Is another book on evolution and the human diet needed? We think so, largely because we know a lot more about the subject than we did just over a quarter of a century ago, when Eaton and Konner (1985) put forward their idea of the 'Stone Age diet'. By collating and quantifying nutrient intakes of contemporary hunter-gatherer groups and showing how similar they were to dietary recommendations for the prevention of chronic diseases, they identified dietary ideals for good health that deviated from the practice of developing dietary norms from foods that are currently available (but not necessarily ideal). While the imposition of a 'Stone Age' ascription to contemporary hunter-gatherer diets is a problematic aspect of their argument, their work was an important deviation from usual nutrition practice. It freed nutritional theorists to think beyond the ecology of food within their particular locale or nation. Public health nutritionists now think broadly about the challenges faced by populations in a rapidly transforming and globalizing world, from trying to construct good nutrition from what the food system delivers, to thinking about what type of food system would deliver the best nutrition. Food production, supply, consumption and education is highly political, and contemporary approaches to public health nutrition may also stress this: various food guide pyramids for the United States (US) have been deconstructed, for example, as representing political, rather than health, interests (Nestle 2002).

Various reformulations of the 'Stone Age diet' theme (Eaton et al. 1988; 2002; Milton 2000a; Cordain et al. 2000a) have been made across the years that followed Eaton and Konner's (1985) original work. Methods and the ideas they inform move on, however, and recent developments in palaeoanthropology, population genetics and epidemiology mean increasing certainty about evolutionary processes related to diet, the time-frames in which they took place, and how they inform our knowledge of contemporary human diet. The 'Stone Age' (often taken to be synonymous with the archaeological Palaeolithic period and the geological Pleistocene epoch) is broadly the period
in human prehistory from the first appearance of stone tools around 2.6 million years ago to the origins of agriculture (around 10,000 years ago), when people lived in foraging (hunting and gathering) societies. But the diets and subsistence strategies of the hominin and primate ancestors of Homo sapiens had already been shaped across the millions of years before then. While much of human nutrition is clearly biological, explanations in this domain usually stop at proximal levels, such as the biochemistry of nutrient requirements and the physiology of energy balance. Up-stream, or more distal, answers to questions such as: "why do humans have a requirement for vitamin C, while most other mammals do not?", and "why are humans protein requirements much lower than those of non-human primates?" are rarely addressed. These are more fundamental questions which can help frame expectations of human physiological responses to diet and dietary change, especially in contemporary societies that are undergoing rapid transformations in the quality and quantity of foods available.

The field of evolutionary medicine frames human illness, disorder, and pathology in a distinct evolutionary framework. It emphasizes that diseases arise from the inevitable compromises of an evolved body interacting with novel environments (Nesse 2008). Many advocates of evolutionary medicine view present-day environments as changing faster than human physiology can (Eaton et al. 2002). Since human form and function change, but slowly and must respond to rapidly altering dietary circumstances, it is important to understand both the evolutionary baggage that humans carry, and the nutritional changes created by the social and economic transformations that they undergo.

Human societies have undergone many transformations, most of which have seen changes in dietary and nutritional circumstances. The Neolithic transformation, for example, was characterized by radical economic, societal and technological change that eventually saw agriculture become the dominant subsistence practice for the majority of the world's populations. European exploration and political expansion from the fifteenth century onwards saw New World plants like tomato, capsicum and potato enter European diets, and European cereal-based crops and livestock such as cattle and sheep enter the Americas. Seventeenth century globalization saw cosmopolitan diets emerge in places affected by colonialism. With the industrial revolution, urbanization saw dietary transformation on a scale previously unknown, including the emergence of fast foods and convenience foods. Globalization in the late twentieth century has seen the amplification of earlier cosmopolitization of diets and the rapid spread of fast and convenience foods.

Human genomes, which have been shaped over hundreds of thousands of generations, interact with the new food environments created by these processes on a daily basis. Any discussion of dietary evolution and dissonance between the human genome and contemporary diet is only half-informed if the creation of new food environments is not considered. Thus we engage with a range of theoretical perspectives, including theories of class, capital, globalization, networks and inequality, as well as human and hominin evolution, biochemistry and nutrition. Disciplinary boundaries make the combined study of these aspects of diet and nutritional health very difficult. We therefore resist any claim of a seamless account of diet and nutrition in human evolutionary and social context. Rather, we have striven to identify disciplinary intersections that are susceptible to collaborative investigation and understanding.

This book considers different aspects of changing human nutrition from evolutionary and social perspectives, and identifies the importance of up-to-date knowledge of human evolution and social theory to public health nutrition practice. The book is framed in three parts: The Animal Within; A Brave New World; and Once Upon a Time in the West. In the first part, we consider what a 'natural' human diet might be and how it may have been shaped across evolutionary time (Chapters 2 and 3), and the extent to which human plasticity in response to changing food availability is a baseline adaptation, especially in relation to climate seasonality (Chapter 4). We also examine the transition in subsistence from hunter-gatherer to agricultural forms of economic life (Chapter 5). In the second part, we discuss the problems of nutritional ill-health created by the dietary change associated with this transition (Chapter 6). We also examine the new nutritional challenges that came with the emergence of new infectious diseases and the increased intensity of infection of existing pathogens (Chapter 7), and the social and economic inequality (Chapter 8) that came after the emergence of agriculture. In the third part, we discuss the modern intensification of food production and its consequences for nutritional health among contemporary nation states. Nutrition transition (Chapter 9) and the high levels of consumption of fats (Chapter 10) and refined carbohydrates (Chapter 11) are all associated with the high prevalence of the relatively recently emerged disorders of cardiovascular disease (CVD) and Type 2 diabetes, as well as a number of cancers. The three parts that form this book are more usually considered separately. However, there is enough understanding of diet and nutrition from a wide range of perspectives to bring these fields together, and this is what we try to do. Before giving a brief outline of the various chapters, it is useful to consider human nutrition within a biological framework that incorporates genetics, physical plasticity and epigenetic change as interactive adaptations to dietary circumstances.
Nutrition, genetics, physical plasticity and epigenetics

Subsistence provides food; food provides nutrition. Nutrients variously supply energy, promote growth and repair of bodily tissues and regulate bodily processes. The requirement for nutrients varies across species, between populations and from individual to individual. All mammals, humans and other primates among them, need a mix of macronutrients and micronutrients (Figure 1.1). Macronutrients include the energy-containing substances carbohydrates, lipids and proteins as well as water and fibre; micronutrients comprise vitamins and minerals. In public health nutrition, the nutritional adequacy of a diet is determined by the extent to which it matches or surpasses the recommended daily allowance (RDA) for various nutrients. This is a population-based statistical estimate of nutrient requirements, based on the premise that, for most nutrients apart from energy, it is sufficient to meet the needs of 98% of healthy individuals in a given population (Kennedy and Meyers 2005). The RDA is set at two standard deviations above the mean for normally distributed nutrient requirements because this takes account of biological variability in individual physiological requirements for different nutrients.

A large proportion of biological variability in nutrient requirement is due to genetic variation that controls the production of enzymes that in turn control the absorption, distribution, retention and utilization of different nutrients (Molloy 2004). Furthermore, genes and gene products act on, or are acted upon, by nutrients, and shape the optimal nutrient intakes of any individual (Stover and Cauldill 2008). Individuals with different polymorphisms in genes coding for the metabolism of any nutrient (via hormones and enzymes) can have different physiological outcomes at equal levels of intake of a particular nutrient. Some of these outcomes may be associated with disease. For example, polymorphisms in genes encoding enzymes involved in folate metabolism influence physiological processes that can lead to aging, cancers and CVD, largely because of the involvement of folate in DNA methylation (Friso and Choi 2002; Ulrich 2005). Alternatively, different apolipoprotein E (ApoE) isoforms (different forms of the same lipoprotein molecule) that are variously involved in fat transport and metabolism are associated with different levels of risk of developing atherosclerosis. Individuals with at least one allele for ApoE4 have an increased chance of developing this condition relative to those with either E2 or E3 isoforms (Greenow et al. 2005), as the former predisposes individuals to serum cholesterol elevation on high-fat diets (Tikkanen et al. 1990). Those with the ApoE4 isoform also have greater reductions in LDL cholesterol with reduced intake of saturated fat and cholesterol than individuals with either the E2 or E3 isoform (Krauss 2001).

Another type of human variability is due to phenotypic plasticity that permits exploitation of changing and changeable environments. For example, human children can undergo growth faltering due to poor food availability and exposure to infection, and show catch-up growth when these stresses are removed. This was probably an adaptation acquired in human evolutionary history in response to seasonal environments, by tuning body size and proportion to food available within any ecosystem (Chapter 4). This remains a fundamental phenotypic response to poor food security across the less developed world in the present-day. However, it may have become maladaptive in parts of the contemporary world experiencing epidemiological and nutrition transition where plastic responses to poor early life environments have left individuals and populations at greater risk of chronic disease later in life. Epidemiological studies and animal dietary interventions show that maternal nutritional imbalance and metabolic disturbances during critical time windows of development have a persistent effect on the health of the offspring and are likely to be transmitted to the next generation (Gallou-Kabani and Junien 2005). The ‘developmental origins of health and disease’ concept hypothesizes that chronic diseases that develop later in life originate in utero by environmental fetal programming (Barker et al. 1992, 2002). Otherwise known as the ‘thrifty phenotype’ hypothesis, it proposes that during development in utero, a child’s physiology responds to nutritional shortages with metabolic adaptations that will maximize its later survival in an environment of chronic food shortage. Such individuals will have smaller body size and lowered metabolic rate. However, if such children go on to live and grow in an environment of ample food energy, they will be more likely to develop metabolic disorders, such as obesity, Type 2 diabetes and the metabolic syndrome (Robinson et al. 2007).

![Figure 1.1. Generalized nutrient typology for mammals.](image-url)
Stoeger (2008) suggests that the mechanism for the developmental origins of health and disease model is an epigenetic one during early development. As a new synthesis of the thrifty genotype (Neel 1962) and thrifty phenotype (Hales and Barker 1992) hypotheses for explaining the rapid emergence of obesity and diabetes among the world’s populations, he put forward the ‘thrifty epigenotype’ hypothesis. In this formulation, metabolic thrift (the capacity for efficient acquisition, storage and use of energy) is seen as an ancient, complex genetic trait, which is encoded by a gene network that is canalized or channelled in a specific direction, such that it is robust against individual mutations and is able to produce the same phenotype in a population regardless of variability in environment and genotype. DNA sequence polymorphisms are seen to play a minor role in the aetiology of obesity and Type 2 diabetes; rather, susceptibilities to these two disorders are seen as being predominantly due to inherited changes in the phenotype or gene expression during the early development of bodily tissues and organs. Epigenetic changes also increase susceptibility to CVD (Waterland 2009) and play a major role in cancer formation (Esteller 2008). Epigenetic changes related to chromatin remodelling and regulation of gene expression have been identified as likely factors involved in the developmental programming of metabolic syndrome (Gallou-Kabani and Junien 2005), as characterized by disturbances in glucose and insulin metabolism, excess abdominal fat mass, dyslipidemia (abnormal amounts of cholesterol and other fats in the blood) and hypertension. Fetal under-nutrition (often manifested as low birth weight) and maternal over-nutrition (in the case of a diabetic mother) increase the future risk of Type 2 diabetes (Yajnik and Deshmukh 2008).

Across recent decades, clinical signs of obesity, Type 2 diabetes and metabolic syndrome have started to appear in childhood, have become more severe from generation to generation, and have come to affect increasing numbers of pregnant women across time. Thus, on top of direct factors like inadequate maternal nutrition, individuals with metabolic syndrome may display trans-generational effects by way of incomplete erasure of epigenetic factors carried by parents and grandparents (Gallou-Kabani and Junien 2005). Epigenetic regulation during fetal programming of the individual in preparation for the environment they expect to enter is likely to be a response to seasonal energy imbalance; changes that favour metabolic efficiency are likely to be adaptive in such circumstances. Removal of seasonal energy stress, as has taken place in contemporary industrialized societies, may turn this efficiency towards pathology. Humans thus have an evolved animal model that can respond genetically (through natural selection), phenotypically (through developmental plasticity) and epigenetically (by a balance of both) to changing dietary and nutritional circumstances. Given this baseline set of adaptations, we can explore how they evolved among the primates (including hominins and humans) and how they may have predisposed (and may continue to predispose) to nutritional ill-health or related pathologies past and present.

The animal within

‘Stone Age’ diets have captured the public imagination because they instruct us in how we might have deviated from a supposed natural diet that we are adapted to eat. In times of rapid change and uncertainty, as with the rapid emergence of chronic disease in the late twentieth century, it is comfortable to hark to a past age when things are thought to have been ideal. But such an ideal past is very unlikely to have existed: there is no ‘Garden of Eden’ diet. Rather, humans, and ancestral hominins before them, are more likely to have muddled through with their diets and adaptations to them. It is precisely this muddle that we attempt to understand better in writing this book.

To understand the type of diets that contemporary humans may be adapted to biologically, we need to dig deeper than the ‘Stone Age’, since many of the attributes of our hominin ancestors, including their diets and behaviours associated with foraging and feeding, were established well before then (Elton 2008a). New methods and perspectives, especially in genetics, epigenetics, archaeological science, network analysis and anthropology, permit this in a way that was not possible 25 years ago. In digging deeper in this book, we use an evolutionary ecology framework (Uljaszek 2002) to examine relationships between mammals, primates, and extinct hominins with respect to their various subsistence environments and adaptations — morphological, physiological and genetic — to them (Chapters 2 and 3). Biologically, humans are primates. The understanding of what natural human diets might be is therefore helped by comparison with modern primates and the types of dietary constraints they live with, including seasonal ones (Chapter 4).

Great species diversity is evident in the human family tree over time (Figure 1.3). The profound morphological differences in hominin skulls, teeth and skeletons hint at considerable interspecific dietary diversity (Chapter 3). As well as diversity between species, the human propensity for intra-specific, and even intra-individual, dietary flexibility probably also has relatively deep evolutionary roots (Chapter 3). Environments change from moment to moment, across the day, across the year, and across decades, centuries and millennia. Humans (and other mammals) are adapted to cope with environmental change and variation. Diet reflects environmental change in its seasonality and year-on-year variation (Chapter 4). Dietary diversity and flexibility were important prerequisites for hominin dispersals out of the tropics, and for
their exploitation of all the major biomes. Human dispersal across the world far surpasses the colonizing abilities seen even in that most tenacious of primates, the macaque (Figure 1.4). Members of the papionin genus *Macaca*, macaques are the only living non-human wild primates to be found in temperate as well as tropical latitudes. Figure 1.2 gives the geographical distribution of different living and extinct macaque species, showing them to range from North Africa into south, east and north Asia as far as Taiwan and Japan, and in the past to have lived in many parts of Europe. Considering what macaques eat and how they obtain this food, as discussed in Chapter 2 alongside data for other non-human primates, allows human dietary adaptation to be viewed through a broader comparative lens.

Whereas most primates are tropical animals, humans are cosmopolitan, inhabiting not only the tropics but also much higher latitudes, including above
the Arctic Circle, a geographic range unknown in any other modern or extinct primate. Humans have used their technological abilities to harvest, process and consume a very wide range of foods, including meat, to help inhabit this very wide range of environments (Elton 2008b). Dietary eclecticism has been profound in shaping human evolutionary history and has allowed humans to exploit varied and variable environments, just as it does now in many Old World monkey species, including macaques. Dietary flexibility is enabled genetically, and it has been critical to human evolutionary success. Gut anatomy is one phylogenetically determined trait essential to feeding success (Chapter 2). The human gut largely follows the general primate plan, but the larger small intestine and the smaller large intestine suggest adaptation to a higher-quality, or higher-energy-density, diet. Meat-eating may have been a selective pressure for this change, starting around the time of Homo erectus (Figure 1.3).

Although humans can eat most things, they vary greatly in their food preferences between and within societies. In Chapter 5 we examine how human food choices and diets have evolved, and how eating behaviours that favour consumption of foods that are either sweet or fatty now emerge as being detrimental to human well-being. Primates, humans included, select food by using vision, smell and taste to discriminate palatable from less palatable potential food items (Ulijaszek 2002). The ability of all mammals to enjoy and want sweetness is separate from Pavlovian learning to consume sweet things (Smith et al. 2011) and is likely to have evolved in response to the emergence of fruit and honey as food sources at the time of angiosperm (flowering plant) diversification between 275 and 65 million years ago (Friis et al. 2010; Magallón 2010). Their consumption would have been driven physiologically by the need for dietary energy. This drive is common to all mammals, but humans have the most highly developed neocortical regulation of appetite and food intake. Hominin encephalization (increased brain to body size, across evolutionary time) mostly involved increases in the size of the neocortex, and later hominins (humans among them) were able to apply aspects of sensory perception, thought and language to the production of associative pleasure with food in ways not possible by other mammals.

Without a large neocortex, humans could not have elaborated cuisines, not only because cooking can be a complex process involving a range of technologies, but also because the neurophysiology for discerning different qualities among foods and appreciating them in an associative manner would not exist. However, the human neocortex allows much more than associative pleasure in food (and other things); it allows complex sociality, intelligence and enskilled engagement with the world and other people through objects (Ingold and Vergunst 2007). Through sociality and kinship, humans have formed societies which undergo change and transformation. Foods and their use also undergo change, whether for nutrition, social or symbolic reasons. These usages result in the production of nutritional health, or nutritional ill-health, often as a side product of social transactions and consumptions.

Our brave new world

For most of human evolutionary history, people have been foragers, subsisting on plants and animals gathered and hunted in the wild. It is only in the past 10 000 years or so that human populations have produced food themselves, growing and rearing domesticated plants and animals in or near to permanent settlements. The eating and foraging behaviours of humans, as one of several primates with the ability and inclination to exploit a wide range of food resources (Chapter 2), were important pre-requisites for the move to food production. Theories to explain the origins of agriculture range from abrupt climate change (Childe 1936) to feasting and social display (Hayden 2009). Regardless of its motivating force, the origin of agriculture was the key evolutionary transformation in the history of our species (Winterhalder and Kennett 2009), with broad-reaching dietary change. Within the evolutionary medicine literature, the origin of agriculture tends to be seen as the primary point at which dietary ‘adaptation’ switches to ‘maladaptation’ within humans (Eaton et al. 1998; Eaton and Eaton 1999).

Agriculture originated independently in several parts of the world, with the best-known early agricultural sites, dated to around 10 000 years ago, being found in the Fertile Crescent in the Near East. Here, the founder crops were einkorn and emmer wheat, barley, lentils, peas, flax, bitter vetch, chickpea and possibly fava (broad) beans (Brown et al. 2009). In eastern China, the origin of rice cultivation in paddy fields, and domestication of pigs, dates to around 8000 years ago (Zong et al. 2007). Bananas and taro formed major parts of diets in New Guinea by 10 000 years ago, with domestication of the banana having taken place by around 7000 years ago (Sandweiss 2007). In Mesomerica, the cultivation of squash and maize originated over 8000 years ago (Piperno et al. 2009). In South America, potato, peanut and manioc were early domesticated crops (Brown et al. 2009), with manioc cultivation possibly having been undertaken around 8000 years ago (Price 2009). In many of these regions, animal domestication also occurred at similar times. The dog, not usually a source of food for humans, was likely to have been the first large animal to live routinely alongside them, with estimates for dog domestication varying widely but probably first occurring between around 12 000 and 14 000 years ago (reviewed in Dobney and Larson 2006). Sheep, pigs and cattle were
other species that were domesticated early, with the best estimates of sheep and pig domestication in the near east being around 12,000 and 9000 years ago respectively, with cattle following at about 8000 years ago (Dobney and Larson 2006).

The process of domestication took many generations and was very probably a gradual process rather than a rapid, dramatic event. Humans are pragmatic, adaptable and behaviourally flexible. In modern populations, the boundary between foraging and cultivation is often blurred (Ellen 1991), and there is no good reason to dismiss similar flexibility in past populations. Well before the earliest evidence of ‘true’ agriculture, human populations exhibited behaviours, including dog domestication, seed processing, fine control of fire, division of labour and food sharing, that formed important precursors of an agrarian existence (Belfer-Cohen and Goring-Morris 2009; Pearsall 2009).

Storage of food is often viewed as a Neolithic innovation and a key element in the development of agrarian economies, but the earliest evidence for pots significantly pre-dates the Neolithic (Boaretto et al. 2009). The earliest fired ceramic storage vessels come from Chita, dated to around 18,000 years ago (Boaretto et al. 2009), while pots dated to around 16,000 years ago have been found in Japan and the far east of Russia (Kuzmin 2006).

Since the process of domestication itself takes time, populations would have shifted their subsistence methods relatively slowly. Initially, domesticated animals would have differed little in size and shape from their wild ancestors, and the earliest changes in their characteristics would have been subtle (Dobney and Larson 2006). Numerous human modifications that pre-dated agriculture included selection of plant species and morphologies (such as larger grain in non-shattering ears), land preparation, and refinement of cultivation practices (Brown et al. 2009). Once established in the Fertile Crescent, agriculture spread through Europe, very probably through the movement of people rather than cultural adoption (Pinhasi et al. 2005; Balaresque et al. 2010). This diffusion took 3000 to 4000 years to reach northwest Europe (Balaresque et al. 2010). A long period of transition to agriculture is also seen in the tropical area of Latin America (Pearsall 2009). Foraging persisted in many cultivating populations across the world, with domesticated crops and animals often supplementing wild-gathered and caught foods (sensu Zong et al. 2007).

A great deal has been written about the decline in health in early agricultural populations (reviewed in Lambert 2009) but often small numbers of case studies are used and extrapolated to all emerging agricultural societies, with few contemporaneous foraging populations being examined (Elton 2008a). It may well be that many populations experienced health challenges, but these were accompanied by increases in fertility rates and population growth (Lambert 2009) which created a Neolithic demographic transition (see Gage and DeWitte 2009 for a circumspect review). Similar phenomena are evident today in developing economies, where poverty, poor health and food insecurity often go hand-in-hand with high reproductive rates (Lambert 2009). This may be one reason why humans are so successful, as they are able to reproduce rapidly in unstable environments (Wells 2010; and Chapter 4). The net evolutionary result of the adoption of agriculture could be therefore an increase in evolutionary fitness (Lambert 2009) rather than ‘maladaptation’, despite some negative effects on individual health. Invoking the emergence of agrarian economies as the start of a slide towards obesity and chronic disease in the contemporary world seems misleading in this context, especially as these are not highly prevalent in present-day populations that are dependent on traditional subsistence practices. It is much more likely that obesity and chronic disease are to some extent products of the industrialization of food production (Strassmann and Dunbar 1998) (Chapters 10 and 11), although the emergence and success of agriculture made grains the obvious source materials for much industrially produced food (Chapter 9).

In Chapter 6, we contrast foraging and contemporary industrial diets in their nutritional properties, and put into sharp relief how far we have come from nature. Contemporary industrial diets exert their effects in part through level of consumption. For example, Figure 1.5 shows the prevalence of obesity and diabetes in a number of nations between the years 2000 and 2010, according to daily dietary energy availability. Although dietary availability does not equate directly with consumption since it does not take account of food wastage and non-food uses, it is a close proxy. Unsurprisingly, there is a relationship between how much dietary energy people have available to them and the prevalence of obesity; below around 3000 kcal per day, national obesity rates are low. However, both Italy and South Korea have lower levels of obesity than might be expected at their levels of energy availability, while Japan has higher levels than expected, although Japanese food intakes are low relative to income. This indicates that other factors are also important in the causation of obesity, and the diseases of civilization associated with it. In Figure 1.5, diabetes rates show little variation according to availability of dietary energy alone. For the US, Gross et al. (2004) have identified relationships between diabetes prevalence and intakes of carbohydrate and high-fructose corn syrup, in addition to intakes of fat and total energy (Gross et al. 2004) (Chapter 10).

Globalization and industrialization have resulted in decreased diversity of fresh plant foods in many populations (Milton 1999a). Human diets constituting a broad range of animal and plant foods in the Pleistocene have been largely displaced by ones based increasingly on processed foods rich in refined grains, sugars and oils. Thus, dietary energy capture has become more efficient than ever before, but micronutrient capture has declined. In 2001, over one
third of the world’s population was estimated to have suffered from micronutrient deficiency (Tonnisru et al. 2002), while less than half this number suffered from chronic energy deficiency (Food and Agriculture Organization 2010a). Well in excess of a billion people currently suffer iron deficiency and its attendant anaemia, while iodine deficiency disorders persist. Cost and availability inhibit many of the world’s poorest people from consuming meat, even though they may prize it as food. Cost and availability also preclude them from obtaining micronutrients from dietary sources. One answer to this problem in recent decades has been the medicalization of nutrition, and the administration of nutrient supplements in non-food forms.

While agriculture emerged as a solution to population problems in different parts of the world at quite similar times, infectious disease added new complexity to an already changed nutritional ecology. That is, agricultural practice changed patterns of human behaviour and organization, concentrating population densities around intensified subsistence. This process aided the spread of pathogens present among humans at low intensity of infection prior to the emergence of agriculture and increased the rate at which pathogens infecting animals could cross the species barrier into humans. Whenever humans change how they do things, responses to existing stressors often allow the emergence of new stressors. The origins of agriculture, industrialization and globalization are all associated with changes that saw the emergence of new infections or increased vigour of infection. Nutrition (with its powerful impacts on immune function) would have been a major environmental mediator of disease susceptibility, exposure and outcome, and of natural selection against infectious agents. The nutritional stresses that followed agricultural intensification would have increased immunological susceptibility to infection, while many diseases would have raised nutritional requirements during the course of infection. In Chapter 7, comparisons are made between the emergence of undernutrition and infectious disease in prehistory and present-day patterns of undernutrition and infection in the modern less economically developed world.

Food security is undermined by poverty, contributing to the interactions between nutritional state and infection that impact on child growth and survivorship. Human populations have differential genetic resistance and susceptibility to past infections, and varied types and levels of nutritional adaptation. Some evolutionary adaptations influence susceptibility to modern diseases. For example, the CCR5 delta 32 deletion which confers resistance to HIV infection is likely to have arisen as a single mutation among a European population around 2000 years ago, and increased in frequency as a result of exposure to a disease that persisted since its origin, perhaps smallpox, but not the plague (Galvani and Novembre 2005). Because of such genetic diversity and its implications for disease susceptibility (an understanding of which is still unfolding), future public health solutions to nutrition-infection interactions will need to be more complex than presently conceived.

Global inequality in nutritional health in the present day is clear to see, with around 13% of the world’s population being undernourished and around 5% of it obese. Variation in food consumption patterns and nutritional health across nations reflects differences in history, ideology, and power relationships, which become expressed in differences in economic development. In Chapter 8, we add anthropological and evolutionary ecological perspectives to the understanding of the emergence of social inequality in humans. We examine how population size is fundamental to the ways in which societies can be structured, and how unequal nutritional health appears to be epiphenomenal to the larger economic project that drives the success or failure of all societies. We argue that social and economic inequalities are likely to have conditioned nutritional health from the origins of agriculture to the present day, and that social inequality and nutritional inequalities are linked through the attribution of symbolic value to goods, including (and sometimes especially) food. Food is the most elementary of symbolic goods because it is used to mark status in all societies. It has been elaborated to a high degree across human prehistory and history, a process that now continues at break-neck pace, with the development of global food cultures and the diversity of shops and restaurants that deliver them. Food preferences and consumption are used to define social class
Once upon a time in the West

In the contemporary world, intensification of food production and the commodification of fats and carbohydrates have allowed growing urban populations in industrialized and industrializing nations to be fed cheaply and generally stably, free of many of the major fluctuations associated with food seasonality. This should be a good thing, except that the consumption of food products developed for mass use has been linked to a number of chronic diseases. Across the twentieth century and especially during the latter half of it, dietary change as an outcome of modernization and globalization has led to the emergence of epidemics of obesity, CVD and Type 2 diabetes. A subset of epidemiological transition theory (Omran 1971), nutrition transition theory has been developed by Popkin (2002a) as an explanatory framework for changes in nutritional health according to stages of dietary consumption types and physical activity patterns, from prehistory to the present. Nutrition transition theory places societal changes towards industrialized diets and sedentary ways of life centrally to the emergence and propagation of chronic disease across the world in recent decades. In Chapter 9, nutrition transition theory is described, and its relationship to broader transformative processes discussed.

Mexico is currently an economically emerging nation affected by globalization and beset with increasing levels of obesity and related chronic diseases. By examining recent nutrition transition in Mexico in relation to inequality, colonialism and globalization, an extension to nutrition transition theory is proposed in which prehistories and histories shape the responses of populations to changing nutritional circumstances. In this chapter we also locate changes in nutritional health in Mexico within a framework of dependency, initially with Spain, as its earliest colonizer, and then with the US. In places like Mexico, the industrialized food system is perhaps an unwitting accomplice of nutritional structural violence. Most broadly, its achievements have transformed human quality of life across the world by delivering predictable, safe and clean food to the billions of people that do not engage in subsistence agriculture. However, its central ethos of profit maximization often promotes consumption in ways that do not promote nutritional health. As an agent of dietary change, the history of the industrialized food system is an important part of the nutrition transition narrative, although one that is not currently widespread.

In Chapter 10, one component of dietary energy intake implicated in nutrition transition is considered. This is the consumption of fat, which has increased in quantity and changed in type in most populations across the past 50 years or so. From an evolutionary perspective, consumption of dietary fat is important because it provides greater energy density than any other macro-nutrient. It allowed the dietary energy budgets of hominins and early humans to be met more easily than with other foods. Only in recent times in industrialized societies, where food availability has been secured and physical work schedules have declined to very low levels, has its importance been questioned. Increased overall consumption of fat with recent nutrition transition has led to increased dietary energy intake, alleviating the problems of undernutrition for some, and creating the new problem of obesity for others. Fat is not just fat: there are many types of fat, varying by carbon chain-length, numbers and types of double bond, and influencing human physiology and health outcomes differently. Saturated fat consumption is associated with CVD, as is that of the industrially-produced trans-fats. Consumption of long chain n-3 polyunsaturated fatty acids (PUFA) is associated with better health outcomes than any other type of fat, as they are protective of many of the chronic diseases that emerged as significant public health problems in the past 50 years or so. However, grain-based n-6 PUFA are less healthy than n-3 PUFA, and they form a dominant part of total fat availability in most industrialized nations. In Chapter 10, we show the importance of the types of dietary fat available for consumption when investigating diet quality in public health.

In Chapter 11 we examine another output of the industrialized food system, that of refined carbohydrates, and how their consumption relates to the production of chronic diseases (Trowell and Burkitt 1981) including Type 2 diabetes and CVD, as well as the predisposing conditions of obesity and insulin resistance. Links between the consumption of refined carbohydrates and the common disorders of gout, acne and myopia are also considered. We link these three conditions as carbohydrate transition disorders, and see them as a subset of nutrition transition. We show that carbohydrate transition is most advanced in the US, where high-fructose corn syrup (HFCS) has become a major source of dietary carbohydrate and sweetness for the population. Human genetics and physiology have evolved in ways that can enhance survivorship through improving fat deposition from the consumption of sugars, especially fructose. These biochemical mechanisms have very deep ancestry, and have been recruited for new tasks, particularly in relation to the digestion of HFCS, often with negative health consequences.
An integrated view

The diversity and flexibility of the human diet is fundamental to our success as a species. However, our taste for food novelty and for creating social signifiers with types of food may be central to our nutritional short-comings. We hope that this book shows that, far from being adapted to a ‘Stone Age’ diet and maladapted to post-agricultural subsistence, humans are suited to consuming a vast range of foodstuffs (Elton 2008a). The fact that novelty is created from a very limited range of food commodities and chemicals by industrialized food systems, and that as a whole, humans respond by buying and eating new food products, places considerable responsibility on the systems of production for maintaining nutritional health. At the time of writing, global food security is in question, while the economic system for delivering adequate nutrition (in both quantity and quality) is in a precarious state. There is no better time to evaluate how food is produced, manufactured, distributed and consumed. This book presents an evolutionary evaluation of how and what we eat. We link nutrition with human form and function as an evolving work in progress, and with the social and political systems that shape human populations and their food systems. It is far from being a complete account, but by showing how prehistoric and historic processes inform and shape present-day nutritional health, we hope to have created a framework for examining other nutritional phenomena in similar ways.

Part I

The Animal Within