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Life History Trade-Offs in Human Growth: Adaptation or Pathology?

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ABSTRACT Human beings growing-up in adverse biocultural environments, including undernutrition, exposure to infection, economic oppression/poverty, heavy workloads, high altitude, war, racism, and religious/ethnic oppression, may be stunted, have asymmetric body proportions, be wasted, be overweight, and be at greater risk for disease. One group of researchers explains this as a consequence of "developmental programming" (DP). Another group uses the phrase "predictive adaptive response" (PAR). The DP group tends to view the alterations as having permanent maladaptive effects that place people at risk for disease. The PAR group considers the alterations at two levels of adaptation: (1) "short-term adaptive responses for immediate survival" and (2) "predictive responses required to ensure postnatal survival to reproductive age." The differences between the DP and PAR hypotheses are evaluated in this article. A life history theory analysis rephrases the DP versus PAR debate from disease or adaptation to the concept of "trade-offs." Even under good conditions, the stages of human life history are replete with trade-offs for survival, productivity, and reproduction. Under adverse conditions, trade-offs result in reduced survival, poor growth, constraints on physical activity, and poor reproductive outcomes. Models of human development may need to be refined to accommodate a greater range of the biological and cultural sources of adversity as well as their independent and interactive influences. Am. J. Hum. Biol. 19:631–642, 2007.

This article reviews some aspects of life history trade-offs in human growth under adverse environmental conditions. Definitions of life history theory and trade-offs are given below. The question guiding this review is: are the costs to human growth and development when living under adversity evidence of accommodation or, even adaptation, or are these costs evidence of suffering and failures of biological competence? The meaning of adaptation is contentious within biology and anthropology. A definition for its use in this article is given below. Adversity within human environments may include malnutrition, exposure to infection, economic oppression/poverty, heavy workloads, high altitude, war, racism, and religious/ethnic oppression. As may be seen from this list, the biological and the economic/sociocultural aspects of hardship are concomitant. Accordingly, it is best to consider human growth—indeed all of human biology—from a biocultural perspective (Goodman and Leatherman, 1998; Lasker, 1969; Stinson et al., 2000; Watts et al., 1975).

WHAT IS ADAPTATION?

The perspective taken in this article is that for mammals as a group, the biological adaptation of the individual has three components. These are: (1) survival, (2) productivity, including somatic growth, behavior (physical activity), and cognition, and (3) reproduction, measured as the quantity and quality of offspring over several generations. These are seen as adaptation because death at any point prior to reproduction negates survival. Insuffi-

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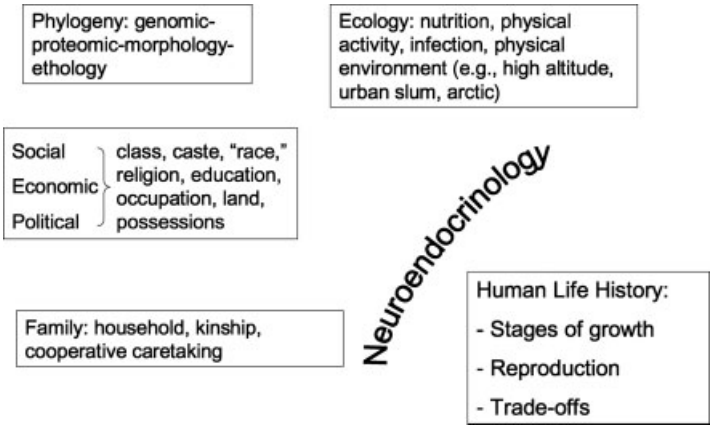

Fig. 1. Biocultural domains of influence on human life history. The "Phylogeny" domain has the most direct impact on genome, on the limits of the functional proteome, and on some aspects of human morphology and behaviors (such as the capacity for bipedalism). However, the "Phylogeny" domain has the most indirect influence on human life history, indicated by its distance from the "Human Life History" box, because it is mediated through the domains of "Ecology," "Social–Economic–Political," and "Family." All human beings share a highly similar "Phylogeny" domain, but live within highly variable ecologies, socioeconomic and political systems, and family groupings. The unevenness in these last three domains interacts with the phylogenetic factors to produce a range of variation in reaction norms of neuroendocrine production and activity. Neuroendocrine products have the most direct influence on the regulation of human life history, directing development through the stages of growth, adjusting the timing and frequency of reproduction, and modulating trade-offs in biology and behavior (original figure, based on Bogin, 2001).

cient productivity at any stage of life may lead to low, or no, reproduction and death. Nonreproduction by an individual mammal or its offspring is, in a genetic sense, equivalent to death of that individual.

Because of the biocultural nature of human beings, we must add additional domains to human adaptation. The individual members of all human societies require technological, sociological, and ideological systems for survival, productivity, and reproduction (Goldschmidt, 2005; White, 1959). Most of human adversity occurs along with disruptions to technological systems of food production and the distribution of other critical resources, disorder to family and other social systems, and the demeaning of local ideology. Warfare, colonialism, slavery, and ethnic/racial discrimination are a few examples of institutions that impose adversity on human populations.

LIFE HISTORY THEORY

Current research and practice in biology views adaptation in a life history theory perspective. One definition of life history theory is the study of the evolutionarily derived strategies used by organisms to allocate, "... energy toward growth, maintenance, reproduction, raising offspring to independence, and avoiding death. For a mammal, it is the strategy of when to be born, when to be weaned, how many and what type of prereproductive 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 histories is one of the most active areas of research in whole-organism biology" (Bogin and Smith, 2000). Detailed discussion of human life history theory may be found in Bogin (1999, 2001), Hawkes and Paine (2006), and Kaplan et al. (2000). A model of the biocultural nature the human species and how this influences human life history is shown in Figure 1. In the figure, the biocultural domains—phylogeny, ecology, social–economic–political, family—that influence human life history are shown as separate text boxes. The human neuroendocrine system is the proximate link between the biocultural domains illustrated and the architecture of human life history (Finch and Rose, 1995; see the legend of Fig. 1 for additional explanation).

A large part of life history theory focuses on the stages of growth from conception to maturity, on the timing of reproductive events, and on the inevitable trade-offs that occur in

growth and reproduction. "Trade-offs occur when two traits compete for materials and energy within a single organism ..." or, "... when selection for one trait decreases the value of a second trait" (Stearns, 1992, p 223).

HUMAN GROWTH UNDER ADVERSITY

Human beings growing-up in adverse environments tend to have reduced survival, productivity, and reproduction—the three aspects of the definition of adaptation given above. In terms of survival, there is a greater risk for fetal wastage as well as infant and child mortality (Bogin, 1999). In terms of productivity, those infants who do survive may begin life with low birth weight or grow to be stunted, have asymmetric body proportions (e.g., relatively short legs for total stature), be wasted during the growth years, be overweight as adults, and be at greater risk for both infectious and metabolic diseases (Bogin and Varela Silva, 2003; Frisancho, 2003; Varela Silva et al., 2007). Additionally, during the years of growth and as adults, the survivors of adversity are at elevated risk for physical and cognitive impairments to work capacity (Martorell, 1989, 1995). Finally, reproduction may be reduced as women with high energy expenditure, low energy intake, and infectious disease burden often have lowered fecundity and fertility (Ellison and O'Rourke, 2000).

The dominant biomedical and clinical paradigm of the past 50 years or more views these outcomes as evidence of pathology. There have been attempts, however, to conceptualize the presence of any fertility, combined with the small body size of women and men, living in these adverse environments as an adaptation. A controversial example is the "small-but-healthy" hypothesis (Seckler, 1982). That hypothesis was dismissed on biological, economic, and humanitarian grounds (Beaton, 1989; Martorell, 1989; Pelto and Pelto, 1989; Sen, 1999). The critics of the "small-but-healthy" hypothesis cite the evidence just discussed that people growing-up under adverse environments have reduced survival, productivity and reproduction. Sen's critique adds a political-economy perspective and shows that people living under adversity are not able to realize their basic desires for their own lives and those of their children.

With the demise of the "small-but-healthy" perspective, the dominant biomedical and clinical paradigm of "small-and-unhealthy" received new invigoration via a field of inquiry called "developmental origins of adult disease." This field originated in the 1920–1930s (Kuzawa, 2005) but became more noteworthy when Ravelli et al. (1976) found evidence of elevated risk for adult obesity among those exposed to the Dutch Famine of 1944–45 during their first trimester of gestation. Building on this key study, many other related findings, and original research, Barker et al. (1989) extended the list of risks of poor early development to heart disease and other metabolic disorders. These relationships have been organized into the "developmental programming" (DP) hypothesis. In essence, the DP hypothesis states that exposure to adverse environments during gestation results in a body that is smaller at birth and will be unhealthy in adulthood. The smallness is a marker of physiological disruption of the prenatal development of one or more physiological systems, including the cardio-circulatory, neuroendocrine, and renal systems. Smaller body size may be in total birth weight, head circumference, body length, organ size, or some combination of these. Barker's group made the important observation that the affected neonates may be within the limits of the clinically accepted range for birth weight or other dimensions, but they are small or disproportionate in size and their early development had been compromised. The DP hypothesis adds that these alterations in growth have permanent maladaptive consequences that place people at risk for disease later in life (Barker, 1997; Barker et al., 2002; Eriksson, 2005).

Soon after the DP hypothesis was first proposed, some researchers found that smaller size at birth plus a greater than expected amount of growth after birth worked synergistically to place people at elevated risk for adult heart disease, glucose intolerance, and other metabolic diseases (Barghava et al., 2004; Eriksson et al., 2001; Roseboom et al., 2006). Gluckman and Hanson (2005; Gluckman et al., 2007) conceptualized the mismatch between reduced fetal growth and accelerated postnatal growth into the hypothesis of "predictive adaptive responses" (PARs). The PAR hypothesis posits that the small size at birth is the result of fetal development under some type of adversity. If that adversity is ameliorated after birth, then there may be a type of over-growth under the relatively better conditions of post-natal life.

Gluckman and Hanson (2005) consider PARs as two levels of adaptation: (1) "short-term

adaptive responses for immediate survival" and (2) "predictive responses required to ensure postnatal survival to reproductive age" (p 68). Fetal growth faltering under conditions of nutritional constraint, such as placental insufficiency or maternal starvation, is an example of the first level. Gluckman and Hanson (2005) state that such short-term responses may be reversible. As an example of the second level PAR, Gluckman and Hanson (2005) cite the work of Chisholm (1999) who reports that human girls, born to women living under adversity, experience relatively rapid growth and early sexual maturation after birth. This is a trade-off between current versus future reproduction. As explained by Coall and Chisholm (2003), "This trade-off underlies the prediction that under conditions of environmental risk and uncertainty (experienced subjectively as psychosocial stress) it can be evolutionarily adaptive to reproduce at a young age" (p 1771). Coall and Chisholm (2003) point out that earlier maturation and reproduction in these girls comes at the expense of their own health quality and that of their offspring (e.g., greater risk for low birth weight). Despite this, these relatively young mothers manage to reproduce, which may be considered adaptive.

Coall and Chisholm's (2003) work, and Gluckman and Hanson's (2005) use of it to defend their PAR hypothesis are controversial, as the predominant view in human biology is that early life adversity may be more likely to delay growth and sexual maturation. An increase in mean stature and a decline in the median age at menarche is the hallmark of secular trend studies of general environmental improvement (Bogin, 1999; Ellison, 2001; Garn, 1987; Greulich, 1976; Hoshi and Kouchi, 1981; Roche, 1979; Tanner, 1962). Indeed, delayed sexual maturation could be viewed as an adaptation, as slower growth and a later age at first reproduction would serve to spread out nutritional demands over a longer period of development.

Part of the controversy may be due to the distinction between physical adversity (e.g., undernutrition and disease), which is assumed under the biomedical and secular trend perspectives, and psychosocial adversity (e.g., unstable family life, emotional abuse), which is assumed under the Coall and Chisholm (2003) hypothesis. Because of the biocultural nature of human beings it seems incumbent to consider both types of adversity, but each may have independent effects on growth and development as well as on adaptive responses. Classic life history theory considers only the physical adversity position. As explained by Pianka (1970), "Natural selection in saturated environments (where demand for resources approximates supply) is density dependent, favoring competitive ability at the expense of slow growth and delayed reproduction. In contrast, in competitive vacuums (resource supplies greatly exceed demand), selection is independent of population density and favors rapid growth, early reproduction, and short life spans" (p 592). A human life history theory may require a broader definition and analysis of Pianka's "saturated" and "unsaturated" environments. Human environments may be saturated in one domain (Fig. 1) such as "Ecology" but unsaturated in another domain, such as "Family" or vice-versa. The biocultural interactions between domains and their levels of saturation are likely to create diverse and unexpected outcomes for human survival, productivity, and reproduction.

Research into the interactions between biocultural domains is a project for the future, but of more immediate concern is a major question about the PAR hypothesis: can embryos, fetuses, and infants make predictive adaptive responses? Indeed, are the terms "predictive" and "adaptive" the correct terms to use? Biological responses will occur when the fetus or infant is exposed to adversity, but are these responses in any way foretelling of the most appropriate path for future development? Are these responses in any way beneficial, or are the responses evidence of deranged developmental physiology and risks for pathology? A discussion of life history theory trade-offs may help approach the answer to these questions.

TRADE-OFFS IN HUMAN GROWTH AND DEVELOPMENT

PARs require trade-offs (TOs), such as reduced fetal growth in favor of survival (Gluckman and Hanson's [2005] first level adaptation), or rapid maturation and early reproduction, but with low quality offspring, versus slower maturation, a healthier mother at first pregnancy, and higher quality offspring (Gluckman and Hanson's [2005] second level adaptation). Gluckman and Hanson (2005; Gluckman et al., 2007) see TOs as beneficial if they occur early in development to bring greater success later in life, such as during the reproductive period. Conversely, they

TABLE 1. Human life history stages and associated growth and development outcomes under adverse conditions

Life history stage—Adverse conditions[a]	Growth/development outcomes[b]
Prenatal-maternal nutritional deprivation; stress of hypothalamic–pituitary–adrenocortical (HPA) and HP-thyroid axes	Insulin resistance, more omental fat, reduced skeletal muscle mass, reduced bone mineralization, reduced capillary density in many tissues, impaired endothelial cells in heart and vasculature, reduced nephron number, reduced negative feedback of HPA axis (greater stress response), elevated adrenocortical and thyroid hormones
Birth/neonate (to 28 days postpartum)-outcomes related to adverse prenatal conditions above	Possible combination of low birth weight, prematurity, reduced brain growth/head circumference, reduced arm and leg length, impaired immune function
Infant[c]: 28 days to 2.9 years, and Child[d]: 3.0–6.9 years, undernutrition, lack of play/stimulation, infection, neglect/abuse; overfeeding and low physical activity	Growth faltering, short extremities especially the legs, infection-malnutrition synergism, motor and cognitive delays, HPA precocious sexual development, infant-child mortality; high BMI with excess fat, incipient diabetes and cardiovascular disease, reduced bone and muscle mass
Juvenile[e]: 7.0–10.0 years (pubarche), as above plus excessive physical labor	Continuation of above responses with possible exacerbation of responses due to additional physical labor and greater exposure to pathogens due to increased independence

[a]The ordering of "Life History Stage" in the left column follows Bogin (1999).
[b]The "Growth/Development Outcomes" in the right column is based on the examples given in Gluckman and Hanson (2005).
[c]Infants are dependent on their mothers for lactation and other primary care.
[d]Children are dependent on older people for feeding and protection.
[e]Juveniles are more independent and exposed to larger biosocial arena (Bogin, 1999).

see TOs as harmful when there is a significant mismatch between the prenatal/neonatal environment and the postnatal environment. In this case, the fetus/neonate makes incorrect adaptive responses that lead to disease later in life (e.g., greater glucose intolerance, heart disease, and obesity in adults exposed to the Dutch Famine as embryos/fetuses). In essence, the PAR hypothesis is attempting to account for the pathology associated with the DP hypothesis and also leave room for adaptation, in the beneficial sense, at the individual or species level.

This discussion of adaptation and trade-offs in human health must be evaluated against the available data for biological outcomes for growth and development. Gluckman and Hanson (2005) provide many human examples of such biological outcomes and some of these are organized into Table 1, following the life history model of human growth stages proposed by Bogin (1999). None of the outcomes seems "good," in fact all are indicators of pathology. The mortality rate for under 5-year-olds (listed as "infant-child mortality" in Table 1) is one of the most sensitive and widely used epidemiological indicators of how "bad" these outcomes are. A specific example is the nation of Guatemala, which in the year 2005 had the highest under 5-year-old mortality rate of all of Central America at 43 deaths per 1000 live born (UNICEF, http://www.unicef.org/infobycountry/guatemala.html, December 30, 2006).

In Guatemala, the majority of the population lives under the conditions of adversity described in the opening paragraph of this article and in Table 1. Poverty is the main correlate of this adversity, with 74.5% of the rural population living below the poverty line (based on the 2003 census, the rural population numbers 7.3 million people and the total Guatemala population is 12.3 million, www.ruralpovertyportal.org, October 11, 2006). An estimated 37.4% of the total Guatemala population lives on less than US$2.00 per capita per day. This is especially true for the Maya ethnic group who comprise 81% of the rural population in Guatemala (ibid). The other major ethnic group is the Ladinos, who are the cultural descendents of the Spanish/Portuguese Conquistadors who arrived in 1500 AD. The contemporary Maya are the cultural descendents of the native people of Guatemala at the time of the European conquest. Since the conquest, the Maya have endured infectious disease epidemics, poor nutrition, forced labor (including slavery), land appropriations, and repeated episodes of military attack by national government forces (Adams,

1970; Handy, 1984; Hoepker, 1998; Manz, 1988). The Maya consists of more than 20 linguistically distinct groups of people living in Guatemala, Southern Mexico, and Belize. Nevertheless, many commonalities of history, social organization, religion, and political economy bind all Maya groups together as an identifiable ethnicity. Together, all the people who collectively self-identify as Maya comprise the largest population of indigenous people in the Americas (Adams and MacLeod, 2000).

The under 5-year-old mortality for the total Maya population is estimated at 46/1000, but in isolated rural areas the number doubles (UNICEF, http://www.unicef.org/infobycountry/guatemala.html, December 30, 2006). In a narrow sense, the women producing these infants and children are "adapted" because these women live long enough to reproduce. But, far too many of their offspring die and those who live are impaired with poor physical growth, reduced cognitive development, and diminished socioeconomic productivity (Martorell, 1995). Given these outcomes, the tradeoff between reproduction versus reduced rates of survival and productivity (i.e., growth and work capacity) indicates a low level of adaptation for the Maya in Guatemala.

INTERGENERATIONAL INFLUENCES OF ADVERSITY

The human biocultural environment influences the well-being of the current generation of people and also has lasting influences on subsequent generations. These effects may be called intergenerational influences. The intergenerational influences hypothesis (IIH) was proposed by Emanuel (1986) and defined as, "... those factors, conditions, exposures and environments experienced by one generation that relate to the health, growth and development of the next generation." The original IIH was proposed to account for the persistence of low birth weight across generations. Both the DP and PAR hypotheses accommodate the existence of the intergenerational influences. Both hypotheses posit that poor quality of the intrauterine and early postnatal environment will result in fetus/infant metabolic and cardiac adjustments, possibly beneficial or harmful. These metabolic and cardiac variations influence the growth, development, and health of individuals for the rest of their life. In the case of women, whose reproductive systems are largely developed during their own fetal period, these variations will also influence their offspring.

There is ample evidence in favor of the IIH as a nongenomic explanation for low birth weight and also cardiovascular disease (Drake and Walker, 2004). Women from ethnic minorities, with histories of several generations of adversity, give birth to higher percentages of low birth weight infants, even when the current generation of mothers grew up under favorable health conditions (Emanuel, 1993; Skjærven et al., 1997). An example is The Netherlands, a nation that provides a high level of general health care, especially prenatal care. Even so, infants born to ethnic minority women are at greater risk for low birth weight and mortality than ethnic Dutch (Drooger et al., 2005). The ethnic minority mothers, or their mothers, were born in other countries and usually grew-up under adverse biocultural conditions. Despite improved health care for the current generation of women in The Netherlands, the intergenerational influences of the past environments are not overcome.

A probable mechanism for intergenerational influences on birth size and other reproductive outcomes is proposed by Price and Coe (2000). They hypothesize that uterine/placental nutrient transport is impaired in mothers who experienced malnutrition during their own gestation. The gestational experience of the mother may be passed on to her daughters through gestation imprinting that takes place during the fetal development of the daughters. Price and Coe's (2000) findings are based on forty years of empirical research with captive rhesus monkeys. Female monkeys descended from large-for-date matrilines produced male and female offspring with relatively greater fetal growth (e.g., greater birth weight and/or length). Females descended from small-for-date matrilines produced male offspring of normal size but female offspring with reduced fetal growth. These small-for-date females had greater risks for poor reproductive outcomes as adults. The research also found that improving the nutritional intake of the small-for-date females led to increased postnatal growth in weight, greater pregnancy weight gain, and heavier newborns. The improvement in maternal growth and reproductive outcomes took four generation to overcome the initial deficits of the great-grandmother generation.

Human research corroborates these nonhuman primate studies. In the 1960s and 1970s, the Institute of Nutrition of Central

American and Panama (INCAP) conducted a longitudinal intervention study in four rural Guatemala Ladino villages. Initial health and anthropometric screening of the residents of the village found high levels of undernutrition, low birth weight, stunting, wasting, infection, and under 5-year-old mortality (Martorell, 1995). During the study, pregnant women and their offspring received one of two types of nutritional supplements. One was a drink called "atole," which was enhanced in protein and energy, the other was a drink called "fresco," which was enhanced with energy, but less than the energy in the atole drink. Both drinks contained vitamin and mineral supplements. The infants and children of the original study received these nutritional supplements until 7 years of age. In the 1990s, women who were part of the original study as children were asked to be part of a follow-up analysis to investigate the intergenerational influences of the original intervention. In one of the follow-up studies, Stein et al. (2004) report on, "... 283 mother-child pairs (mothers born 1969–1977; children born 1996–1999)" (p e270). The mothers had been measured repeatedly from their birth till 3 years of age. The offspring of these women were also measured at similar intervals for 3 years. Stein et al. (2004) find that the current generation of infants grew faster than their mothers. Moreover, infants of women who received atole (protein-energy drink) grew faster than the infants of mothers who received fresco (energy only). In this example, rate of growth in the first 3 years of life is associated with higher quality nutritional supplementation.

This type of outcome is predicted by both the DP and the PAR hypothesis, but is most parsimoniously explained by the DP hypothesis. Malnourished women will give birth to small, slower growing infants and women who received better nutrition in their early life (prenatal and postnatal) give birth to large, faster growing infants. The gestationally imprinted daughters of women who were malnourished in utero are not making any type of predictive or adaptive response; rather, their reproductive outcomes simply reflect the quality of the reproductive environment of their mothers.

The DP and PAR hypotheses differ in their predictions for adult health outcomes of early life intervention. Specifically, the PAR hypothesis predicts that a mismatch between the uterine environment and the postnatal environment will result in clinical symptoms of metabolic disease, such as higher blood pressure, and risk for elevated levels of low density lipoproteins. Additional follow-up studies of the INCAP data by Webb et al. (2005) and Stein et al. (2006) shed some light on the accuracy of these predictions. Webb et al. (2005) studied the blood pressure of 450 men and women of ages 21–29 years who were treated with either "atole" or "fresco" in infancy and childhood. Their mothers had been, generally, undernourished prior to the INCAP intervention, but were given one of the treatments during pregnancy. The intervention continued until the children were 7 years old, but after that age the children were forced to subsist on the prestudy low quality diet, be exposed to health risks of contaminated drinking water, and undertake relatively heavy work loads in these rural villages. The PAR hypothesis predicts that there will be evidence of nonadaptation and health risks under this type mismatch between the prenatal and early life nutrition environment and the environment after 7 years of age. Controlling for birth weight and rate of growth in body length during the first 3 years of life, Webb et al. (2005) find that neither type of supplementation was associated with blood pressure in adulthood. Webb et al. (2005) conclude that the, ".... data do not support the role of maternal nutrition during pregnancy, birth size, or early child growth in programming adult blood pressure" (p 898).

Stein et al. (2006) extend the analysis to 665 men and 790 women, 25–42 years old, who were treated as infants and children to either "atole" or "fresco." The experimental sample is compared with similar aged adults who did not receive any nutritional supplement as infants or children. Treatment is associated with a lower fasting blood, "... glucose level (7.0 mg/dl, 95% confidence interval (CI): 0.5, 13.5) ... lower systolic blood pressure (3.0 mm Hg, 95% CI: 0.4, 5.6) ... a lower triglyceride level (sex-adjusted; 22.2 mg/dl, 95% CI: 0.4, 44.1) and higher high density lipoprotein cholesterol level (males only; 4.7 mg/dl, 95% CI: 1.5, 7.9) ..." (p 1160). Each of the metabolic measures indicates improved health status, despite the mismatch between early life and later life nutrition. The results do not follow the prediction of the PAR hypothesis. These results are more in accord with the DP hypothesis that interventions designed to ameliorate nutritional insufficiency improve birth weight and reduce stunting, and wasting in the early years of life will have beneficial

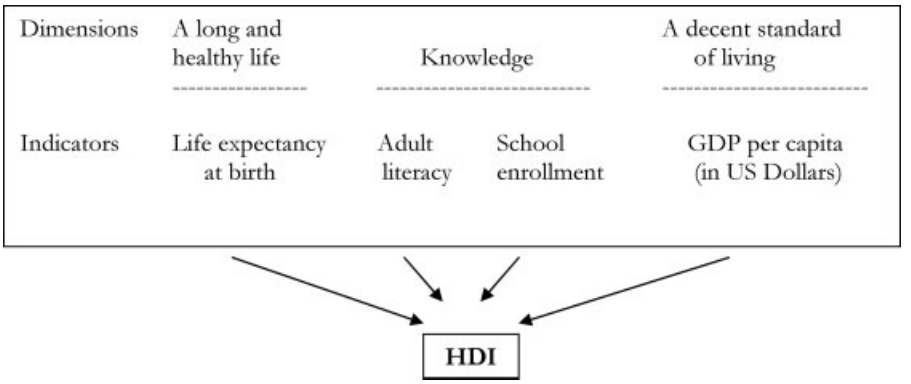

Fig. 2. An overview of the construction of the human development index (HDI). The dimensions, indicators, and final HDI are depicted. Each indicator receives a numerical value and these are weighted in the formula used to calculate the final HDI score. From: http://hdr.undp.org/docs/statistics/indices/technote_1.pdf Technical note 1, United Nation Human Development Report. Accessed August 16, 2006.

consequence throughout life. Viewed in the perspective of life history theory, the results of the INCAP follow-up studies make even more sense. The INCAP nutritional intervention of 1969–1977 moderated, if only a little, the adverse conditions for human development in the four study villages. Living conditions were improved enough to allow for more favorable health and this reduced the need for metabolic and growth trade-offs during the prenatal and early postnatal years of growth and development.

A BIOCULTURAL INSTRUMENT TO ASSESS TRADE-OFFS IN HUMAN DEVELOPMENT: THE HUMAN DEVELOPMENT INDEX

Life history theory and its focus on trade-offs during growth and development may offer a productive alternative to the more narrow predictions of the DP hypothesis and the PAR hypothesis. To achieve its potential, however, the life history theory perspective requires an appropriate means to measure both the biocultural influences on human growth and development and the impact of adverse environments on human development and health. The human development index (HDI) is a measure of human biological well-being, educational/cognitive status, and economic productivity (http://hdr.undp.org, accessed August 17, 2006). The HDI captures some of the aspects of human adaptation as discussed in this article, and the HDI provides a quantitative scale against which human growth, development, and health may be measured. The HDI gauges the average achievements in a country in three basic dimensions of human development: (1) a long and healthy life, as measured by life expectancy at birth, (2) knowledge, as measured by the adult literacy rate (with two-thirds weight) and the combined primary, secondary, and tertiary gross enrollment ratio (with one-third weight), and (3) a decent standard of living, as measured by gross domestic product (GDP) per capita at purchasing power parity (PPP) in United States dollars (USD). The calculation of HDI produces a score that ranges from 1 (most favorable HDI) to 0 (least favorable HDI). Further details of the calculation of the HDI may be found at: http://en.wikipedia.org/wiki/Human_Development_Index. A schematic of the materials and methods used to calculate the HDI is shown in Figure 2. The United Nations Development Report calculates and published the HDI. The most recent data are for 2003 and the report includes 177 countries (http://hdr.undp.org, accessed August 17, 2006).

In the words of the creator of the HDI, "The basic purpose of development is to enlarge people's choices. In principle, these choices can be infinite and can change over time. People often value achievements that do not show up at all, or not immediately, in income or [economic] growth figures: greater access to knowledge, better nutrition and health services, more secure livelihoods, security against crime and physical violence, satisfying leisure hours, political and cultural freedoms, and sense of participation in community activities. The objective of development is to create an enabling

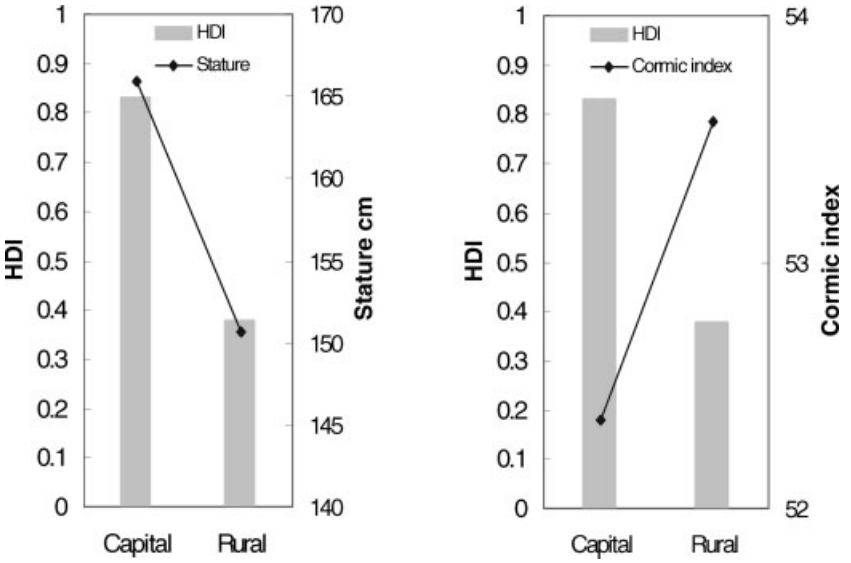

Fig. 3. Differences in Human Developmental Index (HDI) and stature at 15 years of age between urban and rural boys. HDI data from Programa de Naciones Unidas para el Desarrollo (1998); anthropometric data from Luis Ríos, unpublished.

environment for people to enjoy long, healthy, and creative lives" (Mahbub ul Haq, http://hdr.undp.org/hd/, accessed August 17, 2006).

The HDI supersedes traditional economic indicators, such as GDP and median annual salary, which tell us little about how life is lived in any nation. According to the definition of human adaptation given earlier in this article, the HDI is a biocultural measure of the quality of life of people in a given nation. The value of the HDI, and other similar indices, as a biocultural indicator of human well-being has been shown in previous anthropological research (Cameron, 1991; Lee et al., 1997). The HDI is an especially useful measure of both adult and child welfare.

To show how the HDI may serve as an indicator of human adaptation or lack of adaptation, we return to the discussion of living conditions in Guatemala. Nationally, Guatemala has an HDI score of 0.663, which ranks 117 out 177 nations. By contrast, the HDI ranks (and scores) of a few other nations are given here: Norway = 1 (0.963), United States = 10 (0.944), Mexico = 53 (0.814), and Niger = 177 (0.281). The HDI may be calculated for subgroups and regions within one nation. This has been done for Guatemala. As shown in Figure 3, urban regions of Guatemala have a HDI score that is more than twice the value of rural regions. Also shown in Figure 3 are the mean values for stature and cormic index, also called the sitting height ratio ([sitting height/total stature] $\times$ 100), of 15-year-old boys. There are statistically and biologically significant associations between HDI scores and both stature and the cormic index. As mentioned above, the vast majority of the Guatemala Maya population lives in rural areas. Accordingly, the low HDI scores of rural Guatemala affect the Maya disproportionately. Low HDI of the Maya contributes to low birth weight and relatively high under 5-year-old mortality that was discussed above, in addition to short stature and disproportionately short legs.

Existing research demonstrates that stature and the cormic index are sensitive indicators of environmental quality (Bogin et al., 2001, 2002; Frisancho et al., 2001; Gunnell et al., 1998; Gurri and Dickinson, 1990; Lawlor et al., 2003; Leary et al., 2006; Leitch, 1951; Thomas and Duncan, 1954; Wadsworth et al., 2002). Short stature combined with a relatively high cormic index (meaning short legs relative to total stature) is associated with higher risk in adulthood for hypercholesterolaemia, coronary heart disease, impaired glucose and insulin regulation, increased pulse pressure and systolic blood pressure,

and higher fibrinogen levels (Han et al., 1997; Langenberg et al., 2003; Martin Moreno et al., 2003; Smith et al., 2001).

Relatively short legs are also an indicator of reduced human productivity and reproductive quality (Martin et al., 2004; Spurr, 1983). The "Knowledge" and "Descent standard of living" dimensions of the HDI data in Figure 3 indicate that rural Guatemalans (relatively more Maya in ethnicity) have lower productivity than urban Guatemalans (relatively more Ladino in ethnicity). Data independent of HDI show that total reproductive outcome for the urbanites is greater than for the rural Maya—in terms of births surviving to adulthood and number of grandchildren in the following generations. The mortality rate for children 1–4 years old was 14 per 1,000 for all of Guatemala in 2003, but divides into a rate of 9 per 1,000 in the cities and 20 per 1,000 in rural areas (http://www.paho.org/english/dd/ais/be_v25n2-perfil-guatemala.htm, accessed August 17, 2006).

CONCLUSION

Several measures of human health and well-being are used in this article, including traditional epidemiological indicators (birth weight, infant mortality), growth and development, clinical assessment of metabolic disease, and the human development index. Each of these measures, independently and in combination, show that human beings living under adversity display reduced adaptation in terms of survival, productivity, and reproduction. The specific examples discussed above, based on literature review and original research with the rural Maya of Guatemala, show how one human group responds to an adverse biocultural environment. These findings may be extended to the rural poor of many low income nations of the Americas, Africa, and Asia.

These empirical data and the predictions of life history theory may be used to assess the relative merits of the developmental programming (DP) hypothesis and the predictive adaptive response (PAR) hypothesis for human development. At present, it seems that the focus on pathology and disease in adult life of the DP hypothesis has greater merit than the adaptationist view of the PAR hypothesis. In accord with predictions of life history theory, poor quality of life necessitates trade-offs in human growth and development, productivity, reproduction, and health. It seems inappropriate at the present time to consider these trade-offs as predictive responses that lead to adaptation.

Despite this conclusion, we recommend further development of a biocultural human life history theory. As discussed above, the outcomes of interactions between the domains of human biology and culture (Fig. 1) are poorly understood. This is especially the case when there is considerable variation in the "saturation" (i.e., the balance between supply and demand in biological, social, and cultural resources) between domains. Unexpected responses, which may be both predictive and adaptive, may be discovered.

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