factors is preventable. Two other important but unpreventable risk factors for IHD are increasing age and family history of IHD.

There is much overlap between IHD and the major risk factors for stroke, which include smoking, hypertension, lack of exercise, the presence of DM, and greater age. However, abnormalities in the concentrations of blood lipids are of much greater importance for IHD than for stroke. The desirable blood lipid concentrations are as follows:

- Total cholesterol (TC) <5.0 mmol/L
- LDL-C <3.0 mmol/L
- HDL-C >1.2 mmol/L
- TC to HDL-C ratio <6
- TG <1.5 mmol/L

Treatment recommendations for IHD are based on the level of risk of the patient (McPherson et al., 2006): as the number of risk factors increases, so does the level of risk. A relatively low-risk patient – in comparison with other patients at significant risk of IHD – may have one or two risk factors. Treatment can begin when LDL-C >5 mmol/L or TC/HDL-C >6. A moderate-risk patient may have three or so risk factors. Treatment can begin when LDL-C >3.5 mmol/L or TC/HDL-C >5. A high-risk patient has even more risk factors, especially when he or she has already shown clinical signs of IHD.

Clearly, categorizing patients and selecting treatment criteria is a value judgement. It is especially important to assess the overall level of risk rather than be overly concerned by one particular risk factor. The available of drugs and other resources is a major consideration.

Most patients with dyslipidaemia who are at low or moderate risk should receive dietary treatment for a minimum period of 3 months, as described below. If after that period the blood lipids have not reached target values, drug treatment can then be administered. Patients at high risk of IHD should receive drug treatment earlier, and they need medical supervision.

3.3 Screening and Global Risk Assessment

The WHO developed a range of risk assessment charts that can be used in various countries – both developed and developing – to determine CVD risk (WHO, 2007). Furthermore, screening is recommended by the Canadian Cardiovascular Society for the following people (McPerson et al., 2006):

doi:10.15215/aupress/9781927356111.01
• Men over 40 years of age and women over 50 years of age and/or who are post-menopausal (screening recommended every 1 to 3 years)
• Children with a family history of severe hypercholesterolaemia
• Adults with dyslipidaemia, DM, current or recent tobacco smoking, hypertension, abdominal obesity, or family history of premature IHD.

Of course, abiding by these guidelines is only feasible in countries where the medical system has ample resources and/or for affluent patients.

3.4 Dietary Management of Dyslipidaemia

The focus of this section is the dietary management of dyslipidaemia with the goal of preventing IHD, especially in those at relatively high risk of IHD.

3.4.1 Overview of dietary management

The first step in the treatment of dyslipidaemia is to implement lifestyle changes. In this regard, dietary intervention is of major importance and preferably is done with the support of a dietitian. Cultural, ethnic, regional, and religious differences in dietary practices and food choices should be taken into account.

The key objectives of dietary intervention are

• to achieve and maintain the desired blood concentrations of TC, LDL-C, HDL-C, and TG, and
• to achieve and maintain appropriate body weight by balancing energy intake and energy expenditure.

The diet prescribed for those with dyslipidaemia should

• provide enough energy to help achieve and maintain body weight goals;
• include a wide variety of food to ensure that macro- and micronutrient needs are met;
• be low in saturated fatty acids (SFA);
• be very low in or contain no trans-fatty acids;
• contain polyunsaturated fatty acids (PUFA) and monounsaturated fatty acids (MUFA) to replace SFA;
• include foods high in fibre, especially soluble fibre;
• provide enough protein to build and maintain body tissues;
• limit alcohol intake to around 0.5 to 1 drink per day for a woman or 1 to 2 per day for a man (those with high TG concentrations should avoid alcohol); and
• keep sodium to a maximum of about 2000 mg sodium or 5 grams salt per day.

3.4.2 The role of diet in the management of dyslipidaemia

Weight control and energy intake. Obesity has been shown to be an independent risk factor for cardiovascular morbidity and total mortality (McGee, 2005). Obesity is also associated with other risk factors, such as raised blood pressure, glucose intolerance, DM, and dyslipidaemia (Haslam & James, 2005). Research indicates that weight loss induced by a weight-reducing diet and regular exercise reduces TC, LDL-C, and blood pressure, and increases HDL-C (Avenell et al., 2004; Aucott et al., 2005). A body mass index (BMI) of between 18.5 and 25 is regarded as healthy for adults. Obese people (BMI >30) and those who are overweight (BMI 25–30) should be strongly encouraged to reduce their weight. Persons with increased abdominal fat (waist circumference of >102 cm in men or >88 cm in women) should also be encouraged to lose weight (De Backer et al., 2003). An appropriate target is a waist circumference of <94 cm for males and <80 cm for females. Cut-off levels should be lower for people of South Asian or East Asian ancestry (McPherson et al., 2006). Weight-loss strategies are discussed in Chapter 13.

Total fat. Total lipids (total fat) consists of different types of fatty acids, such as SFA, MUFA, and PUFA, as well as other components, such as cholesterol. The type of fat consumed is of far more importance to the
development of IHD than the total amount of fat consumed. This is because the different types of fatty acids affect the blood lipids and lipoproteins differently (Sanders, 2009).

The following discussion on dietary fats applies not only to the management of those with dyslipidaemia, but also to the primary prevention of IHD.

A total fat intake of at least 20% of total energy (20% TE) is regarded as consistent with good health, and women of reproductive age need at least 20% TE from fat (WHO, 2003). In the United States, a total fat intake of 25% to 35% TE is recommended as part of the Therapeutic Lifestyle Changes (TLC) dietary goals recommended by the National Cholesterol Education Program (NCEP) of the Adult Treatment Panel (ATP) III for the prevention of CVD (Krummel, 2008).

**Saturated fatty acids.** A high consumption of SFAs raises total cholesterol and LDL-C, although different types of SFAs have different effects (Sanders, 2009). The major sources of SFA are dairy and meat foods that have a high fat content. Another important source of SFA is tropical oils, namely palm kernel oil and coconut oil. Tropical oils, therefore, are best avoided. In many countries the presence of these vegetable oils is stated on food labels. However, removing these oils from the diet can be problematic as they are an important source of low-cost food energy for poor people in many countries (WHO, 2003).

An SFA intake of <10% TE is recommended by the WHO for the prevention of IHD (WHO, 2003). The TLC dietary goals, which are directed at people with elevated LDL-C concentrations, recommend that SFA intake should be reduced to <7% (Krummel, 2008).

**Trans-fatty acids.** PUFAs are partially hydrogenated in order to increase the shelf-life of foods containing these fatty acids and improve the texture. This process leads to the formation of trans-fatty acids. These fats are mostly found in retail fats (such as hard margarine), spreads, deep-fried fast foods, and baked goods. Naturally occurring trans-fatty acids are found in small amounts in dairy products and in meat from ruminants. Research indicates that trans-fatty acids increase the risk of IHD because they elevate LDL-C and decrease HDL-C (Mozaffarian et al., 2009). Trans-fatty acids are now recognized as the most dangerous type of dietary fat in terms of increasing the risk of IHD. The intake of trans-fatty acids should therefore be minimized, and should be under 1% TE (Krummel, 2008; Lloyd-Jones et al., 2010).

**Mono- and polyunsaturated fatty acids.** MUFAs contain one double bond in the carbon chain. Many foods contain MUFAs, but olive oil is an especially rich source of oleic acid, a MUFA. PUFAs contain two or more double bonds in the carbon chain. There are two families of PUFAs in the diet, the omega-6 (n-6), of which linoleic acid (LA) is the parent fatty acid, and the omega-3 PUFAs (n-3), of which α-linolenic acid (ALA) is the parent fatty acid. Both omega-6 and omega-3 fatty acids are referred to as essential fatty acids because they are required for various body functions but cannot be formed in the body and must therefore be consumed as part of the diet.

**Omega-3 fatty acids and fish oil.** The three most important types of omega-3 fatty acids are ALA, eicosapentaenoic acid (EPA), and docosahexaenoic acid (DHA). Rich sources of EPA and DHA are fatty fish and fish oil. Sources of ALA include flaxseed oil, canola (rapeseed) oil, and walnuts.

Consuming omega-3 fatty acids from fish or fish oil has beneficial effects on cardiovascular risk factors and the prevention of IHD (Zheng et al., 2012; Mozaffarian & Wu, 2011). The effect of ALA on risk of CVD is less clear and is probably much weaker. Omega-3 fats from fish have several actions in the body that are believed to protect against CVD; these include antiarrhythmic effects, lowering TG and blood pressure, reducing platelet function/aggregation, improving vascular function, and decreasing inflammation potential (Micallef & Garg, 2009).

Based on this evidence, an intake of 400–500 mg/day EPA plus DHA is recommended for the prevention of IHD (Hill et al., 2009; Lloyd-Jones et al., 2010). Omega-3 fats should ideally come from fatty fish. An appropriate intake is 2 servings per week of fatty fish, such as salmon, sardines, herring, tuna, and mackerel (Hill et al., 2009). An intake of 1 g/day, in total, of EPA and DHA is recommended for patients with IHD (Hill et al., 2009). If the use of fish oil capsules is considered, this should occur under medical supervision, because large amounts of fish oil from supplements could interact with medications and result in bleeding.

**Recommended essential fatty acid intake levels.** Table 12.10 describes MUFAs and PUFAs in terms of their main food sources and their effect on blood lipids.
Table 12.10: Sources of fatty acids and effect on blood lipids

<table>
<thead>
<tr>
<th>Fatty acid type</th>
<th>Fatty acid name</th>
<th>Major food sources</th>
<th>Effect on risk factors for CVD</th>
</tr>
</thead>
<tbody>
<tr>
<td>MUFA</td>
<td>Oleic acid</td>
<td>Olive oil</td>
<td>↓ TC*</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Canola oil</td>
<td>↓ LDL-C*</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Nuts</td>
<td>↓ TG</td>
</tr>
<tr>
<td>omega-6 PUFA</td>
<td>Linoleic acid</td>
<td>Soya bean oil</td>
<td>↓ TC</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Sunflower seed oil</td>
<td>↓ LDL-C</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>↓ TG</td>
</tr>
<tr>
<td>omega-3 PUFA</td>
<td>Eicosapentaenoic acid (EPA)</td>
<td>Fatty fish</td>
<td>↓ Blood pressure</td>
</tr>
<tr>
<td></td>
<td>Docosahexaenoic acid (DHA)</td>
<td>Fatty fish</td>
<td>↑ Cardiac function</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>↑ Arterial compliance</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>↑ Endothelial function</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>↑ Vascular reactivity</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>↑ Antiplatelet effects</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>↑ Anti-inflammatory effects</td>
</tr>
</tbody>
</table>

* Effect occurs when MUFA replaces SFA in the diet, and is much lower than with PUFA.

Source: Kris-Etherton & Innis, 2007; WHO, 2003; Riediger et al., 2009.

An intake of 2.5% TE from LA and 0.5% TE from ALA is recommended in order to meet essential fatty acid requirements (Elmadfa & Kornsteiner, 2009). The requirement for LA translates to about 2.8 g /1000 kcal (4200 KJ) and for ALA to at least 0.6 g/1000 kcal. However, based on the Dietary Reference Intake (DRI) levels, recommended intake for LA in North America is about twice as high. A total PUFA intake of 6% to 11% TE is recommended for the prevention of chronic disease (Elmadfa & Kornsteiner, 2009).

Dietary cholesterol. The risk of IHD probably rises with an increase in dietary cholesterol, but observational evidence for this association is contradictory. Cholesterol in the blood and tissue comes from either the diet (from egg yolk, organ meat, and dairy fat) or from hepatic synthesis. The evidence regarding the impact of dietary cholesterol on the risk of IHD is contradictory.

It is recommended that the intake of dietary cholesterol be restricted to <300 mg/day for the general public, and on the Therapeutic Lifestyle Changes (TLC) diet an intake of <200 mg/day is recommended (Krummel, 2008).

Carbohydrate. A carbohydrate intake of 55% to 60% TE from a variety of sources – cereals, fruit, and vegetables – is recommended. An increase in refined carbohydrate intake may raise TG concentrations, especially in the short term (Jenkins et al., 2004). However, more fibre in the diet may counteract this undesirable effect. This is one reason, among many, for recommending unrefined rather than refined carbohydrates.

Dietary fibre. Dietary fibre is a combination of polysaccharides and lignin, which cannot be broken down by intestinal enzymes. There are two types of fibre: soluble fibre, such as pectins, gums, mucilages, and hemicelluloses, and insoluble fibre, such as cellulose and some hemicelluloses. Fibre can help lower TC and LDL-C and is linked to reduced risk of IHD (Anderson & Hanna, 1999; Truswell, 2002; Rimm et al., 1996).

While the optimal dietary intake of fibre is uncertain, an intake of approximately 25 to 35 g/day can be recommended for adults. The diet should therefore contain a generous amount of foods high in soluble fibre, such as whole grains, legumes (especially dry beans), fruit, and vegetables.

When fibre is added to the diet, its intake should be increased gradually and accompanied by an adequate fluid intake. An excessively high intake of dietary fibre may induce gastro-intestinal side effects, and it may interfere with the absorption of nutrients such as calcium.

Plant sterols. Dietary plant sterols, such as sitostanol, inhibit cholesterol absorption by up to 50%, thereby reducing blood lipids (Lichtenstein & Deckelbaum, 2001; Micaleff & Garg, 2009). An intake of 2 to 3 g/day has been shown to decrease TC and LDL-C concentrations (Krauss et al., 2000). An intake of 25 g (5 teaspoons) of sterol-enriched margarine per day will provide 2 to 2.5 g of plant sterol and can be used to help lower TC. Foods with added plant sterols are widely available, although the effectiveness of these products still needs to be tested in long-term clinical studies.
**Portfolio diet.** The portfolio diet is a diet strategy that aims to maximize the decrease in LDL-C. It is a vegetarian diet that includes plant sterols and soluble fibre, primarily from oats, barley, and psyllium, in addition to soy protein and almonds. This represents a radical dietary change that many patients may find difficult to sustain. A decrease in LDL-C of around 30% has been achieved when the diet has been used for 4 weeks and patients were strictly supervised (Jenkins et al., 2003), but the benefits were considerably weaker when patients were instructed to follow the diet on their own for a full year (Jenkins et al., 2006). The portfolio diet could be an effective option to control lipid levels if individuals are well motivated.

**General diet.** The management of patients at risk of IHD demands a generally healthy diet, especially with respect to aspects of the diet linked to risk of the disease. This is discussed in more detail in Chapter 13.

### 3.5 Hypertension

Raised blood pressure is a major risk factor for CVD: it is responsible for 62% of all strokes and 49% of all IHD events (He & MacGregor, 2009; Strazzullo et al., 2009).

Hypertension indicates that blood pressure is seriously elevated and has passed a particular cut-off point. Drug treatment is a widely used and effective approach in dealing with this common problem of middle-aged and older adults. Dietary factors play a major role in raising blood pressure and leading to hypertension. A dietary approach is an effective way to both prevent and treat high blood pressure.

There is a strong evidence of an association between sodium intake and high blood pressure (Aburto et al., 2013; He et al., 2013). Intake of salt (sodium chloride) is also strongly linked to risk of both IHD and stroke (Aburto et al., 2013). It is estimated that mean salt intake is about 8 to 10 grams per day in Western countries, but this figure may be much higher in many Eastern European and Asian countries (Strazzullo et al., 2009). The WHO recommends a salt intake of less than 5 grams (about one teaspoon) per day, which yields <2000 mg sodium per day (WHO, 2003). Patients with hypertension and elderly people should aim for a sodium intake of not more than 1500 mg/day (Ayala et al., 2009). Pooled data from a meta-analysis estimated a significant fall in systolic and diastolic blood pressure of 5.4/2.8 mmHg in hypertensive patients and 2.4/1.0 mmHg in normotensive patients, respectively, with a modest reduction in salt intake for 4 weeks or longer (He et al., 2013).

In developed countries, approximately 80% of salt in the diet originates from processed food, fast food, and food consumed away from home (He & MacGregor, 2009). A much smaller amount of salt, about 15%, comes from salt added to food after it has been purchased, either in the kitchen during food preparation or by the person eating the food (He & MacGregor, 2009).

Based on these figures, therefore, a major reduction in salt intake requires a major decrease in the salt content of processed food National strategies to encourage the food industry to work towards that goal have been implemented in countries such as the UK and Australia; other countries should follow this example (He & MacGregor, 2009).

A stepwise approach in reducing the salt content of processed food could be the key to achieving recommendation goals without consumer resistance. It has been shown that a 5% reduction per week in the sodium content of bread, for 6 weeks, could be achieved without losing consumer acceptance, and it resulted in a one-quarter reduction in the sodium content of bread, a major source of sodium in the diet (Girgis et al., 2003).

The Dietary Approaches to Stop Hypertension (DASH) diet is designed to lower blood pressure (Sacks et al., 2001). The DASH diet has increased amounts of fruit, vegetables, low-fat dairy, wholegrain cereals, legumes, fish, and poultry with a reduction in sugar and fat (Appel et al., 1997). Research indicates that this diet regime decreases blood pressure (Appel et al., 1997) as well as TC and LDL-C (Obarzanek et al., 2001; Svetkey et al., 1999). The DASH diet can be combined with a lowered salt intake for even greater effectiveness.

Several other dietary and lifestyle factors also play an important role in hypertension. Obesity is the most important one, followed by lack of physical activity. An excessive intake of alcohol is also harmful in this respect. All of these factors are further discussed in Chapter 13.

Hypertension is a serious and widespread problem and plays a major role in CVD, both for IHD and stroke. However, it must be stressed that an elevated blood pressure, even if well below the range classified
as “hypertension,” is still an important risk factor for CVD. For that reason, the management of risk factors for CVD should include the implementation of the above dietary strategies for patients at risk of CVD, even if they have not been diagnosed with hypertension.

3.6 Management of Patients at Risk of Ischaemic Heart Disease: A Summary

Table 12.11 summarizes the key dietary recommendations for the management of patients at increased risk of IHD.

Table 12.11: Recommendations for the management of patients at risk of ischaemic heart disease

<table>
<thead>
<tr>
<th>Food or food component</th>
<th>Recommendation</th>
<th>Specific advice</th>
</tr>
</thead>
<tbody>
<tr>
<td>Saturated fatty acids (SFAs)</td>
<td>&lt;7% TE (total energy)</td>
<td>Consume less animal fat high in SFAs. Replace high fat with low-fat meat and dairy products. Replace fatty red meat and meat products with chicken without skin, fish, and legumes (pulses). Avoid use of palm kernel oil and coconut oil.</td>
</tr>
<tr>
<td>Trans-fatty acids</td>
<td>&lt;1% TE</td>
<td>Use soft (not hard) margarine.</td>
</tr>
<tr>
<td>Polyunsaturated fatty acids (PUFAs)</td>
<td>10%TE</td>
<td>Use vegetable oils, which are good sources of PUFA. Use these oils in moderation.</td>
</tr>
<tr>
<td>Monounsaturated fatty acids (MUFAs)</td>
<td>Use to replace saturated fat</td>
<td>Use olive oil and canola oil.</td>
</tr>
<tr>
<td>Omega-3 fatty acids</td>
<td>2 servings of fatty fish per week (ideally, 1 g/day omega-3 fatty acids)</td>
<td>Good choices are salmon (both farmed and wild), sardines, herring, tuna, trout, and mackerel.</td>
</tr>
<tr>
<td>Cholesterol</td>
<td>&lt;200 mg/day</td>
<td></td>
</tr>
<tr>
<td>Fruit and vegetables</td>
<td>400 to 500 g/day</td>
<td></td>
</tr>
<tr>
<td>Fibre</td>
<td>25 to 35 g/day</td>
<td>Incorporate oat bran and legumes, especially dry beans, into the diet (in addition to fruit and vegetables).</td>
</tr>
<tr>
<td>Plant stanols/sterols</td>
<td>2 g/day</td>
<td>Note that this is obtained from certain brands of margarine, and it has not yet been properly tested.</td>
</tr>
<tr>
<td>Salt (sodium)</td>
<td>3.8 g/day (1500 mg/day)</td>
<td>Requires action by the food industry.</td>
</tr>
</tbody>
</table>
In addition to the above dietary recommendations, the following are several other important recommendations for the management of patients at increased risk of IHD:

- Smoking is a major risk factor for IHD and stroke. No one should smoke.
- Treat overweight and obesity. Aim for a BMI of <25 and a waist circumference of <94 cm for males and <80 cm for females.
- Encourage regular, moderate physical activity on most days (at least 4 or 5) each week. Ideally, this activity should consist of 30 minutes of moderate exercise; alternatives are 60 minutes of light exercise or 20 minutes of vigorous exercise.
- Try to achieve normal levels of blood lipids. Aim for TC <5 mmol/L; LDL-C <3.0 mmol/l; HDL-C >1.2 mmol/L; and TG <1.5 mmol/L. Use medicinal drugs when appropriate. Today, statins are the most commonly used drugs used in the medical treatment of hypercholesterolaemia. Low-dose aspirin (75 mg daily) is also recommended for patients at high risk of CVD and is far cheaper than statins.
- Control blood glucose. Aim for a fasting blood glucose level of <7 mmol/L. Diabetics require medical treatment.
- Maintain normal blood pressure, or lower blood pressure if it is high. Those with high blood pressure require medical treatment.

3.7 Patient Counselling
An individual with dyslipidaemia should be referred to a dietitian for dietary intervention. Dietitians are encouraged to follow a quantitative approach, especially with high-risk individuals. An individual with high TG needs specialized dietary intervention and should be referred to a lipid clinic. An initial intervention visit should be succeeded by follow-up visits to ensure long-term compliance with dietary guidelines. Practical adaptation of the habitual dietary intake of the patient to meet these guidelines forms the basis of successful dietary management. This requires close co-operation between the individual with dyslipidaemia and the dietitian.

4. CANCER
4.1 Introduction
Cancer is a group of approximately one hundred specific diseases that share certain basic characteristics, including abnormal cell growth, division, and differentiation (Alters & Schiff, 2006; WCRF/AICR, 2007). It is projected that the number of people diagnosed with cancer will double by the year 2030, with most of the increase occurring in the middle- and low-income countries (WCRF/AICR, 2007). Many of the risk factors associated with cancer are modifiable, especially tobacco smoking, alcohol consumption, and poor diet. It can be predicted that, owing to urbanization and changing food systems, the prevalence of lifestyle-related cancers will continue to increase. This will result in a huge economic and social cost.

This section presents information on the most common cancers in various countries. The focus then turns to the role of diet in the treatment of cancer. In Chapter 13 we examine the role of diet and other lifestyle factors in the prevention of cancer.

4.2 Prevalence Rates of the Most Common Cancers
Middle- and low-income countries, especially those in Africa, Asia, and Latin America, generally have higher incidence rates of cancers of the upper aerodigestive tract (mouth, pharynx, larynx, nasopharynx, and oesophagus), stomach, liver, and cervix. In contrast, the higher-income countries and urbanized and industrialized areas of middle- and low-income regions and countries have an increased rate of colorectal cancer and hormone-related cancers (breast, ovary, endometrium, and prostate). Lung cancer is globally the most common cancer, mostly because of high rates of tobacco smoking (Curado et al., 2007).
4.3  Carcinogenesis

The process of carcinogenesis – the initiation of cancer development – takes place over many years. Carcinogenesis occurs in four stages (Arab & Steck-Scott, 2004) as follows:

1. Initiation: Initial changes in the genetic make-up of the cell occur (DNA mutations). These can either be inherited or be due to exposure to specific lifestyle factors over many years (Key, 2005). Initiation leads to unregulated cell growth or damage to tumour suppressor genes. Some chemicals in food, such as aflatoxins and heterocyclic amines, are known mutagens that also cause initiation.

2. Promotion: Initiated cells are changed into populations of altered cells. Although these tumour promoters do not affect DNA directly, they increase cell replication and growth.

3. Progression: The promoted cancer cells develop into an invasive tumour, a stage which is characterized by a variety of abnormalities in the DNA.

4. Metastasis: Cancerous cells migrate to other areas in the body causing secondary growths

4.4  Cancer Treatment

It is essential that cancer is diagnosed as early as possible because this greatly improves the chances of a cure. Various procedures are in use for screening people for early-stage cancer. For example, the Pap test is commonly used for detection of precancerous changes of the cervix.

Different treatment regimens and a combination thereof can be used to treat cancer. The main three regimens are surgery, radiotherapy, and chemotherapy. Surgery is an option if no metastases have occurred and the cancer is still localized. This treatment can have long-term negative effects depending on the cancer site (for instance, surgery for stomach cancer can cause gastro-intestinal problems) (Alters & Schiff, 2006). Radiation, as with surgery, is mostly used in cancer sites where no metastases have occurred. It is often used for cervical and prostate cancer (Alters & Schiff, 2006). Radiation is focused on a specific area in the body, and any side effects will therefore be concentrated at that location (Gregory & Tutt, 2003). Chemotherapy is mostly employed when metastases have occurred, and it can be used in conjunction with either surgery or radiation (Alters & Schiff, 2006). Chemotherapy is an aggressive treatment and may have severe side effects (see Table 12.12).

Side effects and symptoms experienced during cancer treatment might be due to either the tumour itself or the treatment (Broadley, 2003). These symptoms can include nausea, vomiting, loss of appetite, and a dry mouth. Table 12.12 discusses the most common side effects, and their causes and treatment.

<table>
<thead>
<tr>
<th>Side effect</th>
<th>Cause</th>
<th>Treatment</th>
</tr>
</thead>
<tbody>
<tr>
<td>Anorexia</td>
<td>Decreased taste and smell of food, early satiety</td>
<td>Small frequent meals</td>
</tr>
<tr>
<td>Cachexia*</td>
<td>Changes in carbohydrate, protein, and liver metabolism Decreased appetite Nausea and vomiting Bowel obstruction</td>
<td>Small frequent meals Dry food or biscuits Avoiding fatty foods</td>
</tr>
<tr>
<td>Fatigue</td>
<td>Electrolyte disturbances Hypercalcaemia Poor nutrition Cardiac failure Chemotherapy Radiotherapy</td>
<td>Activities suited to ability Rest Balanced diet Psychological support Correction of anaemia Correction of electrolyte imbalances</td>
</tr>
</tbody>
</table>
### Dysphagia (difficulty swallowing)
- **Pain**
  - *Candida*
  - Obstructive lesion
  - Radiotherapy
- **Management of pain**
  - Treat *Candida*
  - Liquidizing normal food
  - Adding food supplements
  - Employing temporary nasogastric feeding

### Xerostomia (dry mouth)
- **Decreased saliva**
  - Decreased saliva
  - Mouth breathing
  - *Candida* infection
- **Management of dry mouth**
  - Artificial saliva
  - Mineral water

### Decreased saliva
- **Drugs**
- Surgery
- Radiotherapy
- Damaged or absent salivary glands
- **Management of reduced saliva**
  - Saliva substitutes
  - Sugar-free gum
  - Good oral hygiene

### Mucositis (inflammation of a mucous membrane)
- **Chemotherapy**
- Radiotherapy
- **Management of mucositis**
  - Good mouth care
  - Enough fluid intake
  - Good nutritional intake
  - Brushing teeth twice a day
  - Gargling with aspirin or saline solution
  - Avoiding mouthwashes containing alcohol
  - Treating *Candida* infection with Nystatin
  - Avoiding drinking spirits (alcohol) and smoking

### Enteritis
- **Reduced bowel mobility**
- **Management of enteritis**
  - Ensuring enough fluid and electrolyte intake
  - Reducing intake of green vegetables

### Nausea and Vomiting
- **Hypercalcaemia**
- *Uraemia*
- Raised intracranial pressure
- Drugs
- Chemo- and radiotherapy
- Constipation
- Bowel obstruction
- Pain
- Anxiety
- **Management of nausea and vomiting**
  - Small frequent meals
  - Dry food or biscuits (Provita etc.)
  - Avoiding fatty foods
  - Eating slowly
  - Small frequent meals
  - Taking liquids between meals
  - Avoiding food items high in sugar

### Cramps, distension, and diarrhoea
- **Gastric surgery**
- **Management of cramps, distension, and diarrhoea**
  - Eating slowly
  - Small frequent meals
  - Taking liquids between meals
  - Avoiding food items high in sugar

### Early satiety (fullness)
- **Gastric surgery**
- **Management of early satiety**
  - Small frequent meals

### Milk intolerance
- **Gastric surgery**
- **Management of milk intolerance**
  - Lactose-free milk and products

### Constipation
- **Medication used and antiemetics**
- **Hypercalcaemia**
- **Management of constipation**
  - Regular laxatives
  - Stimulators such as senna
  - Avoiding bulk-forming laxatives

---

*On cachexia, see Evans et al. (2008); the condition is defined as “a complex metabolic syndrome associated with underlying illness and characterized by loss of muscle with or without loss of fat mass” (2008, p. 794).*

**Sources:** 
- Broadley, 2003; Brown et al., 2001 (for cramps, distension, diarrhoea, early satiety, and milk intolerance); Gregory & Tutt, 2003 (for mucositis and enteritis); Tisdale, 2001 (for the cause of anorexia).

Small, frequent meals might be better tolerated than less frequent, larger meals during cancer treatment. Food items given must be appetizing, easy to chew and swallow, and easily digestible. The most important nutritional...
goal is to provide adequate energy. If available, nutrient-dense commercial shakes are an appropriate food (Brown et al., 2001).

### 4.5 Supplementary Treatment

Physical activity and diet can be employed for the supplementary treatment of cancer. Even small amounts of physical activity, such as walking, can improve a person’s quality of life and help the person deal with side effects of cancer treatment, such as fatigue. Physical activities increase muscle mass, lessen anxiety, improve the appetite, and stimulate digestion (Brown et al., 2001).

A healthy, balanced diet is recommended, and homemade drinks as well as commercially available products (e.g., Ensure, Nutren) can be used to increase nutrient consumption if eating a balanced meal is not possible (Brown et al., 2001). A single multivitamin can be taken after active treatment. However, high consumption of single vitamins and minerals may weaken the immune system. Similarly, consumption of antioxidants during active treatment might decrease the effectiveness of the treatment because cancer treatment often makes use of an oxidative effect to kill the cancerous cells (Brown et al., 2001).

Some cancer survivors follow alternative dietary regimens, such as fasting therapy (to deprive the tumour of nutrients for growth), juice therapy (consumption of large amounts of fruit and vegetable juices), or a macrobiotic diet (a type of vegetarian diet). There is, however, no evidence that any of these alternative dietary regimens are effective (Brown et al., 2001).

### 4.6 Summary

The cancer patient’s diet, whether he or she is currently being treated or post-treatment, should be reviewed on an individual level. Each patient should be treated according to type and severity of symptoms. Over and above being treated at a dietary level, it is important to remember that quality of life is always an important consideration. Not only is nutritional information vital, but constant care, attention, and emotional support are also important.

The most prevalent cancers globally are associated with lifestyle and are therefore preventable. This vital aspect of cancer is examined in Chapter 13. Cancer can be beaten in many cases, if people are aware of how the disease can be prevented, if cancer is diagnosed early, and if the necessary care is provided.

### DISCUSSION QUESTIONS AND EXERCISES

1. What type of snacks would you recommend for low-income diabetic patients?
2. Discuss the short- and long-term advantages of optimal blood glucose control.
3. Plan a 5-day menu for a type 2 diabetic patient taking Diamicron (MR).
4. Plan a 5-day menu for an urban, male, type 2 diabetic patient taking Humalin L.
5. Plan a 5-day diet for a female patient who has a TC of 8 mmol/L, LDL-C of 5 mmol/L, and a normal HDL-C.
6. Suggest three dietary changes that could be suggested for a patient with hypertension to help with his or her condition.
7. Suggest appropriate dietary adaptations that can be made to treat the most common side effects of cancer treatment.

### REFERENCES


doi:10.15215/aupress/9781927356111.01


doi:10.15215/aupress/9781927356111.01


ADDITIONAL RESOURCES


ACKNOWLEDGEMENTS

This chapter is based in part on a chapter previously published in the following book: