Placental weight: Relation to maternal weight and growth parameters of full-term babies at birth and during childhood

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Summary

Objective: Human growth is a continuous process. Studies defining placental effect on prenatal and postnatal human growth are few.
We studied the anthropometric data of hundred mothers who gave birth at term after an uncomplicated singleton pregnancy, and their infants in relation to their placental weight using linear regression analysis. Mother weight, placental weight, and infant length (BL), weight (BW), and head circumference (HC) were obtained at birth and during childhood period (4.5–6 years) of age. At birth, placental weights were correlated significantly with maternal weights (r = 0.21, P = 0.031). Placental weights were significantly correlated with growth parameters of the child at birth and during childhood. Infant BW (r = 0.71, P < 0.001), body mass index SDS (BMI SDS) (r = 0.589, P < 0.001), length SDS (LSDS) (0.567, P < 0.001) and HC (r = 0.699, P < 0.001). During childhood, placental weights were correlated with BMI SDS (r = 0.296, P = 0.002) and HtSDS = (r = 0.254, P = 0.009). LSDS at birth was correlated significantly with HtSDS during childhood (r = 0.445, P < 0.001). Placental weight represents a good marker of fetal growth (at birth) and significantly correlates with early childhood growth in full-term infants.

Key words: placental weight, mother weight, infant length, infant BMI, child height SDS, child BMI.

Introduction

Human epidemiological observations show that the smaller the neonate the less likely it is to survive at birth and the greater its risk of developing adult-onset degenerative diseases, such as hypertension, coronary heart disease and type 2 diabetes mellitus [1–5].

In mammals, the major determinant of intrauterine growth is the placental supply of nutrients to the fetus which occurs primarily by diffusion and transporter-mediated transport [6, 7]. These processes depend upon the size, morphology, blood flow and transporter abundance of the placenta [8]. In experimental mammals, fetal body weight in late gestation correlates positively with placental weight [9].

Placentas can react to circulatory and nutritional environmental changes by adjusting nutrient transfer capacity through altering the relative proportions of different types of nutrients, e.g. amino acids, supplied to the fetus. These adaptive changes may affect the development of individual fetal tissues with pathophysiological consequences long after birth. Therefore placentas may represent a good marker of growth and disease risk in later life than many of the other indices, like birth weight, that are commonly used to indicate exposure to suboptimal conditions during intrauterine development [10, 11].

Reducing placental weight in experimental animals are associated with postnatal abnormalities in physiological function consistent with the human epidemiological data linking impaired intrauterine growth with an increased disease risk in later life [12]. However, when placental growth is compromised experimentally, it appears that more fetus is produced per gram of placenta than in normal circumstances [9]. Transfer of equine embryos between breeds led to either restricted or enhanced growth of the foal, which was directly determined by the growth of the placental allantochorion. Development of the total area of fetomaternal contact was affected by both the maternal and fetal genotypes; the maternal genotype controlled microcotyledonary surface density whereas the fetal genotype appeared to affect gross placental area probably by influencing the length of the fetal villi [5].

Different morphological and functional adaptations in relation to lacental size have been described in animals. Large and small placentas adopt different strategies in supporting fetal growth during late
gestation. Large placentas continue to adapt morphologically during late gestation whereas small placentas, appear to have a more limited capacity for morphological adaptation but can adapt functionally during this period to increase nutrient transfer relative to the large placenta. This adaptability in placental phenotype provides a functional reserve capacity to better match the placental nutrient supply with the fetal nutrient demands but also a potential mechanism of signaling environmental conditions to the fetus, and possibly postnatal events including growth [10, 13].

Little is known about the effects of variations in placental size on the postnatal growth during infancy and childhood periods of normal full-term infants. This study was designed to investigate the relation, if any, between mother weight, placental weight, birth size and postnatal growth of 100 normal full-term children.

Materials and Methods
As of 1 January 2007, 110 consecutive placentas were collected from non-smoker, non-diabetic mothers with normal blood pressure, between the age of 21 and 32, who delivered normally at term (38–42 weeks of gestation) after an uncomplicated singleton pregnancy. Parity ranged between 2 and 4 (3.2 and 3.2). The placenta was collected at spontaneous third stage labor from each mother. The weight of the placenta was performed immediately after trimming the placental disk of membranes and umbilical cord. The cord was clamped at the time of delivery, no effort was made to control for blood drainage from the placenta. The weight of placenta was recorded within 2 min following expulsion of the placenta. Of the 110 consecutive placentas collected, four cases were excluded because the trimmed placental weight was not recorded, and one case was delivered in fragments. Three were excluded because mothers had gestational diabetes. Two children did not come in fragments. Three were excluded because mothers had gestational diabetes. Two children did not come for growth assessment because families left country. This left 100 cases for analysis, 91% of the available placental sample. Placentas were grossly examined and the weight of the disc was recorded after trimming the membranes and cord (within 2 cm of insertion).

Placenta came from 81% Qatari women and 19% non-Qatari women (HMC is the major hospital where the majority (>80%) of all deliveries in Qatar occur). Mother weight (MW), placental weight (PW), infant length (BL), weight (BW) and head circumference (HC) were obtained at birth. Mothers’ weights were obtained within 3 h after giving birth. The newborns were weighed within 1 h after birth before they had nursed from their mothers. The socioeconomic status of all women were above average, as Qatar is one of the rich oil producing countries with good income and living conditions. None was poor or underprivileged. None had preeclampsia or eclampsia. Mothers mean BMI SDS (body mass index SDS) = 0.8 ± 0.6.

Infants’ growth was measured yearly for (4.5 ± 2 years) using accurate Siemens’ scales for weight and length/height. None of the children was undernourished or had a significant systemic disease. The socioeconomic status of their families was good.

BMI was measured by equation: weight (kg)/Height (m²). BMI SDS was calculated based on the WHO reference data (WHO Child growth standards: http://www.who.int/childgrowth/standards/en/).

Linear regression equation was used to investigate any relation between variables. The study was approved by the ethical committee of Hamad Medical Center and all the mothers gave informed consent to participate in this study.

Results
The anthropometric data of mothers, placentas and their full-term children, at birth and at the age of 4.9 ± 2 years, were represented in Table 1.

At birth, placental weights were correlated significantly with infant BW (r = 0.71, P < 0.001) (Fig. 1A), BMI SDS (r = 0.589, P < 0.001), LSDS (0.567, P < 0.001) (Fig. 1B), HC (r = 0.699, P < 0.001) (Fig. 1C) and maternal weight (r = 0.21, P = 0.031) (Fig. 1D) (Table 2).

During early childhood, (age = 4.9 ± 2 years), placental weights were correlated significantly with BMI SDS (r = 0.296, P = 0.002) (Fig. 2A), HiSDS (r = 0.254, P = 0.009) (Fig. 3). The infant length SDS (LSDS) at birth and birth weight were correlated significantly with HiSDS during childhood (r = 0.445, and r = 0.31, P < 0.05) (Fig. 3).

Discussion
Epidemiological studies in several human populations have shown that the pattern of fetal growth

<table>
<thead>
<tr>
<th>TABLE 1</th>
<th>Anthropometric data for mothers, placentas and children</th>
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<tr>
<td></td>
<td>Mean</td>
</tr>
<tr>
<td>Mother age (yr)</td>
<td>26.6</td>
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<tr>
<td>Mother wt (kg)</td>
<td>65</td>
</tr>
<tr>
<td>Placental wt (g)</td>
<td>678</td>
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<tr>
<td>Birth wt (g)</td>
<td>3185</td>
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<tr>
<td>Birth BMI</td>
<td>12.37</td>
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<tr>
<td>Feto-placental ratio</td>
<td>4.77</td>
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<tr>
<td>Birth LSDS</td>
<td>0.34</td>
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<tr>
<td>Birth head circumference (cm)</td>
<td>34.5</td>
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<tr>
<td>BMI (childhood)</td>
<td>16.86</td>
</tr>
<tr>
<td>BMI SDS (childhood)</td>
<td>0.01</td>
</tr>
<tr>
<td>HiSDS (childhood)</td>
<td>−0.16</td>
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<tr>
<td>HC (childhood)</td>
<td>49.4</td>
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</tbody>
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FIG. 1. Regression of placental weight on growth parameters of the newborn and mother. (A) Regression of placental weight on birth weight in 100 full-term infants. (B) Regression of placental weight on birth length in 100 full-term infants. (C) Regression of placental weight on head circumference in 100 full-term infants. (D) Regression of maternal weight on placental weight.
is an important risk factor for various adult-onset diseases. These observations have led to the hypothesis that adult health status may originate in fetal life as a consequence of nutrient provision at critical periods of development [2]. In our study, placental weight was connected significantly with fetal growth, evidenced by the highly significant correlations between placental weights and growth parameters (weight, length, and head circumference) of 100 FT singleton infants at birth.

Placental size accounts for much of the variation in size at birth in mammalian species and placental insufficiency is a major cause of intrauterine growth retardation (IUGR), but its role in later outcomes is unclear [14–18]. In animals, when the functional mass of the placenta has been experimentally reduced using a variety of techniques to reduce uterine blood flow [6, 14, 19] significant retardation of fetal growth and, in several instances, alteration of cardiovascular and metabolic functions occurred, both before and after birth [20, 21]. In human twins, the lighter placental weights of first-borns compared to second-borns were explained by the lighter birth weights of first-borns compared to second-borns [22]. In another human study, not only the weight of the placenta but also the patterns of placental
growth, relating to different functional dimensions of
the placenta, for a given placental weight, accounted
for the different birth weight shared [23].

In this study, the significant correlation between
mothers’ weight and placental weight and between
the placental weights and infants BMI at birth sup-
ported the idea that heavier mothers have larger
placentas and consequently have larger babies. In
addition, placental weights were correlated signifi-
cantly with BMI of these infants during their early
childhood. In support of our findings, a recent re-
search analysed clinical information on 1400 people
regarding the weights of mothers before and during
pregnancy and the weight of their children at birth.
The outcomes of the study revealed a clear influence
of the overweight of the mothers on the overweight
of their children. The children of mothers who put
more than 14 kg during pregnancy had a significantly
higher BMI and hip width as compared to those who
were born to mothers who did not add more than
9 kg during pregnancy [24].

Another human study showed that Large for ges-
tational age (LGA) babies had higher maternal body
mass index (BMI), fasting serum insulin, and cord
blood insulin levels compared to appropriate for ges-
tational age (AGA) group [25]. On the other hand,
experiments in sheep and rats which restricted mater-
nal dietary intake, either in total or of protein, pro-
duced significant retardation of intrauterine growth.
Our data suggested that this effect of maternal weight
(nutrition) on fetal and infantile growth can be
mediated, at least partially, through the placenta
weight and size. In favor of this view, in equids, ma-
ternal size has been shown to interact with both the
maternal and fetal genotypes to control the rate and
extent of fetal growth by influencing the gross area of
the diffuse allantochorion, and the density, complex-
ity and depth of the microcotyledons on its surface.
In mice, the adaptation in placental nutrient transfer
capacity to meet fetal growth demands depends on
placental size [5, 13].

Placental weight, as an indicator of fetal nutrition,
can select thriving genes prenatally and hence can
affect natal and postnatal growth. In this study,
placental weight was correlated significantly with
HtSDS and BMI SDS of children at 4.9 ± 2 years
of their life. The possible mechanism/s of the effect
of placental weight and birth size on postnatal
growth may include low or disturbed growth hormone (GH) secretion rate or pattern, low serum
levels of IGF-1, IGFBP-3 and leptin that have been
shown to be low in children born small for gesta-
tional age (SGA) who remained short during child-
hood [7, 12, 26–31].

In this study birth weight and length and LSDS
were correlated significantly with BMI SDS and HtSDS
during childhood. Literature review showed good evidence of association between birth weight
and subsequent BMI and overweight in children
and young adults, which is linear and positive in
some studies and J- or U-shaped in others [32]. In
addition, in a large cohort study, height in adoles-
cence was predicted by length and weight at birth
and by parents’ height, whereas BMI was predicted
by birth weight and parents’ BMI. An especially high
risk for overweight was found for subjects of average
length but a high weight at birth. These findings sug-
gest that the intrauterine period has enduring effects
on later body size but leave unresolved whether these
effects are genetic or environmental [33]. Another
cohort study in Denmark reported a positive associ-
ation between birth length and adult height that per-
sisted after adjustment for birth weight, gestational
age and other confounders [34].

Although placental weight is easily and reliably
measured and often mirrors problems of fetal
growth and development. The potential weakness
of this study is due to the fact that placenta weights
appear to be influenced by multiple maternal and
fetal processes. Increased weight is associated with
hydrops fetalis, amniotic fluid infection, maternal
diabetes and maternal anemia; while low weights

![Fig. 3. Regression of length SDS at birth on childhood height SDS.](image-url)
are associated with disorders attributed to uteroplacental hypoperfusion (i.e. preeclampsia or karyotype abnormality). Other factors influencing placental weight include parity, maternal height and weight, and serum ferritin concentration. However, in this study, we tried to avoid many of these factors through inclusion and exclusion criteria. We adopted the standard approach to weigh the placenta after the extra placental membranes and the umbilical cord are trimmed from the disk. This limits the measurement to the weight of the placental disk, the actual nutrient exchange part of the placenta. However, Leary et al. suggested that trimmed and untrimmed placental weights are exchangeable, based on their high correlation [35].

In conclusion, placental weight is a good pointer of birth size (weight, length and HC) and helps forecast childhood growth in full-term infants.

References