1 *Introduction: growth, development and the lifespan developmental perspective*

STANLEY J. ULIJASZEK AND C.J.K. HENRY

The Society for the Study of Human Biology Symposium on Long-Term Consequences of Early Environments, held on April 9–10, 1994 at the Pauling Centre, University of Oxford, considered the recent developments in the study of the ways in which early environmental factors can influence human individuals and populations across their lifespan. The topic was examined in a systematic manner by human biologists, auxologists, epidemiologists, anthropologists, physiologists and nutritionists, and the papers discussed at this meeting are published in this volume.

For the past 30 years, human population biology has sought to document and explain processes that have contributed to biological variability in the human species. In particular, it has become clear that the effects of environment upon the genotype are rarely simple, and often not immediately apparent. Environmental effects on human physical and psychological development are considerable and well documented, but the long-term consequences and phenotypic expression in later life of processes taking place during growth and development have only recently become the focus of intense study. Such research has implications for human biology, anthropology, nutrition, clinical science and epidemiology, since many of the long-term outcomes under consideration have consequences for the health and well-being of individuals and populations.

Central to the 'early environment–later outcomes' approach is the understanding that environmental effects on human growth and development, be they nutritional, disease-related, psychosocial or otherwise, can have long-term, often irreversible consequences. Before attempting to address which of the many environmental influences may have irreversible consequences for human form and function, it is important to understand human growth patterns in an evolutionary and life-history context. The

authors of the first three chapters, B. Bogin, S. Ulijaszek, and C. Worthman, offer different perspectives of this framework.

The pattern of human growth is characterized by a prolonged period of infant dependency, an extended childhood, and a rapid and large acceleration in growth velocity at adolescence leading to physical and sexual maturation. This trend is of evolutionary significance since it provides the human species with an extended period for brain development, time for the acquisition of technical skills including tool making and food processing, and for socialization and the development of social roles and cultural behaviour. The chapter by B. Bogin examines the implications of this unique growth pattern for biology and behaviour in adolescence and adulthood. The major health problems of the majority of the world's children are either infections or nutritional deficiencies, often leading to severe and costly physical and psychological complications in adulthood. Bogin argues that physically and psychologically well-adjusted adults are more likely to be the outcome of patterns of development which conform to the evolutionarily derived needs of infancy, childhood, and adolescence.

In the following chapter, S.J. Ulijaszek questions the definition of the term 'early environment', and gives an overview of long-term health and disease consequences of some of the relationships between growth and development, nutrition and disease. Ulijaszek suggests that the definition of early environment varies according to the biological phenomenon under investigation, and may be taken to be any time during the course of growth and development. Using examples of non-insulin-dependent diabetes, schizophrenia, muscular development and energetic efficiency, he concludes that the extended period of adult life experienced in industrialized populations relative to past populations and to the human ancestral state allows the greater expression of degenerative diseases. Most long-term outcomes of early environmental influences on growth and development are due either to distorted morphology, or metabolic programming. The former includes phenomena which may arise from impaired development of a somatic structure, while the second consists of phenomena which may be the outcome of physiological 'setting' of some mechanism, probably hormonal, at some critical time in growth and development.

Following from this, C.M. Worthman examines the adaptive processes which shape the human sex ratio. This has implications for the understanding of early environments, since in many societies humans may adjust each successive reproductive event to track existing environmental conditions in ways that can shape the sex ratio and completed fertilities. In high mortality populations, growth curves represent performance of survivors, and males and females often have different mortality risks, both for biological and socio-behavioural reasons. Thus, in many societies, sex and

gender differences in early environmental experience may have important implications for child survivorship, the understanding of child growth patterns, demographic structure and the basis of economic production. Factors that drive skewed sex ratios include differential parental costs according to the sex of the child, and the cultural conditions that create sex differences in child mortality. Worthman introduces the idea that the early environment may be different according to sex, may be socially constructed, and may have a temporal dimension.

Many factors influence human growth and development. When considering the growth performance of individuals and populations, healthy outcome is usually assessed by some measure of function, be it physical, physiological, psychological or behavioural. All subsequent chapters consider environmental influences upon some aspect of growth and development which have long-term consequences for some aspect of human function.

In the first of these, N. Cameron reviews literature which shows that birthweight is strongly conditioned by the health and nutritional status of the mother, in that maternal undernutrition, ill-health and other deprivations are the most common causes of retarded fetal growth and/or prematurity. Furthermore, there are intergenerational effects of the antenatal environment on adult physique and disease risk, while the associations between fetal environment and disease risk are well known. Drawing on a variety of sources, including studies of various populations in South Africa, Cameron concludes that birthweight is universally the most important determinant of the newborn surviving to experience healthy growth and development, and of the likelihood of any long-term body size consequence of an adverse antenatal environment.

The notion that catch-up growth, if incomplete, suggests a way whereby smaller adult body size and its functional correlates may be influenced by the environment during infancy and childhood. The view that catch-up growth after an early period of growth faltering can only occur in infancy and childhood has recently been challenged, and M.H.N. Golden presents evidence in opposition to the traditional view. He considers genetic–environmental interactions and their effects on growth and development, particularly in relation to the imprinting of gene expression in offspring by the parental nutritional environment. Golden supports Cameron's position about the importance of the uterine environment for postnatal growth and development, when he states that children stunted postnatally may be able to undergo complete catch-up, while those undergoing growth retardation *in utero* may not.

The idea that diet during early life might influence the rate of growth and development at a later stage is not new. Nor is the concept of the 'plane of

nutrition', which involves the regulation of nutritional inputs appropriate to optimal function at various levels including the cellular one. In a largely theoretically oriented chapter, S. Wootton and A. Jackson update and relate these two ideas within the framework of lifespan developmental theory. They develop a model whereby the long-term consequences of nutrition on growth, body size and physiological function are dependent on the programming of metabolic memory, and which includes the process of metabolic change across the lifespan. In support of this model, they describe studies carried out in Southampton which show clear metabolic and dietary behavioural differences between apparently normal but short, and normal and average-statured children. They also cite studies in India which support their view that body size and composition determined in the course of growth and development by interaction with nutritional factors, are likely to play a major role in determining the metabolic characteristics of the body.

There is considerable between- and within-population variability in human nutrient intakes and requirements. Although considerable attention has been paid to defining the range of nutritional requirements at different ages and in different physiological states, almost no attention has been paid to the ways in which the early nutritional environment can shape nutrient needs in later life. In the chapter by C.J.K. Henry, evidence for the existence of such relationships is examined. In particular, the question of whether early manipulation of the nutritional environment of humans can lead to long-term alteration in their nutrient needs later in life due to changes in body composition or organ size, is asked. Using a variety of data from early human studies and animal models, Henry concludes that this may well be the case.

In the chapter which follows, D. Mela and S. Catt examine possible reasons for similarities and differences in food selection. In the context of growth and development, this could be an important factor influencing body size, composition and metabolic characteristics as identified by Wootton and Jackson. Mela and Catt focus on the sensory-affective dimension of food acceptance in relation to the development to taste and smell, and consider evidence for their possible relationships to later food selection. They illustrate the complexity of the dietary environment and of the extent to which sensory acceptance of foods can be associated with the nutritional and physiological properties of foods. Although it is commonly assumed that taste, smell, and food preferences acquired in infancy are maintained through childhood and into adult life, most of the evidence presented in this chapter suggests the opposite. Humans show great plasticity in their food preferences, and it does not appear that specific taste and smell preferences formed early in life track into adulthood.

Perhaps the most dramatic examples of relationships between early environmental impact on growth and development and later biological outcomes are in epidemiology. In the next chapter, D.J.P. Barker reviews recent evidence linking reduced fetal and infant growth with the adult disease. Studies in the UK have shown that babies who were small have, as adults, raised blood pressure, elevated serum cholesterol and plasma fibrinogen concentrations, and impaired glucose tolerance. These are the most important risk factors for coronary heart disease and non-insulin-dependent diabetes mellitus. Barker goes on to describe a model of coronary heart disease causation which puts less emphasis on 'inappropriate lifestyle', and more on environmental factors influencing metabolic programming during growth and development.

It is possible to examine the influence of early environmental factors in past populations, and inferences from such studies may inform our knowledge of more contemporary phenomena. Although models based on the causes and results of physiological disruption or stress have been used to address processual questions in past populations, this approach has only recently been extended to consider the impacts of biological stresses such as undernutrition and infection on child development, and their implications for adult morbidity and mortality patterns. In the chapter by A.H. Goodman, the usefulness of skeletal and other hard tissue memories of early life stress in constructing models of health and disease in past populations is examined.

The development of sexual behaviour differs between males and females, in that the former undergo largely isolated learning of what is desirable in a woman, while the latter initially learn about social relationships, and impose notions of sexuality upon this knowledge. Sex and gender differences in environment, and in social experience across puberty, influence adult sexuality. In a chapter on the childhood environment and the development of sexuality, M.P.M. Richards suggests that existing models assume, on the basis of very little observational data, that sexual behaviour emerges as an inevitable result of the biological changes taking place in puberty. He challenges this notion in the light of recent social research on the development of sexuality. A more realistic view, he claims, is that sexuality has its origins early in life, and continues into adulthood. Puberty, he suggests, should not be viewed as a purely biologically determined event. Rather, it may be seen as a developmental stage whose timing may depend on earlier social relationships, as well as upon nutritional and health circumstances.

Although there is a wealth of information about growth and development in adolescence, and about demographic and biological factors related to adult female fecundity, possible links between the two have not been

sought. L. Rosetta reviews the literature on biological and environmental factors influencing the onset and timing of puberty, in relation to the development of fecundity in late adolescence and into adult life. Although it seems likely that there may be some critical period of development during which an impairment of adult reproductive function might be irreversible, to date, none has been identified. Rosetta suggests that research designs involving studies of migrants might be useful in trying to identify such a relationship.

Another area where epidemiological research involving the lifespan developmental perspective might be useful is that of the study of diseases of long latency and slow progression. These include amylotrophic lateral sclerosis, Alzheimer's disease, multiple sclerosis and parkinsonianism. These are thought to be caused by insults expressed through a common pathway leading to similar neuro-degenerative changes over time. How this takes place is unknown, but the type and sequence of events are worthy of research using studies initiated in early life. R.M. Garruto presents current ideas on the progression of such diseases, and concludes that age, timing, and sequence of events are the most important factors influencing the onset of neuro-degenerative disorders.

Collectively, these chapters set as many new questions as they answer. These are mostly concerned with the relationships between growth and development, nutrition, disease, and functional outcome. Other issues include questioning the measurement and definition of environmental factors which can influence growth and development, and the importance of social factors in understanding biological phenomena. The study of human biology from the lifespan developmental perspective as shown in its various forms in this volume, has a future. Whether it will have long-term consequences, and what these might be, remains to be seen.

2 *Human growth and development from an evolutionary perspective*

BARRY BOGIN

Introduction

The pattern of human growth after birth is characterized by five stages: (i) infancy; (ii) childhood; (iii) juvenile; (iv) adolescence; and (v) adulthood (Bogin, 1988, 1990, 1993). Changes in the velocity of growth from birth to adulthood signal the transitions between these five developmental stages (Figure 2.1). Each of these stages can be defined by distinct biological and behavioural characteristics. Infancy is the period when the mother provides all or some nourishment to her offspring via lactation. Infancy ends when the child is weaned from the breast (or bottle), which in pre-industrialized societies occurs at a median age of 36 months (Detwyller, 1994). Childhood is defined as the stage following weaning, and is a period of time when the youngster is still dependent on older individuals for feeding and protection. Childhood ends when growth of the brain, in weight, is complete. Mathematical modelling of brain growth, using direct measurements from cadavers, indicates that brain growth stops at a mean age of seven years (Cabana, Jolicoeur & Michaud, 1993). The child then progresses to the juvenile stage. Juveniles are defined as, '. . . prepubertal individuals that are no longer dependent on their mothers (parents) for survival' (Pereira & Altmann, 1985, p. 236). In girls, the juvenile period ends, on average, at about the age of 10 years, two years before it usually ends in boys. The adolescent stage begins with some visible sign of sexual maturation, such as pubic hair, which is followed by development of the other secondary sexual characteristics, a growth spurt in height and weight in both sexes, as well as the onset of adult patterns of sociosexual and economic behaviour. Adolescence ends with the attainment of adult stature which occurs, on average, at about age 18 in women and 21 in men.

In the book *Size and Cycle*, J.T. Bonner (1965) develops the idea that the life cycle of an individual organism, a colony, or a society is, '. . . the basic unit of natural selection' (p. 52). Bonner discusses organisms as diverse as

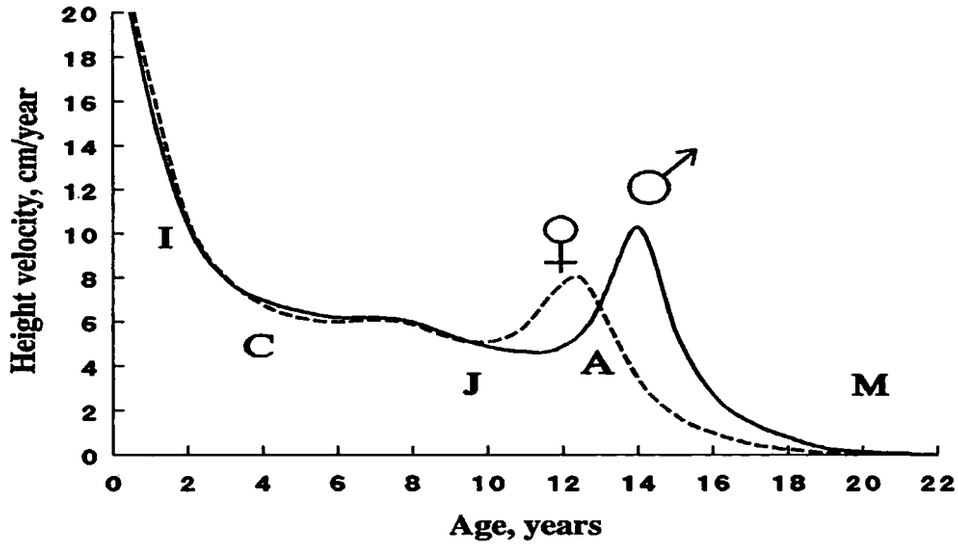

Figure 2.1. Idealized mean velocity curves of growth in height for healthy girls and boys. I-infancy, C-childhood, J-juvenile, A-adolescence, M-mature adult (after Prader, 1984 and other sources).

foraminiferans, slime moulds, algae, volvox, wheat, sequoias, planaria, lichens, blue whales, and colonies of social ants. For each species he shows that its development at different stages of the life cycle, and the duration of each stage, relates to such basic adaptations as locomotion, reproductive rates, and food acquisition. In the 1970s and 1980s many of Bonner's ideas were formalized into the study of life history strategies and evolution. 'A broad definition of life history includes not only the traditional foci such as age-related fecundity and mortality rates, but also the entire sequence of behavioural, physio logical, and morphological changes that an organism passes through during its development from conception to death' (Shea, 1990, p. 325). This chapter focuses on human childhood, in terms of both its evolution and its place in human life history strategy.

The evolution of childhood

Figure 2.2 represents a summary of the evolution of the human pattern of growth and development (the evolution of adolescence is not discussed in this chapter, but see Bogin, 1993, 1994*a*,*b*). This figure must be considered as 'a work in progress', as only the data for the first and last species (*Pan* and *Homo sapiens*) are known with some certainty. The patterns of growth of the fossil hominid species are reconstructions based on the traditional methods of human palaeontology: comparative anatomy, comparative physiology, comparative ethology, archaeology, as well as some speculation. In particular, the work of R.D. Martin and colleagues (1983; Harvey,

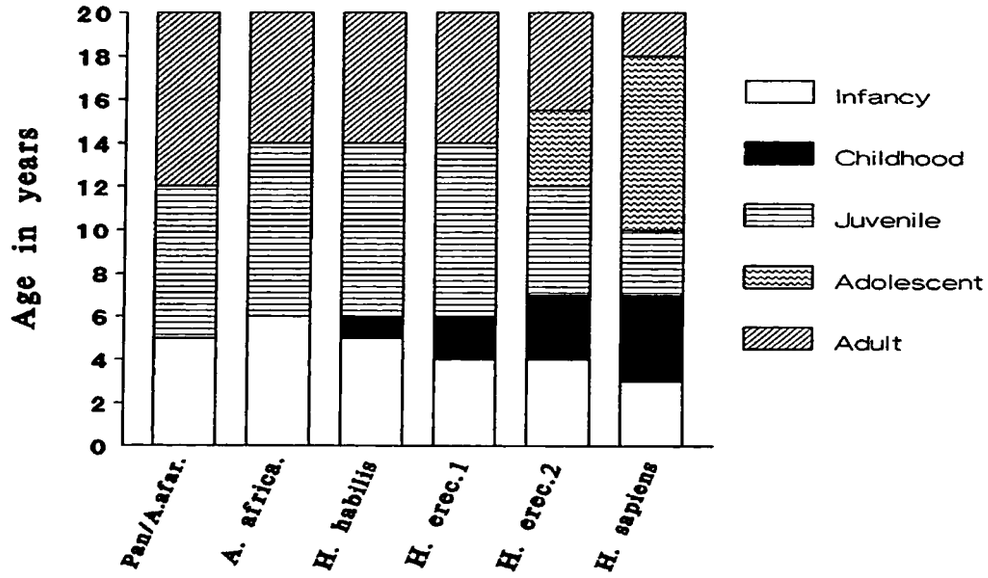

Figure 2.2. The evolution of hominid life history during the first 20 years of life. Abbreviated nomenclature as follows: A.afar.-*Australopithecus afarensis*, A. africa – *Australopithecus africanus*, H. habilis – *Homo habilis*, H. erec. 1 – early *Homo erectus*, H. erec. 2 – late *Homo erectus*, H. sapiens – *Homo sapiens*.

Martin & Clutton-Brock, 1987) on comparative patterns of brain growth in apes and humans is utilized. Martin shows that apes have a pattern of brain growth that is rapid before birth and relatively slower after birth. In contrast, humans have rapid growth both before and after birth. Martin also notes that, from newborn to adult, apes slightly more than double their brain size – a multiplier of 2.3; whereas humans more than triple their brain size – a multiplier of 3.5. Finally, Martin argues that an adult brain size of 873 cm^3 or larger represents a 'cerebral rubicon', separating ape-like and human-like patterns of brain growth. The 873 cm^3 value is calculated by multiplying the average human infant brain size of 384 cm^3 by the ape multiplier of 2.3.

Martin's analysis is elegant and tenable; nevertheless, the difference between ape and human brain growth is not only a matter of velocity; it is also a matter of life history stages. Brain growth for both apes and human beings ends at the start of the juvenile stage, which means that apes complete their brain growth during infancy. Human beings, however, insert the childhood stage between the infant and juvenile stages. Childhood may provide the time necessary to grow the larger human brain. Following this line of reasoning, any fossil human, or any of our fossil hominid ancestors, with an adult brain size above Martin's 'cerebral rubicon' may have included a childhood stage of growth as part of its life history.

Australopithecus afarensis is a hominid, but shares many anatomical features with non-hominid species including an adult brain size of about 400 cm^3 (Simons, 1989) and a pattern of dental development indistinguishable from extant apes (Smith, 1991). Therefore, the chimpanzee and *A. afarensis* are depicted in Figure 2.2 as sharing the typical tripartite stages of postnatal growth of social mammals – infant, juvenile, adult (Pereira, 1993). To achieve the larger adult brain size of *A. africanus* (442 cm^3) may have required an addition to the length of infancy. The rapid expansion of adult brain size during the time of *Homo habilis* (650 to 800 cm^3) might have been achieved with expansion of both infancy and the juvenile period, as Martin's 'cerebral rubicon' was not surpassed. However, the insertion of a brief childhood stage into hominid life history may have occurred. The archaeological evidence for intensification of stone tool manufacture and use to scavenge animal carcases, especially bone marrow (Potts, 1988), may be interpreted as a strategy to feed children. Such scavenging may have been needed to provide the protein, some of the minerals, and the fat (a dense source of energy) that children require for growth of the brain and body (Leonard & Robertson, 1992).

Further brain size increase occurred during *H. erectus* times. The earliest adult specimens have brain size of 850 to 900 cm^3. This places *H. erectus* at or above Martin's 'cerebral rubicon' and may justify an expansion of the childhood period to provide the high quality foods needed for the rapid, human-like, pattern of brain growth. It should be noted from Figure 2.2 that, from *Australopithecus* to *H. erectus*, the infancy period shrinks as the childhood stage expands. As will be discussed below, this gave *H. erectus*, and all later hominids, a reproductive advantage over all other hominoids. Later *H. erectus*, with adult brain sizes up to 1100 cm^3, are depicted with further expansion of childhood and the insertion of the adolescent stage. In addition to bigger brains, later *H. erectus* shows increased complexity of technology (tools, fire, and shelter) and social organization that were likely correlates of the biology and behavior associated with further development of the childhood stage. The transition to archaic and finally modern *H. sapiens* expands the childhood stage to its current dimension.

Why do human beings have childhood?

Brain size of extant and fossil hominoids provides some idea of when human life stages may have evolved, but does not explain why they evolved. To make sense of the pattern of human growth, one must look for the 'basic adaptations' that Bonner describes. The most basic of these adaptations are those that relate to evolutionary success. This is traditionally measured in terms of the number of offspring that survive and reproduce. Biological