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CHAPTER 11

Evolution of the Human Life Cycle

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INTRODUCTION

What Are the Big Questions?

Why should a human biologist study the evolution of the human life cycle? The reason is because, for multicellular organisms, most major evolutionary change proceeds by alterations in life cycles, that is, the patterns of growth, development, and maturation (Bonner 1965). The human species is no exception, which means that the biological and behavioral characteristics of human beings, including those shared with other mammals and those that set our species apart from all others, are derived from the features of our life cycle.

BOX 11.1 CHARLES DARWIN ON THE HUMAN PLACE IN NATURE

The shared features of growth, development, and maturation among mammals are, for the most part, due to a common evolutionary origin. Different species may also share traits due to a process called convergence, an example being convergence toward a streamlined body that minimizes water resistance in both fish and marine mammals. Commonalities in developmental biology and behavior were a source of support for Charles Darwin's hypotheses on human evolution. In his book *The Descent of Man and Selection in Relation to Sex*, Darwin (1871) wrote about growth and development before birth:

> *Embryonic Development.*—Man is developed from an ovule, about the 125th of an inch in diameter, which differs in no respect from the ovules of other animals. The embryo itself at a very early period can hardly be distinguished from that of other members of the vertebrate kingdom. At this period the arteries run in arch-like branches, as if to carry the blood to branchiæ which are not present in the higher vertebrata, though the slits on the sides of the neck still remain (*f*, *g*, fig. 1), marking

(*Continued*)

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their former position. At a somewhat later period, when the extremities are developed, "the feet of lizards and mammals," as the illustrious Von Baer remarks, "the wings and feet of birds, no less than the hands and feet of man, all arise from the same fundamental form." It is, says Prof. Huxley,[10] "quite in the later stages of development that the young human being presents marked differences from the young ape, while the latter departs as much from the dog in its developments, as the man does. Starling as this last assertion may appear to be, it is demonstrably true."

As some of my readers may never have seen a drawing of an embryo, I have given one of man and another of a dog, at about the same early stage of development.

[10] 'Man's Place in Nature," 1863, p. 67.

Quote taken from Darwin Online, http://darwin-online.org.uk/

Darwin's figure 1 is reproduced here as our Figure 11.1. Readers may also consult the work of Dr. Gina Kohts (http://www.kohts.ru/ladygina-kohts_n.n./ichc/html/apes02.html; de Waal 2009), who in the early 20th century carried out detailed comparative studies of growth, development, and behavior between humans and chimpanzees.

Darwin also wrote of some novel features of human development. In his next book, *The Expression of Emotions in Man and Animals*, Darwin (1872) wrote of "Special Expressions of Man: Suffering and Weeping" (p. 147). Darwin was one of the first scientists to include photographs in his books, and these included photographs of human infants crying and screaming. Based on the information available at the time, Darwin believed that only human infants and children expressed distress via long bouts of screaming and crying. Darwin wrote that this special behavior of people is due to human features of anatomy and cognition, which are not shared by other mammals. Since Darwin's time, it has been confirmed that other species of mammals do scream and cry (http://www.janegoodall.org/chimpanzee-crying, http://carta.anthropogeny.org/moca/topics/unconsolable-infant-crying). Research by Ronald Barr finds that crying "results in caregiving responses that can be positive (e.g. increase caregiver investment, nutrition and caring) or negative (abuse)." Barr adds that the shedding of tears during emotional distress may be unique to, or especially well developed in, humans.

These examples from the work of Darwin to the present are provided to explain that the human place in nature, as viewed by evolutionary biologists, balances the physical and behavioral characteristics that are shared with other species against those that are found only in the human species. In this chapter, we are concerned with both the shared and the "human only" characteristics as these relate to the human life cycle.

Several other "big questions" guide this chapter: How can human biologists identify the shared and novel features of the human life cycle? Can the time of origin of the novel features be determined? Can the reasons for the evolution of new growth, development, and maturation patterns be determined?

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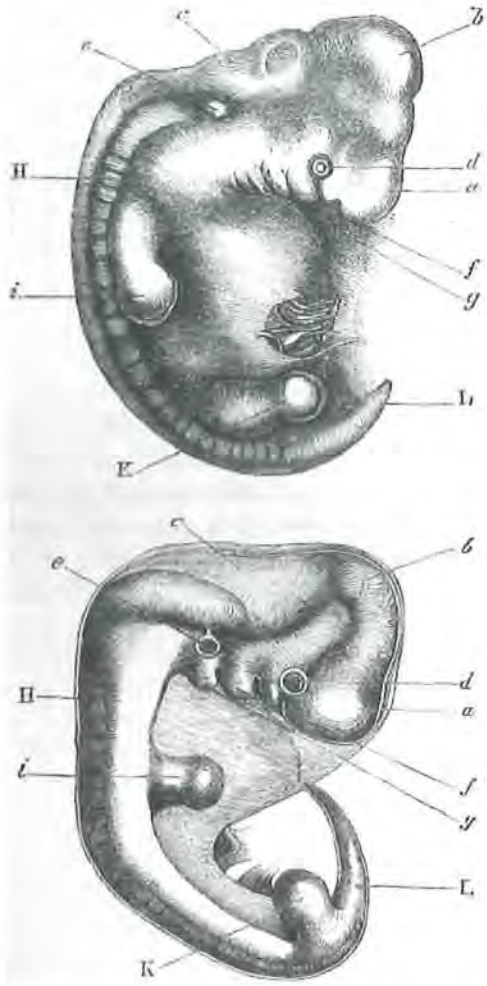

Figure 11.1 Figure 1 from Darwin (1871) *The Descent of Man and Selection in Relation to Sex*. Darwin's legend for the figure reads, "Upper figure human embryo, from Ecker. Lower figure that of a dog, from Bischoff. a. Fore-brain, cerebral hemispheres, &c., b. Mid-brain, corpora quadrigemina., c. Hind-brain, cerebellum, medulla oblongata., d. Eye., e. Ear., *f*. First visceral arch., g. Second visceral arch., H. Vertebral columns and muscles in process of development., *i*. Anterior-extremities., K. Posterior, L. Tail or os coccyx."

The major points of this chapter are the following:

(1) Human beings have a relatively long period of gestation and four stages of growth and development between birth and **adulthood**. These postnatal stages are **infancy**, **childhood**, **juvenile**, and **adolescence**.

(2) Long gestation relative to body size is a trait shared with the other apes. The infant and juvenile stages are shared with most nonhuman primates, social carnivores, elephants, and many cetaceans (e.g., whales, porpoises). Childhood and adolescence, as defined here, are human species-specific features.

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(3) Human childhood and adolescence evolved because they confer reproductive advantages, increasing the **fertility** of parents and reducing the mortality of their offspring. This is classic **natural selection**.

(4) Adolescence may also have evolved by sexual selection, in which sex-specific features of adolescent girls and boys enhance opportunities for survival and mating. The biology and behavior of human adolescence also promotes an apprenticeship-type system of learning and practice of the wide variety of economic, social, political, and sexual skills needed adulthood and successful reproduction.

HUMAN GROWTH AND HUMAN BIOLOGY

The study of human growth has been a part of anthropology and human biology since the founding of these disciplines. European "anthropology" of the early to mid-19th century was basically a combination of anatomy and **anthropometry**, the science of human body measurements. American anthropology of the late 19th century incorporated anthropometry into its foundation, and the early practitioners, especially Franz Boas, are known as much for their studies of human growth as for anything else (see Chapter 2 by Johnston and Little).

An interest in human growth is natural for anthropologists and human evolutionary biologists for two reasons: (1) the way a human being grows is the product of interactions between the biology of our species, the physical environment in which we live, and the social, economic, and political environment that every human culture creates; and (2) because, alteration in the pattern of growth, development, and maturation is a major mechanism of evolutionary change. Human growth and development, therefore, reflect the biocultural nature and evolutionary history of our species. The basic pattern of human growth is shared by all people and is the outcome of the 6–7 million-year evolutionary history of the **hominins** (the term "hominin" has come to replace the earlier term "hominid" as a category including living human beings and those fossil ancestors showing some degree of bipedal locomotion; see Wood and Lonergan 2008 for terminology and taxonomy).

GROWTH AND EVOLUTION

If there is a "secret" to life, it is hidden in the process that converts a single cell, with its complement of deoxyribonucleic acid (**DNA**) into a multicellular organism composed of hundreds of different tissues, organs, behavioral capabilities, and emotions. That process is no less wondrous when it occurs in an earthworm, a whale, or a human being. Because this book is about human biology, in this chapter, we focus on the process of human growth and development; however, the reader must be aware that much of what we know about human growth is derived from research on nonhuman animals, in part due to the ethical limits on the kind of experimental research that may be performed on human beings.

Powerful genomic evidence for the common evolutionary origin of animal development came in 1984 with the discovery of the genomic homeobox (McGinnis

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et al. 1984). The most common definition of a homeobox is a highly conserved sequence of about 180 DNA **base** pairs that code for a 60-**amino-acid** segment of **protein** that regulates patterns of development. **Genes** containing homeoboxes are found in all eukaryotic **genomes** and are associated with cell differentiation and bodily segmentation during embryologic development. Additional evidence for common evolutionary origins was published in 1995 with the identification of PAX6 as a master control gene for eye development (Halder et al. 1995; Callaerts et al. 1997; Gehring 1998) in virtually all organisms that possess one or more eyes. This discovery led to a new hypothesis about the monophyletic origin of the eye in evolution. The PAX6 eye gene is common to species as diverse as marine worms, squid, fruit flies, mice, and humans. In contrast, the unique features of the life cycle of different species, such as metamorphosis in insects and amphibians, or childhood in human beings, attest to the ongoing evolution of life on Earth.

Biological evolution is the continuous process of genomic–phenotypic adaptation of organisms to their environments. Natural selection determines the direction of evolutionary change and operates by **differential mortality** between individual organisms prior to reproductive maturation and by **differential fertility** of mature organisms. Thus, genetic/genomic and **phenotypic** adaptations that enhance the survival of individuals to reproductive age and that increase the production of similarly successful offspring will increase in frequency in a population.

BASIC PRINCIPLES OF HUMAN GROWTH AND DEVELOPMENT

Before we explore the evolution of the human life cycle, let us review some of the basic principles of human growth and development. Human beings, like most animals, begin life as a single cell, the fertilized ovum, via sexual reproduction (see the section on "**Epigenetics**" in this chapter for exceptions). In normal human conception and development, the genetic information provided by each parent, the phenotypic environment of the mother's ovum, and the biocultural environment in which the mother lives interact in complex ways to guide the fertilized ovum to divide, grow, differentiate, and develop through many stages, including embryo and fetus, prior to birth.

Although growth and development may occur simultaneously, they are distinct biological processes. **Growth** may be defined as a quantitative increase in size or mass. Measurements of height or weight indicate how much growth has taken place in a child. Additionally, the growth of a body organ, such as the liver or the brain, may also be described by measuring the number, weight, or size of cells present. **Development** is defined as a progression of changes, either quantitative or qualitative, that lead from an undifferentiated or immature state to a highly organized, specialized, and mature state. **Maturity** is measured by functional capacity. An example is the development of motor skills from crawling to toddling in an infant, to mature human walking in a juvenile. These different levels of maturity in skills of locomotion are dependent on the development of the skeletal, muscle, and nervous systems and their integration. Even though these definitions are broad, they allow us to consider the growth, development, and maturation of organs (e.g., kidneys), systems (e.g., the reproductive system), and the person.

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STAGES IN THE LIFE CYCLE

The life cycle of an organism includes stages of growth, development, and maturation from conception to death. Many of the basic principles of human growth, development, and maturation are best presented in terms of the events that take place during the life cycle. One of the many possible orderings of events is given in Table 11.1, in which growth periods are divided into developmentally functional stages. This is only one possible ordering, because declaring that one moment (e.g., **fertilization**) is the beginning of life is arbitrary in a continuous cycle that passes through fixed stages in each individual person and in generation after generation.

Prenatal Development

The course of pregnancy may be divided into three periods, or **trimesters**. During the first trimester, one of the major events is the multiplication of a single cell, the fertilized ovum, into tens of thousands of new cells. At first, cell division may produce exact copies of the original parent cell. However, within hours of the first division, distinct groups of cells begin to form. The rate of cell division in the separate groups is unequal; these cells have begun to differentiate and will eventually form different kinds of tissue (the "germ layers" of endoderm, mesoderm, and ectoderm) that will constitute the growing embryo. Growth, an increase in cell number, and development (in this case, cellular differentiation) begin almost simultaneously with conception.

Although the human body is composed of dozens of kinds of tissues and organs, their generation and growth during prenatal life, and postnatal life as well, take place through a few ubiquitous processes. Goss (1964, 1978) described two types of cellular growth: **hyperplasia** and **hypertrophy**. Hyperplasia involves cell division by **mitosis**. For instance, epidermal cells of the skin form by the mitotic division of germinative cells, also called stem cells, in the deep layers of the skin. Hypertrophic growth involves the enlargement of already existing cells, as in the case of adipose cells growing by incorporating more lipid (fat) within their cell membranes.

Goss also described three strategies of growth used by different tissues: renewal, expansion, and stasis. **Renewing tissues** include blood cells, **gametes** (sperm and egg cells), and the **epidermis**. Mature cells of renewable tissue are incapable of mitosis and have relatively short lives; for instance, red blood cells (erythrocytes) survive in circulation for ~6 months. New erythrocytes are produced from a reserve of undifferentiated cells located in the bone marrow and lymphatic organs. The rate of replacement of renewing tissues is carefully balanced against the rate of death of the mature cells. **Expanding tissues** include the liver, kidney, and the endocrine glands, the cells of which retain their mitotic potential even in the differentiated state. The liver, for example, has no special germinative layer or compartment, and most liver cells are capable of hyperplasia to replace damaged cells (possibly the source of the Greek myth of Prometheus). **Static tissues**, such as nerve cells and striated muscle, give up the ability to divide by mitosis early in their lives. Once formed, a static tissue can grow in size by hypertrophy but not by hyperplasia. Thus, when an adult person exercises a muscle, its size may increase by enlarging the individual muscle cells already present, not by adding new cells. Also, because the reserve of germinative cells is limited and is usually depleted early in life, the pool

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TABLE 11.1 Stages in the Human Life Cycle and Life History

Stage	Growth Events/Duration (Approximate or Average)
Prenatal development	
Fertilization	
First trimester	Fertilization to 12th week: embryogenesis
Second trimester	Fourth through sixth lunar month: rapid growth in length
Third trimester	Seventh lunar month to birth: rapid growth in weight and organ maturation
Birth	
Postnatal development	
Neonatal period	Birth to 28 days: extrauterine adaptation, most rapid of postnatal growth and maturation
Infancy	Second month to end of lactation, usually by 36 months: rapid growth velocity, but with steep deceleration in growth rate, feeding by lactation to age 6 months and then lactation with gradual introduction of complimentary foods; deciduous tooth eruption; many developmental milestones in physiology, behavior, and cognition
Childhood	Years 3.0–6.9: Moderate growth rate, dependency on older people for care and feeding, midgrowth spurt, eruption of first permanent molar and incisor, virtual completion of brain growth by the end of the stage
Juvenile	Years 7–10 for girls, 7–12 for boys: slower growth rate, capable of self-feeding, cognitive transition leading to learning of economic and social skills
Puberty	An event of short duration (days or a few weeks) at the end of the juvenile stage: reactivation in the hypothalamus of the GnRH pulse generator, dramatic increase in secretion of sex hormones from the ovaries/testes
Adolescence	The stage of development that lasts for 5–10 years after the onset of puberty: growth spurt in height and weight; permanent tooth eruption almost complete; development of secondary sexual characteristics; sociosexual maturation; intensification of interest in and practice of adult social, economic, and sexual activities
Adulthood	
Prime and transition	From age 18–20 years for women to 45 years (end of childbearing) and from age 21–25 years for men to about age 55 years: commences with completion of skeletal growth, homeostasis in physiology, behavior, and cognition; loss of fecundity and menopause for women by age 50, male fecundity may decline with age, but does not drop to zero at any age
Old age and senescence	From end of childbearing years to death: decline in the function and repair ability of many body tissues or systems
Death	

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of static tissues cannot be renewed if damaged or destroyed. The destruction of the central nervous system (CNS) tissue, such as regions of the brain as a result of accident or stroke, often means the permanent loss of the damaged cells and the functions they once performed (although there is evidence that some CNS tissue is renewable or can be induced to renew; Lin et al. 2006). Perhaps as compensation for lack of mitotic ability, many static tissues may live as long as the person survives, unlike renewable tissues that tend to have short lives.

The biological substrate of the individual is not permanent. From embryonic life through adulthood, the human body is in a constant state of decomposition and reorganization. Young adult men renew about 2–3% of their muscle mass each day. In infancy, when new muscle tissue is forming by hyperplasia, the rate of protein renewal is about 6–9% per day. The magnitude of this metabolic renewal may be appreciated by the fact that much of the **basal metabolic rate** of the body (which may be measured by the heat that the body produces when at complete rest) is due to protein turnover. A similar turnover of cellular material occurs in other static tissues, such as nerve cells in the body and in the brain, and in expanding tissues. Tanner (1990, pp. 25–26) wrote,

> This dynamic state enables us to adapt to a continuously changing environment, which presents now an excess of one type of food, now an excess of another; which demands different levels of activity at different times; and which is apt to damage the organism. But we pay in terms of the energy we must take in to keep the turnover running. Enough food must be taken in to provide this energy, or the organism begins to break up.

During the years and decades of life, the turnover and renewal of the molecular constituents of a human being's cells must take place often enough to recreate the entire body many times over.

The metabolic dynamic of the human organism is most active during the first trimester of prenatal life. The multiplication of millions of cells from the fertilized ovum—and the differentiation of these cells into hundreds of different body parts—makes this earliest period of life highly susceptible to growth pathology caused by either the inheritance of genetic **mutations** or exposure to harmful environmental agents that disrupt the normal course of development (e.g., certain drugs, smoking, **malnutrition**, disease, and psychological trauma that the mother may experience). Due to these causes and others, one study estimated that ~10% of human fertilizations fail to implant in the wall of the uterus, and of those that do, ~50% are spontaneously aborted (Werner et al. 1971; see also Chapter 15 of this book by Ellison et al. on "Pregnancy Loss and Female Fecundity"). It is consoling to know, perhaps, that most of these spontaneous abortions occur so early in pregnancy that the mother is not aware that a conception took place.

After the initial embryonic tissues are formed, the first trimester is taken up with organogenesis, the formation of organs and physiological systems of the body. By the eighth week, the embryo is recognizably human. By the start of the second trimester of pregnancy, the differentiation of cells into tissues and organs is complete, and the embryo has become a fetus. During the next 12 or so weeks, most of the growth that takes place in the fetus is in length. At 8 weeks after conception, the embryo is ~30 mm long (~1 in.). By the fourth month, the length from the crown of the head to the bottom of the buttocks (crown–rump length) is ~205 mm (8 in.),

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and by the sixth month, between 356 and 381 mm (~14 in.), which is ~70% of average birth length. An educational web site on human prenatal development, maintained by Dr. Mark Hill, found at http://php.med.unsw.edu.au/embryology/index.php?title=Main_Page, has useful illustrations and animations.

Increases in weight during this same period are much less rapid. Eight weeks after conception, the embryo weighs 2.0–2.7 g (0.08 oz.), and by the sixth month, the fetus weighs only 700 g (1.5 lb), which is ~20% of birth weight. It is during the third trimester of pregnancy that growth in weight takes place at a relatively faster rate. During the third trimester, several physiological systems (e.g., circulatory, respiratory, and digestive) also develop and mature, preparing the fetus for the transition to extrauterine life after birth.

The prenatal stage of the mammalian life cycle encompasses greater amounts of growth, development, and maturation than any other stage. The prenatal gestation of all the apes, including the human ape, takes a long period of time compared with nonprimate mammals, particularly long for our body size. Compare the average gestation length in days for humans = 266, chimpanzees = 240, gorillas = 240, and the orangutan = 260 with, for example, the African lion = 110, which is less than half that of the apes. Female lions have an adult body mass of about 150 kg, which is more than twice the 68 kg average of female body weight for humans and chimpanzees. Even the longest gestation in mammals, the African elephant = 640 days, is not extremely long given the immense size of a female African elephant, about 5000 kg. In proportion to adult female body weight, human and chimpanzee gestation is about five times longer than that of lions (chimpanzee, 240/68 = 3.5; lion 110/150 = 0.73), and about 27 times longer than that of elephants (640/5000 = 0.13).

Indeed, whether we use simple ratios or more complex equations, it has been well demonstrated that the mother's body weight is secondary to the offspring's brain weight in explaining the length of gestation in broad comparisons across mammals (Sacher and Staffeldt 1974). And Primates, as an order of mammals, have both relatively large brains and long gestations. This reaches its extreme in the apes, but it is still not clear if the large brain size of the apes is one of the consequences, or perhaps the cause, of long gestation (Leigh 2004). Prenatal life is, in fact, the most critical period for the formation of most of the body's anatomical and physiological systems. **Critical periods** in developmental biology are times during the life cycle when one or more properties of the organism must grow or develop or when this property develops most rapidly (Cameron and Demerath 2002). Due to the restricted time frame for growth or development, and the velocity of development, these critical periods are usually highly susceptible to alteration by the environment. A well-known example of a critical period is imprinting in birds, which for greylag geese is the 13–16 hours after hatching when the chick will become programmed to follow the first moving object seen, usually its parents (Lorenz 1935). Human prenatal life has many critical periods, for example, maternal exposure to "German measles" (rubella) in the first 3 months of pregnancy is associated with a high risk of birth defects.

Developmental Origins of Health and Disease A very active area of research related to critical periods is the developmental origin of health and disease (Developmental Origins of Health and Disease [DOHaD], http://www.dohadsoc.org/).

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As early as 1927, V.P.A. Derrick, A.R. Davidson, and A.R. Reid found that adult mortality in England and Wales depended on years of birth (see Smith and Kuh 2001). A few years later, Kermack et al. (1934) confirmed the association between year of birth and rates of adult mortality for England, Scotland, and Sweden. Even more to the point, Kermack et al. found that "infantile mortality is dependent in large measure on improvement in maternal health (2001 reprint, p. 683). Kermack and colleagues also suggested that the environmental condition up to age 15 years was key to later health and mortality risk. By implication, improvements in maternal health would have to take place before age 15 of the mother to have an impact **infant mortality** a generation later. Fifty-two years later, Emanuel (1986) formalized these findings into the "intergenerational effects hypothesis" [IEH], defined as "those factors, conditions, exposures and environments experienced by one generation that relate to the health, growth and development of the next generation." Working with British data, Emanuel et al. (1992) found that the birth weight of a woman, her health history during infancy and childhood, and her adult stature (which reflects the total history of her growth and development) are strong predictors of the birth weight of her offspring. Many human **epidemiological** and anthropological studies support the IEH (Drake and Walker 2004; Varela-Silva et al. 2009) and experimental studies confirm the power of intergenerational effects (Drake et al. 2007; Benyshek et al. 2008).

Other DOHaD research focuses on the "fetal origins" hypothesis (Barker 1990; Benyshek 2007; Kuzawa 2007; Gluckman et al. 2009) to explain the origins of several adult chronic illnesses such as coronary heart disease and diabetes. As stated by Kuzawa (2004, p. 194), "The fetal origins hypothesis proposes that intrauterine nutrition influences the development of various hormonal systems and organs, with lasting effects on adult risk for cardiovascular disease." The hypothesis has been expanded to other adult diseases, such as diabetes and obesity. Postnatal environments during infancy and childhood are also proving to be important for the development of health or the risks for disease in later life (Bailey and Schell 2007; Bogin and Varela-Silva 2010; McDade et al. 2010). Human biologists and biological anthropologists are at the forefront of this research, due to their cross-cultural and evolutionary perspectives.

Epigenetics Interactions between the environment and genome of the mother and her embryo/fetus are now known to influence both the risks for disease (Kuzawa and Sweet 2009) and the normal range of growth, including development of adipose tissue (Mostyn and Symonds 2009) and muscle tissue (Du et al. 2010). Epigenetics refers to changes in gene expression caused by mechanisms other than changes in the underlying DNA sequence. Epigenetic mechanisms such as DNA methylation, histone acetylation, and micro **RNA** interference (Fig. 11.2) can affect gene activation and inactivation; methylation, for example, inactivates or represses gene expression. Epigenetic mechanisms may be activated by exposure to temperature extremes, exposure to disease, excess or lack of dietary factors, and many behavioral practices including physical activity, smoking, and alcohol consumption. Segars and Aagaard-Tillery (2009, p. 349) wrote that epigenetic mechanisms "are increasingly understood to have a profound effect in altering an individual's appearance, transmission of a specific **congenital** abnormality ('birth defect'), and even one's lifetime risk of common diseases such as obesity and cancer."

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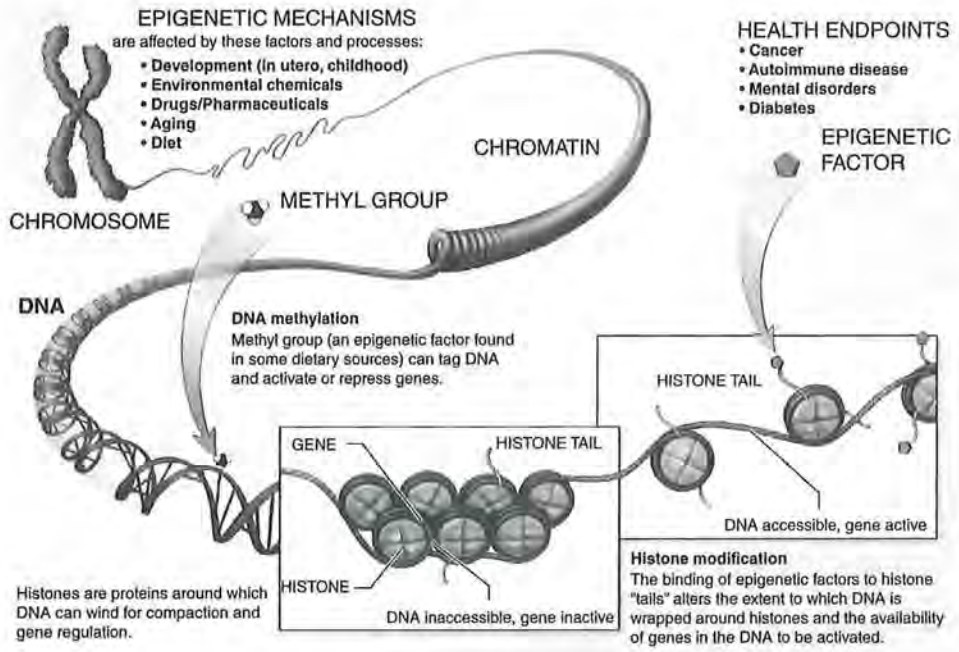

Figure 11.2 Two epigenetic mechanisms: DNA methylation and histone acetylation (Image: http://nihroadmap.nih.gov/EPIGENOMICS/epigeneticmechanisms.asp). See color insert.

Epigenetic expression in the phenotype may be a heritable change in biology or behavior, but a change that does not alter DNA sequence. In this sense, epigenetic biology is a departure from the traditional genetic dogma of

$$\text{DNA} \rightarrow \text{amino acid} \rightarrow \text{polypeptide chain} \rightarrow \text{protein.}$$

The flow of information in epigenetic biology may begin with a social factor, such as the decision of families to migrate from a poorer country to a richer country or the choice by a woman to deliver an infant via Caesarian section (C-section). In the first case, second-generation Bangladeshi women, that is, the daughters of women who migrated to the United Kingdom, have higher levels of salivary **progesterone** and higher ovarian function than first-generation migrants. The difference in progesterone level is in part due to greater methylation of the progesterone receptor protein in the first-generation migrants, who grew up in Bangladesh (O'Connor et al. 2009). Why this is so, is not known, but a consequence of elevated progesterone levels is greater risk for breast cancer in the second-generation women (Nuñez-De La Mora et al. 2008). In the second case, women giving birth by C-section deliver infants with greater DNA methylation in general (Schlinzig et al. 2009). Why this is so, and how it influences later health is unknown, but infants delivered by C-section have an, "increased risk for allergy, diabetes and leukaemia" (Schlinzig et al. 2009, p. 1096). In a more general sense, the traditional "gene-to-protein" dogma is changing to a perspective of greater environmental control of genomic programming and DNA expression. Several nutrients, such as vitamins A, C, niacin, and D are known

to regulate DNA activity and be related to diseases such as diabetes, **atherosclerosis**, and cancer (Kato et al. 2007; McGrane 2007). A socioeconomic factor such as poverty can influence the availability of vitamin D due to limited food choices. Vitamin D is found in a small number of foods, such as expensive oily fish, for example, salmon, in liver, and in eggs, which poor families may not eat on a regular basis. Low **socioeconomic status** (SES) may also lead to a lack of exposure to sunlight due to the need to work at low-paid indoor jobs. Humans get most of their vitamin D from sunlight (**ultraviolet radiation**) striking the skin and converting **cholesterol**-based substances into precursors of vitamin D (see Chapter 6).

In this case, the flow of epigenetic information is as follows:

Social–economic–political forces producing poverty
→ inability to purchase vitamin-D-containing foods / low sunlight exposure
→ low bioavailability of vitamin D → low transactivation of DNA expression
→ low amino acid production → insufficient protein → possible harm to health.

An important human example is risk for the disease multiple sclerosis (MS). Many studies show that people living at northern latitudes, with low exposure to sunlight, low vitamin D intake, and with a specific genetic variant of the major histocompatibility complex (MHC) on **chromosome** 6, are at greater risk to MS (Ramagopalan et al. 2009). People with the same MHC genetic variant but with adequate vitamin D bioavailability have significantly lower risk for MS.

Other nutrients such as methionine and vitamins B_6, B_{12}, and folate are known to be related to DNA methylation, and the availability of these nutrients during fetal development may influence susceptibility to complex diseases, such as diabetes and obesity (Chmurzynska 2010). Via this nutrient route, there is a connection between intergenerational effects, developmental origins, and epigenetic events.

Another epigenetic mechanism is genomic imprinting, a process that restricts gene expression to only the **allele** inherited from the mother or the father (also called parental imprinting). Earlier in this chapter, we discussed the sexual nature of most animal reproduction. Among vertebrates, some fish and a few reptiles may reproduce via parthenogenesis, the development of an unfertilized ovum into a sexually mature adult female. In nature, successful parthenogenesis is prevented in mammals, probably via the process of genomic imprinting. Most genes are nonimprinted and have biallelic expression (Lobo 2008). Healthy mammalian development seems to require allele expression from both the maternal and paternal alleles, although a parthenogenic mouse has been produced in the laboratory (Kono 2006).

Some human examples of the deleterious effects of genomic imprinting are the congenital conditions Prader–Willi and Angelman syndromes. Remarkably, both are metabolic disorders due to the same DNA deletions in chromosome 15 (Knoll et al. 1989). But, whereas the features of the Prader–Willi syndrome are short stature, mental retardation, poor muscle tone, hyperphagia (overeating), and obesity, those of Angleman syndrome are developmental delay, lack of speech, seizures, and walking and balance disorders (http://www.angelman.org). The phenotypic expression of these two disorders comes from parental imprinting: The paternal alleles produces the Prader–Willi syndrome, and the maternal allele produces the Angelman syndrome. It is important to understand that the phenotypes of these

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two syndromes arise from the same DNA sequence, modified by two different epigenetic pathways of gene expression. More detail on these syndromes and other aspects of human epigenetics may be found at http://embryology.med.unsw.edu.au/MolDev/epigenetic.htm, an educational web site on the epigenetics of human development.

Birth

Birth is a critical transition between life in utero and life independent of the support systems provided by the uterine environment. The **neonate** moves from a fluid to a gaseous environment, from a nearly constant external temperature to one with potentially great volatility. The newborn is also removed from a supply of oxygen and nutrients (provided by the mother's blood and passed through the placenta, which also handles the elimination of fetal waste products) to reliance on his or her own systems for digestion, respiration, and elimination.

The difficulty of the birth transition is illustrated by data presented in Figure 11.3. As may be seen, in the year 1950 in the United States, nearly one-half of all neonatal deaths occurred during the first 24 hours after birth. Of course, most of these deaths are not attributable to the birth process itself; rather, the leading factor associated with neonatal death is **low birth weight** (LBW, defined as a weight < 2500 g [5.5 lb] for a full-term birth). LBW is the result of growth retardation during fetal life. The cause of this growth retardation may be congenital (hereditary or inborn) problems with the fetus, placental insufficiency, maternal **undernutrition**, disease, smoking, alcohol consumption, exposure to environmental toxins, or other causes.

An index of relative mortality during the neonatal period by birth weight is given in Figure 11.4. Relative mortality is defined as the percentage of deaths in excess of the number that occur for infants within the normal birth weight range of 3.0–4.5 kg

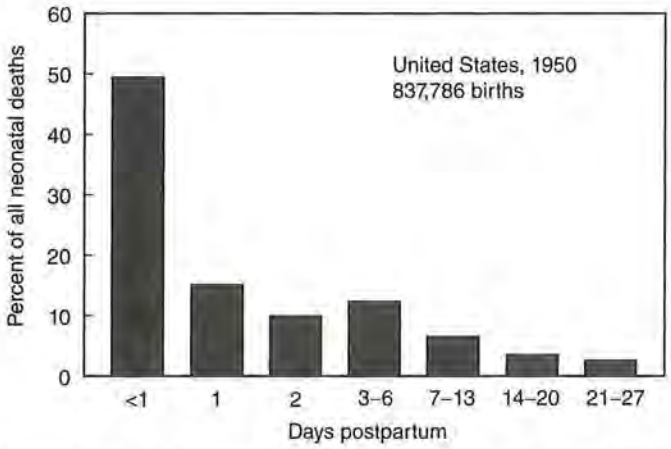

Figure 11.3 Percent of deaths occurring during the neonatal period (birth to day 28). Data from the United States, all registered births for 1950. These data are presented in lieu of more recent data because the technology for extraordinary neonatal medical care in existence today reduces neonatal deaths.

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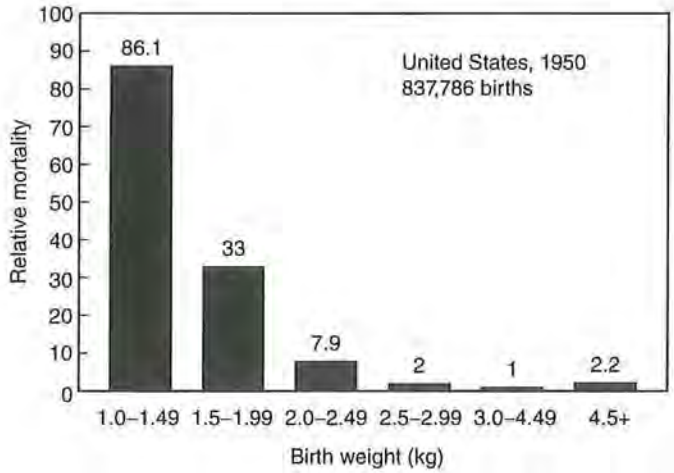

Figure 11.4 Index of relative mortality by birth weight during the neonatal period. Data from the United States, all registered births for 1950. Relative mortality is calculated as the risk of death for an infant born at a given weight. Infants of normal birth weight, 3.0–4.5 kg (6.5–10 lb), have a relative mortality of 1.0. Infants born at 2.5–3.0 kg (5.5–6.5 lb) have twice the risk of death as infants with normal birth weights and so on. Data for the year 1950 are displayed here because the mortality rates for more recent data may be influenced by medical technologies that can sustain the life of newborns with conditions that are eventually lethal.

(6.5–10 lb). These data are for infants at all gestational ages. **Prematurity**, defined as birth before 37 weeks' gestation (full term is 40 weeks), may cause additional complications that increase the chances of neonatal death. However, an infant small for gestational age—that is, of LBW—is usually at greater risk of death than a premature infant of the expected weight for gestational age.

There is also a strong relationship between LBW, with or without prematurity, and SES. SES is a concept devised by the social sciences to measure some aspects of education, occupation, and social prestige of a person or social group. In the year 2000, the incidence of LBW in the wealthy, higher-SES, developed nations was 7% of all live births; in the poor, lower-SES, developing nations, the incidence was 19%, but as high as 27% in South–Central Asia (UNICEF 2004). Even in the United States, the socioeconomic relationship with birth weight is strong. When educational attainment is used to estimate general SES, researchers found that 10.1% of births to women with less than 12 years of schooling had LBW, compared with 6.8% of births to women with 12 years and 5.5% to those with 13 or more years of formal education (Taffel 1980). African-Americans show consistently lower average birth weights and a greater percentage of LBW full-term newborns compared with European-Americans. In the year 2005, the mean birth weight and percent of LBW for U.S. blacks was 3105 g and 11.9%. For U.S. whites, these statistics were 3364 g and 5.32% (Jasienska 2009). Part of this difference is due to the lower SES of the blacks. However, when white and black women are matched for age, education, and SES, black women still give birth to a higher percentage of LBW infants.

Some researchers suggest that ethnic or genetic factors determine the mean difference in birth weight. However, evidence shows that the lower birth weight of the

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black infants is more likely due to the effects of slavery and several generations of discrimination in the postslavery period. Emanuel et al. (1992, 2004) and Jasienska (2009) made use of the IEH, which we discussed above. The researchers explained that SES matching for the current generation of adult black and white women ignores the social and biological history of the United States, Great Britain, and other nations that practiced slavery. The mothers and grandmothers of these black and white women were less likely to be equally matched for SES. The IEH predicts that the poor growth and development of women from older generations will have a lasting effect on the current generation.

Experimental research with rats shows that protein deficiency in the first generation has harmful effects on **glucose** metabolism for at least the following two generations, even when the daughters and granddaughters are adequately fed (Benyshek et al. 2006). Closer to home in an evolutionary sense, Price and Coe (2000) analyzed the birth weight of rhesus monkeys descended from small-for-date (i.e., weight relative to gestation length) and large-for-date birth weight matrilines. Newborn males and females descended from the large-for-date mothers showed greater than normal birth weight. Females, but not males, descended from small-for-date mothers had lower than average birth weight. Price and Coe also found that the lower birth weight female newborns "were at higher risk of poor reproductive outcomes in adulthood, and they perpetuated the matrilineal birth weight pattern by selectively constraining the fetal development of their daughters" (p. 452). From these results, they conclude that (1) "female family members overwhelmingly account for the intergenerational transmission of birth weight in monkeys" (p. 456), and (2) "one pregnancy should not be viewed as an independent event, but as a manifestation of the reproductive health of the female's lineage overall" (p. 456). These conclusions for monkeys are also found for human beings in many studies (Jasienska 2009).

It is clear that birth weight is influenced by many factors. Several lines of research estimate the statistical **variance** in birth weight due to fetal genotype to be 10–31%, the variance due to maternal genotype to be 22–24%, and the variance due to nongenetic maternal and environmental factors to be 47–66% of the total variance (Robson 1978; Lunde et al. 2007). Because of the predominance of nongenetic factors, public health workers often use birth weight statistics as one indication of the well-being of a population.

Weight at birth is only one measurement that is commonly taken to indicate the amount of growth that took place during prenatal life. **Recumbent length** (length of the body when lying down); circumference of the head, arm, and chest; and **skinfolds** are other measurements. Circumferences measure the contribution made by various tissues to the size of different body parts. For example, head circumference measures the maximum girth of the skull, which includes bone and, more importantly, the size of the brain. Some representative data on size at birth and at 18 years of age for several measures are given in Table 11.2. At birth, **sexual dimorphism** (a difference in appearance or behavior between males and females) in size is biologically insignificant. At 18 years of age, however, there is considerable dimorphism between the sexes in average stature, body weight, and fatness as measured by **triceps** and **subscapular** skinfolds.

From birth to adulthood, humans experience many changes in body size, shape, composition (e.g., fat, muscle, and water content), proportions, and skeletal formation (Figs. 11.5 and 11.6). The importance of studying these contrasts between early

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TABLE 11.2 Size at Birth and at Age 18 Years for Children Born in the United States

	Birth		18 Years	
	Boys	Girls	Boys	Girls
Recumbent length/stature (cm)	49.9	49.3	176.6	163.1
Weight (kg)	3.4	3.3	71.4	58.3
Head circumference (cm)	34.8	34.1	55.9	54.9
Triceps skinfold (mm)	3.8	4.1	8.5	17.5
Subscapular skinfold (mm)	3.5	3.8	10.0	12.0
Arm muscle area (mm^2)	20.4[a]	19.6[a]	75.0	57.2

Data from various sources of nationally representative statistics of the 1960s and 1970s.
[a] Measured at 1 year old.
Source: Arm muscle area data are from Frisancho (2008); other data sources are cited in Bogin (1999).

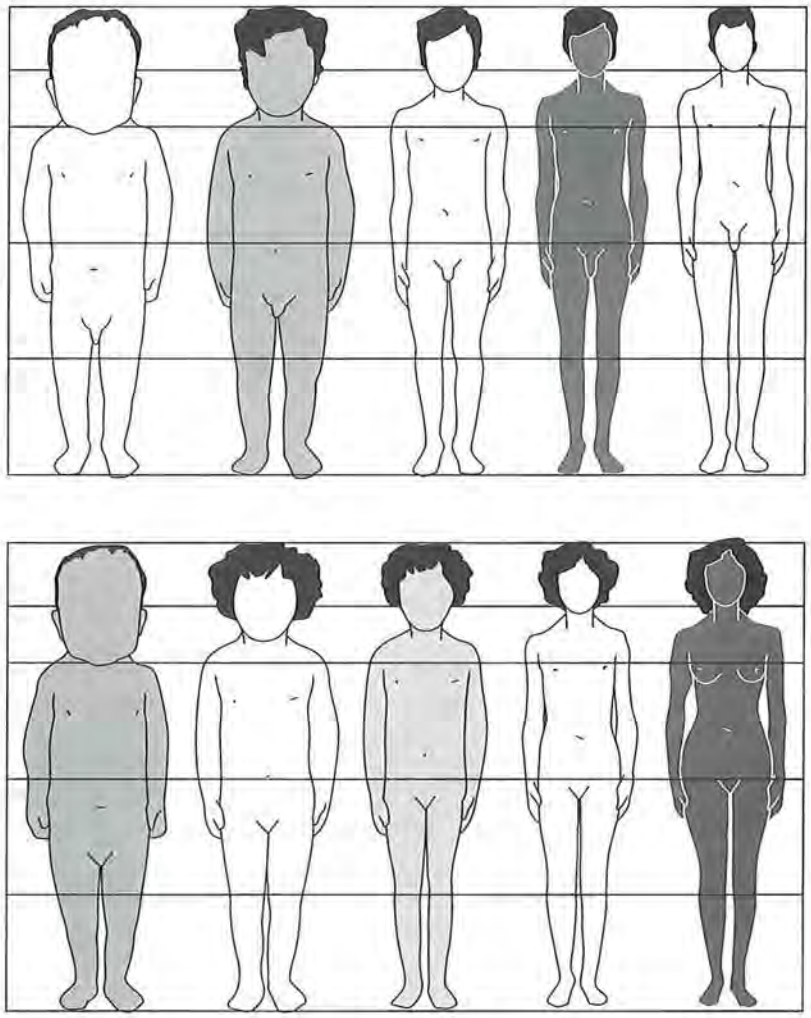

Figure 11.5 Changes in body proportion during human growth after birth. Ages for each profile are, from left to right, newborn, 2 years, 6 years, 12 years, 25 years. Leg length increases relative to total stature relatively more rapidly from birth to age 12 years and then more slowly to adulthood. The hair style and shading of the cartoon silhouettes are for artistic purposes and is not meant to imply any ethnic or eco-geographical phenotypic characteristics of the human species. Courtesy of Dr. J. V. Basmajian.

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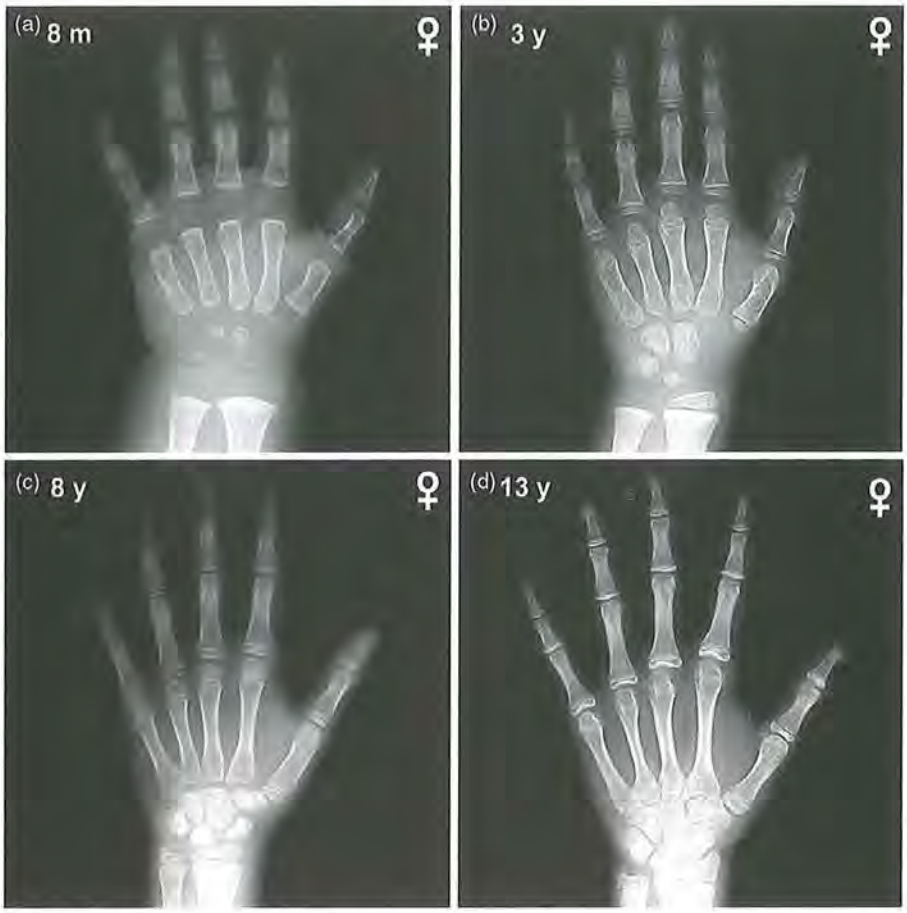

Figure 11.6 Radiographs of the hand and wrist at different skeletal ages, illustrating the sequence of bone maturation events. All radiographs from healthy girls. (a) At 8 months after birth, most wrist bones and the growing ends of the finger bones (called epiphyses) are formed of cartilage. At certain X-ray exposures, this cartilage is "invisible." The image shows few **centers of ossification** (i.e., places where bone is present) in the wrist and few visible epiphyses. (b) At 3 years old, some wrist ossification centers are present, epiphyses of the radius are present, and most epiphyses of the hand are calcified (i.e., forming bone). (c) At 8 years old, all ossification centers are calcified. (d) At 13 years old, all bones have assumed final shape, but growth in size remains to be completed. *Source*: Gilsanz and Ratib (2005), with kind permission from Springer Science + Business Media and the authors.

and later life is twofold. First, they allow clinicians and researchers to assess a child's stage of biological maturation for different organs, tissues, or the body as a whole independent of chronological age. Biological maturation is used to help determine whether a child is developing too slowly or too quickly, either of which may indicate the presence of some disorder. Second, the contrasts between early and later life are also conceptually important. They show that the infant may take one of several different paths for growth, maturation, and functional development.

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Plasticity refers to the ability of an organism to modify its biology or behavior to respond to changes in the environment, particularly stressful changes (Lasker 1969; Frisancho 1993). Adult human morphology, physiology, and behavior are plastic and in no way rigidly predetermined. Of course, people cannot sprout wings or breathe under water, but the sizes, shapes, colors, emotions, and intellectual abilities of people can be significantly altered by environmental stress, training, and experience. When the biology and behavior of people are considered together (i.e., in a biocultural perspective), it seems that human beings are, perhaps, the most plastic of all mammalian species, hence one of the most variable and adaptable.

Postnatal Development

Life after birth can be divided into distinct periods in many ways. In this chapter, as explained earlier, we use a four-stage model of human postnatal growth and development—infant, child, juvenile, adolescent—between birth and adulthood (outlined in Table 11.1). The rationale for this model begins with an analysis of the amount and rate of growth from birth to adulthood (more detailed explanation for the four-stage model is found in Bogin 1999).

To visualize the amount and rate of growth that takes place during each of these stages, the growth in height (or length) for normal boys and girls is depicted in Figure 11.7; growth in weight follows very similar curves. In Figure 11.7, the **distance curve** of growth, that is, the amount of growth achieved from year to year, is labeled on the right *y*-axis. The **velocity curve**, which represents the rate of growth during any 1 year, is labeled on the left *y*-axis. Below the velocity curve are symbols that

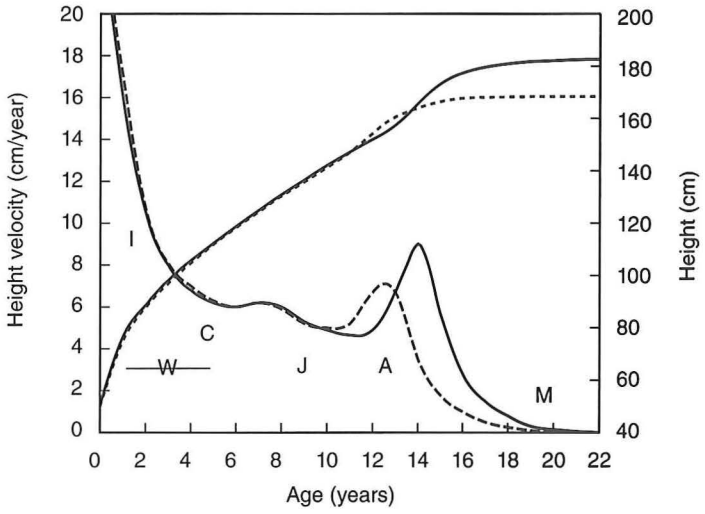

Figure 11.7 Idealized mean velocity and distance curves of growth in height for healthy girls (dashed lines) and boys (solid lines) showing the postnatal stages of the pattern of human growth. Note the spurts in growth rate at mid-childhood and adolescence for both girls and boys. The postnatal stages: I, infancy; C, childhood; J, juvenile; A, adolescence; M, mature adult; –W–, range of age for human weaning. *Source*: Data from Prader (1984) and Bock and Thissen (1980).

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indicate the average duration of each stage of development. Clearly, changes in growth rate are associated with each stage of development. Each stage may also be defined by characteristics of the dentition, changes related to methods of feeding, physical and mental competencies, or maturation of the reproductive system and sexual behavior.

Infancy Infancy is characterized not only by the most rapid velocity of growth of any of the postnatal stages, but also by a steep decline in velocity, a deceleration. The infant's changing growth velocity continues the fetal pattern, in which growth rate in length peaks in the second trimester and then begins a deceleration that lasts until childhood (Fig. 11.8). As for all mammals, human infancy is the period when the mother provides all or some nourishment to her offspring via lactation or some culturally derived imitation of lactation. During infancy, the deciduous dentition (the so-called milk teeth) erupts through the gums. Human infancy ends when the child is weaned from the breast, which in preindustrialized societies occurs between 24 and 36 months of age (Sellen 2006). By this age, all the deciduous teeth have erupted, even for very late maturing infants.

Motor skills (i.e., what a baby can do physically) develop rapidly during infancy. At birth, states of wakefulness and sleep are not sharply differentiated, and motor coordination is variable and transient. By 1 month, the infant can lift its chin when prone, and by 2 months, lift its chest by pushing up with the hands and arms. Developmental milestones for sitting, standing, and walking are shown in Figure 11.9. These motor development milestones are averages for human infants. Some infants may skip a stage, such as "hands-and-knees crawling," all of the stages overlap, and each stage is quite variable in duration. On average, by 3 years of age, the end of infancy, the youngster can run short distances, pour water from a pitcher, and manipulate small objects, such as blocks, well enough to control them. There is a similar progression of changes in the problem solving, or cognitive, abilities of the infant.

Accounting for all of these motor and cognitive advancements is the development of the skeletal, muscular, and nervous systems, especially brain growth and development. The human brain grows rapidly during infancy, much more rapidly than almost any other tissue or organ of the body (Fig. 11.10). All parts of the brain seem to take part in this fast pace of infant growth and maturation, including the **hypothalamus**, a center of neurological and endocrine control (Fig. 11.11). During fetal life and early infancy, the hypothalamus produces relatively high levels of **gonadotropin-releasing hormone** (GnRH). This **hormone** causes the release of **luteinizing hormone** (LH) and **follicle-stimulating hormone** (FSH) from the **pituitary gland**. These hormones, in turn, travel via the bloodstream to their sites of action, which include the developing ovaries or **testes**.

The endocrine system interacts in complex ways with the environment and the genome to direct the course of growth, development, and maturation by release of hormones. In addition, changes in the operation of the endocrine system seem to play a primary role in the evolution of life cycles (Finch and Rose 1995). The operation of the primate hypothalamic system is a case in point. In 1975, Grumbach and colleagues reported that GnRH has an on–off–on pattern of activity during postnatal development in humans. Nonprimate mammals, such as rodents, do not show this pattern; instead, these animals have a progressive and uninterrupted increase

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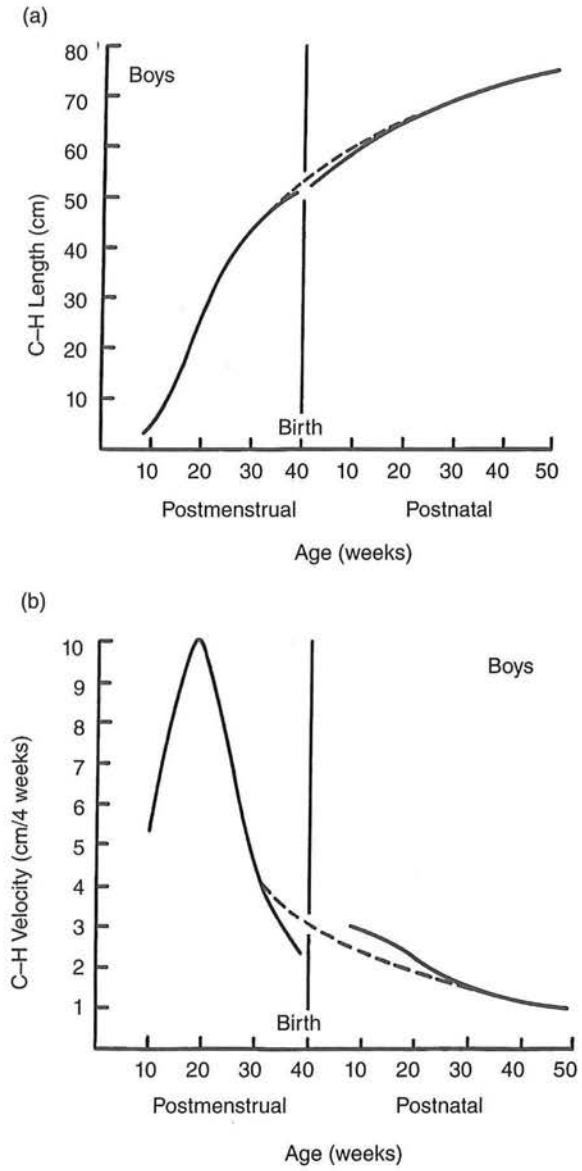

Figure 11.8 Distance (a) and velocity (b) curves for growth in body length during human prenatal and postnatal life. The graphs are diagrammatic, because they are based on several sources of data. Dashed lines depict the predicted curve of growth if no uterine restriction takes place. In fact, such restriction does take place toward the end of pregnancy and may impede the flow of oxygen or nutrients to the fetus. Growth rate slows but rebounds after birth and returns the infant to the size he or she would be without any restriction. C–H, crown–heel. *Source*: Tanner (1990).

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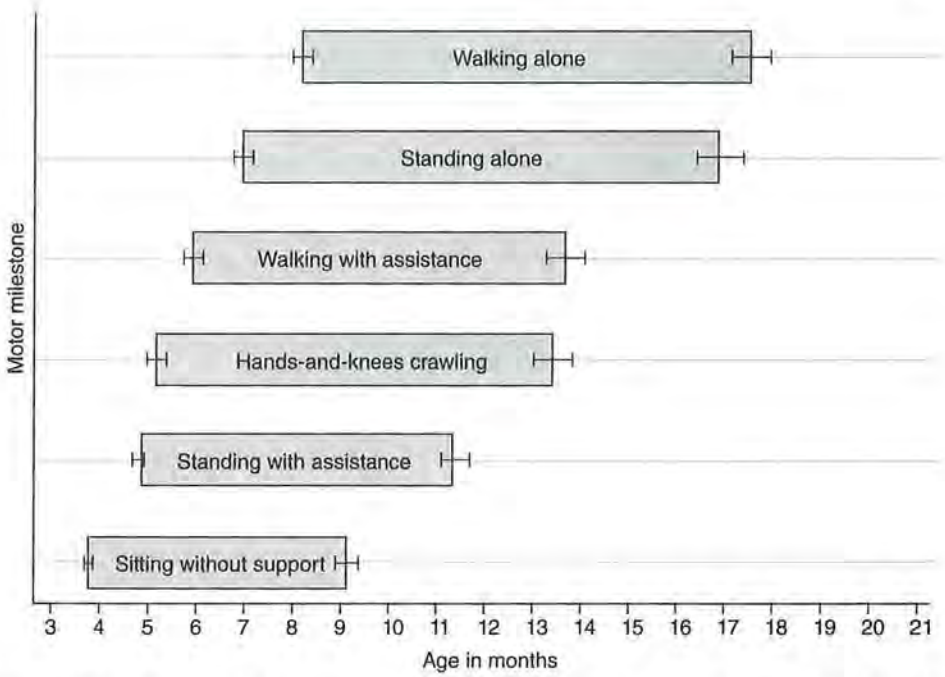

Figure 11.9 Windows of achievement for six gross motor milestones. *Source*: World Health Organization Multicentre Growth Reference Study Group (2006).

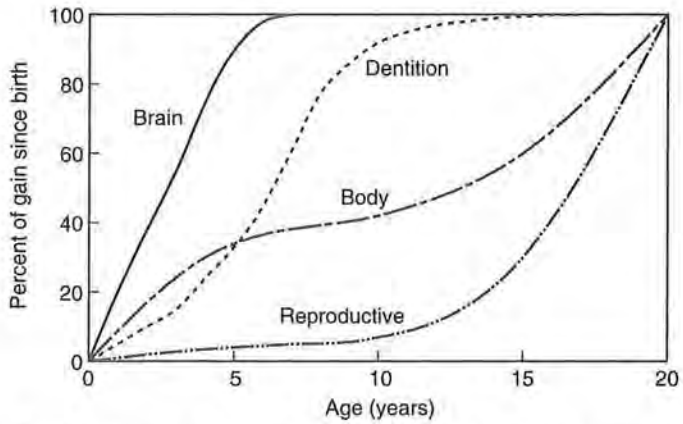

Figure 11.10 Growth curves for different body tissues. The "brain" curve is for the total weight of the brain (Cabana et al. 1993). The "dentition" curve is the median maturity score for girls based on the seven left mandibular teeth (I1, I2, C, PM1, PM2, M1, and M2) (Demirjian 1986). The "body" curve represents growth in stature or total body weight, and the "reproductive" curve represents the weight of the gonads and primary reproductive organs (Scammon 1930).

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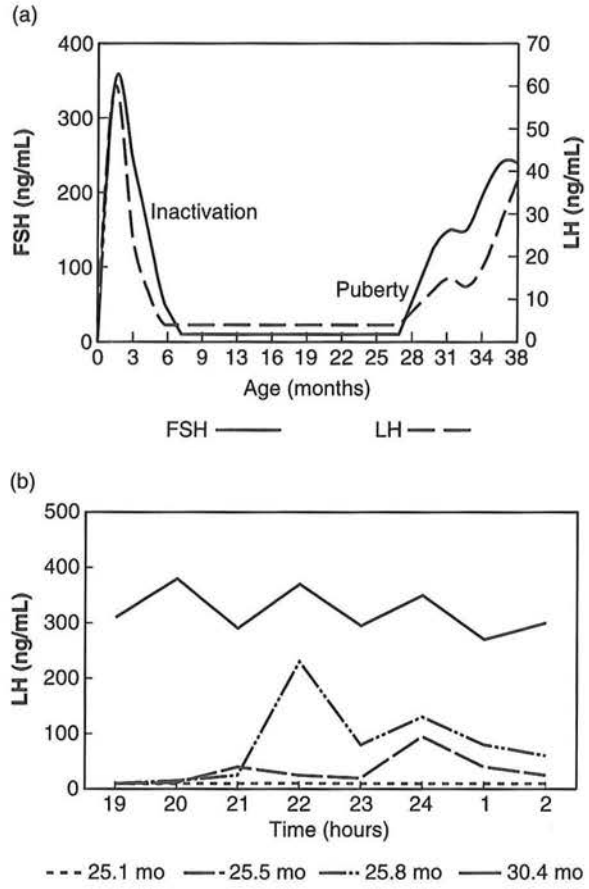

Figure 11.11 (a) Pattern of secretion of FSH and LH in a male rhesus monkey (genus *Macaca*). The testes of the monkey were removed surgically at birth. The curves for FSH and LH indicate the production and release of GnRH from the hypothalamus. After age 3 months (i.e., during infancy), the hypothalamus is inactivated. Puberty takes place at ~27 months, and the hypothalamus is reactivated. (b) Development of hypothalamic release of GnRH during puberty in a male rhesus monkey with testes surgically removed. At 25.1 months (mo) of age, the hypothalamus remains inactivated. At 25.5 and 25.8 months, modest hypothalamic activity is observed, indicating the onset of puberty. By 30.4 months, the adult pattern of LH release is nearly achieved. This pattern shows increases in both the number of pulses of release and the amplitude of release. In human beings, a very similar pattern of infant inactivation and late juvenile reactivation of the hypothalamus takes place. *Source*: Adapted, with some simplification, from Plant (1994).

in GnRH production from birth to sexual maturation. Since 1975, much research has been focused on the mechanisms that control this on–off–on pattern (Fig. 11.11). The current understanding is that one, or perhaps a few, centers of the brain change their pattern of neurological and endocrinological activity, and their influence on the hypothalamus. The hypothalamus becomes, basically, inactive in terms of sexual development by about age 2–3 years. The "inhibitor" has not been identified but

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likely is located in the brain and certainly not in the gonads. Human children born without gonads, as well as rhesus monkeys and other primates whose gonads have been surgically removed at birth, still undergo both GnRH inhibition in infancy and hypothalamus reactivation at age when **puberty** would occur normally (Plant 2008; Plant and Ramaswamy 2009).

It is accepted that the GnRH on–off–on pattern has a regulatory effect on pituitary LH and FSH production and release. In turn, LH and FSH regulate body growth and sexual maturation. Released from the pituitary, LH and FSH travel in the bloodstream to the gonads (ovaries or testes), where they stimulate the production and release of **estrogen** or **androgen** hormones (Fig. 11.12). These gonadal hormones are, in part, responsible for the rapid rate of growth during early infancy. By late infancy, however, the hypothalamus is inhibited. GnRH secretion almost stops, and the levels of the sex hormones fall, which seems to relegate body growth to a steady 5–6 cm/year and suspends reproductive maturation (Fig. 11.10, "reproductive" curve). The hypothalamus is reactivated just before puberty, the event of development that marks the onset of sexual maturation. The age at which this activation takes places varies between primate species, and this variation is part of the control of the transition between stages of the life cycle.

Childhood The childhood stage follows infancy, encompassing the ages of about 3.0–6.9 years. Childhood is a period of postweaning dependency on adults that may also be defined by its own pattern of growth, feeding behavior, and motor and cognitive development. In humans, the rapid growth deceleration of infancy begins to moderate as childhood begins. Growth rate declines from about 7–8 cm/year at age 3 years to ~5–6 cm/year by age 6 years and then levels off. This moderation of growth rate decline and the leveling off in growth rate are unusual for mammals, because almost all other species continue a pattern of relatively rapid deceleration after infancy (look ahead to Figs. 11.18 and 11.19).

This slower and steady rate of growth maintains a relatively small-sized body during the childhood years. In terms of feeding, children are weaned from the breast or bottle but still depend on older people for food and protection. In contrast, most mammalian species move from infancy, and its association with dependence on nursing, to a stage of independent feeding. Some months of postweaning dependency is a characteristic of the social carnivores (such as lions, wild dogs, and hyenas) and a few species of primates, such as marmosets and tamarins (Ewer 1973; Goldizen 1987).

Given this, postweaning dependency is, by itself, not a sufficient criterion to distinguish human childhood. Dental traits, digestive systems, and brains add to the features of human childhood. Human children require specially prepared foods because of the immaturity of their dentition, the small size of their stomachs and intestines, and the rapid growth of their brain (Fig. 11.10). Again, we emphasize that the human brain is especially important. The newborn uses 87% of its **resting metabolic rate** (RMR) energy for brain growth and function. By the age of 5 years, the percent RMR usage is still high at 44%, and higher than any other mammal, whereas in the adult human, the figure is between 20% and 25% of RMR. At comparable stages of development, the chimpanzee devotes about 45%, 20%, and 9%, respectively, of its RMR to brain growth (Leonard and Robertson 1994). Chimpanzee energy needs are lower than those for humans for three reasons: (1) smaller size of

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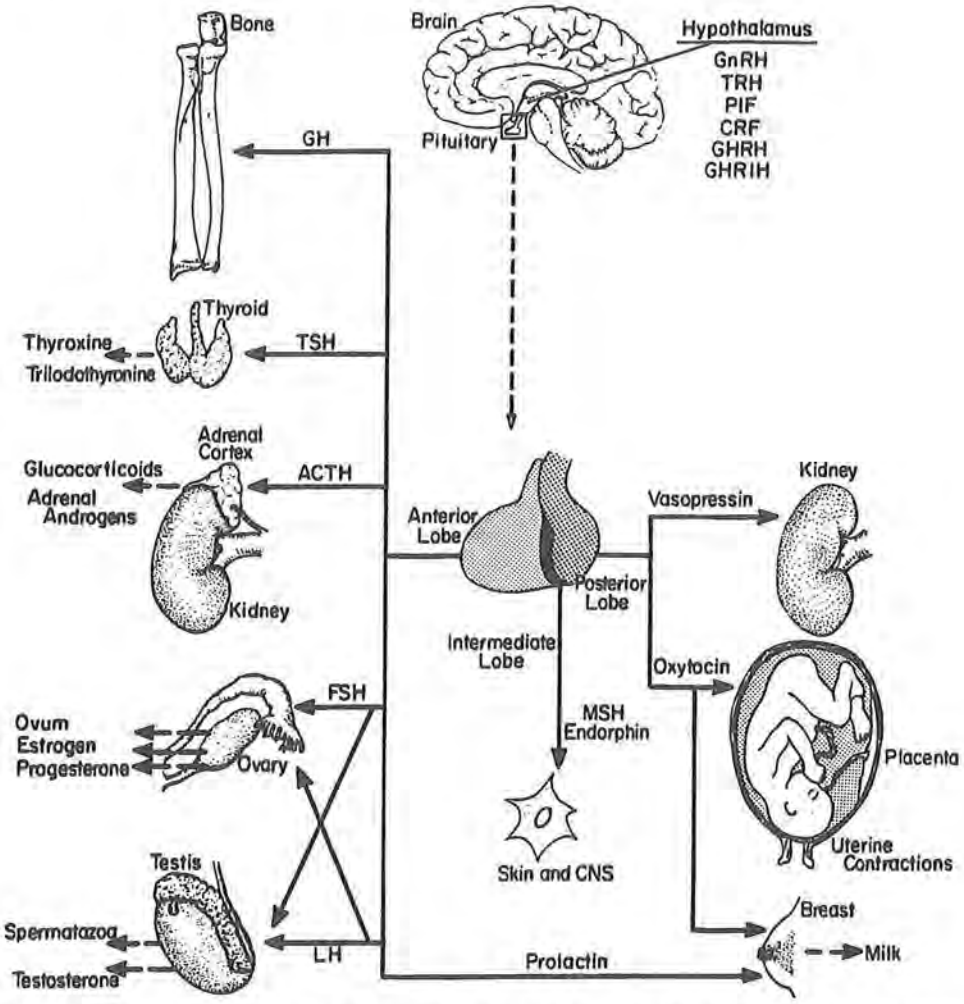

Figure 11.12 Diagram of the location of the hypothalamus and pituitary within the brain, and a schematic illustration of the target organs and tissues of the pituitary hormones. GnRH, gonadotropin-releasing hormone; TRH, thyrotropin-releasing hormone; PIF, prolactin-release inhibiting factor; CRF, adrenocorticotropin-releasing factor; GHRH, growth hormone-releasing hormone; GHRIH, growth hormone release-inhibiting hormone; GH, growth hormone; TSH, thyroid stimulating hormone; ACTH, adrenocorticotropic hormone; FSH, follicle-stimulating hormone; LH, luteinizing hormone; MSH, melanocyte-stimulating hormone. *Source*: Redrawn from Schally et al. (1977).

the brain at all ages and (2) slower rate of growth of the brain after birth, and (3) the brain stops growing about 2 years earlier in chimpanzees than in humans (Leigh 2004; Fig. 11.13).

The constraints of immature dentition and small digestive system mean that human children need a diet that is easy to chew and swallow and low in total volume. The child's relatively large and active brain, almost three times the size of an adult

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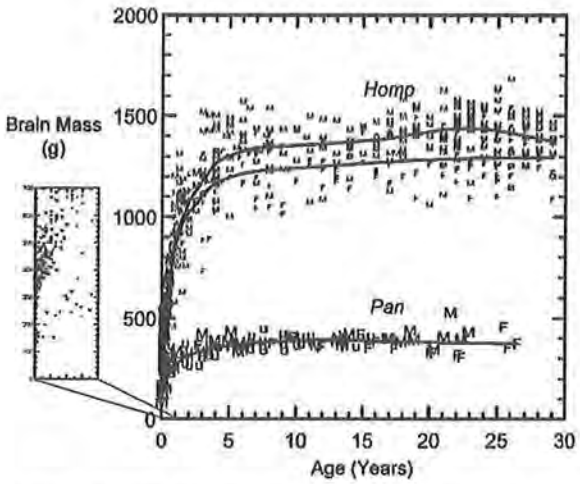

Figure 11.13 Brain-mass growth data for humans (*Homo sapiens*) and chimpanzees (*Pan troglodytes*). Brain mass increases during the postnatal period in both species. Lines represent best-fit Lowess regressions through the data points. M, males; F, females; U, sex unidentified (Vrba 1998). The human regressions separate into male (upper) and female (lower) curves. The inset shows brain-mass growth for each species during the first postnatal year (reproduced from Leigh 2004, with kind permission of the author).

chimpanzee's brain, requires that the low-volume diet be dense in energy, lipids, and proteins. Children do not yet have the motor and cognitive skills to prepare such a diet for themselves. Children are also especially vulnerable to predation and disease and thus require protection. Children will not survive in any society if deprived of the care provided by older individuals. The so-called wolf children and even street children, who are sometimes alleged to have lived on their own, are either myths or not children at all. A search of the literature finds no case of a child (i.e., a youngster under the age of 6 years) living alone, either in the wild or on urban streets (Panter-Brick et al. 1996; Bogin 2006).

Two of the important physical developmental milestones of childhood are the eruption of the first permanent teeth, replacement of the deciduous teeth, and completion of brain growth (in weight). First molar eruption takes place, on average, between the ages of 5.5 and 6.5 years in most human populations. Eruption of the central incisor quickly follows, or sometimes precedes, the eruption of the first molar. By the end of childhood, usually at the age of 7 years, most children have erupted the four first molars, and permanent incisors have begun to replace "milk" incisors. Along with growth in size and strength of the jaws and the muscles for chewing, these new teeth provide sufficient capabilities to eat a diet similar to that of adults.

A close association between human dental development and other aspects of growth and maturation was noted in the early 20th century by anatomists and anthropologists. More recently, Smith (1991) analyzed data from humans and 20 other primate species and found that age of eruption of the first molar is highly associated with brain weight (**correlation** coefficient, $r = 0.98$, where 1.00 is a perfect

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correlation) and a host of other growth and maturation variables. Other research, based on direct measurements of victims of accidents and disease, shows that human brain growth in weight is virtually complete at a mean age of 7.0 years (Cabana et al. 1993; Fig. 11.13). Thus, at this stage of development, not only is the child capable dentally of processing an adult-type diet, but the nutrient requirements for brain growth also diminish. Moreover, cognitive and emotional capacities mature to new levels of self-sufficiency. Language and symbolic thinking skills mature rapidly, social interaction in play and learning become common, and the 7-year-old individual can perform many basic tasks, including food preparation with little or no supervision (Locke and Bogin 2006).

Another feature of the childhood phase of growth associated with these physical and mental changes is the modest acceleration in growth velocity at about 6–8 years, called the **midgrowth spurt** (shown in Fig. 11.7). Some studies note the presence of the midgrowth spurt in the velocity curve of boys but not girls, while other studies find that up to two-thirds of boys and girls have midgrowth spurts. The midgrowth spurt is linked with an endocrine event called **adrenarche**, defined as the postnatal onset of secretion of the androgen hormones dehydroepiandrosterone (DHEA) and DHEA sulfate (DHEA-S) from the **adrenal gland**. Among the primates, these hormones are produced in a novel histological region called the zona reticularis. In humans and chimpanzees, adrenarche occurs between the ages of 6–10 years. In some other primates, such as the rhesus monkey, the upregulation of DHEA and DHEA-S begins perinatally (Bogin and Campbell, http://carta.anthropogeny.org/moca/topics/adrenarche). It is unclear, however, if adrenal androgens produce the midgrowth spurt in height, but these androgens seem to cause the appearance of a small amount of axillary and pubic hair.

Adrenal androgens also seem to regulate the development of body fatness and fat distribution, and in humans, adrenarche has been related to the "adiposity rebound" at the transition between the childhood and juvenile stages of the life cycle. The adiposity rebound describes the increase in the body fatness that takes place between the ages of 5 and 7 years. The adiposity rebound is usually measured as the **body mass index** (BMI), which is calculated as [weight in kilograms/height in square meters]. In many human populations, the BMI is a reasonable proxy measure of body fatness. From late infancy through childhood, there is usually a decline in BMI, but then in late childhood, fatness begins to increase (Cole 2004; Hochberg 2009).

The mechanism controlling adrenarche is not understood because no known hormone appears to cause it. Adrenarche is found only in chimpanzees and humans, and the midgrowth spurt, and perhaps the adiposity rebound, is apparently unique to human beings. The physical changes induced by adrenarche and increased fatness are accompanied by a change in cognitive function, called the "5- to 7-year-old shift" by some psychologists, or the shift from the preoperational to concrete operational stage, using the terminology of Piaget. This shift leads to new learning and work capabilities in the juvenile. Very little is known about the consequences of adrenarche for chimpanzee development. It is proposed that adrenarche and DHEA-S may play a role in hominin evolution in terms of extended brain development and prolonged **life span** compared with other primates (Campbell 2006). These ideas need to be tested scientifically, but they make sense given the importance of body fat stores to both cognitive function and physical work (see Chapter 7 by Leonard and Chapter 8 by Snodgrass of this book).

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In summary, human childhood is defined by the following traits: (1) a slower rate of growth than during infancy and relatively small body size; (2) a large, fast-growing brain; (3) higher brain RMRs than any other mammalian species; (4) immature dentition; (5) motor immaturity; (6) cognitive immaturity; and (7) adrenarche, the midgrowth spurt, and adiposity rebound. We know of no other mammalian species with this entire suite of features.

Juvenile Stage Juvenile mammals may be defined as "prepubertal individuals that are no longer dependent on their mothers (parents) for survival" (Pereira and Altmann 1985, p. 236). This definition is derived from ethological research with social mammals, especially nonhuman primates, but applies to the human species as well. The phrase "no longer dependent on their mothers" means that the young are weaned from feeding by lactation. However, some juvenile mammals are, to a greater or lesser extent, dependent on their parents (see above). Even so, the major difference between infant and juvenile mammals is that it is possible for the juveniles to survive the death of their adult caretakers. Human infants and children cannot survive without assistance from older people. Ethnographic research shows that juvenile humans have the physical and cognitive abilities to provide much of their own food and to protect themselves from predation and disease. The so-called street children mentioned above are in fact street juveniles!

The human juvenile stage begins at about 7.0 years of age. In girls, the juvenile period ends, on average, at about the age of 10 years, 2 years before it usually ends in boys, the difference reflecting the earlier onset of adolescence in girls. The juvenile stage is characterized by the slowest rate of growth since birth.

The evolution of the juvenile stage of primates is associated with both social complexity, especially larger social groups, and diet complexity, including foraging for fruits and seeds (Walker et al. 2006a). Studies of juvenile primates and human juveniles in many cultures indicate that much social learning takes place during this stage, and a "learning hypothesis" has often been proposed to account for the evolution of the juvenile stage. It has also been argued that the primary reason for the evolution of juvenility appears to be a strategy to avoid death from competition with older individuals while living in a social group (Janson and van Schaik 1993).

Adolescence Human adolescence is the stage of life when social and sexual maturation takes place. Adolescence begins with puberty, or more technically with **gonadarche**, which is the final "on" of the on–off–on pattern of the GnRH pulse generator of the hypothalamus (Fig. 11.11a). The transition from juvenile to adolescent stages requires not only the renewed production of GnRH but also its secretion from the hypothalamus in a specific frequency and amplitude of pulses (Fig. 11.11b).

None of these hormonal changes can be seen without sophisticated technology, but the effects of gonadarche can be noted easily as visible and audible signs of sexual maturation. One such sign is a sudden increase in the density of pubic hair (indeed, the term "puberty" is derived from the Latin pubescere, "to grow hairy"). In boys, the increased density and darkening of facial hair and the deepening of the voice (voice "cracking") are other signs of puberty. In girls, a visible sign is the development of the breast bud, the first stage of breast development. The pubescent boy or girl, his or her parents, and relatives, friends, and sometimes

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everyone else in the social group can observe one or more of these signs of early adolescence.

The adolescent stage also includes the development of **secondary sexual characteristics**, such as development of the external genitalia, sexual dimorphism in body size and composition (Fig. 11.14), and the onset of greater interest and practice of adult patterns of sociosexual and economic behavior. These physical and behavioral changes occur with puberty in many species of social mammals. What makes human adolescence unusual among the primates are two important differences. The first is the length of time between age at puberty and age at first birth. Humans take, on average, some 10 years for this transition (Bogin 1999, 2001; Walker et al. 2006b). On a worldwide basis, and throughout history, the average age for the first external manifestations of puberty for healthy girls is 9 years and first birth is at 19 years. On the same basis, boys show external signs of puberty, on average, at 11 years and fatherhood at 21–25 years. The evolutionary reasons for delay between puberty and first birth or fatherhood are discussed below. The point to make here is that monkeys and apes typically take less than 3 years to make the transition from puberty to parenthood.

The second human difference is that during this life stage, both boys and girls experience a rapid acceleration in the growth velocity of almost all skeletal tissue—

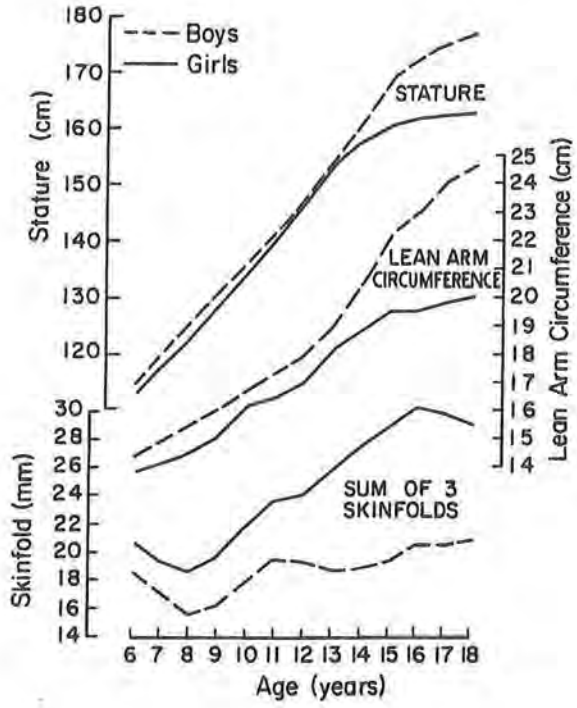

Figure 11.14 Mean stature, mean lean arm circumference, and median of the sum of three skinfolds for Montreal boys and girls. Notice that sexual dimorphism increases markedly after puberty (~12–13 years old). *Source*: Baughn et al. (1980).

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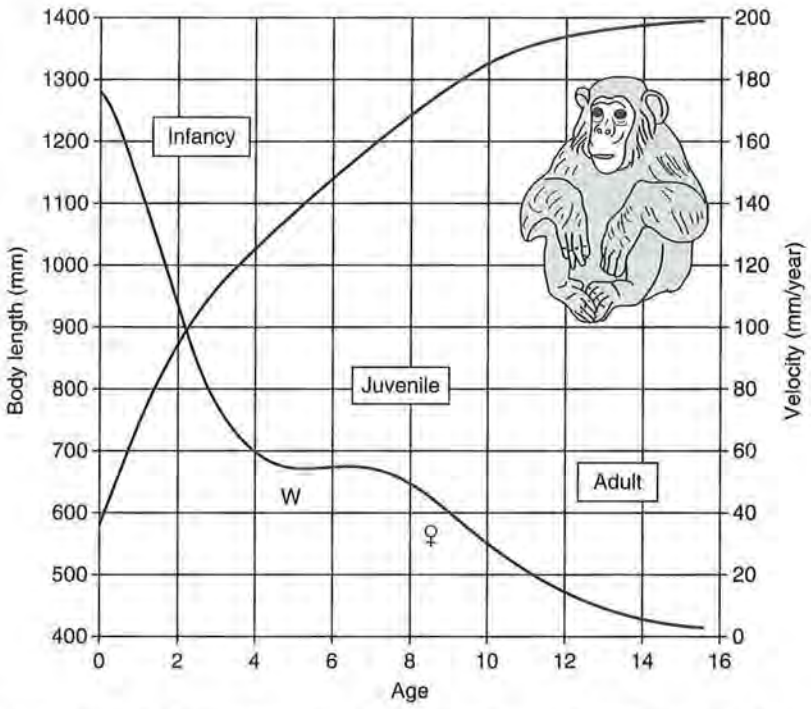

Figure 11.15 A model of distance and velocity curves for chimpanzee growth in body length. This is based on the longitudinal study of captive chimpanzee growth conducted by Hamada and Udono (2002). In the wild, weaning (W) usually takes place between 48 and 60 months of age (Pusey 1983). The female symbol "♀" indicates the average age at first sexual swelling in captivity. In the wild, age at first sexual swelling is 12 years.

the **adolescent growth spurt** (see Fig. 11.7, velocity curve). The magnitude of this acceleration in growth was calculated for a sample of healthy Swiss boys and girls measured annually between the ages of 4 and 18 years. At the peak of their adolescent growth spurt, the average velocity of growth in height was +9.0 cm/year (3.5 in./year) for boys and +7.1 cm/year (2.8 in./year) for girls (Largo et al. 1978). Similar average values are found for adolescents in all human populations. No other primate species, not even chimpanzees, exhibits this pattern of skeletal growth (Fig. 11.15). It is important to point out that these are average human values and that there is considerable variation in the size of the spurt at peak height velocity (the maximum rate of growth during adolescence) and in the duration of adolescence. In some studies of adolescent growth, up to 10% of seemingly healthy girls showed no discernable growth spurt. The absence of an adolescent growth spurt in healthy, well-nourished boys is rare.

Most primate species have rapid growth in length and body weight during infancy and then a declining rate of growth from weaning to adulthood. Some primate species may show a rapid acceleration in soft tissue growth at puberty, especially of muscle mass in male monkeys and apes. Sexually maturing nonhuman primates may have skeletal spurts in the face, for example, due to the eruption of large canine teeth in male baboons (Bogin 1999; Leigh 2001). However, unlike humans, other

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primate species either have no adolescent acceleration in total skeletal growth or a very small increase in growth rate (Watts and Gavan 1982; Hamada and Udono 2002). The velocity of human long bone growth may be five times as rapid as that of the ape (Cameron et al. 1982; Watts and Gavan 1982). The human skeletal growth spurt is unequaled by other species, and when viewed graphically, the duration and intensity of the growth spurt define human adolescence (Fig. 11.7)—It is a species-specific characteristic. Human adolescence, however, is more than skeletal growth. It is also a stage of the life cycle defined by several changes in behavior and cognition that are found only in our species. We discuss these changes in later sections.

Adulthood Adolescence ends and early adulthood begins with the completion of the growth spurt, the attainment of adult stature, and the achievement of full reproductive maturity, meaning both physical and psychosocial maturity. Height growth stops when the long bones of the skeleton (e.g., femur and tibia) and the vertebral bodies of the backbone lose their ability to increase in length. Usually, this occurs when the **epiphysis**, the growing end of the bone, fuses with the **diaphysis**, the shaft of the bone. As shown in Figure 11.6, the process of epiphyseal union can be

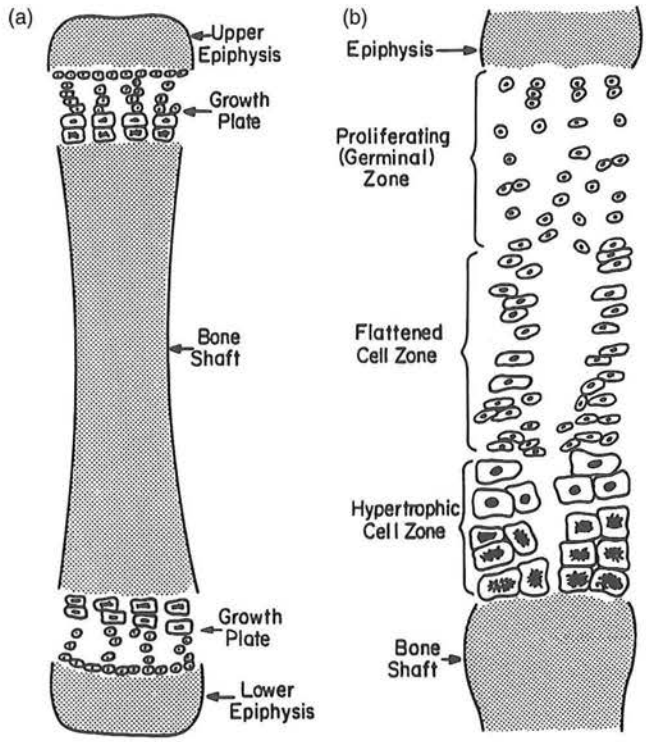

Figure 11.16 (a) Diagram of a limb bone with its upper and lower epiphyses. (b) Diagrammatic enlargement of the growth plate region: New cells are formed in the proliferation zone and pass to the hypertrophic zone to add to the bone cells accumulating on top of the bone shaft. *Source*: Tanner (1990).

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observed from radiographs of the skeleton. The amount of growth that occurs late in adolescence is a function of **skeletal maturation**; thus, late maturers can grow more than average maturers and much more than early maturers. This fact has been known for many years, and an estimate of skeletal maturation, often called skeletal age, is incorporated into equations used to predict the adult height of children and juveniles. The fusion of epiphysis and diaphysis (Fig. 11.16) is stimulated by the gonadal hormones, the androgens and estrogens. However, it is not merely the fusion of epiphysis and diaphysis that stops growth, because children without gonads or whose gonads are not functional do not have epiphyseal fusion, even though they stop growing. Rather, it is a change in the sensitivity to growth stimuli of cartilage and bone tissue in the **growth plate region** (Fig. 11.16) that causes these cells to lose their hyperplastic growth potential.

Reproductive maturity is another hallmark of adulthood. The production of viable spermatozoa in boys and viable **oocytes** in girls is achieved during adolescence, but these events mark only the early stages, not the completion, of reproductive maturation. Socioeconomic and psychobehavioral maturation must accompany physiological development. All of these developments coincide, on average, by about age 19 in women and 21–25 years of age in men. We discuss possible reasons for the human sequence of biological, social, and psychological development toward adulthood, in terms of the evolutionary background of human reproductive development, later in this chapter.

The transition to adulthood is marked by several events, including the cessation of height growth and achievement of full reproductive maturity. In contrast, the course of growth and development during the prime reproductive years of adulthood are relatively uneventful. Most tissues of the body lose the ability to grow by hyperplasia, but many may grow by hypertrophy. Exercise training can increase the size of skeletal muscles, and caloric over sufficiency certainly will increase the size of adipose tissue. However, the most striking feature of the prime adult stage of life is its stability, or **homeostasis**, and its resistance to pathological influences, such as disease-promoting organisms and psychological stress. It contrasts with the preceding stages of life, which were characterized by change and **susceptibility** to pathology.

Late Life Stage Old age and **senescence** follow the prime years of adulthood. The aging period is one of gradual or sometimes rapid decline in the ability to adapt to environmental stress. The pattern of decline varies greatly between individuals. Although specific molecular, cellular, and organismic changes can be measured and described, not all changes occur in all people. Unlike the biological regulation of growth and development prior to adulthood, the aging process appears to follow no species-specific uniform plan. **Menopause** may be the only event of the later adult years that is experienced universally by women who live past 50 years of age; men have no similar event. We discuss the biology and possible value of menopause later in this chapter.

There are many theories about the aging process and about why we must age at all. Chapter 13 of this book discusses some of them and shows that aging is a multicausal process. We may state here simply that the inability of all cell types—renewing, expanding, and static—to use nutrients and repair damage leads to aging, senescence, and death.

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BOX 11.2 COLLECTION AND ANALYSIS OF GROWTH AND LIFE HISTORY DATA FROM MAMMALIAN SPECIES: A CLASS EXERCISE

The concept of **life history** and its importance in the study of human growth and development are easier to understand if we compare several features of human growth and the human life cycle with those of other species. In this exercise, we confine such comparisons to the mammals.

Each student in the class should select a species of mammal for study. The mammals chosen should include both large and small species and some that are terrestrial, aquatic, and capable of flight. Try to choose species that live in different habitats, such as tropical, temperate, desert, and arctic zones.

Complete the following data list for each chosen mammal:

Species: _____
Habitat: _____
Adult body size: _____ kilograms
Adult brain size: _____ grams (or cubic centimeters)
Length of gestation: _____ days
Age at weaning: _____ months
Age at reproductive maturity: _____ months
Number of offspring per pregnancy: _____ (mean litter size)
Interval between births: _____ (mean number of months)
Body temperature: _____ (mean adult value)
Mean life span: _____ months
Maximum life span: _____ months

This information can be found online at PanTHERIA, http://www.esajournals.org/doi/abs/10.1890/08-1494.1, and in encyclopedias such as *Walker's Mammals of the World* (Nowak 1999) and in books devoted to the various families and genera of mammals. The Primate Life Histories Database (http://plhdb.org/) is under construction by the National Evolutionary Synthesis Center. As of this writing, there are some demonstration data available. After you have collected the data, perform several analyses. As a start, enter all of the data into a database and use any statistical software package to compute correlations between each of the variables. Discuss the meaning of the pattern of correlations. Consult review articles on life history (e.g., Harvey et al. 1987) to help interpret the meaning of the data analysis. Use the correlation analysis to frame new questions about the pattern of life history variables. Then, devise a way to analyze the data to answer your questions. More advanced exercises include **phylogenetic** analyses; see some methods and software at http://evolution.genetics.washington.edu/phylip/software.html#Comparative.

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HUMAN POSTNATAL GROWTH IN COMPARATIVE PERSPECTIVE

In contrast to the process of aging and death, growth and development follow a predictable pattern from conception to adulthood. During the evolutionary history of our species and of those species ancestral to ours, selective pressures operated to shape our pattern of growth. Thus, to understand why we grow the way we do, we must examine some of the events that occurred during human evolution.

Life History Theory

Life history theory is a branch of biology that studies the selective forces that have guided the evolution of the schedule and duration of key events in an organism's lifetime related to investments in growth, reproduction, and survivorship. Variation between species in these key events constitutes evolutionarily derived strategies to promote increased reproductive **fitness**. Each species has its own **life history**, that is, a pattern of allocation of energy toward growth, maintenance, reproduction, raising offspring to independence, and avoiding death. It is important to repeat that this pattern, or life history strategy, is not conscious or planned. Rather, it is the set of biological and behavioral traits that characterize a species and which evolved via a series of **trade-offs**, that is, costs to an organism's fitness in one trait due to investment in another trait (Kozlowski and Wiegert 1986; Stearns 1989, 1992; Roff 1992; Charnov 1993). An example of a human trade-off between adult height of men and women and age at first reproduction is given in Figure 11.17. The data are for 16 small-scale hunter–gatherer and horticultural human societies (data kindly provided by Dr. Robert Walker). In these societies, which do not use contraception, investment in greater body height tends to delay the age at first birth. This type of negative

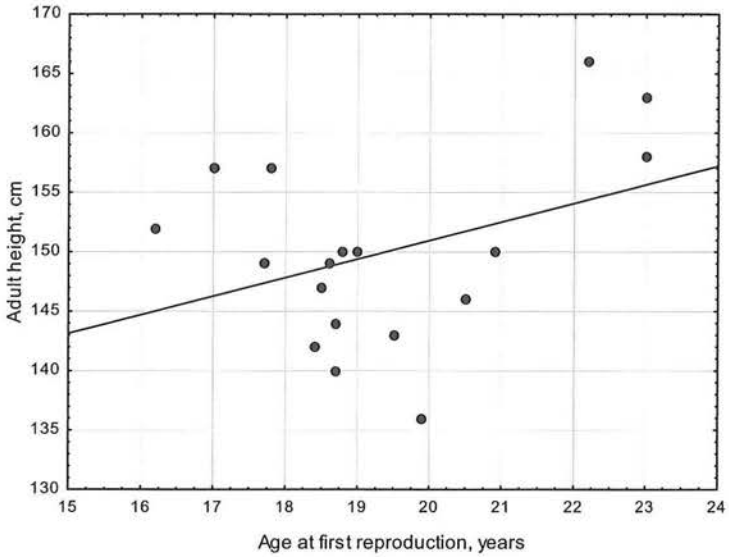

Figure 11.17 Trade-off between adult body height of men and women and age at first reproduction as seen in 16 small-scale hunter–gatherer and horticultural human societies (data from Walker et al. 2008).

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relationship makes sense, as the simultaneous investment in both the growth of the parent's own body and that of a fetus may be beyond the limits of available energy (food) for these human groups. For a mammal, a life history strategy includes when to be born, when to be weaned, how many and what type of pre-reproductive stages of development to pass through, when to reproduce, and when to die. Living things on earth have greatly different life history strategies, and understanding what shapes these is one of the most active areas of research in whole-organism biology.

The origins of the life history theory go back at least to Fisher's (1930) mathematical analyses of reproductive value, which is defined as an organism's expected contribution to its population through both current and future reproduction. Fisher's mathematical insight was to model reproductive value in terms of the trade-offs between reproduction, growth, and survivorship (for more on the history of these ideas, see Caswell 1982 and Stearns 1989, 1992). Some species live life in the "fast lane," meaning that they grow to adulthood quickly after birth, leave many offspring, and die young. Other species, such as human beings and many primates, live life in the "slow lane," meaning that newborns grow relatively slowly to adulthood, have fewer births, and live to older ages.

Anthropologists and human biologists have become increasingly interested in explaining the significance of human life history in relation to the life cycle. This interest is due to the discovery that the human life cycle stands in sharp contrast to other species of social mammals, even other primates. Anthropological and biological theory need to explain how humans successfully combined a vastly extended period of offspring dependency and delayed reproduction with helpless newborns, a short duration of breast-feeding, an adolescent growth spurt, and menopause. A central question is, did these characteristics evolve as a package or a mosaic? The present evidence suggests that the human life cycle evolved as a mosaic and may have taken form over more than a million years.

Understanding the human life cycle and the life history trade-offs that shaped it requires a comparative approach. Biologists employ at least two methodologies. One compares many species to identify general adaptive patterns via trait–environment and trait–trait correlations. A trait–environment correlation, for example, is the relationship of environmental temperature to body mass or reproduction schedules (see Chapter 6 by Beall et al. and Chapter 15 by Ellison et al.). An example of a trait–trait correlation is the relationship of brain size to **diet quality** or gut size (see Chapter 7 by Leonard). The second methodology studies "selection in action," meaning that researchers measure fitness costs and benefits in current environmental contexts of different life history strategies. Studies of the timing of a woman's first birth are a human example, which is an important life history event, on the women's own health and mortality and that of her offspring (Kramer and Lancaster 2010 and Chapter 15). In this chapter, we present and discuss the results of research using these two methodologies, with examples from Primates and other mammals.

Mammalian Life Cycles

The majority of mammals progress from infancy to adulthood seamlessly, without any intervening stages, and puberty occurs after the peak velocity of their postnatal growth. This pattern of postnatal growth is illustrated in Figure 11.18 using data for

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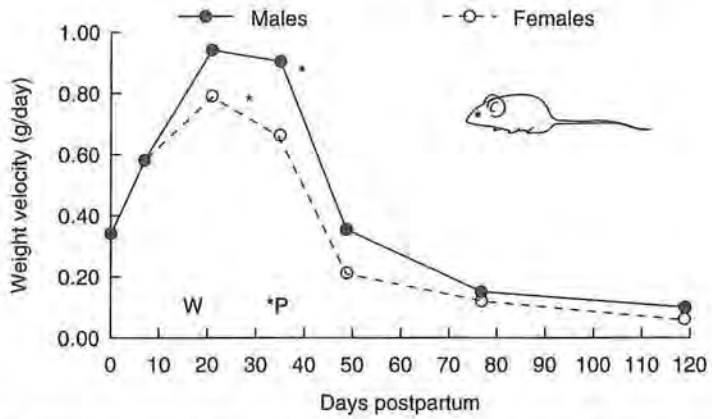

Figure 11.18 Velocity curves for weight growth in the mouse. In both sexes, puberty (*P, vaginal opening for females or spermatocytes in the testes of males) occurs just after weaning (W) and maximal growth rate. Weaning takes place between days 15 and 20. Sexual maturity follows weaning by a matter of days.

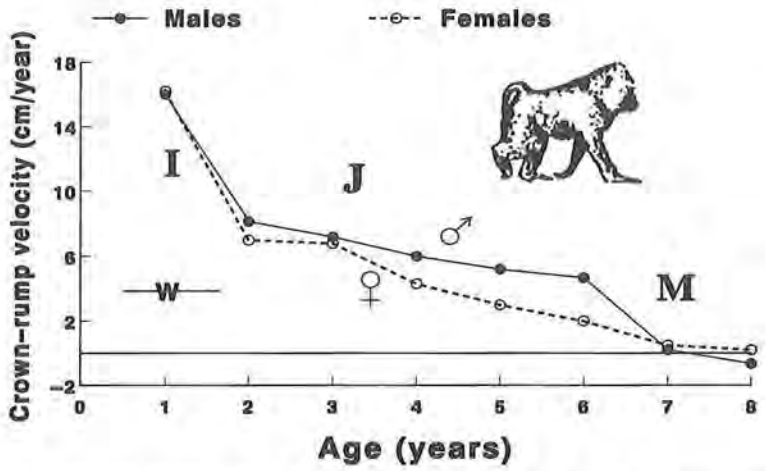

Figure 11.19 Baboon crown–rump length velocity. Letters indicate the stages of growth as in Figure 11.6. Weaning (W) may take place anytime between 6 and 18 months. *Source*: Adapted from Coelho (1985). I, infant; J; juvenile; M, mature adult.

the mouse. Highly social mammals such as wolves, wild dogs, lions, elephants, and the primates (e.g., the baboon [Fig. 11.19] and humans) postpone adulthood by inserting a period of juvenile growth and behavior between infancy and adulthood.

As we mentioned earlier, for many years, a "learning hypothesis" was assumed to be the reason for the juvenile stage. Indeed, a juvenile life stage allows for the extended period of brain growth and learning that are common to species of group-living social mammals. Learning is certainly important in relation to feeding skills such as hunting, attaining mating skills, and negotiating the intricacies of interpersonal relationships within a complex social group. All this said, the evolution of the

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mammalian juvenile stage must first be explained in terms of its immediate benefits, especially for survival to the adult stage.

Janson and van Schaik (1993) reviewed the data and explanations for the juvenile stage and found that a "juvenile risk aversion hypothesis" appears to be a reasonable explanation. Juveniles must forage for their own food in competition with adults. Janson and van Schaik noted that this competition becomes clear during times of food scarcity, when juvenile primates die in greater numbers than infants or adults. Juveniles are smaller than adults and are growing slowly. These traits reduce energy and nutrient requirements. A human juvenile boy of age 9–10 years old has an average body weight of 29.7 kg and average growth velocity of 5.0 cm/year. This boy requires 66.6 kcal/kg/day energy intake. A 4- to 5-year-old boy child, of 17.7 kg body weight and 6.5 cm/year growth velocity, requires 76.8 kcal/kg/day energy intake (FAO/WHO/UNU 2004). The difference is about 13% less energy required by the juvenile.

Janson and van Schaik also cited research with wild and captive primates showing that high-ranking individuals in the social hierarchy can suppress and inhibit the reproductive maturation of low-ranking individuals. The inhibition may be due to the stress of social intimidation acting directly on the endocrine system, or the suppression may be secondary to inadequate nutrition due to feeding competition. Several lines of research, including with human juveniles, show that younger, less mature looking individuals are less likely to be subjected to social intimidation and more likely to be more integrated into the social group (reviewed in Bogin 1999, pp. 193–198). Juveniles, with their slow growth and delayed reproductive maturation after infancy and childhood, may have evolved in part to withstand intimidating adults and survive to adulthood more often than individuals with rapid growth and maturation.

Charnov (1993, 2001) took another perspective and explained slow juvenile growth as a trade-off, the by-product of time needed to grow to larger size and reap benefits in terms of greater survival and fertility against the risks of mortality before reaching reproductive age and size. Life history data for dozens of species from many classes of animals, but not primates or bats, conform to the expected negative correlations (trade-offs) between adult mortality rates and length of the pre-adult period, such that species with higher adult mortality rates have shorter periods of growth (Charnov 2001).

Charnov's correlation methodology and its findings have been central to much of life history theory. There is, however, a growing body of criticism to this methodology (Kozlowski and Weiner 1997; Nee et al. 2005; and de Jong 2005, with a reply by Savage et al. 2006). One reason for the criticism is that correlation is a mathematical–statistical technique that can estimate the strength of a relationship but cannot establish causation. We can determine that adult mortality rates and length of the pre-adult period are related, but it is not possible to be sure which variable causes the other, or if other variables we did not measure are, in fact, causing the relationship between mortality and rate of development. Another source of criticism focuses on the assumptions of Charnov's models, including the assumption of stable population size, an unchanging environment, and constant rates of mortality imposed by the physical environment. These assumptions are often not found in real biological populations. A discussion of these criticisms is beyond the scope of this chapter. Interested readers may consult Caswell (1982), Tuljapurkar (1990), and Tuljapurkar and Steiner (2010).

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Juvenile Human Beings: An "Atypical Species?"

Another criticism centers on Charnov's exclusion of primates and bats from analysis. He does this because these species are, in his words, "atypical mammals" (2001, p. 521). In an earlier paper, Charnov and Berrigan (1993) described primates as living "in the slow lane," meaning that, "Contrasted to most other mammals, primates have long average adult life spans and few babies per year for their adult body size" (p. 191). The human species lies at the extreme end of the range of this primate life history pattern.

In the model of Charnov and Berrigan, "Selection acts on the age at maturity to maximize lifetime reproductive success in the face of adult mortality rates imposed by the environment." By this they seem to mean that longer pre-adult stages of life can evolve only with reduced adult mortality, as species with high adult mortality must develop relatively quickly from conception to reproductive age. Although Charnov and Berrigan recognized that the juvenile period also has mortality risks, they offered no tangible benefits of slow growth and delayed maturation to the juveniles themselves. Charnov and Berrigan suggested that the juvenile stage may have something to do with large primate brains, but they seem to reject the learning hypothesis. They wrote that, "One possible explanation for the [slow growth] in primates is the greater (?) energy demands for growing and supporting big brains; there is a fair bit of irony in this suggestion because it implies that the long primate lifespan follows from the cost, rather than some cognitive benefit, of having a big brain" (p. 193, text in square brackets added). It would certainly make primates atypical if they evolved a long juvenile stage in the face of substantial costs rather than benefits.

In contrast, the risk aversion hypothesis of Janson and van Schaik offers immediate benefits to the juvenile, which seems more consistent with traditional natural selection theory. Whatever the cause, Alexander (1990) pointed out that in broad perspective, "juvenile life has two main functions: to get to the adult stage without dying and to become the best possible adult." We conclude that, given its potential costs in terms of risk for death and delayed onset of reproduction, adding a juvenile stage to mammalian, and especially primate, life history must have served this purpose well.

Once part of the life cycle, the juvenile stage does allow for learning, and human boys and girls learn a great deal about important adult activities, including the production of food and methods of infant and child care. The completion of growth in weight of the brain and the onset of new cognitive competencies at the end of the childhood stage allow for this increased intensity of learning. Because juveniles are prepubertal, they can attend to this kind of social learning without the distractions caused by sexual maturation (Del Giudice et al. 2009). As an aside, the start of the juvenile stage coincides with entry into traditional formal schooling in the industrialized nations. The connection is hardly a coincidence, because the juvenile stage allows for the kinds of learning and socialization found in school environments.

WHY DO NEW LIFE STAGES EVOLVE?

During mammalian evolution the juvenile stage evolved, inserted between birth and adulthood, for many social species. In the course of human evolution, childhood and

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adolescence were added as new life stages, just before and just after the juvenile stage. Universally, human females who live long enough experience menopause followed by a decade or more of vigorous life. This postreproductive stage is a new life history period for primates. These new stages presumably add additional security and value to the whole of life history. In the sections that follow, we offer some current ideas as to why and when these additional stages of the human life cycle evolved.

In his book *Size and Cycle*, Bonner (1965) developed the idea that the stages of the life cycle of an individual organism, a colony, or a society are "the basic unit of natural selection." Bonner's focus on life-cycle stages follows in the tradition of many of the 19th-century embryologists who proposed that **speciation** is often achieved by altering rates of growth of existing life stages and by adding or deleting stages. Bonner showed that the presence of a stage, and its duration, in the life cycle relates to such basic adaptations as locomotion, reproductive rates, and food acquisition. From this theoretical perspective, it is profitable to view the evolution of human childhood, adolescence, and perhaps menopause/postreproduction as adaptations for both feeding and reproduction.

Why Childhood?

Consider the data shown in Figure 11.20, which depict several **hominoid** developmental landmarks. Compared with living apes, human beings experience developmental delays in eruption of the first permanent molar, age at **menarche**, and age

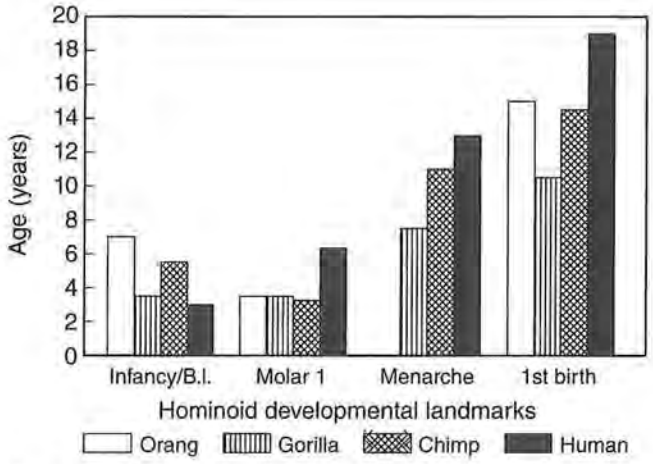

Figure 11.20 Hominoid developmental landmarks. Data based on observations of wild-living individuals or, for humans, healthy individuals from various cultures. Infancy/B.I. is the period of dependency on mother for survival, usually coincident with mean age at weaning and/or a new birth (where B.I. is birth interval); molar 1 is mean age at eruption of first permanent molar; menarche is mean age at first estrus or menstrual bleeding; 1st birth is mean age of females at first offspring delivery. Orang, *Pongo pygmaeus*; gorilla, *Gorilla gorilla*; chimp, *Pan troglodytes*; human, *Homo sapiens*. *Source*: Bogin and Smith (1996).

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at first birth. However, humans have a shorter infancy and a shorter birth interval. In apes and traditional human societies, the infancy stage, that is, the time of feeding by lactation, is virtually equal to the interval between births.

We discussed earlier that dental development is an excellent marker for life history in the primates. Many mammals, including most primate species, wean infants and terminate the infancy stage about the time the first permanent molar (M1) erupts. This timing makes sense, because the mother must nurse her current infant until it can process and consume an adult diet, which requires at least some of the permanent dentition.

Earlier in this chapter, we discussed primate and social carnivore species that provide food to their young during the postweaning period. In these species, the molar and premolar teeth may erupt before the postweaning dependency period ends. These primates and carnivores are similar in that both are predatory (the primates tend to hunt insects) and the young need time to learn and practice hunting skills. The effect of postweaning dependency on the mother is a delayed return to reproduction. In a review of reproductive behavior of the carnivores, Ewer (1973) found that female lions, hyenas, the sea otter (a fisher–hunter), and many species of bears wait 2 or more years between pregnancies, even though the infants are weaned months before. Lions, for example, wean their infants at about 7 months of age and the birth interval for adult females averages 20–25 months. The delay between weaning and the next birth is, in large part, explained by the fact that the newly weaned juveniles are dependent on the mother for continued care until about age 35–36 months (Creel and Creel 1991). Female lions that lose a litter before the cubs are weaned have a shorter interbirth interval of between 4 and 6 months (Pusey and Packer 1987). The same birth interval delay holds for the more insectivorous primates, such as marmosets and tamarins, and for the same reasons.

The data for the social carnivores and some nonhuman primates show that the correlation between eruption of the first permanent molar (M1) and weaning can be modified by other factors, especially the feeding independence of the young. Such modification is most extreme for chimpanzees and orangutans. The interval between successful births for chimpanzee females averages nearly 5 years, and for orangutan mothers, who may be under nutritional stress, it averages 6–8 years in the wild (van Schaik 2004). But, first permanent molar eruption in these apes takes place at about age 3.1 years in captivity. Even in the wild chimpanzee M1 eruption occurs at about age 4.0 (Zihlman et al. 2004). Chimpanzee and orangutan mothers continue to nurse their young until near the end of the interbirth interval. From these data, we may conclude that birth spacing in the social and predatory mammals may be delayed until well after the age at which the first permanent teeth erupt in the current infant.

The human species is a striking exception to this relationship between permanent tooth eruption and birth interval. Women in traditional societies wait, on average, 3 years between births, not the 6 years expected on the basis of the average age at M1 eruption. The short birth interval gives humans a distinct advantage over other apes, because human women can produce and rear two offspring through infancy in the time it takes chimpanzees or orangutans to produce and rear one offspring. By reducing the length of the infancy stage of life (i.e., lactation) and by developing the special features of the human childhood stage, humans have the potential for greater lifetime fertility than any ape.

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The answer to the question "Why childhood?" is that the *evolution of childhood gave human beings a reproductive advantage over other apes*. Childhood comes with a significant trade-off: Human children are still dependent on older individuals for feeding, needing foods that are specially chosen and prepared, and protection. Without sufficient care, children will suffer and die. We discuss the risks of childhood near the end of this chapter. Humans deal with the special needs of children by spreading the needs for feeding and protection across a number of people, including juveniles, adolescents, and adults. The mother of the child does not have to provide 100% of offspring nutrition and care directly. Indeed, traditional societies deal with the problem of child care by spreading the responsibility among many individuals.

Cooperative child care, also called cooperative breeding, seems to be a human universal (Bogin 1999; Hrdy 1999). Key features of human cooperation are food sharing and division of labor by age and sex. The contribution of food and labor may be quantified in terms of energy (**kilocalories**). Another way is to quantify the food and labor sharing in terms of reproductive outcomes, such as birth and survival of offspring to adulthood. Reiches et al. (2009) did just this by proposing a "pooled energy budget" hypothesis. They defined the pooled energy budget as

> the combined energetic allocations of all members of a reproductive community that might result in direct or indirect reproductive effort. These transactions can take many forms at one time and vary across the life course. . . . Individuals draw on the pooled energy budget by consuming calories and by diverting time and energy [to] reproduction. They contribute by diminishing their own energetic costs and by contributing to the energy budgets of others. The output of the pooled energy budget is the production of new individuals. (p. 424)

According to Reiches and colleagues, the pooled energy budget allows human women to sustain a higher fertility and greater survival of their offspring than would be possible for energetically isolated individuals. Energetic isolation, relative to the human condition, is the case for adult female chimpanzees, bonobos, gorillas, and orangutans. Females in these ape species are, generally, competitors. In terms of survival, about 50–60% of infants and children in human forager societies reach adulthood. In the great apes, the survival averages less than 40% (Kaplan et al. 2000).

One human example of pooled energy budgeting are the Hadza society, African hunters and gatherers, where grandmothers and great aunts supply a significant amount of food and care to children (Blurton Jones et al. 1992, Hawkes et al. 1998). In Agta society (Philippine hunter–gatherers), women hunt large game animals but still retain primary responsibility for child care. They accomplish this dual task by living in extended family groups—two or three brothers and sisters, their spouses, children, and parents—and sharing the child care. Among the Maya of Guatemala (horticulturists and agriculturists), many people live together in extended family compounds. Women of all ages work together in food preparation, clothing manufacture, and child care (Bogin, field observations, Fig. 11.21). In some societies, fathers provide significant child care, including the Agta and the Aka pygmies, hunter–gatherers of central Africa. Summarizing the data from many human societies, Lancaster and Lancaster (1983) called this kind of cooperative child care and feeding "the hominin adaptation," because no other primate or mammal does all of this.

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Figure 11.21 Cooperative care of children by women and juvenile girls. The example is from the Kaqchikel-speaking Maya region of Guatemala. The women perform household and food preparation duties, while the juvenile girls play with and care for the children. Photograph by Barry Bogin.

Various types of cooperative breeding, meaning care of offspring by nonparents, are found in some species of birds and other mammals (e.g., wolves and hyenas), and it works to increase net reproductive output (Bergmuller et al. 2007). In those species, and in many but not all human groups, the cooperative breeders are close genetic relatives of the mother (Clutton-Brock 2002). By assisting the mother to care for her offspring, the helpers increase their own **inclusive fitness**, meaning that they help to ensure that their genetic kin survive to reproductive age (Hawkes et al. 1998; Paine and Hawkes 2006).

Human societies define kinship relations on the basis of genetic and social ties. Humans are the only species to use language and the cultural institution of marriage to define kinship categories. The overarching importance of kinship for the human species is that in traditional societies (foragers, horticulturalist, pastoralists, and pre-industrial agriculturalists), kinship is the central organizing principle for economic production, social organization, and ideology (e.g., moral codes, religious behavior). Many societies make use of fictive kinship, the application of kinship names to people unrelated by marriage or descent, to enhance social relations, including rights and responsibilities toward each other's offspring. An example is calling the close friend of one's mother by the name "Aunt Maria" instead of Mrs. Smith. "Aunt Maria" may provide food, supervision, protection, gifts, and other types of **parental investment** to her "niece," and the "niece" is expected to behave in accordance with the rules of interaction between family members. Human cooperative breeding, therefore, is biocultural in nature—explained by both genetic and fictive kinship (Kramer 2007). Human cooperative breeding may better be called **biocultural**

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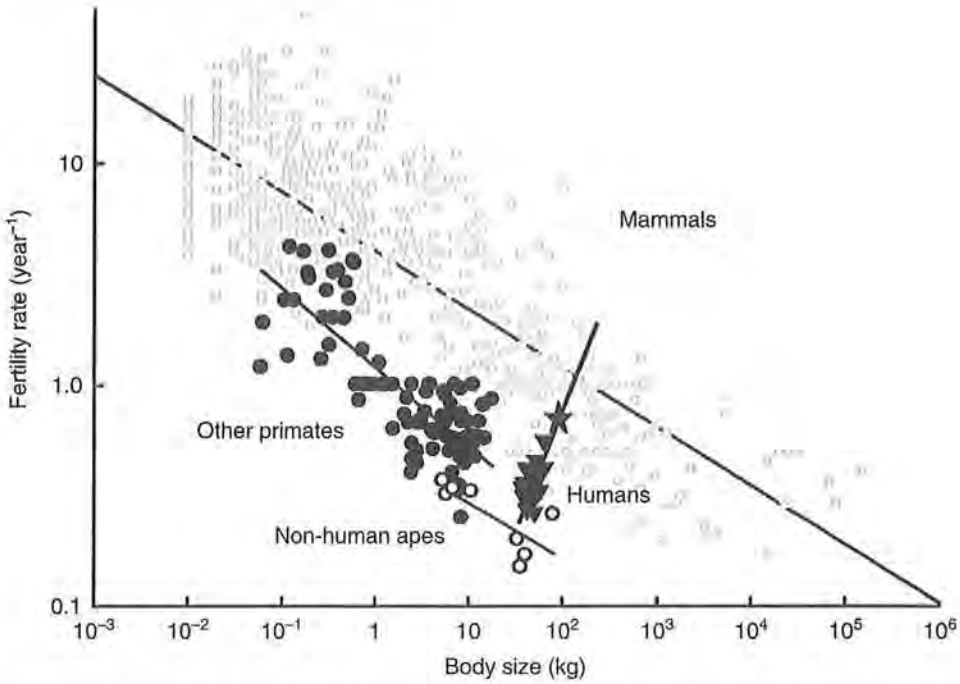

Figure 11.22 Trade-off between fertility rate and female adult body size in placental mammals (modified from Walker et al. 2008). Fertility tends to decline as adult female body size increases, a negative correlation. Human beings are the exception and show a positive correlation between body size and fertility. "Natural-fertility human societies are the only sample here where fertility increases with body size" (Walker et al. 2008). Why this is so is not completely known, but likely relates to energy available to support both body growth of the mother and her reproduction in adulthood. The text of this chapter explains this in greater detail in terms of human "pooled-energy budgets" (Reiches et al. 2009).

reproduction, because it enhances the social, economic, political, religious, and ideological "fitness" of the group as much as it contributes to genetic fitness.

The life history impact of biocultural reproduction may be seen in Figure 11.22. Illustrated in the figure is the trade-off between fertility rate and female adult body size in placental mammals (modified from Walker et al. 2008). The data are for 610 species of placental "mammals," excluding primates and bats; 101 species of "other primates," including lemurs, lorises, tarsiers, and New World and Old World monkeys; 13 species of "nonhuman apes," including gibbons (9 species), orangutans, chimpanzees, bonobos, and gorillas (1 species each); and 17 natural fertility human societies (no effective contraception) including hunter–gathers, horticulturalists, one pastoral group, and the Hutterites (star symbol). The Hutterites are a Christian religious sect of North America, traditionally rural farmers, who prize high fertility and do not practice contraception. During the 1940–1950s, the Hutterites achieved the highest average fertility per women of all human societies (Eaton and Mayer 1953).

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The fertility of mammals, including the nonhuman primates tends to decline as adult female body size increases, a negative correlation. Note that the nonhuman primates and apes are downshifted, meaning that they have a slower life history including a longer period of growth and a later age at first reproduction than other mammals of the same body size. Human beings are the exception and show a positive correlation between body size and fertility. This human life history relationship likely relates to energy available to support both body growth of the mother and her reproduction in adulthood via "pooled-energy budgets" and other sociocultural strategies.

Phenotypes and Plasticity

Childhood may also be viewed as a mechanism that allows for more precise "tracking" of ecological conditions by allowing more time for developmental plasticity. The fitness of a given phenotype (i.e., the physical features and behavior of an individual) varies across the range of variation of an environment. When phenotypes are fixed early in development, such as in mammals that mature sexually soon after weaning (e.g., rodents), environmental change and high mortality are positively correlated. Mammals with more plasticity in biological and social characteristics (carnivores, elephants, primates) prolong the developmental period by adding a juvenile stage between infancy and adulthood. Adult phenotypes develop more slowly in these mammals because the juvenile stage lasts for years. These social mammals experience a wider range of environmental variation, such as seasonal variation in temperature and rainfall. They also experience years of food abundance and food shortage as well as changes in the number of predators and in the types of diseases. The result on the phenotype may be a better conformation between the individual and the environment.

There is a correlation between the presence of additional life history stages and evolutionary fitness. As with all correlations, it is not possible to determine causation; however, the comparison between species is thought provoking. For example, ~4% of infant Norway rats (no juvenile stage) born in the wild survive to adulthood versus 14–16% of lions, which have a juvenile stage. Monkeys and apes have juvenile periods that last as long, or longer, than those of social carnivores, and these primates rear between 12% and 36% of their offspring to adulthood.

In addition to the primate juvenile stage, humans have a childhood stage of life. The human childhood stage adds another 4 years of relatively slow physical growth and allows for behavioral experience that further enhances developmental plasticity. The combined result is that humans in traditional societies (hunting and gathering, or horticultural groups) rear ~50–60% of their offspring to adulthood. Whichever the direction of cause and effect, long childhood and juvenile periods are associated with higher human survival to adulthood. In addition to life history stages, other social, economic, and political factors are crucial to human survival. In the technologically advanced nations today, survival to adulthood is appreciably higher. For the United States in the year 2004, an estimated 98.7% of infants born alive will survive to adulthood (age 20 years, Arias 2007). For the Central American nation of Costa Rica, this figure is 98.4% (2008 estimate of the World Health Organization, http://apps.who.int/ghodata). Costa Rica enjoys a high standard of living and social services. Human survival declines when warfare, civil unrest, and other disturbances

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to well-being occur. In the nation of Guatemala, a neighbor of Costa Rica, the estimate for survival to age 20 years is only 95.1% of live-born infants (World Health Organization, http://apps.who.int/ghodata). The difference amounts to 3267 more deaths per 100,000 people in the population, or nearly 33,000 more deaths for Guatemala, with a total population of 11 million. Human biocultural reproduction has many risks, and phenotypic plasticity has its limits.

The childhood stage has its benefits when the biocultural environment is favorable. Human childhood decreases the interbirth interval for women and increases the survival of the young. The relatively slow rate of body growth and small body size of children reduces competition with adults for food resources. Slow-growing small children require less food per day than larger juveniles, adolescents, and adults (Gurven and Walker 2006). Thus, although provisioning children is time-consuming, it is not as onerous a task of investment as it would be, for instance, if both brain growth and body growth were rapid simultaneously. Moreover, biocultural reproduction makes provisioning less onerous for the mother, who would otherwise have to supplement each offspring by nursing for much longer time. Finally, the additional time for development added by childhood increases the quality of the young and its chance of surviving to adulthood.

Why Adolescence?

An adolescent stage of human growth may have evolved by the processes of natural selection and sexual selection. Both types of selection were identified by Charles Darwin. Where natural selection operates to increase the frequency of genotypes and phenotypes, which confer reproductive advantages on the individuals possessing them, sexual selection "depends on the advantage which certain individuals have over other individuals of the same sex and species, in exclusive relation to reproduction"[1] (Darwin 1871, vol. 1, p. 276). Darwin also wrote that "There are many other structures and instincts that must have been developed through sexual selection—such as the weapons of offence and the means of defence possessed by the males for fighting with and driving away their rivals—their courage and pugnacity—their ornaments of many kinds—their organs for producing vocal or instrumental music—and their glands for emitting odours; most of these latter structures serving only to allure or excite the female" (Darwin 1871, pp. 257–258). It is known today that sexual selection also works for females, meaning that female-specific physical and behavioral traits may evolve via competition between the females for mating opportunities with males.

Both childhood and adolescent stages must ultimately be shaped by natural selection. Childhood could be selected because it allows hominin females to give birth at shorter intervals, but producing offspring is only a small part of reproductive fitness. Rearing the young to their own reproductive maturity is a surer indicator of success. Adolescence may be a key in helping the next generation rear its own young successfully. The adolescence stage may provide boys and girls a life history strategy to survive to adulthood, the immediate benefit, and as a longer-term benefit practice complex social skills required for effective mating and parenting. Sexual selection

[1] Today, we would replace the word "reproduction" with "mating." In many primate species, a good deal of mating is more about social relations rather than a reproductive event, such as fertilization.

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may have acted on the "mating," and natural selection on the "parenting." There are, of course, trade-offs associated with the adolescence stage, such as a delay in the onset of reproduction and risks for damage and death. These risks, it seems, have been outweighed by the reproductive advantages of adding adolescence to human life history.

Studies of yellow baboons, toque macaques, and chimpanzees show that between 50% and 60% of first-born offspring die in infancy, and more die before reaching adulthood (see Bogin 1999 for references for these statistics and those that follow in this section). By contrast, in hunter–gatherer human societies, between 39% (the Hadza of eastern Africa) and 44% (the !Kung of southern Africa) of offspring die in infancy. Studies of wild baboons by Altmann (1980) show that whereas the infant mortality rate for the first born is 50%, mortality for second born drops to 38%, and for third and fourth born reaches only 25%. The difference in infant survival is, in part, likely due to experience and knowledge gained by the mother with each subsequent birth.

Such maternal information is internalized by human females during their juvenile and adolescent stages, giving the adult women a reproductive edge. The initial human advantage may seem small, but it means that up to 21 more people than baboons or chimpanzees survive out of every 100 first-born infants—more than enough over the vast course of evolutionary time to make the evolution of human adolescence an overwhelmingly beneficial adaptation.

In human societies, juvenile girls are often expected to provide significant amounts of child care for their younger siblings, whereas in most other social mammal groups, the juveniles are often segregated from adults and infants. Thus, human girls enter adolescence with considerable knowledge of the needs of young children. Adolescent girls gain knowledge of sexuality and reproduction because they look mature sexually, and are treated as such, several years before they actually become fertile. The adolescent growth spurt serves as a signal of maturation. Early in the spurt, before peak height velocity is reached, girls develop pubic hair and fat deposits on breasts, buttocks, and thighs. They appear to be maturing sexually. About a year after peak height velocity, girls experience menarche, an unambiguous external signal of internal reproductive system development. Menarche, however, does not signal fertility as most girls experience 1–3 years with partial **anovulatory menstrual cycles**. The frequency of these cycles that do not release an egg cell from the **ovary** after menarche is high at first and then declines over time. One study of the fertility development of 200 normal girls in Finland, aged 7–17 years, examined the participants on two occasions between 1 and 5 years apart. **Ovulation** was determined by a hormonal assessment. It was found that, "In postmenarcheal girls, about 80% of the cycles were anovulatory in the first year after menarche, 50% in the third and 10% in the sixth year" (Apter 1980, p. 107). These findings have been replicated in other studies (Ibáñez et al. 1999). Pregnancy is possible in the immediate postmenarche years, but its likelihood is low compared with later ages (see Chapter 15 by Ellison et al.). Nevertheless, the dramatic changes of adolescence stimulate the girls to participate in adult social, sexual, and economic behavior, and stimulate the adults around these girls to encourage this participation.

It is noteworthy that female chimpanzees and bonobos, like human girls, also experience up to 3 years of postmenarcheal infertility, so this time of life may be a shared hominid trait. Like human adolescents, the postmenarcheal but infertile

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chimpanzees and bonobos participate in a great deal of adult social and sexual behavior. Primate researchers observing these apes point out that this participation, without pregnancy, allows for the common practice of transferring from the female's natal social group to a new social group and securing her place there, as well as improving many key behaviors that will be needed to successfully rear an infant.

Even if practicing to become an adult has a future benefit, does adolescence also have an immediate benefit for the adolescent? Most evolutionary benefits relate to feeding or reproduction and, as adolescents do relatively little of the latter, the benefits seem more likely to relate to feeding. Human juveniles may hunt, gather, or produce some of their own food intake, but overall they require provisioning to achieve energy balance. This is the case in many traditional societies, such as the Ache, Hiwi, !Kung foragers (Kaplan et al. 2000), and the Maya farmers of Mexico and Guatemala (Kramer 2002), as well as many historical and contemporary urban–industrial societies (Bogin 1999, 2001). In contrast, human adolescents are capable of producing sufficient quantities of food to exceed their own energy requirements. Some of the food that adolescents produce may be used to fuel their own growth and development, creating larger, stronger, and healthier bodies. The surplus production is shared with other members of the social group, including younger siblings, parents, and other immediate family members (defining families in the broad ethnographic sense). Adolescents are economically valuable and for their services in food production, they receive care and protection, as an immediate benefit, to safeguard their health and survival. This is important because adolescents are immature in terms of sociocultural knowledge and experience (Schlegel and Barry 1991; Bogin 1993; Kaplan et al. 2000).

Adolescent Growth and Development of Girls

Full reproductive maturation in human women is not achieved until about 5 years after menarche. For example, the average age at menarche for girls living in the United States is 12.4 years, which means that the average age at full sexual maturation occurs between the ages of 17 and 18 years. Although adolescents younger than these ages can have babies, both the teenage mothers and the infants are at added risk because of the reproductive immaturity of the mother, especially for LBW infants, premature births, and high blood pressure in the mother. The likelihood of these poor outcomes declines, and the chance of successful pregnancy and birth increases markedly, after age 15 years and dropping to a minimum after age 18 years (Kramer and Lancaster 2010).

Another feature of human growth not found in the African apes is that female fertility tracks the growth of the pelvis. Ellison (1982) and Worthman (1993) found that age at menarche is best predicted by biiliac width, the distance between the left and right iliac crests of the pelvis. A median width of 24 cm seems to be needed for menarche in American girls living in Berkeley, CA, Kikuyu girls of East Africa, and Bundi girls of highland New Guinea, even though the pelvic width constant is attained at different ages in these three cultures, about 13, 16, and 17 years old, respectively. Differences in nutrition and disease account for the substantial spread in growth rates and age of sexual maturity across these human groups. Moerman (1982) also reported a special human relationship between growth in pelvic size and reproductive maturation. She found that the crucial variable for successful first birth

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is size of the **pelvic inlet**, the bony opening of the birth canal. Moerman measured pelvic X-rays from a sample of healthy, well-nourished American girls who achieved menarche between 12 and 13 years, although they did not attain adult pelvic inlet size until 17–18 years of age. Quite unexpectedly, she found that the adolescent growth spurt, which occurs before menarche, does not influence the size of the pelvis in the same way as the rest of the skeleton. Rather, the female pelvis has its own slow pattern of growth, which continues for several years after adult stature is achieved. Cross-cultural studies of reproductive behavior suggests that human societies acknowledge (consciously or not) this special pattern of pelvic growth. The age at marriage and first childbirth clusters around 18–19 years for women from such diverse cultures as the Kikuyu of Kenya, Mayans of Guatemala, Copper Eskimos of Canada, and both the colonial and contemporary United States (Bogin 1999, 2001). Why the human pelvis follows this unusual pattern of growth is not clearly understood. Nor is it known if any of the nonhuman primates show this pattern of pelvis-to-body growth.

Unlike human beings, females of many nonhuman primate species give birth successfully before the mother's own body growth terminates (http://pin.primate.wisc.edu/factsheets). Perhaps another human attribute, bipedal walking, is a factor contributing to human pelvis-to-body growth and its relationship to delayed reproduction. Bipedalism is known to have changed the shape of the human pelvis from the basic apelike shape. Apes have a cylindrical-shaped pelvis, but humans have a bowl-shaped pelvis. The human shape is more efficient for bipedal locomotion but less efficient for reproduction because it restricts the size of the birth canal. The required pelvic architecture, in terms of size and shape, for successful reproduction may take longer to develop in bipedal humans than in the nonhuman primates. The time of waiting allows adolescents to invest in their own growth and development, which enhances their current health and survival, as well as work at food production and other tasks that enhances the survival of other members of the social group. These other people are often biological (genetic) relatives of the adolescent. This type of helping behavior toward biological kin is found in many species of insects, birds, and mammals and is able to evolve by **kin selection**, which is a special case of natural selection (Hamilton 1964; West and Gardner 2010); in large-brained species, such as the primates, mechanisms such as contingent reciprocity may amplify favoritism to kin (Silk 2009).

Thus, a point to emphasize here is that the learning benefits of adolescence were grafted on to the primary gains in fitness to the adolescent and her kin. There are also several risks associated with human adolescence, which we discuss later in this chapter.

Why Do Boys Have Adolescence?

The adolescent development of boys is quite different from that of girls. Boys become fertile well before they assume the size and the physical characteristics of men. Analysis of urine samples from boys 11–16 years old shows that they begin producing sperm at a median age of 13.4 years (Muller et al. 1989). Yet cross-cultural evidence indicates that few boys successfully father children until they are into their third decade of life. The National Center for Health Statistics of the United States, for example, reports that only 3.09% of live-born infants in 1990 were fathered by

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men under 20 years of age. In Portugal, for years 1990, 1994, and 1999, the percentage of fathers under 20 years of age was always below 3% (Instituto Nacional de Estatística 1999). In 2001, Portugal stopped presenting results concerning the percentage of fathers below 20 because there were too few of them (Instituto Nacional de Estatística 2001). Among the traditional Kikuyu of East Africa, men do not marry and become fathers until about age 25 years, although they become sexually active after their circumcision rite at around age 18. Among the Ache, traditional foragers of the forests of Paraguay, adolescent boys do not become net food producers until age 17 years, and they do not marry until about age 20 years (Hill and Kaplan 1988). In the Central Canadian Arctic, Inuit people living as traditional hunters did not even consider an adolescent boy ready for marriage until he was 17–18 years old (Condon 1990). Even then, the adolescent had to provide bride service to his prospective in-laws for several years before he became a father. These delays in fatherhood occurred despite the fact that there was considerable pressure to reproduce because of "the slim margin of survival in the precontact period" (p. 270).

The explanation for the lag between sperm production and fatherhood is not likely to be a simple one of sperm performance, such as not having the endurance to swim to an egg cell in the woman's fallopian tubes. More likely is the fact that the average boy of 13.4 years is only beginning his adolescent growth spurt (Fig. 11.7). Growth researchers have documented that in terms of physical appearance, physiological status, psychosocial development, and economic productivity, a 13-year-old boy is still more of a juvenile than an adult. Anthropologists working in many diverse cultural settings report that few women (and more importantly from a cross-cultural perspective, few prospective in-laws) view the teenage boy as a biologically, economically, and socially viable husband and father.

The delay between sperm production and reproductive maturity is not wasted time in either a biological or social sense. The obvious and the subtle psychophysiological effects of **testosterone** and other androgen hormones that are released after gonadal maturation may "prime" boys to be receptive to their future roles as men. Alternatively, it is possible that physical changes provoked by the endocrines provide a social stimulus toward adult behaviors. Whatever the case, early in adolescence, sociosexual feelings including guilt, anxiety, pleasure, and pride intensify. At the same time, adolescent boys become more interested in adult activities, adjust their attitude to parental figures, and think and act more independently. In short, they begin to behave like men. However—and this is where the survival advantage may lie—they still look like boys. One might say that a healthy, well-nourished 13.5-year-old human male, at a median height of 160 cm (62 in.) "pretends" to be more childlike than he really is (Smith 1993).

During the adolescent years, boys are even shorter than girls of roughly the same chronological age, furthering an immature image (Fig. 11.7; Tobias 1970). Even more to the point is that the spurt in muscle mass of adolescent males does not occur until an average age of 17 years (Fig. 11.14; Malina 1986). At peak height velocity during the skeletal growth spurt, the typical adolescent boy has achieved 91% of his adult height, but only 72% of his adult lean body mass. Since most of the lean body mass is voluntary muscle tissue, adolescent boys cannot do the work of men. This is one important reason why adults of the Kikuyu, the Inuit, and many other cultures do not even think of younger adolescent boys as manlike.

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As Schlegel and Barry (1991) found in their cross-cultural **survey**, adolescent boys are usually encouraged to associate and "play" with their age mates rather than associate with adult men. During these episodes of "play," these juvenile-looking adolescent males are protected from many dangers of adult male life and they can practice behaving like adult men before they are actually perceived as adults. The activities that take place in these adolescent male peer groups include the type of productive, economic, aggressive/militaristic, and sexual behaviors that older men perform. But the sociosexual antics of adolescent boys are often considered to be more humorous than serious. Yet, they provide the experience to fine tune their sexual and social roles before either their lives or those of their offspring depend on them. For example, competition between men for women favors the older, more experienced man. As such competition may be fatal, the juvenile-like appearance of the immature, but hormonally primed, adolescent male may be life-saving, as well as educational.

When Did Childhood and Adolescence Evolve?

The stages of the life cycle may be studied directly only for living species. However, we can postulate on the life cycle of extinct species. Such inferences for the hominins are, of course, hypotheses based on comparative anatomy, comparative physiology, comparative ethology, and archaeology. Examples of such methods are found in the work of Martin (1983) and Harvey et al. (1987) on patterns of brain and body growth in apes, humans, and their ancestors.

We may only estimate at which point in human evolution childhood appeared, although we can even see a hint of it in chimpanzees, where young are known to stay in close association with their mothers for another 2 years after weaning (Goodall 1986). It is most likely that childhood and its extension coevolve with the increase in brain size and concomitant slowing of overall rate of growth and development so evident in our genus *Homo*.

Apes have a pattern of brain growth that is rapid before birth and relatively slower after birth. In contrast, humans have rapid brain growth both before and after birth (Fig. 11.23). This difference may be illustrated by comparing simple **ratios** of brain weight divided by total body weight. The data are given in Table 11.3. At birth, this ratio averages 0.09 for the great apes and 0.12 for humans, showing that in proportion to body size, humans are born with brains that average 1.33 times larger than those of the apes. At adulthood, the ratio averages 0.006 for the great apes and 0.028 for humans, meaning that the difference between apes and humans in the brain-to-body size proportion has increased to 4.7 times. It is the faster rate of human brain growth after birth that accounts for most of the difference. Indeed, the rate of human brain growth exceeds that of most other tissues of the body during the first few years after birth (Fig. 11.10).

Martin's analysis of ape and human trajectories of growth suggests that a "human-like" pattern of brain and body growth becomes necessary after adult hominin brain size reaches ~850 cc. This biological marker is based on an analysis of the size of the head of the fetus and the size of the pelvic inlet (birth canal) of the mother across a wide range of social mammals, including the living primates and fossil hominins. Given the mean rate of postnatal brain growth for living apes, an 850-cc adult brain size may be achieved by all hominoids, including extinct hominins, by

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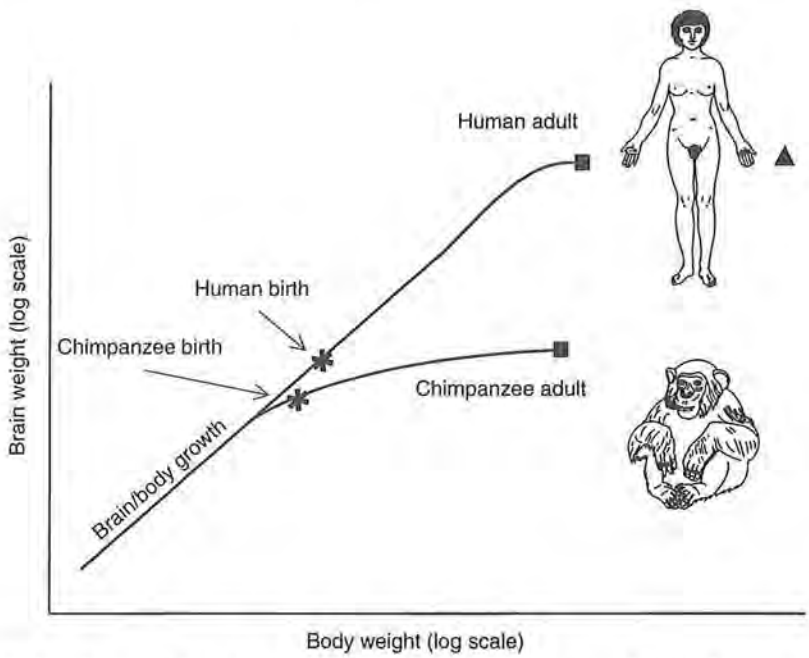

Figure 11.23 Brain and body growth curve for humans compared with chimpanzees. The length of the human fetal phase, in which brain and body grow at the same rate for both species, is extended for humans. In chimpanzees, brain growth slows after birth, but in humans, the high rate of brain growth is maintained during the postnatal phase. In contrast, the rate of human body growth slows after birth. If human brain and body growth rates were equal to those of chimpanzees, then adult humans would weigh 454 kg (998.8 lb) and stand nearly 3.1 m tall (9.9 ft). That body size is indicated by the "▲" symbol.

TABLE 11.3 Neonatal and Adult Brain Weight and Total Body Weight for the Great Apes and Human Beings

Species	Neonatal Weight (g)		Adult Weight (g)	
	Brain	Body	Brain	Body
Pongo (orangutan)	170.3	1728.0	413.3	53,000.0
Pan (chimpanzee)	128.0	1756.0	410.3	36,350.0
Gorilla	227.0	2110.0	505.9	126,500.0
Homo sapiens	384.0	3300.0	1250.0	44,000.0

Adult body weight is the average of male and female weight. The averages obscure the sexual dimorphism that exists in each species. The values given here are useful only in the context of the discussion of brain-to-body size presented in this chapter.

Source: Data from Harvey et al. (1987).

lengthening the fetal stage of growth. At brain sizes >850 cc, the size of the pelvic inlet of the fossil hominins and living humans does not allow for sufficient fetal growth. Thus, a period of rapid postnatal brain growth and slow body growth—the human pattern—is needed to reach adult brain size.

From this analysis, we can see clearly why so much of human postnatal growth and development is intimately associated with brain size. We presented earlier the

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figures on the percent of RMR due to brain growth and activity. The relation of human life history to our large and active brain can be looked at as an energetic problem. Large brains are costly investments; recall that the adult human brain uses 20% of RMR, whereas the chimpanzee uses only 9% and an average marsupial uses only 2%. Moreover, larger brains have lower tolerances for temperature extremes, blood pressure, and oxygenation. The large human brain may increase obstetric risks (birth defects and maternal death). The costs are potentially high, but what is the payoff? The explanation we favor here is that a large brain is an investment that pays off immediately to infants, children, juveniles, and adolescents, as well as on a long timescale to adults. An organism recoups its energetic "investment" in a large brain through complex behavior, which is itself a combined product of large brains, slow development, extended care by older individuals, enhanced learning, and phenotypic plasticity, among other influences (Smith and Tompkins 1995). The greatest benefits of large brains probably accrue slowly over a long life, by building embodied capital (an individual's physical and social resources; Kaplan et al. 2000) as well as cognitive **reserve capacity** (Bogin 2009). There are also immediate benefits of a large brain in terms of complex behaviors that elicit care, feeding, and protection, such as smiling, social engagement, and language. These behaviors develop soon after birth and become well established in human infants and children (Kagan et al. 1980; Kagan 2003; Locke and Bogin 2006). For primates in general and humans in particular, much of life history may support a substantial investment in brains and what they can do in terms of food production and successful reproduction (Smith 1990; Bogin 1993).

We are, perhaps, fortunate that brains are so important, because after teeth and jaws, skulls are one of the more common pieces of fossil evidence preserved in the record of primate evolution. Having skulls, or at least sufficient skull parts to reconstruct the whole, allows paleontologists to estimate brain size. Having teeth and jaws in relative abundance is also fortuitous because of the strong correlation between tooth formation and eruption with so many other life history events.

Given this background, we present in Figure 11.24 our summary of the evolution of the human pattern of growth and development. This figure must be considered as "a work in progress," because only the data for *Pan* (chimpanzees) and *Homo sapiens* are known with some certainty. Known ages for eruption of the M1 are given for *Pan* and *H. sapiens*. Estimated ages for M1 eruption in other species were calculated by Smith and Tompkins (1995). Brain size is another crucial influence on life history evolution, and known or estimated adult brain sizes are given at the top of each bar. These values are averages based on skulls from the fossil record.

The hominin fossil record now appears to stretch back more than 6 million years with the appearance of *Sahelanthropus*, *Orrorin*, and *Ardipithecus* in Africa (Wood and Lonergan 2008; White et al. 2009). These earliest known forms appear to have had brains no larger than the living bonobo and chimpanzee, a mere 300–350 cc in *Ardipithecus ramidus* (ca. 4.4 MYA) and less than 400 in *Sahelanthropus*. We begin to know something more directly of infant and juvenile life by the appearance of *Australopithecus afarensis* about 3.9 MYA. Here the Dikika infant, about 3 years of age at death by its teeth, already has a brain size of 330 cc; adult *A. afarensis* reached an adult brain size of about 400 cc with a pattern of dental development a little different from extant apes. Therefore, the chimpanzee and *A. afarensis* are depicted as sharing the typical tripartite stages of postnatal growth of social

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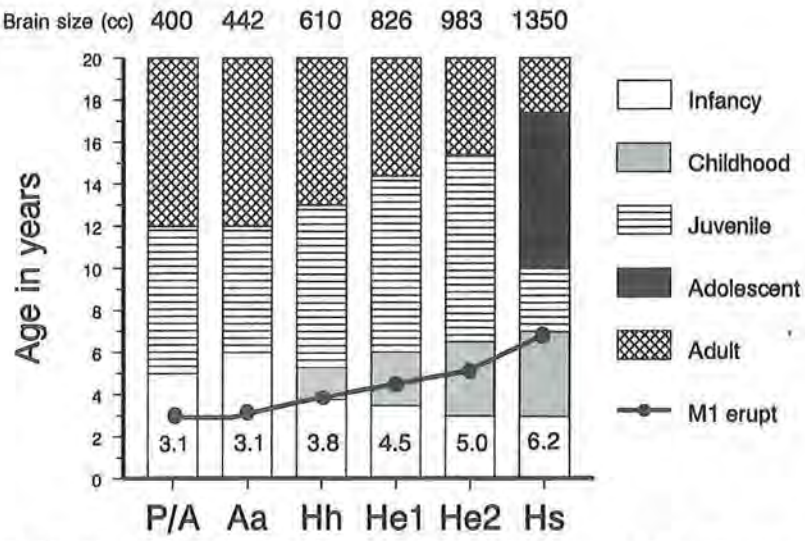

Figure 11.24 The evolution of hominin life history during the first 20 years of life. Abbreviation of the pongid and hominin taxa are P/A, *Pan, Australopithecus afarensis*; Aa, *Australopithecus africanus*; Hh, *Homo habilis*; He1, early *Homo erectus*; He2, late *Homo erectus*; Hs, *Homo sapiens*. *Source*: Bogin (1999).

mammals—infant, juvenile, and adult. In Figure 11.24, the duration of each stage and the age at which each stage ends are based on empirical data for the chimpanzee.

A probable descendant of *A. afarensis* is the fossil species *Australopithecus africanus*, dating from about 3.0 MYA. To achieve the larger adult brain size of *A. africanus* (average of 442 cc) may have required an addition to the length of the fetal and/or infancy periods. Figure 11.24 indicates an extension to infancy of 1 year.

The first permanent molar (M1) of the chimpanzee erupts at about 3–4 years, but chimpanzees remain in infancy until about age 5 years. Until that age, the young chimpanzee is dependent on its mother, and is highly unlikely to survive if the mother dies or is otherwise not able to provide care and feeding. After erupting M1, the young chimpanzee may be able to eat adult-type foods, but is still learning how to find and process foods. Learning to successfully open fruits that are protected by hard shells and to extract insects from nests (such as ants and termites) requires more than 1 year of observation and imitation by the infant of the mother. For these reasons, chimpanzees extend infancy for more than 1 year past the eruption of M1.

With a body and brain size near that of the chimpanzee, it is likely that these early hominins followed a pattern of growth and development very similar to chimpanzees. Moreover, detailed study of incremental growth in teeth confirms that in *A. africanus*, the M1 erupted at about 3.1 years of age (Bromage and Dean 1985), and we know that tooth emergence is an excellent indicator of the pace of development of the individual and the species (Smith 1989). Behavioral capacities of these fossil hominins also seem to be similar to living chimpanzees. Like the living chim-

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panzee, the early hominins may also have extended infancy for at least 1 year beyond the age of M1 eruption.

At about 2.2 MYA, we find fossils with several more humanlike traits, larger cranial capacities, and greater manual dexterity. Also dated to about this time are stone tools of the Oldowan tradition. To date, manufacture of stone tools can be traced approximately 2.5 million years ago, although use of (possibly natural) stone to cut carcasses has been claimed to extend earlier (McPherron et al. 2010). Given the biological and cultural developments associated with these fossils, they are considered by most paleontologists to be members of the genus *Homo* (some researchers split early *Homo* into two species, *Homo habilis* and a larger *Homo rudolfensis*). The rapid expansion of adult brain size during the time of *H. habilis* (650–800 cc) might have been achieved with further expansion of both the fetal and infancy periods, as Martin's "cerebral Rubicon" was not surpassed. However, the insertion of a brief childhood stage into hominin life history may have occurred. Tardieu (1998) showed that *H. habilis* has a pattern of growth of the femur that is distinct from that of the australopithecines, but consistent with that of later hominins. The distinctive femur shape of the more recent hominins may be due to the addition of a prolonged childhood stage of growth. *H. habilis*, then, may have had a short childhood stage of growth.

For these reasons, a brief childhood stage for *H. habilis* is indicated in Figure 11.24. This stage begins after the eruption of M1 and lasts for about 1 year. That year of childhood would still provide the time needed to learn about finding and processing adult-type foods. During this learning phase, *H. habilis* children would need to be supplied with special weaning foods. There is archaeological evidence for just such a scenario. *H. habilis* seems to have intensified its dependence on stone tools. There are both more stone tools, more carefully manufactured tools, and a greater diversity of stone tool types associated with *H. habilis*. There is considerable evidence that some of these tools were used to scavenge animal carcasses, especially to break open long bones and extract bone marrow. This behavior may be interpreted as a strategy to feed children. Such scavenging may have been needed to provide the essential amino acids, some of the minerals, and, especially the fat (dense source of energy) that children require for growth of the brain and body.

A childhood stage of growth for the earliest members of the genus *Homo* is also supported by a comparison of human and ape reproductive strategies. There are limits to the amount of delay possible between birth and sexual maturity, and between successful births, that any species can tolerate. The great apes are examples of this limit. Chimpanzee females in the wild reach menarche at 11–12 years of age and have their first births at an average age of 14 years—later than any other mammal on earth, excepting elephants and living humans. The average period between successful births in the wild is 5.6 years, as infant chimpanzees are dependent on their mothers for about 5 years. Actuarial data collected on wild-living animals indicate that between 35% and 38% of all live-born chimpanzees survive to their mid-20s. Although this is a significantly greater percentage of survival than for most other species of animals, the chimpanzee is at a reproductive threshold. Goodall (1983) reported that for the period 1965–1980, there were 51 births and 49 deaths in one community of wild chimpanzees at the Gombe Stream National Park, Tanzania. During a 10-year period at the Mahale Mountains National Park, Tanzania, Nishida et al. (1990) observed "74 births, 74 deaths, 14 immigrations and

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13 emigrations" in one community. Chimpanzee population size in these two communities is, by these data, effectively in equilibrium. Any additional delay in age of females at first birth or the time between successful births would likely result in a decline in population size.

The great apes and extinct hominins, such as *Australopithecus*, may have reached a demographic limit by extending the length of the infancy stage and requiring enormous direct maternal investment in each offspring and long interbirth intervals. Somewhere in our history, however, hominins began to reverse the trend, producing offspring in more rapid succession. An often cited example, the !Kung is a traditional hunting and gathering society of southern Africa. A !Kung woman's age at her first birth averages 19 years and subsequent births follow about every 3.6 years, resulting in an average fertility rate of 4.7 children per woman (Short 1976; Howell 1979). Women in another hunter–gather society, the Hadza, have even shorter intervals between successful births, stop nursing about 1 year earlier, and average 6.15 births per woman (Blurton Jones et al. 1992). The key seems to be that humans wean infants before they can feed themselves, freeing mothers from the demands of nursing and the physiological brake that frequent nursing places on ovulation, allowing mothers to reproduce again much sooner. These early-weaned infants are by definition "children," still dependent on others for feeding, but no longer supplemented by mother's milk.

Further brain size increase occurred during *Homo erectus* times, which began about 1.8 MYA. The earliest adult specimens have mean brain sizes of 826 cc, but many individual adults had brain sizes between 850 and 900 cc. As shown by an adult female *H. erectus* pelvis 1.8 MYA from Gona, Ethiopia, pelves are now more obstetrically capacious for giving birth to a larger-brained infant, as large as 315 cc (Simpson et al. 2008). Judging from Gona, *H. erectus* may give birth to an offspring with 35% of adult brain size, intermediate between chimpanzees (40%) and humans (28%) (Simpson et al. 2008). With adult *H. erectus* at or above Martin's "cerebral Rubicon" and new more direct evidence for an intermediate more helpless neonate, we expect that *H. erectus* required some degree of a postnatal rapid catch-up in brain growth. Although a more helpless infant would require even more intense care and an even longer infancy, at some point, hominins actually shrink infancy, substituting and expanding childhood rather than ever-increasing the infant stage, eventually dropping the transition from infancy down to before the eruption of M1 and the permanent teeth.

From other fossils of *H. erectus*, we even know that the timing of M1 has evolved, changing from the ca. 3–3.5 years of earliest hominins to about 4.5 years (Dean and Smith 2009). In all, the fossil record indicates that *H. erectus* evolved slowed general maturation, more helpless infants, larger adult brains, and increasing sophisticated tools—all of which adds to an adaptive network of higher-quality offspring, long and intense learning, and reliance on complex behavior. Taken together with evidence of larger body size in females, Aiello and Key (2002) pointed out the enormous energy demands for reproduction in an *H. erectus* female, demands that necessitate change in energetic strategies. Whenever it did evolve, a childhood period would at first diminish the reproductive cost of this "high-quality strategy," and eventually allow the strategy to become even more extreme. At some point, perhaps even with *H. erectus*, hominins shrank the infancy period to below that of chimpanzees, which would have given them a greater reproductive advantage than any previous hominin. The fact that *H. erectus* populations certainly did increase

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in size and began to spread throughout Africa and into other regions of the world suggests that fundamental changes in life history had already begun.

Later *H. erectus*, with average adult brain sizes of 983 cc, are depicted in Figure 11.24 with further expansion of the childhood stage. In addition to bigger brains (some individuals had brains as large as 1100 cc), the archaeological record for later *H. erectus* shows increased complexity of technology (tools, fire, and shelter) and social organization. These techno-social advances, and the increased reliance on learning that occur with these advances, may well be correlates of changes in biology and behavior associated with further development of the childhood stage of life (Bogin and Smith 1996). The evolutionary transition to archaic, and finally, modern *H. sapiens* expands the childhood stage to its current dimension. Note that M1 eruption becomes one of the events that coincides with the end of childhood. This is roughly the point that many mammals become independent juveniles, and as we discussed earlier, is the period in humans that introduces significant biological, cognitive, behavioral, and social changes.

With the appearance of *H. sapiens* comes evidence for the full gamut of human cultural capacities and behaviors. The technological, social, and ideological requisites of culture necessitate a more intensive investment in learning than at any other grade of hominin evolution. The learning hypothesis for childhood, while not sufficient to account for its origins, certainly plays a significant role in the later stages of its evolution.

The *H. sapiens* grade of evolution also sees the addition of an adolescent stage to postnatal development. The single most important feature defining human adolescence is the skeletal growth spurt that is experienced by virtually all boys and girls. There is no evidence for a humanlike adolescent growth spurt in any living ape. There is no evidence for adolescence for any species of *Australopithecus*. There is some tentative evidence that early *Homo*, dating from 1.8 MYA, may have a derived pattern of growth that is leading toward the addition of an adolescent stage of development. As mentioned above, this evidence is based on an analysis of shape change during growth of the femur (Tardieu 1998). Modern humans have highly diagnostic shape to the femur, a shape that is absent in fossil ascribed to *Australopithecus*, but present in fossils ascribed to *H. habilis*, *H. rudolfensis*, *Homo ergaster*, or early African *H. erectus*. The human shape is produced by growth changes during both the prolonged childhood stage and the adolescent stage, but whether the two are inextricably linked remains conjectural.

A remarkable fossil of early *H. erectus* is both of the right age at death and complete enough to allow for an analysis of possible adolescent growth. The fossil specimen is cataloged formally by the name KMN-WT 15000, but is called informally the "Turkana boy" as it was discovered along the western shores of Lake Turkana in 1984. This fossil is 1.6 million years old, and clearly an early variety of *H. erectus*. The skeletal remains are almost complete, missing the hands and feet and a few other minor bones. Smith (1993) and more recently Dean and Smith (2009) analyzed the skeleton and dentition of the Turkana fossil and ascertained that, indeed, it is immature and most likely a male. The youth's deciduous upper canines were still in place at the time of death, and he died not long after erupting the second permanent molars, an event that occurs about the time of puberty in male higher primates (Smith 1993; Fig. 9.2). These dental features place him firmly in the juvenile stage by comparison with any primate. Even though we have most of his skeleton, we cannot be exact about his stature at death: earlier estimates of 160 cm have been

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lowered recently to 154 cm (Graves et al. 2010). Even so, the Turkana boy is one of the tallest fossil youths or adults ever found.

The Turkana youth is sufficiently complete to study his pattern of growth and development, and ask "Did early *H. erectus* have an adolescent growth spurt?" At present, the best answer seems to be "no." Judged according to modern human standards, the Turkana boy's dental age of 10.5–11 years is in some conflict with his bone age (skeletal maturation) of 13 years and his stature age of 15 years. If the Turkana boy grew along a modern human trajectory, then dental, skeletal, and stature ages should be about equivalent. Skeletal and dental ages this discrepant are known in less than 1.5% of normal boys age 6–15, and it is particularly rare to see the skeleton advanced over the dentition (Dean and Smith 2009). By chimpanzee growth standards, however, the boy's dental and bone ages are in perfect agreement, both suggesting 7 years of age. So was he 13 or 7? Recently, Dean and Smith (2009) answered this question by counting evidence of time passing in growth increments preserved in the teeth of the Turkana boy (analogous to counts of growth rings in trees). They found that dental microanatomy could account for about 8.5 years of life for the youth. If this is correct, death at age 8.5 means the Turkana boy followed a timing of growth that is neither that of a modern human nor that of a chimpanzee. His relatively large stature for age becomes understandable if we suppose that the distinct human pattern of moderate to slow growth prior to puberty followed by an adolescent growth spurt had not yet evolved in early *H. erectus*. Rather, the Turkana boy followed a more apelike pattern of growth in stature, completing a fairly high proportion of adult growth by the onset of puberty and emergence of second molars (see also Graves et al. 2010). At the time of puberty, the chimpanzee has usually achieved 88% of stature growth, while humans have achieved only 81%. "Because of this, any early *H. erectus* youth would seem to us to be too large" (Smith and Tompkins 1995, p. 273).

Unfortunately, there are no appropriate fossil materials of later *H. erectus* available to analyze for an adolescent growth spurt. There are several fossils of a species called *Homo antecessor*, found in Spain and dated to about 800,000 BP (Bermudez de Castro et al. 1999). *H. antecessor* has been proposed as a possible last common ancestor between modern *H. sapiens* and Neanderthals (Bermudez de Castro et al. 1997), although recovery of new material has led the research group to emphasize links with eastern hominins, with increasing uncertainty as to their contribution to later European populations (Carbonell et al. 2005). Based on an analysis of tooth formation, this species seems to have a pattern of dental maturation much like modern humans, but there is as yet no juvenile with both teeth and skeleton for an in-depth analysis of growth patterns. Even if we are left only with teeth, at some point, dental microanatomy should give us real answers to the ages that these and other hominins attain growth markers, although such information is slow to appear because it often involves destructive methods such as cutting fossilized teeth into sections.

For later hominins like the Neanderthals, we have much more complete material. There is one fossil of a Neanderthal in which the associated dental and skeletal remains needed to assess adolescent growth are preserved. Le Moustier 1, found in 1908 in western France, is a juvenile, most likely a male. The specimen is dated at between 42,000 and 37,000 years BP (before present). Thompson and Nelson (2000) used information on crown and root formation of the molar teeth to estimate a

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dental age of 15.5 ± 1.25 years. His dental development—beyond M2 emergence and with third molars well developed—clearly indicates a late adolescent. Compared with modern human standards for length of the long bones of the skeleton, Thompson and Nelson estimated that Le Moustier 1 has a stature age of about 11 years and had achieved about 87% of adult femur length. The dental age of 15.5 years and the stature age of 11 years are in very poor agreement, and indicate that like the Turkana boy, Le Moustier 1 may not have followed a human pattern of adolescent growth. In contrast to the Turkana boy, however, he seems short for his dental age. Although we expect death and cemetery samples to preferentially represent the undernourished and growth delayed, Thompson and Nelson cannot match the Le Moustier pattern of development even in cemetery samples of prehistoric Inuit.

What is clear is that the pattern of adolescent growth found in modern humans does not seem to be present in either the Turkana boy or in the Le Moustier 1 fossil. The future promises that we will soon be able to solve some of the mysteries about Neanderthal life history. New technical advances allowing nondestructive dental anatomy are beginning to yield ages of death for Neanderthal juveniles long considered too precious for destructive studies. Recent work by Smith et al. (2007b) finds evidence for more rapid growth and development in Neanderthals than expected for modern humans. And indeed, work of the last decades on Neanderthals, including DNA analysis, has tended to emphasize their differences from modern peoples and it is increasingly common to see Neanderthals placed in their own separate species *Homo neanderthalensis* (e.g., Rak et al. 2002 and Green et al. 2008). Even so, as more of the Neanderthal genome has become known, comparisons find more genetic variants of Neanderthals shared with Eurasian than sub-Saharan African modern populations, suggesting that Neanderthals and early moderns did interbreed to some degree (Green et al. 2010). These recent more robust genetic studies of ancient DNA promise both to challenge paleontology and ultimately to lead to synthesis of the findings of the two fields. In modern humans, certain diseases, prolonged undernutrition, and unusual individual variations in growth may produce similarities to the skeletal and dental features of the Turkana boy and Le Moustier 1 fossils. While it is possible that these two specimens fall into one of these categories of unusual growth, the most parsimonious conclusion that one may draw from these findings is that all the features of the modern human adolescent stage of the life cycle evolved only in the *H. sapiens* line. Quite likely this would be no earlier than the appearance of archaic *H. sapiens* in Africa, and to date, this is our earliest hard evidence of more modern growth and development. An archaic *H. sapiens* fossil child from Jebel Irhoud, Morocco, dated at about 160,000 YBP, has become the earliest fossil for which the assigned human dental age matches the actual age of death (Smith et al. 2007a).

The Valuable Grandmother, or Could Menopause Evolve?

In addition to childhood and adolescence, human life history has another unusual aspect: menopause. One generally accepted definition of menopause is "the sudden or gradual cessation of the menstrual cycle subsequent to the loss of ovarian function" (Timiras 1972). The process of menopause is closely associated with but distinct from the adult female postreproductive stage of life. Reproduction usually ends before menopause. In traditional societies, such as the !Kung, the Dogon of Mali,

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and the rural-living Maya of Guatemala, women rarely give birth after age 40 and almost never give birth after age 44. Even in the United States from 1960 to today, with modern health care, good nutrition, and low levels of hard physical labor, women rarely gave birth after age 45. This is even true among social groups attempting to maximize lifetime fertility, such as the Old Order Amish.

As among the !Kung, Dogon, and Maya, menopause occurs well after this fertility decline, at a mean age of 49 years for living women in the United States. After age 50, births are so rare that they are not reported in the data of the U.S. National Center for Health Statistics or by the Amish (even if sensationalized in the tabloids sold at supermarket checkouts!). We report these ages for the onset of human female postreproductive life versus the ages for menopause because some scholars incorrectly equate menopause with the beginning of the postreproductive stage, so one must read the literature carefully to interpret in what sense the term "menopause" is used. Menopause, and a significant period of life after menopause, has a unique effect on the life of human females. Although elderly primates also experience a degradation of fertility and may even cease reproductive cycling (e.g., Margulis et al. 2007), their menopause accompanies senescence. In a review of the data for mammals, Packer et al. (1998) reported that "reproductive cessation has also been documented in non-human primates, rodents, whales, dogs, rabbits, elephants and domestic livestock" (p. 807). Packer and colleagues found that these declines in nonhuman mammals are best interpreted as a normal part of senescence (see also Chapter 13 by Crews and Ice of this book). Many body systems are failing in these nonhuman females and death follows relatively soon after reproduction ceases. Wild-living nonhuman primate females do not share the universality of human menopause, and human males have no comparable life history event.

In contrast to the nonhuman female primates, the human female reproductive system is "shut down" well before other systems of the body. Human women may be healthy at menopause and may have 20 or more years of relatively vigorous and active life following menopause. Why are human women different from the females of other primate species?

The building of reserve capacity, that is, healthier bodies with greater physical, cognitive, and emotional resilience, is one hypothesis for why humans live long past the point of reproductive decline (Bogin 2009). There are several other hypotheses for the evolution of menopause and a postreproductive life stage, including various versions of the "grandmother hypothesis." In the chapter by Crews and Ice, some of these hypotheses are reviewed in detail. Suffice it to state here that in terms of basic biology, it appears that a 50-year age barrier exists to female primate fertility because by that age, all oocytes are depleted. Total body size seems to matter, because very large mammal females, such as elephants and some whales, are fertile beyond age 50 years. In terms of natural selection, the loss of fertility hardly matters because by the age of 50 years, the females of most primate species, indeed most mammalian species, are dead. The few exceptions are, again, elephants, some whales, and humans. Much ethnographic evidence shows that significant numbers of women in almost every human society, traditional and industrial, live for many years after oocyte depletion (menopause). The historical evidence shows that this was possibly true in the human past for at least 50,000 years (Caspari and Lee 2006; see Hawkes and O'Connell 2005 for an alternative interpretation). Given the biological age limit to fertility, one reproductive strategy open to postmenopausal human women is to

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provide increasing amounts of aid to their offspring and their grandoffspring. The ethnographic evidence shows that human grandmothers and other postreproductive women are beneficial to the survival of children in many human societies. Grandmothers provide food, child care, and a repertoire of knowledge and life experiences that assist in the education of their grandchildren. In sum, the inevitabilities of primate biology, combined with the creativity of human culture, allow women, and men, of our species to develop biocultural strategies to take the greatest advantage of a postreproductive life stage. Viewed in this biocultural context, human grandmotherhood may be added to human childhood and adolescence as distinctive stages of the human life cycle.

BOX 11.3 COLLECTING AND COMPARING ETHNOGRAPHIC DATA ON THE TREATMENT OF CHILDREN, ADOLESCENTS, AND GRANDMOTHERS IN SEVERAL CULTURES

To better understand the concept of a biocultural model of human growth presented in this chapter, we recommend the following exercise. Seek out official government agency reports, or analyses that reference such reports, concerning the treatment of children, adolescents, and the **elderly**—especially grandmothers. We emphasize these life history stages because they are highly developed and important to the human species, at least among the primates. Look for data regarding health, the most common diseases and conditions, any incidence of abuse and neglect, and economic and political status. Students concentrating in subject areas relating to children, adolescents, or the elderly may think of other topics for research.

The data for this exercise may be found in various places. Many large university libraries are repositories for official government reports. There are many electronic and Internet sources for these data. The reports of the United States National Center for Health Statistics (NCHS; http://www.cdc.gov/nchs/Default.htm), the Demographic and Health Surveys (http://www.measuredhs.com/start.cfm), The World Bank Data Catalog (http://data.worldbank.org/data-catalog), and the United Kingdom Data Archive (UKDA; http://www.data-archive.ac.uk/) are useful. Social service agencies and charitable organizations devoted to children or the elderly may have these reports in addition to their own publications.

When searching for data, each student or group may chose a life history stage, a particular country in the world, or an ethnic or social group found in one or more countries. Devise a method to record the data from each study so that you can compare findings. Use the data collection form from Box 11.2 as an example to assist in this task. Some of the data will be statistical in nature. You may enter these values into a database for statistical analysis, as you did for the life history data. Other information may be in the form of narratives, which will require a nonstatistical analysis.

Be creative when thinking about ways to make sense of all the data. Remember, the goal of this exercise is to see how human biology and culture interact and influence each other in terms of growth, development, and life history stages.

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LIFE CYCLE TRADE-OFFS AND RISKS FOR CHILDREN, ADOLESCENTS, AND POSTMENOPAUSAL WOMEN

The evolution of new human structures, functions, and stages of life history may have brought about many biocultural benefits; however, these also incur trade-offs and risks. In earlier sections of this chapter, we mentioned these risks and promised to discuss them in more detail. We do so in this section. Bipedalism, the method of human locomotion unique among the primates, is one example of evolutionary benefits and risks. Often considered to be one of the crucial feeding and reproductive adaptations of our species, bipedalism also brings about many physical ailments, including lower back pain, fallen arches, and inguinal hernias. Similarly, the benefits of childhood need to be tempered against the hazards of this developmental stage. Dependency on older individuals for food and protection, small body size, slow rate of growth, and delayed reproductive maturation convey liabilities to the child. In the past 20 years, the global **epidemic** of HIV/AIDS has claimed the life of millions of young adults, often leaving their children with no biological parent. Africa has been burdened most severely, with at least 50 million orphans as the legacy of AIDS and other diseases, as well as war and high rates of death in pregnancy and childbirth (*New York Times*, June 25, 2009). The "charms" of children and childhood do not provide for total security.

To illustrate this point, one can examine traditional societies of both historic and prehistoric eras. In such societies, including hunter–gatherers and horticulturists, ~35% of live-born humans died by age 7—that is, by the end of childhood (Bogin 2001; see also Chapter 14 by Gage et al. of this book). Even if two-thirds of these deaths occurred during infancy, the childhood period still had an appreciable risk of death. In hunter–gatherer societies, starvation, accidents, and predation accounted for most childhood deaths.

Today, hunter–gatherer and traditional horticultural societies account for <1% of human cultures, but trade-offs and risks for children remain. Even in the wealthiest nations, and with programs of legal protection and welfare for children enacted in the 20th century, many risks remain; abuse and neglect are two. One estimate of the worldwide mortality from abuse and neglect is between 13 and 20 infants and children per 1000 live births (Bogin 2001). The incidence of all suffering from abuse and neglect is probably higher but very difficult to estimate because data are not reported by most nations. Some industrialized nations do maintain statistics for nonfatal abuse and neglect of children. In the United States, for example, "in 1991 2.7 million abused or neglected children were reported to child protection agencies" (Kliegman 1995). These numbers constitute a rate of 38.6 children per 1000. In a worldwide survey of data from 153 countries, Helander (2008) reported that some form of violence, ranging from physical punishment at home to regional and global warfare, "has victimized about half of the world's population" (p. xi) of children. The Kliegman and Helander studies use the term "children" in reference to anyone under 18 or 15 years of age. Even so, their findings encompass a large number of children as defined in this chapter.

Some cases of child abuse, neglect, and violence may result from a severance between the biology of childhood and the rapid pace of technological, social, and ideological change relating to families and their children. It is now technologically possible to nourish infants without breast-feeding, and this development allows

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parents (mothers) an opportunity to pursue economic activities or have another baby. Among the poor populations of the developing countries, short birth intervals (<23 months) may compromise the health of both the infant and the mother (Dewey and Cohen 2007). A major negative effect on the infant is LBW, which impairs both physical growth and cognitive development during childhood and later life stages.

In the populations of the more developed nations, such as among the U.S. middle class, ~20% of infants are breast-fed through 6 months of age. The weaning process—from bottles and formula—may begin by 3 months of age, severely curtailing infancy. These "premature children" present a problem for care, because they are still biologically in the infancy stage of development. The problem is often "solved" by relegating these young to restraining devices such as high chairs, playpens, and cribs/cots or segregating them from the family by placement in daycare centers or preschools. When the employees of these centers and preschools are trained properly, these arrangements are suitable. However, if not well trained, and especially if the infants react poorly to these arrangements, the frustrated parents or caregivers may respond with abusive or neglectful behavior. In addition, the tendency to live in nuclear families in middle class Western societies, away from grandmothers and aunts, deprives young mothers of other caretakers and shared experience of raising children.

Rapid culture change can introduce considerable discord into a previously harmonious relationship between human biology and behavior. Two brief examples that impact growth and development may be offered here. First, Kenyan mothers with more formal (European style) education believe that sibling care responsibilities teach juveniles to be passive and that domestic work, including child care, is menial (Weisner 1987). Second, forced settlement of the Inuit (hunters of North America) resulted in loss of their nomadic hunting lifestyle. This change required them to acculturate to settled life, the economics of wage labor—parents at work and children at school—and the social values of television (Condon 1990). Such shifts in values and behavior may present significant changes for future generations of Kenyans and Inuit. For example, the average juvenile may not learn about human growth and development until after the birth of his or her first infant. The consequences of these changes in social learning for the health, nutritional status, physical growth, and development of the next generation are less clear.

Human adolescence comes with a new set of risks. Among the most common and serious of these are psychiatric and behavioral disorders. The onset of such problems tends to peak during the adolescent life history stage (Paus et al. 2008). Most mammalian species terminate all brain growth well before sexual maturation, but human adolescents show enlargement and pruning of some brain regions leading to structural changes in the cerebral cortex. One hypothesis for the increase in brain-related disorders posits that these cortical changes leave the adolescent brain "more or less sensitive to reward" (Paus et al. 2008, p. 947).

The evolution of human menopause is associated with several risks for older women. The hormonal changes and bone loss that occur with the cessation of ovarian function (reviewed by Crews and Ice) are one kind of risk. These biological changes may bring about several degenerative diseases, such as **osteoporosis**. Postmenopausal women must often assume new social and economic roles for which they need adequate training and social support. But grandmothers, like their adolescent grandchildren, may no longer receive appropriate training, or have the

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desire, for evolutionarily traditional postreproductive sexual, social, and economic expectations in some "modern" societies.

The **elderly** may also be denied a productive social role and even be segregated away from productive society—in "retirement communities" for those who can afford it and "old age homes" for those of limited means. The social isolation that these sequestered elderly people may experience exacerbates the normal degenerative process of aging. Moreover, research in the wealthy nations shows that children living in households with little or no contact with grandparents suffer more abuse and neglect than children in multigenerational households—another testament to the value of grandparents.

CHAPTER SUMMARY

In this chapter, we have taken a life history approach to the study of human growth and development. We reviewed several of the basic principles of human growth and development and set these basic principles in their evolutionary context. The older "genocentric" view of the regulation of DNA is giving way to a new perspective of epigenetic, environmental, and social regulation of the genome (Varki et al. 2008). This is especially the case during embryonic and fetal development, giving rise to the field of developmental origins of health and disease (DOHaD, www.mrc.soton.ac.uk/dohad/). The postnatal life cycle of the social mammals, including the nonhuman primates, has three basic stages of postnatal development: infant, juvenile, and adult. The human life cycle, however, is best described by six stages: infant, child, juvenile, adolescent, adult, and postreproductive woman. It is hypothesized that the new life stages of the human life cycle represent feeding and reproductive specializations of the genus *Homo*. Hominins prior to the genus *Homo* probably had life histories more similar to living apes than humans. These hominins seem to have lived at a pace nearly twice as fast as modern humans. The evidence from fossil remains of *H. habilis*, *H. erectus*, *H. neanderthalensis*, and early *H. sapiens* suggests that the elements of human life history evolved as a mosaic over more than 1 million years. It is increasingly evident that the complete "package" of modern human life history took shape with the evolution of *H. sapiens* and not before.

We have tried to take a biocultural perspective of human development, a perspective that focuses on the constant interaction taking place during all phases of human development, both between genes and hormones within the body and with the sociocultural environment that surrounds the body. Research from social anthropology, developmental psychology, endocrinology, primate ethology, physical anthropology, and human biology shows how the biocultural perspective enhances our understanding of human development.

We also discussed the risks of the new stages of human development, especially when these stages impact with culture change. Malnutrition, child abuse, and neglect of both infants, children, and the elderly are some of the risks. With this knowledge, we hope that some readers of this chapter will conduct new biocultural research on human growth and development. We also hope that the findings of your research may help bring about peaceful improvement in the social, economic, and political conditions of life and will lead to good growth for all human beings.

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