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Developmental Origins of Life History: Growth, Productivity, and Reproduction

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ABSTRACT There is now much evidence that early life undernutrition elevates risk of diseases like cardiovascular disease. Less clear is whether the underlying developmental plasticity in metabolism and physiology evolved to serve an adaptive function, beyond these effects on pathophysiology. This review builds from principles of life history theory to propose a functional model linking early environments with adult biology. An organism has metabolic potential in excess of survival requirements, called productivity, that supports growth before being shunted into reproduction after growth ceases. This concept from inter-specific studies leads to the prediction that plasticity in growth rate will be positively correlated with components of future adult reproductive expenditure. Consistent with this idea, evidence is reviewed that early nutrition or growth rate predict offspring size in females, and increased somatic investment related to reproductive strategy in males. Thus, population birth weight and sexual size dimorphism are predicted to increase in response to improvements in early nutrition. A striking feature of the continuity of metabolic production is its perpetuation not merely during the life-cycle but across generations: in females, growth rate predicts future nutritional investment in reproduction, which in turn determines fetal growth rate in the next generation. Growth and reproduction serve as mutually-defining templates, thus creating a phenotypic bridge allowing ecologic information to be maintained during ontogeny and transmitted to offspring. Resetting of metabolic production in response to maternal nutritional cues may serve a broader goal of integrating nutritional information within the matriline, thus providing a more reliable basis for adjusting long-term strategy. Am. J. Hum. Biol. 19:654–661, 2007.

INTRODUCTION

Pathophysiology and metabolic productivity

Environmental conditions experienced early in the life cycle can profoundly influence an organism's biology and long-term health. Among the best documented examples include the role of early life nutrition and stress as influences on adult risk for developing metabolic diseases like diabetes, stroke, and heart attack (Barker, 1994). Early findings that individuals born small have elevated risk for cardiovascular mortality and associated risk factors have been widely replicated in human populations, and are now backed by an extensive experimental literature. Although findings vary by study and animal model, smaller size at birth and constrained prenatal or maternal nutrition during pregnancy are associated with adverse changes in nutrient metabolism and cardiovascular function (Adair et al., 2001; Kensara et al., 2006; Kuzawa and Adair, 2003; Louey and Thornburg, 2005). This research has highlighted the importance of maternal nutrition to health in the next generation, while underscoring the need to adopt a life cycle approach when studying chronic diseases and the processes of aging.

While the public health significance of these findings have received considerable attention, the emphasis of this research on identifying mechanisms of pathophysiology has left important questions unaddressed. It is unknown, for instance, if this developmental plasticity in response to early environments has a purpose, and if so, what function it evolved to serve. It has been proposed that the fetus or infant could use nutritional or endocrine cues as

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a basis for setting long-term biological strategy, such as its level of "nutritional expectations," in response to local ecologic or social conditions (Bateson et al., 2004; Bateson, 2001; Gluckman and Hanson, 2005; Gluckman et al., 2007; Kuzawa, 2001, 2005). Rapid changes in nutrition during one's lifetime could then lead to "mismatch" and metabolic disease (Bateson et al., 2004; Gluckman and Hanson, 2005). It has further been proposed that intergenerational influences on nutrition and growth stabilize the nutritional signal experienced *in utero*, a process of phenotypic "inertia" that could increase the reliability of the intrauterine cue as a predictive signal (Kuzawa, 2005).

Building from this work, this article considers the utility of Charnov's (1991, 1993) concept of productivity as a potential link between plasticity in early nutrition and growth and adult biology and function. The article first reviews the scaling of metabolic processes to body mass across species, which reveals tight links between mass and expenditure on growth and reproduction. Second, animal model and human epidemiologic research is reviewed suggesting that early nutritional experiences and plasticity in growth rate have lingering effects that drive sex-specific patterns of adult reproductive effort. Evidence for a continuity in metabolic production across the lifecycle, manifesting first in growth and later in reproduction, suggests a link between developmental responses to early environments and adult biology that is grounded in functional relationships rather than pathology. These relationships are broadly consistent with the assumptions of interspecific models of mammalian life history, and complement current research on the disease impact of early environments. Perhaps most intriguingly, they also provide insights into a potential mechanism for the hypothesized capacity for ecologic information to be maintained and integrated across generations (Kuzawa, 2005).

Metabolic scaling and productivity

The deep connection between an organism's size and rate of energy use have long been appreciated. As a general rule, smaller species have lower total requirements consistent with their smaller size, but their rate of energy use per unit body mass is increased. Thus, each gram of the 4.2 gram pygmy shrew consumes roughly 7.8 kcal/h, while an elephant consumes less than 0.5 kcal g^{-1} h^{-1}. Kleiber (1961) showed that species differences in BMR are linked to body size, with total BMR scaling as a power function of body mass with an exponent of 0.75, and with mass-specific BMR scaling with an exponent of −0.25. Recent work derives such "quarter power" scaling from the fractal geometry of the vasculature that supplies cells with metabolic substrate (West et al., 1997).

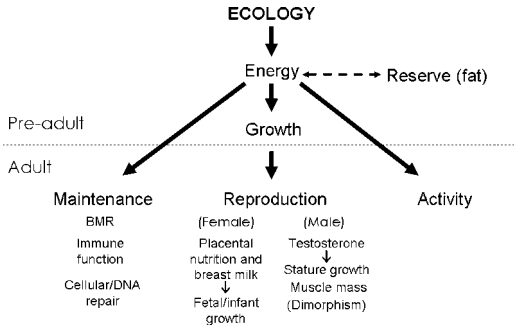

Fig. 1. Organismal allocation and the continuity of production in growth and later in reproduction (after Kuzawa, 2005).

These links between body mass and metabolic rate place fundamental constraints on an organism's life history strategy, because they dictate the size of the pool of metabolic resources available to be allocated across the organism's functions, classically including maintenance, growth, and reproduction (Charnov, 1991, 2001; Gadgil and Bossert, 1970). When maintenance expenditures are subtracted from total respiration, the balance of excess energy, defined as *production*, is available to support expenditures like growth and reproduction (Klieber, 1961; Lavigne, 1982). After Kozlowski and Wiegert (1986, 1987), Charnov (1991, 1993) has formalized the concept of production into his model of comparative life histories, which assumes that production is first used to support growth before being used to support reproduction once growth ceases at reproductive maturity (Fig. 1).

Developmental changes in production are described by an ontogenetic growth law that reflects such factors as the decline in mass-specific metabolic rate as body size increases (see Charnov, 2001; West et al., 2001). Evidence that growth and reproductive effort are expressions of a common metabolic fraction come from allometric analyses of mammalian metabolism and life history variation. Just as

metabolic rate scales with body mass, specific components of metabolic expenditure also scale tightly with body mass with similar quarter power scaling. For instance, species-specific growth rates scale to body mass to the power of 0.75 (Case, 1978), as does daily milk energy yield, egg size (birds), and litter size (reviewed in Lavigne, 1982). Thus, moving from small- to large-bodied mammals, not only does mass-specific metabolic rate decline, but the fraction of expenditure devoted to self-growth and later to offspring production also decline as similar power functions of body size.

These regularities in metabolic processes show that a species' energy metabolism and its strategy of energy allocation vary tightly in relation to body mass, and suggest deep connections between body mass and both growth rate and adult reproductive biology (Charnov, 2001). It is important to note, however, that these relationships are observed on species averages. Less attention has been given to evaluating whether similar connections among body size, growth expenditure, and reproduction hold when variation in growth rate is between individuals of a single species tracing not to genes, but to developmental plasticity in response to environmental influences like nutrition.

Evidence for plasticity in productivity in females

Longitudinal studies document connections between early life plasticity in growth rate and adult stature, which in turn is related to reproductive performance (Bogin, 1999; Tanner, 1990). In humans, growth rate is most sensitive to nutrition during the period spanning fetal life to roughly 2–3 years after birth (Billewicz and McGregor, 1982; Martorell et al., 1995). This can be explained, in part, by the fact that growth rate is most rapid and accounts for its largest fraction of the organism's metabolic budget at this age (Butte, 2005). Compared to childhood or adolescence, the hormones that drive linear growth during fetal life, infancy, and early childhood are also more acutely sensitive to nutrition, making growth rate a more direct outcome of nutritional intake at this than at other periods of the life cycle (Gicquel and Le Bouc, 2006; Gluckman and Pinal, 2003). As a result of this limited window of nutritionally-mediated growth attainment, the majority of environmental influences on adult standing height are present by 2–3 years of age (Billewicz and McGregor, 1982; Martorell et al., 1995).

To the extent that adult size is a predictor of birth outcome (Institute of Medicine, 1990), the connection between infant or early childhood growth attainment and final adult size might be expected to yield correlated changes in offspring birth size. This is indeed the case. However, the most widely-documented evidence for a persistent effect of growth in one generation on growth in the next comes from intergenerational cohort studies that include information on birth weight across multiple generations (reviewed by Ramakrishnan et al., 1999). These studies find that the mother's own birth weight is a strong, and often the strongest, predictor of offspring birth weight. These relationships are strongest when adjusted for gestational age, indicating that it is fetal growth rate, rather than differences in size due to prematurity, that is important (Alberman et al., 1992). Moreover, these relationships are typically independent of maternal adult stature, suggesting that they are not merely capturing an effect of birth size on later adult size (Ramakrishnan et al., 1999).

While a correlation between fetal growth rate in mother and offspring must partly reflect an effect of shared genes, there is also evidence for an epigenetic contribution to these correlations. As one well-known example, women whose mothers experienced the Dutch Famine Winter while pregnant gave birth to offspring (the grandoffspring of the pregnant women who lived through the famine) who were themselves smaller (Lumey, 1992). This is consistent with the idea of continuity of productivity from early life into adulthood: the nutritional experiences and fetal growth rate of these women prior to birth influenced the nutrients that their bodies had available to invest in reproduction later in adulthood, as reflected in the fetal growth rate of their own offspring.

Although taller women tend to give birth to larger babies, the specific components of maternal linear growth that correlate most strongly with offspring size also suggest a lingering impact of early postnatal nutrition on adult reproduction and offspring fetal growth. Several studies find that leg length is the component of adult stature that is strongest as a predictor of offspring birth weight (Lawlor et al., 2003). Data from the 1958 British birth cohort study have been used to evaluate whether birth weight is predicted by the mother's adult trunk and leg length, and also

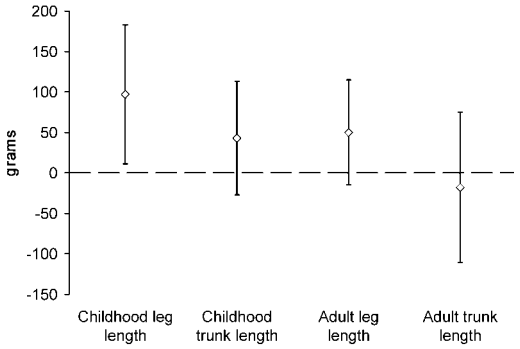

Fig. 2. Relationships between maternal trunk and leg length measured in adulthood and childhood, and birth weight in offspring. Values indicate the change in birth weight (grams) for a one standard deviation change in each predictor adjusting for the mother's socioeconomic variables, birth weight, and energy intake (data from Martin et al., 2004).

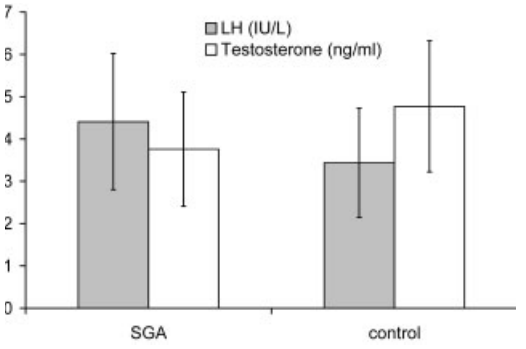

Fig. 3. Testosterone (T) and luteinizing hormone (LH) in males born small for gestational age (SGA) compared to controls (data from Cicognani et al., 2002).

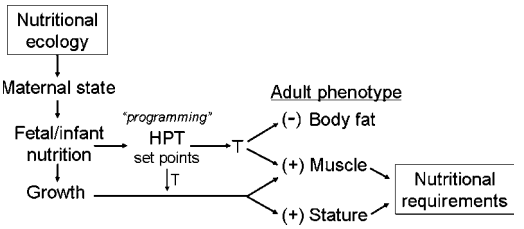

Fig. 4. Hypothetical model linking early nutrition with hypothalamic–pituitary–testicular function and downstream effects on energy requirements in males.

her trunk and leg length measured when she was 7 years of age (Martin et al., 2004). In these analyses, adult trunk length was the weakest predictor of birth weight, while the strongest was leg length measured in childhood (Fig. 2). Because childhood leg growth is the component of linear growth most sensitive to nutrition (Scrimshaw and Behar, 1965), these findings once again point to early life nutrition as an influence on later reproduction.

Early growth and reproductive expenditure in adult males

Like females, males cease linear growth at maturity, thus freeing up metabolic resources for use in other functions. Unlike females, however, males do not invest these resources directly in the production of offspring. Models of male life history assume instead that they invest reproductive effort in traits and behaviors that benefit reproductive fitness indirectly by boosting qualities like competitive ability and attractiveness (Bribiescas, 1996; Ellison, 2001). Testosterone—the primary male sex steroid—is central to this model, as it stimulates the growth and maintenance of sexually dimorphic traits that tend to be metabolically costly, such as stature, lean mass, and strength (Bribiescas, 2001). It is thus of interest that testicular production of testosterone, and expression of testosterone-sensitive traits like stature and lean body mass, have been shown to be sensitive to early life nutrition, not unlike the sensitivity of offspring size to early nutrition and growth in females (Cicognani et al., 2002). Although data are once again sparse, testosterone concentrations are reduced in individuals born SGA, and are particularly low among individuals who failed to experience catch-up growth (Cicognani et al., 2002) (Fig 3). In Guatemala, birth weight is a positive predictor of the testosterone-sensitive traits of stature and lean mass in both males and females, but this effect is twice as strong in males (Li et al., 2003). Other studies find that birth weight is positively related to grip strength in both sexes, but that the relationship is stronger in males (Kuh et al., 2002; Sayer et al., 2002, 2004). Although awaiting more definitive tests with longitudinal data, these findings in males suggest a capacity to calibrate expenditure on costly sexually-dimorphic traits through effects of early nutritional environments on the hypothalamic–pituitary–testicular axis (Fig. 4).

DISCUSSION

Longitudinal studies in humans and the experimental animal model work reviewed

here tentatively support the idea that nutrition-mediated developmental plasticity in early growth rate has a lingering effect on physiology and metabolism, influencing future expenditure on reproduction in adulthood. In females, this continuity is evidenced by correlations between growth rate and nutritional conditions experienced early in life and the level of future investment in reproduction as reflected in offspring birth size. In males, there is limited evidence that traits that reflect male somatic investment, such as stature, lean mass and strength, and the testicular hormones that mediate this pattern of investment, are positively correlated with birth weight. Because reproduction is costly, this sensitivity of reproductive expenditures to early life nutrition may provide an example of long-term requirements being set by developmental plasticity in response to cues that are correlated with local nutritional ecology (Ellison, 1990; Gluckman et al., 2007; Kuzawa, 2005).

Relationships between early nutrition and growth and the anatomic and physiologic characteristics of the reproductive axis have been documented in both animal models and human studies, providing clues into the potential mechanisms contributing to these associations. There is limited evidence from a Spanish cohort that females born small for gestational age (SGA) have smaller ovarian and uterine size, reduced cycling as adolescents, and reduced gonadal steroid production (Ibanez et al., 2000, 2002, 2003). Similarly, a recent study of Polish women finds that estrogen is positively related to the ponderal index measured at birth (Jasienska et al., 2006a), and that the well-documented sensitivity of ovarian steroid production to energy stress is contingent upon birth size (Jasienska et al., 2006b). Prenatal nutritional manipulations have been shown to have similar effects on the developing reproductive axis in both rats and sheep (reviewed in Rhind et al., 2001).

Although greater male than female linear growth responses to improved nutrition have been noted (Bielicki and Charzewski, 1977; Gray and Wolfe, 1980; Greulich, 1951; Stinson, 1985; Wolanski and Kasprzak, 1976; but see Holden and Mace, 1999), the studies reviewed here suggest that plasticity in postnatal sex differences may partially reflect processes initiated *in utero* (Kuzawa, 2005). Consistent with this, experimental restriction of fetal nutrition has been shown to have a greater negative impact on the growth of male fetuses in rodents, with the effect of reducing sexual dimorphism at birth (Oyhenart et al., 1998; see also Kuzawa and Adair, 2003). In humans, the magnitude of the secular trend in adult stature is greater in males than females, and the increase in stature over the same-sex parent has been shown to relate to prior fetal growth rate (Alberman et al., 1991). The model presented here will be supported if future longitudinal studies find that growth rate correlates positively with measures of adult reproductive effort, including birth size or weaning weight of offspring in females, and in males, stature, lean mass, and strength. Through these sex-specific effects, improvements in early nutrition are predicted to yield an increase in population birth weight and sexual size dimorphism (Fig. 1).

If more widely documented, a connection between growth rate and reproductive effort could shed light on the now well-documented connections between birth weight or early life nutrition and adult biology and health (Barker, 1994). The most important pathophysiologic outcomes associated with early life nutrition prominently include changes in the supply and distribution of metabolic resources, including blood pressure, glucose, and lipids. Modifications in these systems through developmental plasticity may be particularly important around the time of weaning in light of the high brain energy requirements, frequent nutritional disruption, and mortality at this age (Kuzawa, 1998; Kuzawa et al., in press). It will be important to consider how adjustments in productivity and reproductive effort might also change the demands placed upon physiology and metabolism, including the partitioning of energy and energy substrate, thereby potentially contributing to the long-term health and disease consequences of early nutritional exposures or birth weight.

Intergenerational inertia and productivity: Chance or Lamarckian strategy?

The data reviewed in females suggests reciprocal influences between growth rate and reproductive effort. If nutrition-mediated changes in early growth rate persist into adulthood to influence investment in reproduction in generation 1, and adult reproductive effort in turn predicts growth rate in generation 2, then the components of an epigenetic inheritance system are in place (e.g., Jablonka and Lamb, 2005; see also Drake and Walker, 2004); in this case, this system allows a partic-

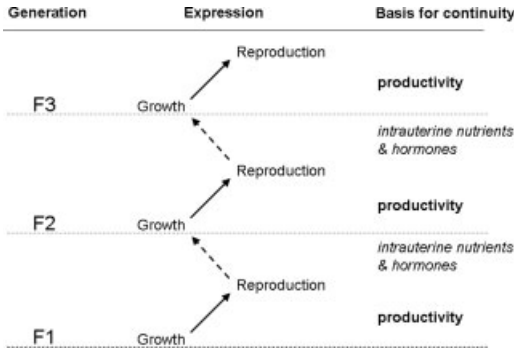

Fig. 5. Model for the intergenerational inheritance of metabolic information through the matriline.

ular phenotypic pattern of metabolic expenditure and allocation to persist beyond the current generation (Fig. 5). This transfer of information does not involve nucleotide sequences, and thus is not inheritance as classically defined. Instead, a pattern of metabolic expenditure manifests within the first generation as a correlation between growth rate and later reproduction, while adult reproductive effort, in turn, influences nutrient investment and thus growth rate in the next generation, starting the cycle anew. In this way, growth rate and reproduction serve as mutually-defining templates, and create a fascinating example of what has been described as a "bridging phenotype" allowing nongenetic information to be conveyed across generations (West-Eberhard, 2003). Although the mechanisms underlying the intergenerational influences on growth and production remain poorly characterized, the molecular basis of similar examples of epigenetic inheritance have recently been described in detail (see Drake et al., 2005; Lillycrop et al., 2005; Weaver et al., 2004).

Because the early nutritional experiences of females have a lingering imprint that transcends the current generation, the metabolism and strategy of energy partitioning in any given generation is contingent not merely upon current conditions, but also upon the ecology experienced by prior matrilineal ancestors during their rearing. As an adaptive strategy, blunting the response to acute change in this fashion may provide important advantages: if abrupt ecologic shifts are more likely to be transient, a system that is designed to capture and harness information about typical conditions should buffer or ignore abrupt changes to the extent possible, while allowing more gradual baseline adjustments (in e.g. growth rate and size) in response to sustained trends in factors like nutrition or demography. It has been hypothesized that this phenotypic "inertia" allows the matriline to recalibrate expenditure in response to predictive information that is made more reliable as a result of averaging across generations (Kuzawa, 2005). Although speculative, the resetting of long-term production in response to intrauterine nutritional cues—reflected first in growth rate and later in reproductive expenditure—may form a central component of the conduit that allows nutritional information to be maintained and integrated across adjacent matrilineal generations.

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