

Michael Gleeson, PhD

BEATING TYPE 2 DIABETES

A Healthy
Lifestyle
Guidebook

Natural and
Simple Methods to
Reverse Diabetes for Good

Beating Type 2 Diabetes

The contents of this book were carefully researched. However, readers should always consult a qualified medical specialist for individual advice before adopting any new nutrition or exercise plan. This book should not be used as an alternative to seeking specialist medical advice.

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Michael Gleeson, PhD

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Meyer & Meyer Sport

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CONTENTS

Chapter 1	What Is Type 2 Diabetes?	9
	Introduction.....	9
	Who Am I?.....	11
	The Main Purpose of This Book.....	12
	Type 1 and Type 2 Diabetes: What Is the Difference?.....	13
	The 21st Century Disease.....	15
	How Many People Currently Have Type 2 Diabetes?.....	16
	Insulin Action and the Control of Blood Sugar.....	17
	What Goes Wrong in Type 2 Diabetes?.....	21
	What Are the Consequences of Type 2 Diabetes?.....	22
	What Is the Metabolic Syndrome?.....	25
	Are Type 2 Diabetes and the Metabolic Syndrome Reversible?.....	27
Chapter 2	How Do I Know If I Have Type 2 Diabetes, and How Will It Be Treated?	30
	Symptoms of Type 2 Diabetes.....	30
	Diagnosis of Type 2 Diabetes.....	32
	What Happens After the Diagnosis?.....	35
	Treatment of Type 2 Diabetes.....	36
	Medication.....	36
	Insulin Therapy.....	38
	Changes to Diet and Lifestyle Behavior.....	40
Chapter 3	How Is Type 2 Diabetes Monitored and Managed?	42
	Monitoring of People With Type 2 Diabetes.....	43
	Monitoring By Your Healthcare Practitioner.....	43
	Self-Monitoring at Home.....	46
	Management of Type 2 Diabetes.....	48
	Keeping Your Feet Healthy.....	48
	Practicing Good Personal Hygiene.....	49
	Practicing Good Food Hygiene.....	53
	Changing Your Diet and Eating Less.....	53
	Choosing Low Glycemic Index or Low Glycemic Load Foods.....	58
	Becoming More Physically Active.....	62
	Blood Glucose Self-Monitoring.....	66
	Will I Get Better?.....	68

BEATING TYPE 2 DIABETES

Chapter 4	What Causes Type 2 Diabetes?	71
	Possible Mechanisms Leading to Insulin Resistance in Prediabetes and Type 2 Diabetes.....	73
	Reduced Numbers of Insulin Receptors in Target Tissues.....	73
	Inhibition of Insulin Action Resulting From Inflammation in Adipose Tissue ...	73
	Decreased Production of Adiponectin.....	74
	Elevated Levels of Plasma Free Fatty Acids.....	74
	Elevated Levels of Plasma Triglycerides.....	74
	Chronic High levels of Plasma Insulin	75
	Elevated Levels of Methylglyoxal	75
	Possible Mechanisms Leading to Impaired Insulin Secretion in Type 2 Diabetes....	77
	β -Cell Exhaustion	77
	Genetic Factors.....	77
	Risk Factors for Type 2 Diabetes.....	79
	Sex Differences in Type 2 Diabetes and Its Relation to Body Fat Mass and Distribution.....	82
	Sex Differences in Insulin Resistance and Blood Glucose Levels	83
	Sex Differences in Risk of Cardiovascular Disease	83
	How to Evaluate Your Own Diabetes Risk Based on Your Personal Details and the Known Risk Factors	84
Chapter 5	A Weighty Problem	88
	What Causes Obesity?	89
	The Obesity Problem	91
	The Role of Genetics	92
	Why What We Eat Is Important.....	94
	The Problem With Snacking.....	101
	The Role of Food Addiction	102
	The Links With Sedentary Behavior.....	104
	The Links With Poor Sleep Quality	105
	Socio-Economic Factors in the Development of Obesity	107
	Too Many Calories Ingested, or Too Few Expended?	108
Chapter 6	How Do I Know If (or By How Much) I Am Overweight or Overfat?	110
	Normal Levels of Body Fat	111
	How Can I Tell If I Am Overweight or Overfat?.....	112
	Height–Weight Relationship and Body Mass Index.....	112
	Waist Circumference and Waist-to-Hip Ratio.....	114
	Estimating Percentage Body Fat By Bioelectrical Impedance Analysis.....	116
Chapter 7	What Can I Do to Reduce My Risk of Complications If I Have Type 2 Diabetes?	118
	Eat a Healthier Diet.....	119
	Recommendations for Healthy Eating	119

Diets That Are Known to Be Very Healthy.....	122
Food Shopping Tips and How to Use Nutrition Facts Labels on Packaged Foods.....	128
Exercise More.....	130
Recommendations for Physical Activity	131
Sleep Better.....	132
Recommendations for Improved Sleep Quality.....	133
Lose Some Weight.....	135
Common Mistakes.....	135
Defining Goals	136
Recommendations for Weight Loss by Dieting	136
Ways of Losing Body Fat and Weight	138
If You Are a Smoker, Stop Smoking.....	140
The Take Home Message.....	141
Chapter 8 What Can I Do to Beat Diabetes?.....	144
Losing Sufficient Weight Is the Key to Reversing and Getting Rid of Type 2 Diabetes.....	145
About Food Calories and the Effects of Energy Restriction	148
Common Diets for Weight Loss and the Ones That Diabetics Should Avoid.....	148
Fasting and Crash Dieting	149
Fad Diets	149
Very Low-Carbohydrate Diets.....	150
Very Low-Fat Diets.....	151
Food-Combining Diets.....	152
The Paleo Diet.....	152
The Zone Diet.....	153
Diets That Work for Weight Loss and Are Suitable for People With Prediabetes or Type 2 Diabetes.....	154
Low Glycemic Index Diets	154
Very Low Energy Diets.....	155
Intermittent Fasting Diets	156
Reduced Fat Diets	158
High-Protein Diets.....	158
Low Energy Density Diets.....	161
Comparisons of the Different Diets for Weight Loss in People With Type 2 Diabetes	164
Exercise for Weight Loss.....	166
Aerobic Exercise	167
Resistance Exercise	172
High-Intensity Interval Exercise.....	172
How Much Exercise Is Needed to Achieve Significant Body Weight and Fat Loss?..	175
Why Combining Dieting and Exercise Is Best for Both Weight Loss and Health.....	178

BEATING TYPE 2 DIABETES

Chapter 9	How to Personalize Your Weight Loss Plan to Beat Type 2 Diabetes.....	182
	The Combination 1,000GPW (Grams of Fat Per Week) Weight Loss Plan for Diabetics.....	183
	Decide Your Target Weight and How Much Weight You Need to Lose.....	183
	Why Stick to Just One Weight Loss Diet When You Can Pick and Mix From Several Diets That Are Effective and Safe?.....	184
	The Exercise Part of the Weight Loss Plan	186
	What If I Want to Lose More Weight Than This?.....	192
	Weight Maintenance Following Successful Weight Loss	192
	How Will I Know If I Have Beaten Type 2 Diabetes?.....	193
Chapter 10	Meal Ideas for Diets for People Who Are Trying to Manage or Beat Type 2 Diabetes.....	195
	Meals for the Low Glycemic Index Diet for Managing Blood Sugar Levels	198
	Low GL Breakfasts (250-400 kcal)	199
	Low GL Lunches (300-400 kcal).....	200
	Low GL Dinners (700-900 kcal, Including Dessert).....	202
	Meals for Days on the Very Low Energy Diet and “Fasting Days” on the Intermittent Fasting 4:3 Diet.....	204
	Breakfasts Containing About 125 kcal	205
	Breakfasts Containing About 250 kcal.....	206
	Lunches Containing About 125 kcal.....	208
	Lunches Containing About 250 kcal.....	208
	Dinners Containing About 250 kcal.....	210
	Dinners Containing About 400 kcal.....	212
	Meals for “Nonfasting Days” on the Intermittent Fasting 4:3 Diet.....	215
	Meals for the Mediterranean Diet	215
	Meals for the Japanese Diet.....	218
	Desserts for the Japanese or Mediterranean Diets (All Less Than 200 kcal) .	221
	Meals for the Vegetarian Diet	222
	Meals for the Reduced Fat Diet	225
	Breakfasts (250-350 kcal).....	225
	Lunches (300-400 kcal)	226
	Dinners (600-800 kcal).....	227
	Daily Meal Plans for the High-Protein Diet	228
	Daily Meal Plans for the Low Energy Density Diet.....	230
	Breakfasts With Low Energy Density and Fewer Than 250 kcal.....	230
	Lunches With Low Energy Density and Fewer Than 250 kcal	231
	Dinners With Low Energy Density and Fewer Than 800 kcal.....	231
	My Final Message to You	234
	Glossary.....	235
	References.....	254
	About the Author.....	275

Chapter 1

What Is Type 2 Diabetes?

Objectives

After studying this chapter, you should:

- Understand the scope of this book.
- Know something about the author.
- Have a basic understanding of what diabetes is and the difference between type 1 and type 2 diabetes.
- Understand the role of insulin in the control of blood sugar.
- Know the basics of what goes wrong in type 2 diabetes.
- Appreciate the potential long-term health problems caused by being overweight and having type 2 diabetes.
- Know what having the "metabolic syndrome" means and its impact on cardiovascular disease risk.
- Appreciate that type 2 diabetes can be reversed and effectively cured.

INTRODUCTION

Firstly, I want to thank you for buying this book and congratulate you on your decision. The book contains everything that I think you should know about **type 2 diabetes** which includes not only explanations about the diagnosis of the condition, what causes it, how it is treated, managed, and monitored but also how you can reduce your risk of health consequences, and how – if you are determined enough – you can beat it. In this book I provide advice and recommendations about living with type 2 diabetes and how to get rid of it. I also explain the scientific reasons for the guidance that is given in a way that any reasonably intelligent person can understand.

BEATING TYPE 2 DIABETES

In this book I use my extensive experience of working with elite athletes and games players to help people with both **prediabetes** (the prelude to type 2 diabetes, sometimes also referred to as borderline diabetes) and type 2 diabetes learn how to safely and effectively lose weight, get fitter, sleep better, avoid illness, and live healthily for longer. Much of my career has been spent researching the impact of food and exercise on health and performance in sport. I have conducted research projects on how athletes can stay healthy and in peak condition so that they can perform at their best, which includes training to maximize adaptation but avoid burnout, avoiding being underweight or overweight, improving the quality of sleep, promoting robust immunity, and eating the right foods at the right time to maximize performance. This means I have an excellent understanding of energy metabolism, nutrition, exercise, and fitness. What's more is that I have had type 2 diabetes myself and have successfully reversed it by applying the science that I have learned in my work with athletes. The advice I give in this book is all based on the latest scientific evidence, and my aim is to explain in clear and simple terms how this can be applied to help others get rid of their type 2 diabetes. I do not promote any fad diets or exercise regimens, just ones that have been proven to be safe and effective. From me you will learn the principles of healthy eating and what you can do to make your health optimal...that is as good as your health can possibly be.

Although this book is mainly for people who have been diagnosed with either prediabetes or type 2 diabetes it will also be a useful resource for their family members, friends, carers, or healthcare practitioners who want to be able to help them with their condition. If other members of your family have prediabetes, type 2 diabetes, or suspect that they may be at risk of the condition, pass this book on to them. If you are interested in how to actually avoid type 2 diabetes altogether, then you should read my previous book *Eat, Move, Sleep, Repeat*, published by Meyer and Meyer January 2020.

Type 2 diabetes is known as a metabolic disease. This is because it affects how your body deals with **glucose**, the primary source of **energy** that our **cells** rely on. This change in your body's handling of glucose results in the main dangers of type 2 diabetes: substantially higher risks of health complications that can include **coronary heart disease**, **stroke**, kidney disease, infections, ulcers, and blindness.

Please note that any word that appears in **bold** font in this book is defined in the glossary which you will find at the end of the book. I have tried to identify some of the key words or terms, as well as words that may be unfamiliar to some readers, and those that it is simply useful to know the exact meaning of.

One of the main problems with type 2 diabetes is that it creeps up on you, and there are probably many people out there that have the condition but aren't actually aware of it. In fact, we know for sure that there are millions of people who have high blood glucose levels but don't actually realize they have a developing metabolic problem because they have not had a blood test to diagnose it, and nor do they yet feel ill or abnormal in any way. This is because, especially in the early stages of the disease, the symptoms may be absent or not obvious with no overt signs of feeling unwell. But high blood **sugar** levels are the first real indication that something is wrong and always precede the onset of type 2 diabetes.

However, the lack of obvious symptoms is no reason to ignore high blood sugar levels. The big danger with type 2 diabetes is that it increases the risk of associated major health problems only a few years down the line. The biggest single risk factor for diabetes – by far – is being overweight. Therefore, it is important, particularly if you are overweight or have high blood pressure, to have regular health checks by your local medical practitioner, including an annual blood test which can reveal if you have prediabetes or type 2 diabetes. More detail about these conditions will follow shortly, but first let me tell you a little about myself and why I have written this book.

WHO AM I?

I am a recently retired university professor who has spent the last 40 years of my life teaching and researching in the field of exercise physiology, metabolism, immunology, and health with a particular interest in sport nutrition. So essentially much of my work has been spent examining the body's reactions to food and exercise. The last 20 years of my career were spent working at two of the top universities in the world for sport, exercise, and health science (Birmingham and Loughborough in the UK). I have coauthored several books on metabolism, immunology, and nutrition in sport and exercise, and have published over 200 research papers in scientific and medical literature journals; much of which has been focused on the well-being of athletes and the factors influencing their health and performance. After retiring and joining the ranks of the aging general public I turned my attention to the issue of living a healthy lifestyle (in part, to improve my own quality of life and longevity) and spent the first couple of years of my retirement putting together the material for my healthy lifestyle guidebook *Eat, Move, Sleep, Repeat*.

About 10 years ago I was diagnosed with high blood pressure (**hypertension**) and high serum **cholesterol**, both of which are major risk factors for coronary heart disease and stroke. This was not too surprising because despite working in a university sport, exercise, and health science department, my working days were mostly sedentary with much of my time spent sitting at a desk or standing to deliver a lecture. Also, my mother suffered from hypertension from her mid-50s, and some conditions like hypertension tend to run in families. Although both conditions can be managed to some degree by alterations to diet and lifestyle, some medication is required to get them back down into the normal "healthy" range. So since then I have had a daily tablet (what is known as an angiotensin converting enzyme or ACE inhibitor) to lower my blood pressure and a daily statin tablet to lower my cholesterol. These work relatively quickly for most people, and my values were back in the normal range within a few months.

However, five years ago, after another annual blood test I was told that I had developed type 2 diabetes and was advised to change my diet and do more exercise. I researched the literature on type 2 diabetes and realized that it is possible to cure the condition without the need for medication, and that is what set me on the road to writing this book. I also discovered for myself that losing some weight by being more active and eating a healthier diet does actually work. Five years ago my body weight (also known as **body mass**) was close to 80 kg with a **body mass index (BMI)** of 27.0 kg/m² (BMI is just your weight in kilograms divided by your height in meters, and

BEATING TYPE 2 DIABETES

then divided again by your height in meters; more on this in chapter 6). Within a few months I got my weight down to 70 kg and my BMI dropped to 23.7 kg/m²; my diabetes went into remission, and I intend to keep it that way.

I did not achieve this 10 kg weight loss by drastic dieting. My main dietary changes were to reduce my **fat** intake; eat fruit with yogurt rather than pies, sweets, or puddings for dessert; cut down on **alcohol** to just one glass of wine per day; and eat more low **energy density** vegetables, rice, and noodles in place of boiled potatoes, French fries, and bread. I enjoy both Mediterranean and Asian cuisine so fish, stir-fry, tagine, and curry are all on the menu at least once per week and some kind of salad features at least twice per week. I also now have a much more active lifestyle: I aim to walk at least six miles (10 km) on average per day. I play the occasional game of doubles tennis and do two or three 10- to 15-minute **resistance exercise** sessions (at home, not in the gym) each week. So, if you were wondering if it is actually possible to reverse and essentially get rid of type 2 diabetes after it has been diagnosed, the answer is yes. If it wasn't, I wouldn't have written this book!

THE MAIN PURPOSE OF THIS BOOK

I decided this book was needed while researching the literature on type 2 diabetes. I found many websites on the subject of diabetes, and some gave excellent advice about the condition and its management. But much of the advice about diabetes was concerned with its diagnosis and treatment and how to live with the condition. There was little information about how to get rid of it for good. I decided that many people with type 2 diabetes, prediabetes, or who are at risk of the condition would appreciate a single, reliable, evidence-based resource on the subject. Therefore, in this book I have tried to include everything that I think you should know about type 2 diabetes and how you can reverse it.

I begin by explaining exactly what type 2 diabetes is (and how it differs from type 1 diabetes), how the condition is diagnosed, how it is treated (with medication and encouraging changes to lifestyle behavior), and how the condition is monitored and managed. Then I explain what is known about the causes of type 2 diabetes, the main risk factors, and why it is linked so strongly to having an excess of **body fat** (i.e., being overweight or obese), how you can tell if (or by how much) you are overweight or overfat, what you can do to reduce your risk of serious health complications, and – if you are determined enough – how you can beat the condition.

I will take you through a novel and effective weight loss plan that uses sensible, varied, non-extreme dieting, combined with enjoyable and exhilarating (but not exhausting) exercise that should kick your diabetes into remission and improve many other aspects of your health in the process. The great thing about the diet part of my weight loss plan is that it does not require you to stick with the same boring diet week after week. I utilize a variety of different, but equally effective diets that are well suited to people with type 2 diabetes and that you can change on a weekly basis. And none of the exercises I recommend will leave you with aching muscles, or feeling sick, or tired. Finally, I have devoted a chapter to describing some meal ideas for the dieting part of the weight loss plan.

TYPE 1 AND TYPE 2 DIABETES: WHAT IS THE DIFFERENCE?

The full clinical name for diabetes is **Diabetes mellitus** which is derived from the Greek word diabetes meaning siphon – to pass through – and the Latin word mellitus meaning honeyed or sweet. This is because in diabetes, excess sugar is found in both the urine and the blood, making it taste sweet – if you were daft enough to drink it that is! Having said that, tasting urine to see if it was sweet was actually how diabetes was diagnosed in the old days. It wasn't until the 1800s that scientists developed chemical tests to detect the presence of sugar in the urine. There are two main forms of diabetes mellitus, and both result from a problem related to the hormone insulin.

Type 1 diabetes mellitus, also known as insulin-dependent diabetes mellitus is a **chronic disease** (one that persists over a long time, like arthritis, cancer, coronary heart disease, stroke, etc.) in which the pancreas (an accessory digestive organ located below the liver in the abdomen) produces little or no insulin. Type 1 diabetes usually results from a highly specific immune-mediated destruction of pancreatic islet β -cells – specialist cells that detect the level of blood glucose and secrete the hormone insulin when it rises above normal. This is commonly described as an autoimmune type of disease. Insulin stimulates the tissues to take up insulin from the blood such that the blood glucose concentration returns to its normal fasting level within a few hours following a meal. For type 1 diabetics, the absence of insulin production results in chronic elevated blood glucose concentration (known as hyperglycemia). The condition usually occurs in early childhood and can lead to many serious complications if not properly managed. The patient and their physician must work together to optimize glucose control involving both regular insulin injections and management of food intake. Around 5-10% of people who are diagnosed as diabetic have type 1 diabetes mellitus. Although much rarer (less than 1% of cases), type 1 diabetes can also be caused by a genetic defect in β -cell function that prevents normal levels of insulin production.

Another form of type 1 diabetes is the acquired type which develops when there is damage to the pancreas caused by infection or **inflammation** resulting in a condition called **pancreatitis**. In some cases, this can be caused by stones that are formed in the gall bladder coming down the shared pancreatic duct and getting trapped in the pancreas and later forming a cyst. This form of diabetes may resolve itself within a year or two as the inflammation disappears and the pancreas repairs itself. It depends on the extent of damage to the β -cells; if the damage is too extensive, the affected person may be stuck with diabetes for life just like other type 1 diabetics.

Type 2 diabetes mellitus, also known as **non-insulin-dependent diabetes mellitus**, usually comes on slowly, is relatively rare below the age of 40, and most often develops in people who are overweight and sedentary. The main problem in type 2 diabetes is that the body tissues (e.g., **adipose tissue**, liver, and skeletal muscle) become resistant to the action of insulin which normally stimulates these tissues to take up blood-borne glucose when the blood glucose level is elevated such as after a meal. The lack of tissue response to insulin – often referred to as **insulin resistance** – results in sustained hyperglycemia (higher than normal blood glucose concentration) with many serious complications including reduced life expectancy if it is not properly managed.

BEATING TYPE 2 DIABETES

These consequences are described in detail later in this chapter. The main differences between type 1 and type 2 diabetes are illustrated in figure 1.1.

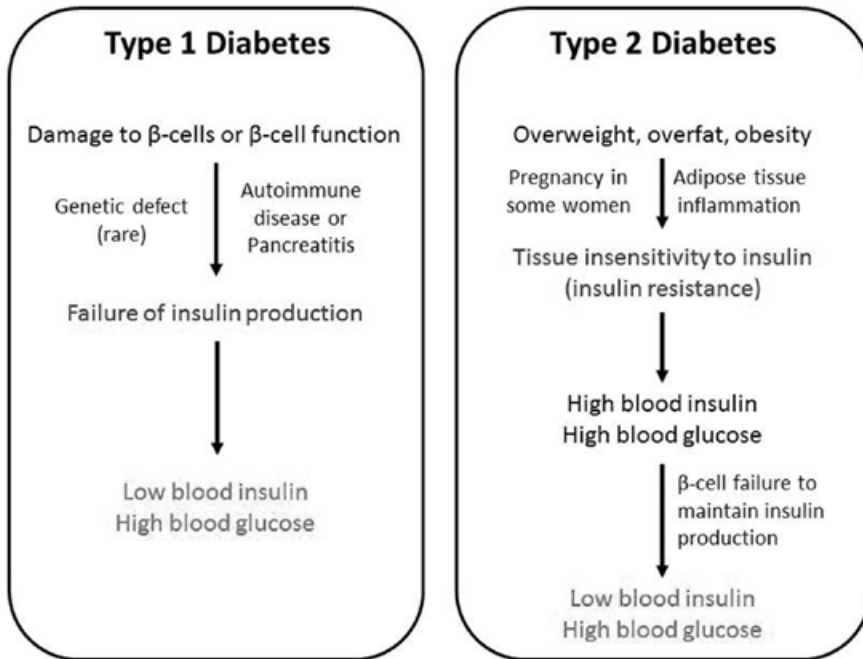


Figure 1.1 A simplified illustration of the main differences between type 1 diabetes and type 2 diabetes. A form of type 2 diabetes called gestational diabetes that develops during pregnancy in 6-8% of women is usually temporary but puts the mother at higher risk of type 2 diabetes later in life.

A temporary form of diabetes called **gestational diabetes** that has some similarities to type 2 diabetes can occur during pregnancy, particularly in overweight women. The hormones produced by the placenta during pregnancy (estrogen, **cortisol** and human placental lactogen) can block the actions of insulin putting the mother-to-be at an increased risk of insulin resistance, and some women are not able to produce enough insulin to overcome this resistance. This makes it difficult for the tissues to take up glucose from the blood, so the glucose remains in the blood and the levels rise, leading to gestational diabetes. The condition develops during the second and third trimester and currently affects 6-8% of pregnant women, though its prevalence is on the increase and is higher in 35- to 45-year-old women than in younger women. Some women diagnosed with gestational diabetes may already have had type 2 diabetes before they conceived. If they didn't have type 2 diabetes already, having gestational diabetes means that there is a higher chance of developing type 2 diabetes later in life. That's why mothers are usually offered a blood test for diabetes at their postnatal check, and annually from then on.

THE 21ST CENTURY DISEASE

Type 2 diabetes has exhibited increasing prevalence in the adult population since the late 1950s. In 1960 the percentage of the US population with diagnosed diabetes was close to 1.0%, by 1990 it had risen to 2.7%, in 2000 it was 4.7%, and in 2018 it stands at 9.0% (figure 1.2). It is projected to rise to close to 12% by 2030, and this does not include people (estimated to be approximately 3% of the population) who may have type 2 diabetes but have not yet had a confirmed diagnosis. Alarming, about 34% of the US population in 2018 were confirmed to have prediabetes, a condition that if not treated often leads to Type 2 diabetes within 5 years. So it is rapidly becoming the disease to beat in the 21st century.

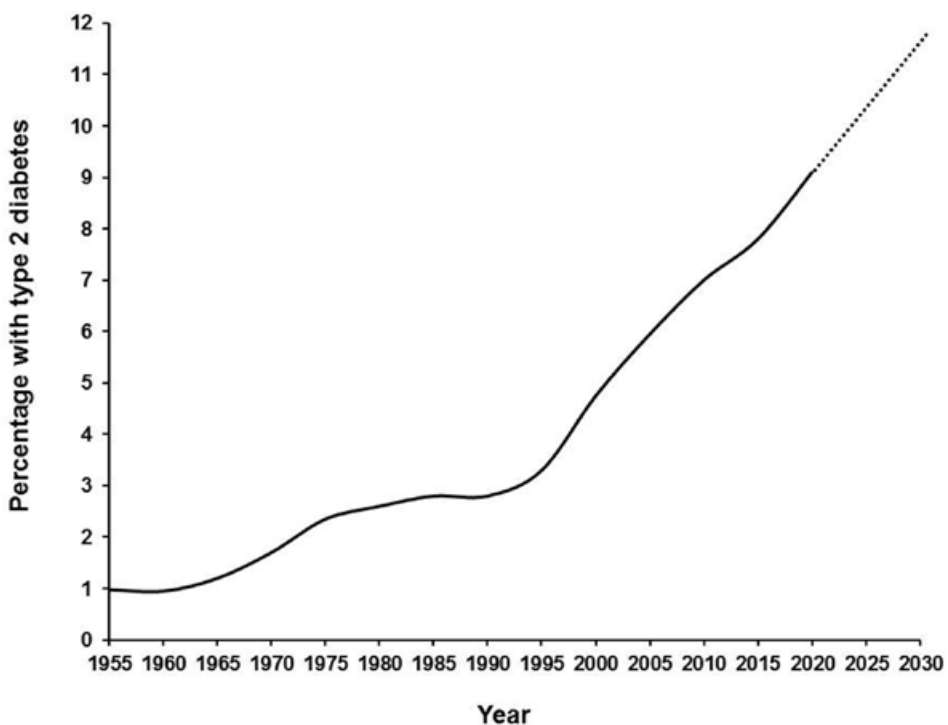


Figure 1.2 Percentage of the US population with diagnosed diabetes since 1958 (solid line) and the projected increase to 2030 (dashed line). From US Center for Disease Control Division of Diabetes Translation. Available at <http://www.cdc.gov/diabetes/data>.

In the past we have beaten other diseases. Before the 19th century the major diseases were infections caused by viruses (e.g., measles, mumps, polio, smallpox) or bacteria (e.g., bubonic plague, tuberculosis, typhoid, whooping cough). The introduction of vaccines in the 19th century and antibiotics in the 1940s has resulted in these diseases being eliminated or greatly reduced in incidence. Improved **nutrition**, better medications, and improved sanitation and hygiene practices have also contributed to the much lower prevalence of infectious diseases that we now take for granted. Lung cancer became a big problem in the 20th century due to the smoking of

BEATING TYPE 2 DIABETES

tobacco products and increasing air pollution, but in recent years the incidence of lung cancer has dropped as many people have quit smoking or switched to less harmful vaping. In the early 1980s we had the start of the Acquired Immunodeficiency Syndrome (AIDS) epidemic caused by the Human Immunodeficiency virus (HIV) which attacks and destroys crucial **white blood cells** (called CD4+ T-helper **lymphocytes**) and is transmitted through sexual contact or blood. These cells direct the body's immune responses to other viral and bacterial infections. The loss of these T cells leads to the point where opportunistic infections can become fatal; although, nowadays, while there is still no cure for HIV, there are very effective treatments that enable most people with the virus to live a long and healthy life.

Becoming much more prevalent now are other forms of what we could call "self-inflicted disease", namely obesity, type 2 diabetes and hypertension and the health complications that result from them including increasing fatality from cardiovascular disease and cancers. For these diseases of modern society that affect increasing numbers of people in developed countries, medication will almost certainly not be the answer. Although there may be some improvements to drug treatment to help the management of type 2 diabetes, it is very unlikely that a drug will be found to cure the condition. Changes to diet and lifestyle are the things that are needed. Simply put, weight loss is the key to curing type 2 diabetes, and avoiding excessive weight gain in the first place is the key to its prevention. This will require government investment in health and nutrition education, the introduction of taxes on high **calorie** and high sugar food products to discourage people from buying them, and healthcare programs to help people get rid of type 2 diabetes by dieting and/or doing more exercise. Most importantly, it needs people to take these messages on board and make a conscious decision to change their unhealthy lifestyle.

Current projections of the incidence of type 2 diabetes suggest that the numbers of people with the condition and those that die prematurely from it (we're talking about living 5-10 years less than average here) will increase considerably more in the next 10-30 years unless something is done about it very soon. The good news for people with type 2 diabetes, and especially those who have only recently been diagnosed with the condition, is that you can get rid of it through your own actions. In this book I will explain how.

HOW MANY PEOPLE CURRENTLY HAVE TYPE 2 DIABETES?

Globally, the estimated diabetes prevalence for 2017 was 425 million and is expected to affect about 629 million people by 2045 if current trends continue. According to a 2017 report from the US Centers for Disease Control and Prevention (CDC) over 30 million Americans – almost 10% of the US population – have type 2 diabetes. Another 84 million have prediabetes, a condition that if not treated often leads to type 2 diabetes within five years. In the UK about 9% of adults (aged over 16) have diabetes. This means that, including the number of undiagnosed people who have the condition but don't yet realize they have it (estimated to be about one million), there are about five million people living with diabetes in the UK at present. Furthermore, an estimated 12.3 million people (that is almost 20% of the total population) in the UK are currently

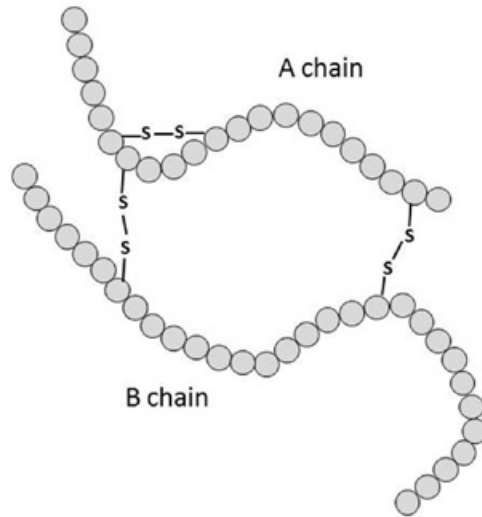
considered to be at increased risk of the disease. The situation is similar in many other developed countries including other parts of Europe, Canada, and Australasia. There are huge economic costs to society. In 2018 it was estimated that the cost of medications to treat type 2 diabetes alone for the National Health Service in the UK was over one billion pounds sterling.

The proportion of people who have diabetes increases with age: 9% of people aged 45 to 54 have diabetes, but for over 75s it is 24%. Diabetes at older ages has even bigger health implications as people are more likely to be suffering from other health problems, particularly cardiovascular diseases. Diabetes is more common in men (10% compared with 8% for women) and people from south Asian, Hispanic, and black ethnic groups are nearly twice as likely to have the disease compared with people from white, mixed, or other ethnic groups (15% compared with 9%).

INSULIN ACTION AND THE CONTROL OF BLOOD SUGAR

Let me explain a little more about what insulin is and what it actually does in a normal healthy person. Insulin is a small **protein** hormone consisting of 51 **amino acids** in the form of two linked chains (figure 1.3 and photo 1.1). Insulin is synthesized by specialized **endocrine** β -cells which are arranged in small clusters (called the islets of Langerhans) in the pancreas. The name insulin comes from the Latin *insula* for island. The main function of the pancreas is as a digestive accessory organ and it is located just behind and below the stomach. The pancreas produces pancreatic juice containing **enzymes** that break down protein, fat, and **carbohydrate** when the juice is secreted into the small intestine. The pancreas's β -cells can detect the concentration of glucose (blood sugar) in the circulation and increase their production and release of insulin when the blood glucose concentration rises above its normal fasting level of about 5 **millimoles per liter (mmol/L)** or 900 milligrams per liter (mg/L). Another hormone called **incretin** that is produced and released by cells in the small intestine after eating helps the body produce more insulin when it is needed and reduces the amount of glucose being produced by the liver when it is not needed. Insulin travels in the circulation to its target tissues which include adipose tissue, the liver, and skeletal muscle where it binds to specific **insulin receptors** located on the surface of the cells. The binding of insulin to its receptor activates **insulin receptor substrate 1 (IRS-1)** which then initiates a cascade of reactions inside the cell that constitute a signaling pathway that brings about the actions of insulin in these target tissues as illustrated in figure 1.4. Insulin increases glucose uptake by causing glucose transporter proteins (e.g., **GLUT4** in muscle and adipose tissue; GLUT2 in the liver) which are located in vesicles (small bags of liquid encased within a lipid membrane) in the **cytoplasm** of the target cells to move to the cell membrane which then allows glucose transport from the blood into the target tissue cells. This causes the blood glucose concentration to fall back to its normal (pre-feeding or fasting) level and the stimulus for insulin secretion is no longer present. The insulin gets cleared from the circulation and when **plasma** insulin levels fall, the glucose transporters move back into the cytoplasm and tissue glucose uptake drops.

BEATING TYPE 2 DIABETES



*Figure 1.3 Diagrammatic illustration of the structure of the human insulin molecule. It consists of two linked chains of amino acids: an A chain containing 21 amino acids and a B chain containing 30 amino acids. The circles in the diagram represent individual amino acids which are linked together by **peptide** bonds. The A and B chains of the insulin molecule are linked together by disulfide bonds formed between cysteine (a sulfur containing amino acid) residues in the two chains.*

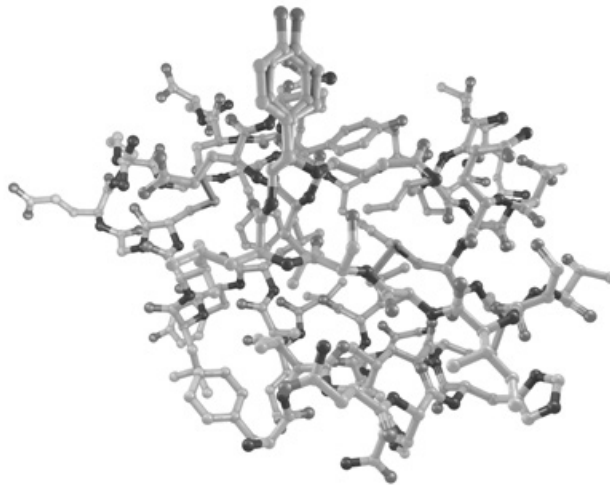


Photo 1.1 A three-dimensional picture of the human insulin molecule.

The main action of insulin is to promote the uptake of glucose (blood sugar) from the blood into the cells of its target tissues. When glucose is taken up into adipose tissue cells (**adipocytes**) it is converted into fat. Glucose taken up by skeletal muscle is converted to **glycogen** (a glucose polymer that acts as a store of carbohydrate for later use) or is used as a fuel for muscle contraction (i.e., exercise). Glucose that enters the liver is mostly converted to glycogen with excess amounts being converted to fat. This uptake of glucose by various tissues in response to insulin returns the blood

glucose concentration back to its normal level. Other actions of insulin are to promote the storage of glucose into glycogen and increase amino acid uptake from the blood into skeletal muscle when the blood concentration of certain amino acids rises above normal such as after eating a meal containing protein (e.g., from meat, fish, eggs, milk, cheese, nuts, beans, or **legumes**). Insulin action also inhibits the breakdown of fat in adipose tissue.

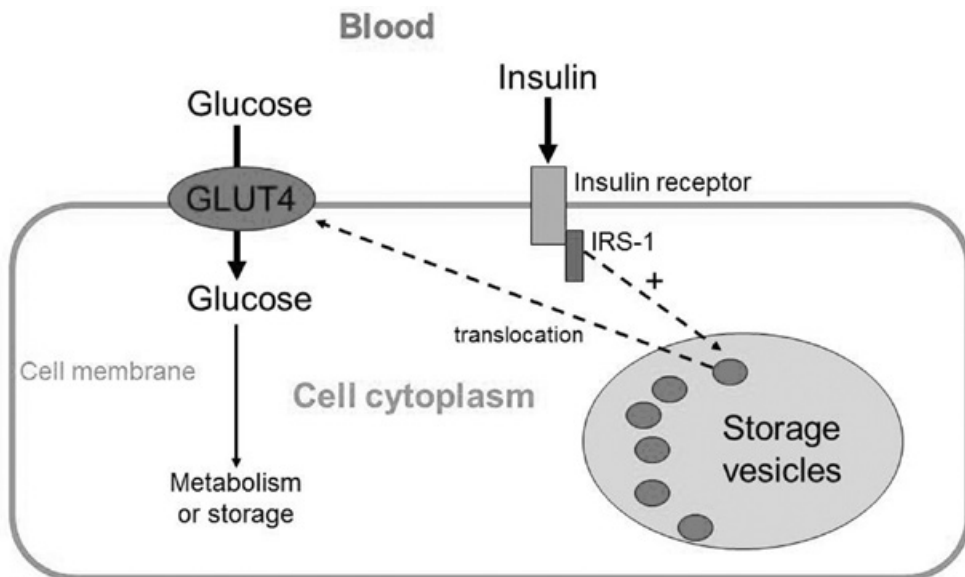


Figure 1.4 The mechanism of insulin action. See text for details. IRS-1: Insulin receptor substrate 1.

Insulin secretion is increased as soon as the blood glucose concentration starts to rise above about 5 mmol/L. The carbohydrate in our diet comes mostly from **starch**, a glucose polymer in plants that has a similar structure to glycogen, and is abundant in potatoes, rice, corn, and foods made from cereal grains such as bread, breakfast cereals, and pasta). The other major source of dietary carbohydrate is in the form of free sugars (e.g., **sucrose**, **lactose**, **maltose**, and **fructose**). Our digestive system breaks down the **starch** into glucose which is absorbed into the blood together with free glucose released from the enzymatic breakdown of sucrose (cane sugar which is formed of two sugars – glucose and fructose – linked together), lactose (milk sugar formed from glucose and galactose), and maltose (malt sugar formed from two linked glucose **molecules**). The glucose in the gut is then absorbed via the intestinal cells into the blood. Fructose is also absorbed separately and is converted into glucose by the liver. After eating a meal containing carbohydrate, the blood glucose concentration rises – usually to a peak of about 7-9 mmol/L – as its rise is countered by the action of insulin. Figure 1.5 illustrates the change in the blood glucose and insulin concentrations that occurs following a meal containing 75 grams (g) of glucose. As you can see, the blood glucose concentration remains elevated for several hours after a meal and the insulin concentration changes according to the level of blood glucose.

BEATING TYPE 2 DIABETES

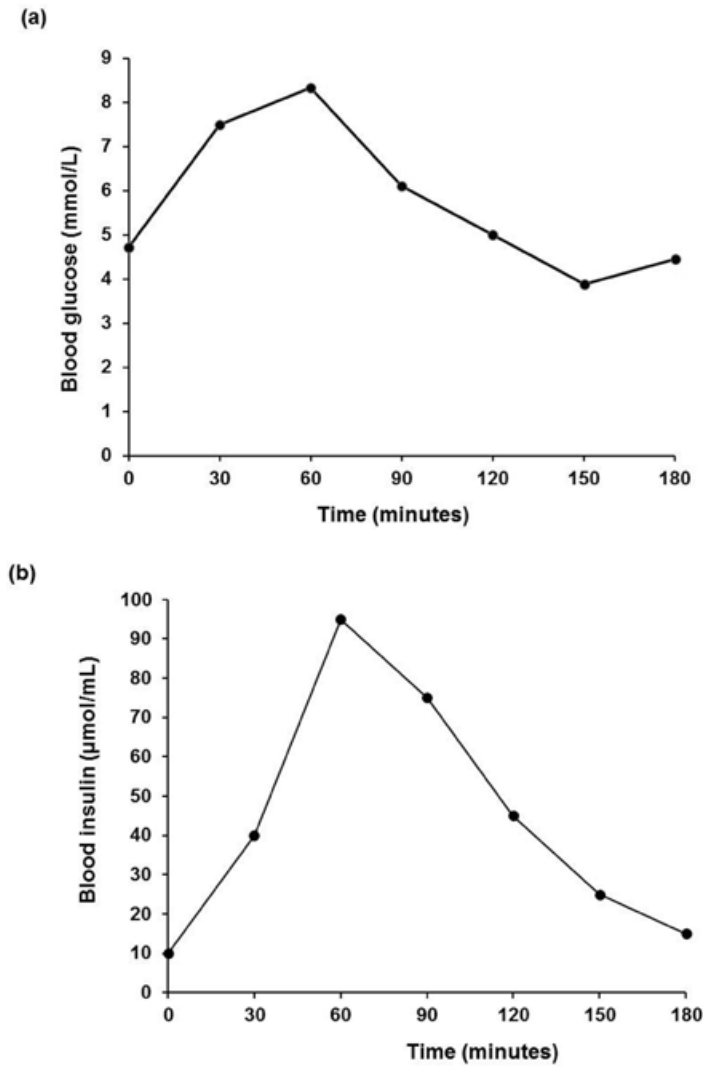


Figure 1.5 Changes in the blood concentration of (a) glucose and (b) insulin following a meal containing 75 g of glucose in a healthy adult.

Apart from the gut, the only other main source of glucose in the blood is the glucose that is released from the liver, an organ which can make glucose and stores it as a glucose polymer called glycogen. When you haven't eaten for quite some time (e.g., when you wake up in the morning after a night's sleep), your blood glucose levels will be lower than normal and the liver responds by breaking down stored glycogen into glucose and releasing it into the circulation to keep your blood glucose level within the normal range. This response occurs because when your blood glucose level drops below normal (e.g., due to a prolonged fast or performance of prolonged exercise without prior food intake) the drop in glucose availability is detected and your **neuroendocrine system** secretes several other hormones (**epinephrine** and cortisol from

the adrenal glands and **glucagon** from the α -cells of the pancreas) that stimulate greater liver glucose production and glycogen breakdown. These hormones essentially prevent the blood glucose levels from falling too low (known as **hypoglycemia** or just “hypo” for short) which is important as the brain, nerve cells, and blood cells all rely on glucose as their main source of energy. Hypoglycemia (a blood glucose level below 3 mmol/L) causes symptoms of fatigue, dizziness, lack of coordination, impaired cognitive function, and fainting. If too prolonged, hypoglycemia can result in coma and death.

WHAT GOES WRONG IN TYPE 2 DIABETES?

In prediabetes and at least in the early stages of type 2 diabetes, normal (or above normal) amounts of insulin are still usually produced when the blood glucose concentration rises above the normal fasting level of 5 mmol/L. So the problem is not a lack of insulin production as in type 1 diabetes but is one of insulin resistance. In other words, the tissues are less sensitive (or more resistant) to insulin and do not respond in the usual way to increase their uptake of glucose from the blood (some uptake will occur but nowhere near as much as normal). Exactly why this happens is unknown, although genetics and environmental factors, such as excess weight and inactivity, seem to be contributing factors. What this means is that after a carbohydrate containing meal the blood glucose concentration will rise much higher than normal (e.g., to something like 11-50 mmol/L) and will only gradually return back towards normal over many hours as illustrated in figure 1.6. As most people eat three or four meals per day, in type 2 diabetics the blood glucose concentration remains chronically high. Even after a night's sleep with a 10-12 hour period of fasting it is usually still above 7 mmol/L. Type 2 diabetes usually begins with insulin resistance, a condition in which muscle, liver, and fat cells cannot take up glucose from the blood as normal, and at this stage it is referred to as prediabetes. As a result, your body needs more insulin to help glucose enter cells. At first, the pancreas makes and releases more insulin to keep up with the added demand. Over time, the β -cells of the pancreas become worn out and can't make enough insulin to meet the body's demands and blood glucose levels rise even more. When this occurs full-blown type 2 diabetes has developed, and medication will soon be needed to treat the condition and slow its progression.

BEATING TYPE 2 DIABETES

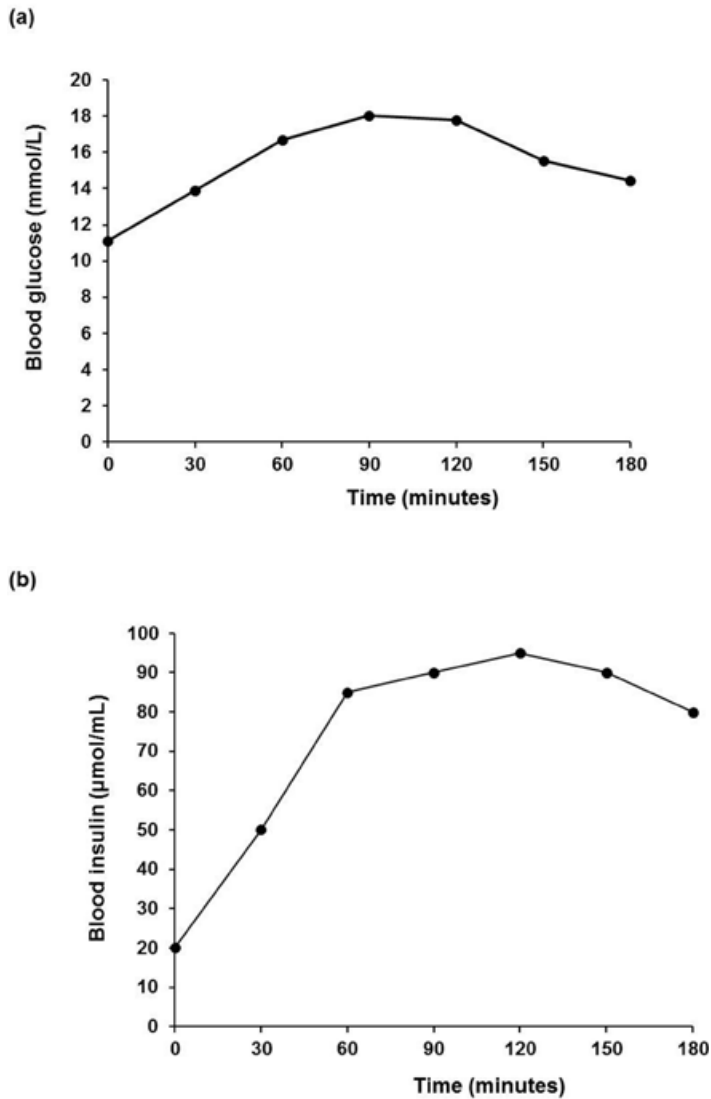


Figure 1.6 Changes in the blood concentration of (a) glucose and (b) insulin following a meal containing 75 g of glucose in an adult who is a type 2 diabetic.

WHAT ARE THE CONSEQUENCES OF TYPE 2 DIABETES?

If uncontrolled blood glucose stays much higher for longer after feeding and even in the fasting state, blood glucose concentrations can remain extremely high. Fat breakdown and mobilization is normally inhibited by insulin, and reduced insulin action therefore results in increased **fatty acid** concentrations in the blood. Some of these fatty acids are taken up by the liver and incorporated into **triglycerides** or converted into cholesterol and then released as **low-density lipoprotein**

(LDL) particles which contribute to the development of fatty deposits (called **plaques**) on the walls of blood vessels. This reduced insulin sensitivity (which is also called increased insulin resistance) has far-reaching consequences and may result in many serious clinical complications including hypertension, coronary heart disease, **atherosclerosis**, **peripheral vascular disease**, **cerebrovascular disease**, kidney disease, nerve damage, blindness, leg ulcers, more frequent infections, and poor wound healing (figure 1.7). Being a type 2 diabetic typically reduces life expectancy by 5-10 years. For further details of these complications see the sidebar.

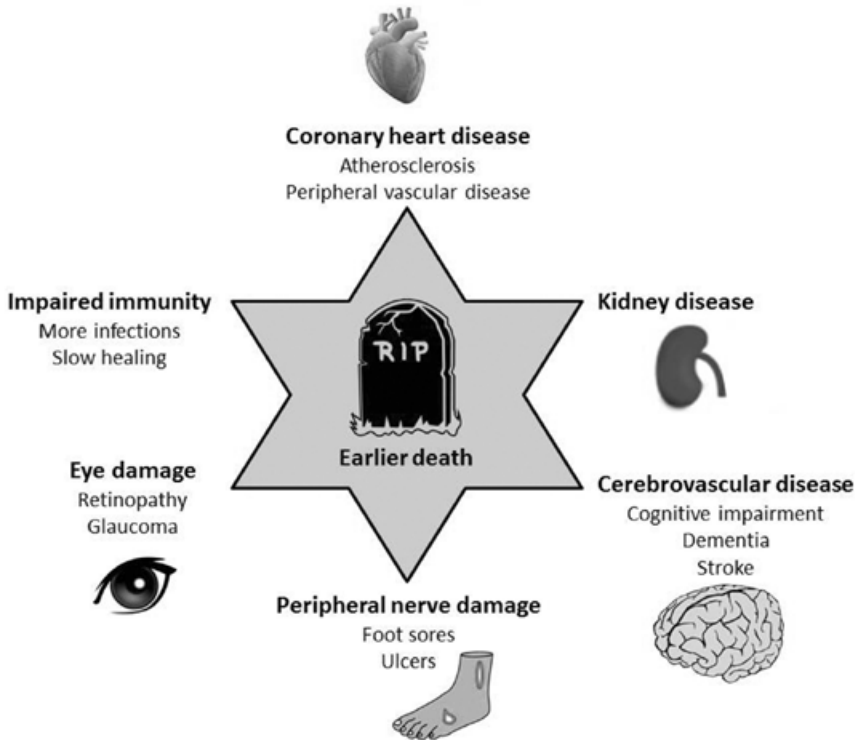


Figure 1.7. The serious and life-threatening health complications that come with type 2 diabetes.

The complications that can come with diabetes

Too much glucose in your blood and other body fluids, together with the associated changes in blood fats (higher cholesterol and triglyceride), can damage organs, blood vessels, and nerves which can lead to long-term health problems such as:

- Hypertension (high blood pressure) which increases the risk of stroke and coronary heart disease.
- Coronary heart disease which can lead to heart attacks that can be fatal. In diabetics the relative risk of a fatal heart attack is double that of the nondiabetic population.

(continued)

BEATING TYPE 2 DIABETES

(continued)

- Atherosclerosis (or **arteriosclerosis**), known as hardening or clogging up of the arteries is the build-up of cholesterol, fatty cells, and inflammatory deposits (called plaque) on the inner walls of the arteries that restrict blood flow to the heart and organs such as the brain and kidneys.
- Peripheral vascular disease is a type of atherosclerosis that can affect the arteries in the arms, legs, and feet. It is also known as peripheral arterial disease and lower extremity vascular disease. If the arteries become narrowed or blocked, blood cannot get through to supply oxygen to the tissues, causing the muscles of the lower extremities to cramp; when this occurs during walking it is called “intermittent **claudication**”. In diabetics the relative risk of an obstruction (claudication) in a peripheral artery is eight-fold higher in women and four-fold higher in men than in nondiabetics.
- Cerebrovascular disease. This is when blood vessels in the brain that are damaged due to atherosclerosis cause cognitive impairment, dementia, and higher risk of stroke. About one third of people with type 2 diabetes suffer from cerebrovascular disease and the risk of stroke is 50% higher than in nondiabetics.
- Kidney disease which can lead to kidney failure with life-threatening **toxemia** (a build-up of toxins in the blood) and the need for a kidney transplant. In diabetics the relative risk of kidney disease is 3-fold and 2-fold higher in women and men, respectively than in nondiabetics.
- Diabetic retinopathy which is a condition in which the back of the interior of the eyes (called the **retina**) is damaged which leads to impaired vision and often results in total blindness. In diabetics the relative risk of blindness is five times that of the nondiabetic population. You can see a simulation on your phone or tablet of how this condition affects your vision as it develops over time if you download the Royal National Institute of Blind People (RNIB) app (it’s called the RNIB Diabetic app) from the App Store.
- **Glaucoma** which is a common eye condition where the optic nerve, which connects the eye to the brain, becomes damaged. It’s usually caused by fluid building up in the front part of the eye, which increases pressure inside the eye. Glaucoma can lead to loss of vision if it isn’t diagnosed and treated early. In diabetics the relative risk of glaucoma is about 40% higher than that of the nondiabetic population.
- Nerve damage (known as neuropathy) which impairs sensation (loss of feelings of touch and pain) in the skin that increases the risk of complications such as increased risk of damage, infections, and slow healing of cuts, abrasions, and wounds. This is most noticeable for the hands and feet and can also affect the penis or vagina of sufferers.
- Leg and foot ulcers caused by poor circulation. These are slow to heal and prone to infection.
- Poor wound healing in general due to poor circulation and nerve damage.

- Foot problems because of poor or obstructed circulation and nerve damage which result in injuries, wounds, and ulcers that are prone to worsen and become infected. In extreme cases this can lead to the need for toe, foot, or even lower leg amputations. In diabetics the relative risk of having a lower limb amputation is 13 times higher than in the nondiabetic population. Among 65- to 75-year-olds it is over 23 times higher.
- More frequent infections (e.g., the common cold) due to impaired immunity. Also, having the flu (influenza) is more dangerous for diabetics as there is a 25% higher risk of developing pneumonia which is the seventh leading cause of death in the US.
- Gingivitis (gum inflammation) caused by bacterial infection which can progress to periodontitis – a condition in which the tissues that surround and support the teeth become infected and inflamed – resulting in the breakdown of connective tissue, bone, and the base of the tooth, leading to tooth loss.
- Men who have diabetes are two to three times more likely to develop erectile dysfunction (the inability to get or maintain an erection firm enough for sex). Although diabetes and erectile dysfunction are two separate conditions, they tend to go hand-in-hand. Like the other vascular (blood vessel) problems that develop with type 2 diabetes, erectile dysfunction is caused by damage to nerves and blood vessels caused by poor long-term blood sugar control. Sexual dysfunction can also affect females with type 2 diabetes and studies have found that the prevalence in women is similar to that in men. Sexual dysfunction in women can affect sexual desire and arousal and can also lead to pain during sex. Vascular damage can limit blood supply to the vagina and clitoris which can cause problems with dryness and arousal. Neuropathy (nerve damage) can have a similar effect in that it can reduce sensitivity. Type 2 diabetes can also lead to lower than normal estrogen levels which can also affect lubrication of the vagina. However, it does not affect the ability of a woman to become pregnant.

WHAT IS THE METABOLIC SYNDROME?

Type 2 diabetes also forms part of the **metabolic syndrome**, a term that is used by scientists and clinicians to describe the common co-occurrence of several known cardiovascular disease risk factors that include elevated blood glucose levels and insulin resistance that are main features of type 2 diabetes. The metabolic syndrome is not actually a disease but a group of characteristics. These characteristics include:

- Obesity.
- Hypertension (high blood pressure).
- Elevated blood sugar levels.

BEATING TYPE 2 DIABETES

- Insulin resistance.
- **Dyslipidemia:** A collective term for non-healthy levels of blood **lipids** (fats) including high levels of triglycerides (a storage form of fat), cholesterol, and LDL, and low levels of **high-density lipoproteins** (HDL).

Each of these characteristics is an individual risk factor for cardiovascular diseases including atherosclerosis, coronary heart disease, and stroke. Having just one of these characteristics doesn't mean you have metabolic syndrome; you need to have three or more of them. However, having any of them increases your relative risk (see the following sidebar about what relative risk means) of serious disease and the more you have increases your risk even further. The following factors increase your chances of having metabolic syndrome:

- Age: Your risk of metabolic syndrome increases with age.
- Race: In the US, Mexican Americans appear to be at the greatest risk of developing metabolic syndrome. In the UK it is the South Asians who are at highest risk for metabolic syndrome.
- Obesity: Carrying too much weight (fat) increases your risk of metabolic syndrome. People with central obesity – which is when you store fat around your middle (abdomen), rather than around the hips and thighs – and people with a BMI of 30 kg/m² or more are particularly at greater risk of metabolic syndrome.
- Type 2 diabetes: Your risk of metabolic syndrome increases the longer you remain diabetic.
- Gestational diabetes: You're more likely to develop metabolic syndrome if you have had diabetes during pregnancy (known as gestational diabetes). In 90% of women gestational diabetes will resolve after the baby is born but leaves the mother with an increased risk of developing both type 2 diabetes and metabolic syndrome.
- Other diseases: Your risk of metabolic syndrome is higher if you've ever had cardiovascular disease, nonalcoholic fatty liver disease, or polycystic ovary syndrome.

What does relative risk really mean?

In this book you will read about relative risks to health of certain characteristics such as being obese or over the age of 50 and certain lifestyle behaviors such as being sedentary, eating too much sugar, smoking, or drinking alcohol. It is important to realize that the comparisons of risk are made relative to the average person who, for example, does some exercise, consumes an average amount of sugar, does not smoke, or does not drink alcohol. It is not to say that the risk of health problems in these people with healthy habits is zero. Everyone has a certain degree of risk for a particular health problem like type 2 diabetes, coronary heart disease, or cancer but the risk of some diseases can be increased above average by unhealthy lifestyle behaviors such as consuming too much fat, sugar, or alcohol.

Sometimes the risk is not simply linearly related to the intake of “unhealthy” food items or the degree of other bad behaviors such as lack of exercise or smoking tobacco products.

Let’s take alcohol as an example. A massive global study published in the *Lancet* in 2018 concluded that there is no safe level of drinking alcohol. In other words, even drinking one glass of wine or beer per day increases the risk of health problems such as cancer, injuries (e.g., accidents, burns, falls, violence, and road traffic incidents), and infectious diseases. But the increased risk to health of having just one alcoholic beverage per day is only 0.5% and this would probably not dissuade people from having a daily glass of wine with their meal. Specifically for the risk of developing type 2 diabetes, moderate alcohol consumption is actually beneficial. A **meta-analysis** (a pulling together of multiple studies in an effort to increase statistical power and help resolve uncertainty) of 20 studies concluded that moderate alcohol consumption was associated with a 30% reduced risk of developing type 2 diabetes in both men and women. Optimal alcohol consumption (compared with being teetotal) was 22 g/day for women and 24 g/day for men (20 g of alcohol is equivalent to one 175 mL glass of wine or a 330 mL bottle of beer). However, alcohol intake worsened the risk of type 2 diabetes when it exceeded 50 g/day for women and 60 g/day for men. Note that many food and health agencies report intakes as **units of alcohol**. One unit of alcohol is equivalent to 8 g or 10 mL of pure alcohol, so a 175 mL glass of wine or a 330 mL bottle of beer both contain just over two units of alcohol, and the maximum recommended intake for adult is 14 units per week.

So relative risk depends on the nature of the risk factor, its magnitude or dose, and on the specific outcome measure that is under examination. Studies that only concentrate on type 2 diabetes may be misleading with regard to the risks of other diseases such as coronary heart disease, cancer, or infectious illness so bear this in mind when you read about studies on characteristics or behaviors that influence the risk of developing type 2 diabetes. You also need to take into account the magnitude of any relative risk before making any decisions on changing your current lifestyle.

ARE TYPE 2 DIABETES AND THE METABOLIC SYNDROME REVERSIBLE?

The latest research indicates that type 2 diabetes can be reversed and gotten rid of by dieting that results in a sufficient loss of body weight and abdominal fat. Increased physical activity can also assist with weight loss and both acute and chronic exercise is known to improve insulin sensitivity. In other words, going for a run or a long walk will not only burn some fat but will also temporarily increase your insulin sensitivity and doing that sort of activity regularly will further improve your insulin sensitivity. Type 2 diabetes gets more difficult to reverse the longer you have it so it is best to act as soon as you are diagnosed with the condition. Now many people may think that because there are no immediate symptoms and there is some medication available to help