

Prologue

Abdul was born on 22nd October 1961 in Pakistan, in a village close Rawalpindi. The family was poor. His father worked on the land but his mother did not work **outside, staying** in the house most of the time. As far as he can remember his mother wore a head-scarf but she did not cover her face. Abdul did not go to school but he would spend his days playing outside with the other boys.

In 1971 the family emigrated to England in search of a better life. They settled in Oswaldtwistle in Lancashire, where his father obtained employment in a textile mill. Abdul went to school in Oswaldtwistle but he did not play out very often. They lived in a terraced house without a garden. The textile mill in which his father worked closed down but he was able to obtain work as a labourer in a cement factory and the family moved, again living in a small terraced house. After leaving school with no qualifications, Abdul worked in a factory as a battery filler. In England two more children were born to the family but they were not as healthy as Abdul was in childhood. One suffered from learning disabilities and the other was congenitally deaf.

Abdul had always been of low weight and small in stature but in 1993, at the age of 32 years, his health deteriorated. He visited his family doctor with symptoms of lethargy. He was 52 kilograms in weight and initial blood tests showed that he was anaemic, and further investigation showed that he had advanced renal (kidney) failure. He was clearly critically ill and his family doctor therefore arranged for him to be admitted to the hospital as a matter of urgency. Full assessment included an ultrasound scan that showed the kidneys to be small and shrunken, the result of progressive inflammation over a period of perhaps ten years, usually driven by an immune process. The disease was not reversible. Abdul was started on the life-saving dialysis and was put on a maintenance programme. In 1996 he received a kidney transplant.

On further investigation he was shown to have a low serum calcium level in the blood, and the serum parathyroid hormone (PTH) concentration was very high. The combination of the two indicates biochemical features of osteomalacia, due to vitamin D deficiency, and he was given the vitamin D supplement that was necessary. The vitamin D supplement had to be stopped quite early as the blood calcium level rose to above the normal range; he had developed tertiary hyperparathyroidism, a rare but well-recognised complication of advanced chronic renal failure.

In 1995 Abdul sustained a fracture of the scapula, an unusual fracture that was almost certainly a manifestation of continuing osteomalacia.

The next development was in 1997 when he developed enlarged lymph glands in the neck, cervical lymphadenopathy. Chest X-ray showed more enlarged lymph glands, within the chest. Biopsy of the neck glands confirmed the clinical suspicion of tuberculosis and he was given a six-month course of treatment with rifampicin, isoniazid and pyrazinamide.

In 1998 Abdul developed sudden onset of weakness of his left arm and this was due to a cerebral infarction, a stroke.

In 2001 he was admitted to hospital as an emergency on account of sudden onset of chest pain. ECG showed features of myocardial infarction. On that admission he was found to have a high blood glucose and a diagnosis of diabetes was made. He later went on to receive treatment with oral hypoglycaemic agents.

In 2002 his kidney function deteriorated, and it appeared that the immunological / inflammatory process (glomerulonephritis) that had damaged his natural kidneys was now damaging the transplanted kidney.

In October 2004 he was admitted as an emergency on account of sudden onset of weakness of the right leg. He had sustained a further cerebral infarction, or stroke. Blood tests showed deteriorating renal function. Despite this huge burden of illness during the past ten years, Abdul remained cheerful and optimistic. This was the last time that I saw Abdul.

In December 2004 he developed a staphylococcal septicaemia. Despite full intensive care support his kidney function deteriorated rapidly and he died. His age was 43 years. The good life that Abdul's parents expected for their children had failed to materialise.

Frank was born in Accrington in 1943, into a household of significant socio-economic deprivation. His father had been born in 1910 but had a great deal of illness. He had spent quite a lot of time as a young man in a sanatorium being treated for tuberculosis. This meant that he was never able to establish regular employment and at the time Frank was born he was working as a part-time caretaker. Frank's mother did some work as a part-time cleaner.

Frank passed through school without education making much of an impact on him and he left at the age of 15 without any qualifications. He had a succession of jobs in unskilled work usually as a store-man. He married at the age of 24 and had 2 children. The cycle of social economic deprivation continued and there was never much money in the family. They lived in a small house close to the centre of Accrington.

In his early twenties Frank developed dyspepsia and was found on barium X-ray to have a duodenal ulcer. At the age of 31 years, because of worsening symptoms, surgical treatment (vagotomy and pyloroplasty) was undertaken in 1974, shortly before effective medical treatment became available.

He became unemployed in 1980 at the age of 37 and did not work afterwards. By that time ill health had developed, mainly in the form of low-grade bronchitis. He had smoked 20 cigarettes per day during his adult life but did not drink very much alcohol

At the age of 40 Frank was admitted to hospital as an emergency on account of severe chest pain and this was found to be due to myocardial infarction. Appropriate medical treatment was given and he made a good immediate recovery but subsequently developed angina. By the age of 48 he was experiencing pain in his calf muscles on walking, and peripheral atherosclerotic vascular disease was diagnosed. This progressively worsened causing quite severe restriction of mobility. He continued to smoke despite advice to stop.

Although he was thin as a child and young adult, his weight increased when he stopped work and this also affected his mobility. In 1996 at the age of 53 years, he had a stroke, a cerebral infarction, causing a right hemiplegia and a severe speech disturbance. He spent 2 months in hospital and recovery was only partial.

In 2002 he was admitted to hospital on account of severe chest pain, the result of a further myocardial infarction, but he survived this further development. He was left severely disabled and was unable to go outside. He was not able to manage stairs and slept downstairs.

In 2004 he had a second stroke, which was fatal. His age was 61 years.

Stuart was doctor, a director of public health in Lancashire. He was careful about his personal health, not smoking, moderate in alcohol, eating the right food, taking exercise, having a normal cholesterol and blood pressure. It came as surprise to him when he developed chest pain at the age of 58 years, but he realised that he was having a heart attack. He was admitted to hospital and the diagnosis of myocardial infarction was diagnosed, and he went

on to make a good recovery. He was so puzzled by this unexpected turn of events that he wrote an article 'Why me?'.¹ He could not understand his predisposition to an illness that he, like others, was taught to be due to a faulty lifestyle, and his was faultless. But, he remembered when I was in conversation with him, that as a child he was badly sunburnt, and for the rest of his life he was very careful to avoid exposure of his skin to the sun. Stuart died at the age of 70.

Leila's skin is pale and immaculate, soft as a baby's bottom. The facial colour changes between white, yellow and pale grey. The life she leads is reflected in her childlike skin that never sees the sun, and her hands – rough and worn like an old woman's. For a long time, Leila felt dizzy and weak – when she eventually went to see the doctor, he said she needed sun and vitamin D.

Paradoxically Kabul is one of the sunniest towns in the world. The sun shines nearly every day of the year, 1,800 metres above sea level. The sun makes cracks in the earth, dries up what were once moist gardens, burns the children's skin. But Leila never sees it. It never reaches the first-floor flat in Mikrorayon, nor in behind her burka. Not one single curative ray gets past the grille. Only when she visits her big sister Mariam, who has a backyard in her village house, does she allow the sun to warm her body. But she goes there only on rare occasions.

Aimal is Sultan's youngest son. He is twelve years old and works twelve hours a day. Every day, seven days a week, he is woken up at daybreak. At eight in the morning, Aimal opens the door to a little booth in the dark lobby of one of Kabul's hotels. Here he sells chocolate, biscuits, soft drinks and chewing gum. He counts the money and is bored. He calls the shop "the dreary room". His heart bleeds and his tummy churns every time he opens the door. This is where he must sit until he is fetched at eight o'clock in the evening, when it is already dark outside. He goes straight home to eat supper and go to bed².

Åsne Seierstad. *The Bookseller of Kabul*.

Introduction

During almost 40 years of clinical practice, I have seen many patients with a variety of illnesses, the causes of which have frequently remained rather a mystery. This was generally accepted without a great deal of professional or lay curiosity. Perhaps the most notable is a myocardial infarction, which with angina is generally called ischaemic heart disease or coronary artery disease. There is a recent international tendency to call it coronary heart disease and this is perhaps the best term in that it indicates that it is a disease, that has an effect on the heart and that it involves the coronary arteries that supply blood to the heart muscle. It has been a major cause of death in Western Europe and North America during the latter half of the 20th century, and it is becoming so in what we might call the newly industrialising world.

Somehow we gain the impression that coronary heart disease is understood, that it is simply a multi-factorial disease, and that most of those factors are contained within genetically determined family risk, faulty diet, faulty lifestyle, too little exercise, too much cholesterol in the blood, and cigarette smoking. However, this is far from satisfactory as people can have the disease without any of these factors, for example Stuart who we met in the Prologue. I believe that there is a great ignorance concerning the cause of coronary heart disease and this leads me to look critically at the way thinking has developed over the years.

Clinical medicine and the research that develops from it tend to be specialist based. People who look at heart attacks tend to become experts in the condition and see no other type of illness. They therefore see an increasingly small picture and they never have sight of the much bigger picture - the totality of illness that surrounds them. My clinical practice has been more general and this gives me a great advantage. I am not a heart specialist, and although I see a large number of people with coronary heart disease, I see the condition within the perspective of a much wider range of disease and illness. It is quite clear that the big picture gives a great deal more information, but can also expose ignorance. As has been observed by Thomas Kuhn: ¹

“Professionalism leads....to an immense restriction of the scientist’s vision and to a considerable resistance to paradigm change.”

It is the resistance to paradigm change that particularly interests me. During the past 35 years or thereabouts, I have read widely and I have collected many published papers on subjects that have interested me. I have always taken the view that it is important to examine original texts rather than summaries that often contain erroneous conclusions of individuals. It is thought that medical knowledge has more than doubled during my working life-time. This means that present-day medical students must learn more than twice what was necessary when I was a medical student. Correspondingly, I have been able to absorb this additional medical knowledge slowly and as it has been produced. This has been a great help to understanding, and I feel that I have been fortunate to have practised clinical medicine during the last 40 years of the 20th century.

Many of the papers I collected have been on the subject of the sociology and geography of disease and I will review many of them in the chapters that follow. I suppose that my interest in these topics was my origin in Manchester and Salford, and we will see that this is a conurbation of high disease incidence, especially among the poor. The sufferers of

disease are often blamed for their misfortunes, on the basis of a faulty life-style. I feel that this is unjust and without strong foundation; a search for the truth requires further investigation.

In addition to wide reading, I have a busy clinical practice, specialising in gastroenterology but with a strong foundation in general and acute medicine. I have seen many patients with a variety of conditions; Abdul and Frank are two of them. The details and implications of their tragic long-term illnesses will become apparent during the course of this book. In fact I effectively dedicate the book to them and people like them, for whom the burden of illness is disproportionately great and for whom death comes far too early. Their experience contrasts so much with my personal health and life, which started in the same year as that of Frank and only 20 miles distant. Our passages through life could not have been more different.

Many of the papers that I will review have been published in the Lancet and the British Medical Journal. We are fortunate in having such excellent journals available to us and I have been an avid reader of both since I was a medical student in Manchester. To achieve publication in these journals, papers must be reviewed very critically. The same applies to two other excellent publications, the New England Journal of Medicine and the Quarterly Journal of Medicine (QJM). The latter has published many excellent review articles during the past few years in particular. The New Scientist also gives a frequent supply of a wide range of up-to-date reviews.

These journals provide each week what I can only call clues to the mysteries of the causation of disease. Some of the clues are very important but that importance might not be appreciated at the time and out of context. They can also be regarded as pieces of a large jigsaw puzzle, the importance of an individual piece being realised only when it is placed alongside others. I am attempting in this book to put the pieces together to form what I think is a picture that makes sense of the clues, pointing to the influence of the environment, especially the sun, in the causation or susceptibility to disease. Most of the work reviewed has been conducted by others, but I have used their results rather than their conclusions. Many people can be shown in retrospect to have misinterpreted their work and may also have underestimated its value. Once again I quote the wisdom of Thomas Kuhn:

“What a man sees depends on both what he looks at and also upon what his previous visual-conceptual experience has taught him to see.”

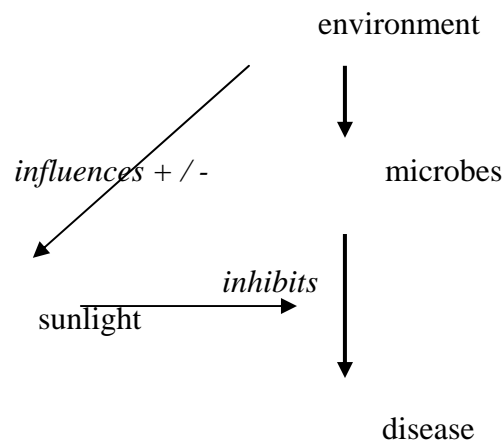
We will see many examples of this and I will try to produce a vision somewhat different from that of many, but by no means all of the original research workers. The process is that of

“...handling the same bundle of data as before but placing them in a new system of relations with one another by giving them a different framework.”²

Although we think about the cause of a disease, there is a failure to separate the concept of cause from the concept of susceptibility of individuals or populations to a given disease. For example death from the impact of a car can be regarded as caused by the car, but susceptibility to the death might be a high blood alcohol level of either the pedestrian or the driver. It is important to recognise that these two factors are different. Death or disease can occur not only because the individual is exposed to “the cause” but also because he or she is susceptible to it. We thus see the basic model:

cause + susceptibility → disease or death

The conclusion that I draw is that coronary heart disease and several other important diseases affecting the health of the general public in Europe and North America are due to microbes, and that the susceptibility to these microbes is modulated by the powerful environmental factor of sunlight – the less the sunlight exposure, the greater the susceptibility. Various environmental factors influence the exposure of populations and individuals to the sun, as we have seen with Leila in Kabul. We will meet several of these influences later, but the general model that emerges is as follows:



This book looks at the development of ideas and published information leading to the conclusion that the sun is extremely beneficial to the human body, and that a large burden of illness, especially among the poor and the ethnic minorities of the UK, is the result of inadequate exposure to sunlight. This is particularly important for populations living on the north-west fringes of Europe, a long distance from the equator and with a climate that creates a great deal of cloud cover. Environmental, geographical and cultural factors are thus very important in determining the protective effect of the sun, and thereby the incidence of disease.

I will develop the idea that much of the improvement of the health of the population in Western Europe during the 20th century has been due to the controls over atmospheric pollution that were introduced in its latter half. Correspondingly, I propose that the emergence of many “western diseases” in newly industrialising countries at the present time is due to atmospheric pollution that results from industrialisation, as took place in Western Europe in the 19th century.

Within the UK, there is a disproportionate burden of illness borne by the poor and the South Asian ethnic groups; I believe that this is explained by their low exposure to sunlight, compared to the white middle classes. Furthermore within Europe the high level of illness and reduced life expectancy in the north-western parts can also be explained by climatic features that result in a low exposure to sunlight.

In other words, I am presenting disease as a result of the ecosystem within which human beings exist. We share our environment with microbes, many of which have the ability to kill us, either as individuals or on pandemic proportions. This ability is influenced by variations in the environment, and the variation of sunlight reaching ground level is one of the most important factors. We also influence our environment by housing characteristics, patterns of work practice, cultural variations including indoor life and clothing, pollution of

water and the air. We move from rural environments to cities, and we move from one country or continent to another. These all influence the delicate ecosystem in which we live.

There is at present within our society an obsession with cholesterol and diet that is out of proportion to the evidence available, but with both of these there is a close relationship to sunlight exposure that is not generally appreciated. The evidence on which this obsession is based will be dealt with in detail in later chapters of this book.

These ideas obviously have great implications internationally for the health of individuals and populations, and particularly for those who shape health policy. Health is a subject that is of importance to individuals and to governments. Health issues feature in national press and magazines; government and opposition parties argue over many of them. Unfortunately health is not a simple subject; it is the culmination of about two hundred years of thought and scientific development, based on physico-chemical, biological and behavioural sciences. The understanding of health and illness as we know it requires an understanding of science based on the western scientific tradition. There are of course many alternative or complementary medical practices, the basis of which is not scientific but phenomenological. Sometimes they are based on what we must call pseudo-science, often derived from alleged Chinese tradition.

In this book I attempt to explain the scientific basis of the story that I am about to unfold. I have tried to make the concepts and examples as clear as possible but there are bound to be technical words that might be unfamiliar to a non-medical reader. However it is necessary to learn the language of science to be able to understand it. I also provide access to original work in some detail, rather than simply providing an interpretation or personal opinion. The journey through the book might be difficult at times, but I hope that persistence will be rewarded by a better understanding of the relationship between human disease and the environment in which we live.