1 *Introduction*

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 human 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 all 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 cosmopolitanization 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 climatic 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 Caudill 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

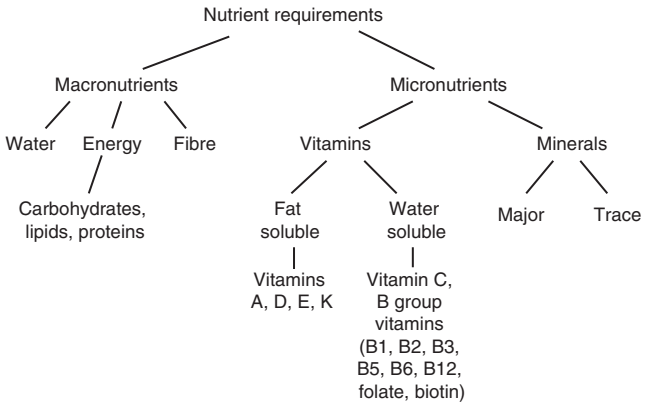

Figure 1.1. Generalized nutrient typology for mammals.

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).

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 thifty 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 (Ulijaszek 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

Figure 1.2. Global distribution of macaques.

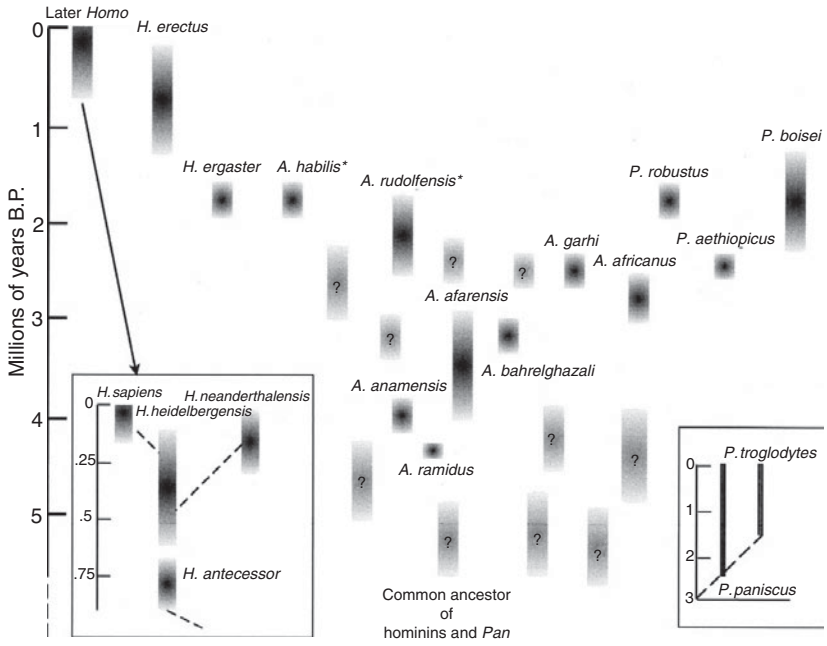

Figure 1.3. Time-line of hominin evolution (from Wood and Richmond 2000). Horizontal axis separates species according to the relative size of brain and chewing teeth. Taxa with large molar and premolar crowns are to the right, while those with smaller postcanine teeth are to the left. The hypothetical taxa marked with a question mark indicate that in the less explored period of between 2 and 6 million years ago, the number of taxa are likely to increase. Although the two taxa marked with asterisks are, or have been, conventionally assigned to *Homo*, it is likely that they are more closely related to *Australopithecus* species.

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

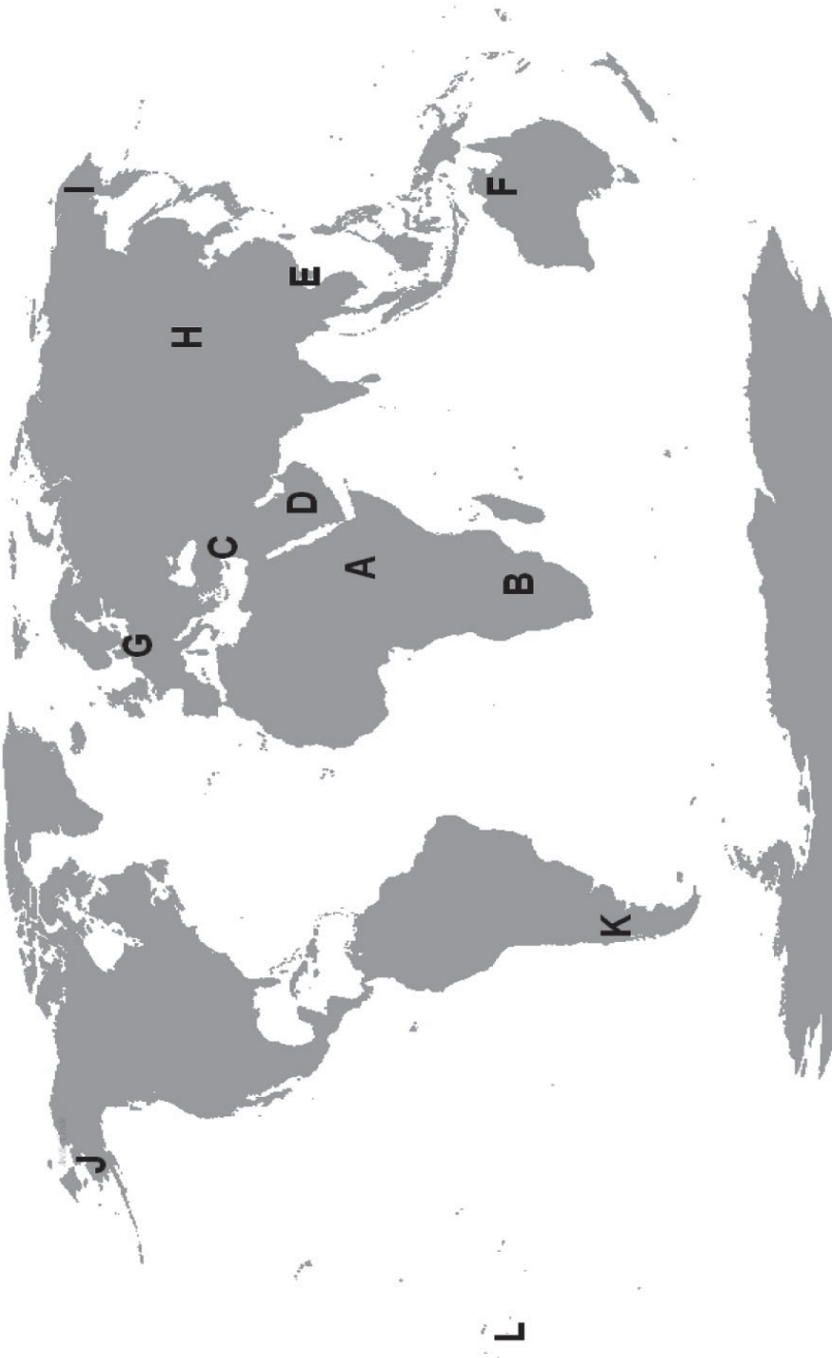

Figure 1.4. Schematic of modern human global dispersal. Data taken from Dillehay *et al.* 2008; Goebel 1999; Moodley *et al.* 2009; Oppenheimer 2009; Beyin 2011.