

Original Research Article

Why Are Human Newborns So Fat? Relationship Between Fatness and Brain Size at Birth

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ABSTRACT The plumpness of the human newborn has long been recognized as a trait in need of explanation among researchers. Using a linear regression analysis, we find that head circumference is significantly and positively associated with BMI at birth, after gestational age and birthlength were controlled for, in a sample of 1,069 healthy liveborn routinely delivered at the University Hospital of Coimbra (partial correlation $r = 0.409$, $P < 0.0001$). This significant association is consistent with the idea that newborn fatness is related to the higher need of lipids in newborn humans as an energetic and plastic substrate during its accelerated brain growth period. As birthweight and birth head size are associated with head size and cognitive abilities in childhood and adult life, it could be postulated that these cognitive abilities could have acted as selective pressure responsible for the newborn fatness increase in our lineage. *Am. J. Hum. Biol.* 16:24–30, 2004. © 2003 Wiley-Liss, Inc.

The evolutionary mechanism of acquiring greater than predicted (by allometric rules for hominoids) human newborn's amount of fat is still unknown. Most mammals, including nonhuman primates (Schultz, 1969; Lewis et al., 1983), do not begin to deposit white fat until after birth (Adolph and Heggeness, 1971). The precocious condition of adipose tissue development at birth, despite the human newborn's otherwise altricial state (Watts, 1990), highlights the timing of fat deposition as an atypical feature of human somatic development and raises questions about the evolutionary origins and function of this developmental shift. In fact, the plumpness of the human newborn has long been recognized as a trait in need of explanation among researchers. Human newborns have a fat mass roughly four times that predicted for a mammal of their body size at birth (Kuzawa, 1998; Pawlowski, 1998). The percentage of fat tissue of the human newborn is similar to mammals living in arctic conditions and not to a mammal living in Africa (Forbes, 1987), exceeding that of even the pinniped seals (Oftedal et al., 1989).

While explanations for the fat layer of human neonates have commonly assumed that it serves as insulation to compensate for hairlessness (e.g., Hardy, 1960; Pawlowski, 1998), empirical support for this hypothesis

is presently weak (Kuzawa, 1998). The greater adiposity of human neonates as energy storage might be also explained as an important life-history strategy and a means to modify mortality risk during the nutritional and immunological turbulent period of infancy (Kuzawa, 1998). Although this assertion waits to be confirmed with better data, nutritional stress and heightened susceptibility to infection are not unique features of humans, but characteristic of mammals generally. Finally, it has been suggested that the plumpness of the human newborn is at least partially explainable as an accompaniment of the enlarged human brain, which demands a larger energy reserve to ensure that its obligatory needs are met when the flow of resources from mother or other caretakers is disrupted. The data reviewed (Kuzawa, 1998) and a high correlation between newborn size and the brain size of an adult within the *Primate* order (Lynch et al., 1983) and in

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mammals (Frost, 1987) provide support for this hypothesis, albeit only indirectly.

In order to achieve better insight into the association between brain size and fatness at birth in humans, the relationship between head circumference and body mass index was investigated in a sample of healthy human neonates.

MATERIALS AND METHODS

Sample

A total of 1,069 healthy liveborn singletons (553 males and 516 females) routinely delivered at the University Hospital of Coimbra between September 2002 and February 2003 took part in this study. Newborns of smoking or diabetic mothers, nonwhites, and not from an intermediate socioeconomic background family (using culturally appropriate criteria: from parents years in school and parents occupation—in accordance with the Census of Population Classification of Occupations) were excluded from the analysis, as were those with known developmental defects. All selected births were the first ones for all the mothers and occurred between 38 and 42 weeks of gestation. Data on birthweights, head circumferences, gestational ages (postmenstrual weeks) and recumbent lengths were obtained from birth records. All newborns were measured anthropometrically during the first 24 hours after birth by the same anthropometrist. All information regarding the pregnancy, delivery, baby's condition at birth, health, and socioeconomic status of the parents were obtained from the hospital clinical files.

Data analysis

Descriptive statistics, distribution curves, and correlations between the variables were analyzed. The data were analyzed separately for boys and for girls. Using a linear regression analysis, we investigated the relation between body mass index (BMI) at birth (weight (Kg)/ recumbent length (m)²), a simple, accurate, and valid measure of fatness (e.g., Billewicz et al., 1962), and head circumference at birth, a convenient measure of a baby's brain size (e.g., Cooke et al., 1977; Epstein and Epstein, 1978). Because the distributions of variables are skewed (Shapiro-Wilk's W-test, $P < 0.05$), analyses were repeated after we transformed variables to

natural logarithms; the results were similar (data not presented). Linearity was tested using a quadratic term for birth BMI. The quadratic term was significant, indicating nonlinearity. In order to reduce possible multicollinearity, we did not include birthweight (which is used to calculate the BMI) with BMI in the regression analysis. To analyze whether BMI is relatively independent of birthlength, we used a linear regression analysis, with BMI as the dependent variable and gestational age and birthlength as the independent variables. In order to investigate whether the correlation between head circumference and BMI is special and is not simply the result of fetal growth, we used a linear regression analysis, with BMI as the dependent variable and gestational age, birthlength, and head circumference as the independent variables.

RESULTS

The means and standard deviations of the variables by sex are given in Table 1. Pearson's product-moments between variables are given in Table 2.

Regression analysis shows that birthlength is independent of BMI, adjusting for gestational age (Table 3) and head circumference is significantly and positively associated with BMI at birth, after gestational age and birthlength were controlled for (partial correlation $r = 0.409$, $P < 0.0001$) (Table 4).

DISCUSSION

A limitation of this study is that it does not use a direct measure of adiposity, but rather uses the BMI as a measure of body fatness in human newborns. However, measurement of body fat in the living human can be accomplished only by indirect methods, such as weight/height ratios, circumference measure-

TABLE 1. Descriptive statistics for gestational age (postmenstrual weeks), birthweight (g), birthlength (cm), birth head circumference (cm), and birth BMI (Kg/m²)

	Males	Females
Gestational age	39.57 ± 1.1	39.58 ± 1.03
Birthweight	3,385.07 ± 444.12	3,262.36 ± 430.92
Birthlength	49.66 ± 2.56	48.84 ± 1.86
Birth head circumference	34.89 ± 1.47	34.38 ± 1.45
BMI	13.8 ± 2.1	13.7 ± 1.4

Means ± standard deviations.

TABLE 2. Correlation coefficients for the anthropometric measurements for both sexes combined and by sex

	Gestational age	Birthweight	Birthlength	Head circumference	Birth BMI
<i>Both sexes</i>					
Gestational age	—	0.86*	0.85*	0.83*	0.74*
Birthweight		—	0.89*	0.87*	0.87*
Birthlength			—	0.87*	0.63*
Head circumference				—	0.74*
Birth BMI					—
<i>Males and Females^a</i>					
Gestational age	—	0.86*	0.84*	0.83*	0.71*
Birthweight	0.87*	—	0.88*	0.87*	0.86*
Birthlength	0.87*	0.90*	—	0.87*	0.56*
Head circumference	0.85*	0.87*	0.87*	—	0.71*
Birth BMI	0.77*	0.90*	0.72*	0.78*	—

^aCorrelation coefficients for males appear above the diagonal and for females below the diagonal.

*Two-sided $P < 0.001$.

TABLE 3. Results of a linear regression analysis with birth BMI as the dependent variable and gestational age and birthlength as the independent variables ($n = 1,069$, both sexes)

	Partial correlation	Two-sided P
Gestational age	0.509	<0.000001
Birth length	0.007	0.810169

TABLE 4. Results of a linear regression analysis with birth BMI as the dependent variable and gestational age, birthlength, and birth head circumference as the independent variables ($n = 1,069$, both sexes)

	Partial correlation	Two-sided P
Gestational age	0.400	<0.000001
Birthlength	-0.236	<0.000001
Birth head circumference	0.409	<0.000001

ments, skinfold thickness measurements, isotope dilution, hydrostatic weighing, gas displacement weighing, radiographic studies, inert gas inhalation, photon absorption, and total-body potassium (K) measurements by in vivo counting using the 1.46 MeV gamma-ray emission of the naturally occurring potassium isotope, ^{40}K . Each of these has its limitations and each relies on a certain number of assumptions (Lloyd and Mays, 1987). BMI is often used as a proxy measure for body fatness because it is correlated with body mass and percentage of body fat in the general population (e.g., Bouchard, 1994). The BMI has been proposed as a simple, accurate, and valid measure of fatness in childhood and adolescence that could be used worldwide

(e.g., Poskitt, 1995). The advantages of BMI are that it is easy to compute, is relatively independent of stature (e.g., Billewicz et al., 1962; Rolland-Cachera et al., 1982), and has one of the highest correlations with other measures of body fat (e.g., Micozzi et al., 1986). Due to its simplicity and high correlation with total body fat, it has been the method of choice in both pediatric clinics and research over the years (He et al., 2000). BMI is highly correlated with many skinfold thicknesses (e.g., Schroeder and Martorell, 1999). The correlation between BMI and percentage of body fat estimate from underwater weighing reported is high ($r = 0.8-0.9$) (e.g., Knowler et al., 1991). Estimates of body fat by weight/height ratios or circumference measurements only in unusually fit, muscular individuals, especially in typical athletes, classify them incorrectly as obese (e.g., Flint et al., 1977). Obviously, in this study such a problem does not exist.

The few evolutionary hypotheses that attempt to explain the ponderous condition of humans generally and human infants in particular similarly assume a connection between the loss of fur during hominid evolution and a parallel need for compensatory insulation from subcutaneous fat stores. There is some evidence supporting this insulation hypothesis. For example, the birthweight of female babies born after warm winters is significantly lighter than those born during years after cold winters (e.g., Wells, 2002). However, Pond (1997) has reviewed the literature on body-fat insulation in vertebrates, including humans, and concludes that very few data support the insulation theory, even in the case of some aquatic

mammals. Most but not all research among circumpolar human population reports show comparable or even thinner subcutaneous fat stores compared to temperate-latitude peers, suggesting that the tissue's properties as insulation may play only a minor role in human adaptation to cold, at least among children and adults (e.g., Eveleth and Tanner, 1976), and there is surprisingly little evidence that fat stores influence body temperature in human newborns (Johnston et al., 1985). On the other hand, the support for the immune theory proposed by Kuzawa (1998) is presently weak and a recent study suggests that immune function is not related to birth size (Moore et al., 2001).

However, the brain size hypothesis seems to be more consistent. Recent evolutionary perspectives on hominid encephalization hypothesize that the metabolic needs of the enlarged human brain required a suite of dietary adaptations to sustain its energy requirements (e.g., Foley and Lee, 1991; Bogin, 1997), and by implication the human infant—whose brain devours fully half of total metabolic expenditure (Epstein, 1999)—may face energetic challenges unique among mammals. This fat storage, as an adaptation made by the fetus in response to conditions that retard its growth, may be largely successful in maintaining brain development (Martyn et al., 1996). It is widely accepted that the primary function of mammalian body fat is to serve as an energy store (e.g., Norgan, 1997), and developmental changes in adiposity during other stages of the human life cycle are understood as preparation for future energetic challenges, a notable example being the rapid fat deposition of females at puberty and the subsequent contribution of this tissue to the energetics of pregnancy and lactation (McFarland, 1997). Additionally, breast-fed neonates have the capacity of using the ketone bodies acetacetate and D-3-hydroxybutyrate, in addition to glucose, as energy substrates for the brain (e.g., Girard et al., 1992). This capacity is an interesting example of a developmentally regulated adaptive mechanism, because maternal milk is highly enriched in lipids, resulting in a lipid-to-carbohydrate ratio much higher than that present in postweaning nutrients (Smith and Abraham, 1975). Indeed, lipids account for ~55% of the total calories contained in human milk, in contrast with 30–35% for a balanced postweaning diet (Smith and Abraham, 1975). Another consideration regarding the lipid-rich diet provided

during the suckling period is related to its contribution to the process of myelination (Cuzner and Davison, 1968).

The results of some studies suggest that birth fatness also has a plastic role in brain growth. For example, Loesch et al. (1999) found that birthweight and birth BMI are significantly related to head circumference at school age. It has been also ascertained that the largest increases in brain weight in both sexes seems to occur during the first 2 (Voigt and Pakkenberg, 1983) or 3 years of life, when the value quadruples over the one at birth, while during the next 15 years the brain weight quintuples over the one at birth (Dekaban, 1978). Studies by Reiss et al. (1996), using MRI techniques, reveal also that both boys and girls show little change in the total cerebral volume after the age of 5 years. Thus, this accelerated growth of brain size during the first years may help explain why newborns devote roughly 60% of growth expenditure to fat deposition during the early postnatal months (Dufour and Sautner, 2002). It has been suggested that head circumference is relatively unaffected by malnutrition (Jelliffe, 1969). However, there is considerable evidence that malnutrition in early life can have an adverse effect on the developing brain (Morgane et al., 1993). Some researchers have demonstrated smaller head circumference in malnourished populations and among the malnourished in portions of the United States (e.g., Garn and Clark, 1975).

During various critical periods in the development of the central nervous system, malnutrition has been shown to affect the morphology, physiology, and neurochemistry of the brain, and this may have important implications for future cognitive function (Winick et al., 1972). In humans, birthweight (e.g., Matte et al., 2001) and birth head size (e.g., Hack et al., 1991) have been associated with cognitive abilities. A small head circumference at birth was associated with an increased risk of minor neurological dysfunction (Hadders-Algra et al., 1988). A small head circumference at 1 year has been also associated with a lower score on intelligence tests in early childhood (Nelson and Deutschberger, 1970). Additionally, studies by Lynn and Hattery (1990) reveal that heavier twins at birth have significantly higher IQs at the age of 12, suggesting that prenatal nutrition exerts a significant effect on intelligence. Some animal studies also support this idea. For

example, studies in rats demonstrated that undernutrition during proliferative growth of the brain retards the rate of cell division and causes a permanent reduction in numbers of brain cells (Winick and Noble, 1966). Others animal studies suggest that such nutritional privation during fetal life affects later performance on tests of learning (Smart, 1977).

As a positive correlation between brain size and intelligence has been found in many studies using or not the modern technology of magnetic resonance imaging (see review by Jensen and Sinha, 1993; Tramo et al., 1998), it could be suggested that the newborn brain size and fatness increase were associated with a greater brain size and intelligence in our lineage. These results and the relation between birthweight and cognitive abilities support the brain myelination hypothesis (Miller, 1994), which contends that much variance in intelligence reflects myelination differences.

Consistent with earlier studies (e.g., Palti and Adler, 1975), in this sample newborn anthropometric variables are typically highly correlated. The statistical analysis shows a significant association between BMI and head circumference in human newborns. It could be hypothesized that newborn BMI and head circumference are simply indices of the adequacy of fetal growth, and that conditions leading to impaired growth in one would also tend to lead to impaired growth in the other. Both measures are positively correlated with birthlength, for example (Table 2). However, it is postulated that mechanisms controlling subcutaneous fat are largely independent of those controlling bony growth (e.g., Palti and Adler, 1975).

In this sample, BMI is not significantly correlated with birthlength (controlling for gestational age) as an index of fetal growth (Table 3). In contrast, the head circumference is positively and highly correlated with BMI (controlling for gestational age and birthlength), suggesting a functional connection between the two (Table 4). The negative correlation between birthlength and BMI, controlling for gestational age and head circumference, suggests that fat deposition seems to increase at the expense of the length growth.

The significant association between BMI and head circumference in human newborns, controlling for gestational age and birth length, found in the present study supports the idea that the newborn fatness is related to the higher need of lipids in new-

born humans as an energetic (Armstrong, 1983) and plastic (Martin, 1981) substrate during its accelerated brain growth period. Newborns with bigger brains require higher amounts of energy and fat substrate; thus, they need more fat storage to secure their accelerated brain growth, especially in nutritional stress conditions.

With the results of this study, it is tempting to speculate that, in our evolution, expansion of fat stores in human females during gestation (Aiello and Key, 2002) was also accompanied by an extraordinary fat deposition in the human fetus. This strategy seems to reduce the costs of lactation, the most energetically expensive phase of the reproductive cycle (Aiello and Key, 2002), in order to support the increased energy demands of encephalization (e.g., Leonard and Robertson, 1994; Aiello, 1998) and also to accommodate the inferred increase in the energy requirements of the encephalized hominid brain during ontogeny (Foley and Lee, 1991). Probably, this strategy was associated with an energy-rich diet as an adaptation to the high metabolic cost of our large brain (Leonard and Robertson, 1994). Since the majority of brain growth in humans occurs prenatally and early in the postnatal period, it seems possible that the extraordinary fat storage in newborns was a consequence of the selection for larger brain size in hominid evolution.

As birthweight, which may reflect high levels of fat deposition in the neonate, and birth head size have been associated with cognitive abilities, which are a domain-general attribute that would be favored by natural selection because it enables humans to attain evolutionary goals (MacDonald, 1997), it could be postulated that these cognitive abilities to solve problems, to learn, and to remember could have acted as a selective pressure responsible for the newborn fatness increase in our lineage.

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