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Distribution of Adipose Tissue in the Newborn

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ABSTRACT

Regional differences in adipose tissue distribution are associated with differences in adipocyte metabolism and obesity-related morbidities. Intrauterine growth restriction appears to place individuals at greater risk of obesity associated morbidities in later life. Despite this, little is known regarding the quantity and distribution of adipose tissue in infants during early development. The aim of this study was to compare total and regional adipose tissue content in appropriate-for-gestational-age (AGA) and growth-restricted (GR) newborn infants born at or near term. Whole body adipose tissue magnetic resonance imaging (MRI) was performed as soon as possible after birth. Total and regional adipose tissue depots were quantified. A total of 35 infants (10 GR; 25 AGA) were studied. Mean (SD) total percentage adipose tissue was lower in GR infants than AGA infants [GR: 17.70% (2.17); AGA: 23.40% (3.85); $p = 0.003$]. This difference arose from differences in subcutaneous adipose tissue mass [mean (SD) percentage subcutaneous adipose tissue mass, GR: 16.13% (2.20); AGA: 21.44% (3.81); $p = 0.004$], but not intra-abdominal adipose tissue mass [mean (SD) percentage intra-abdominal adipose tissue, GR: 0.42% (0.22); AGA: 0.61% (0.31); $p = 0.45$]. In contrast to subcutaneous adipose tissue, intra-abdominal adipose tissue is not reduced in infants with intrauterine growth restriction. This suggests that subcutaneous and intra-abdominal adipose tissue compartments may be under different regulatory control during intrauterine life. **(*Pediatr Res* 55: 437–441, 2004)**

Abbreviations
AGA, appropriate for gestational age
AT, adipose tissue
GR, growth restricted
DEXA, dual-energy x-ray absorptiometry
MRI, magnetic resonance imaging

Epidemiologic evidence suggests that poor growth *in utero* is a risk factor for the later development of insulin resistance and type-II diabetes (1). These conditions are strongly associated with obesity and, in particular, intra-abdominal obesity. Many of the studies that have examined this hypothesis have suggested that small size at birth is a marker of the fetal adaptations that program for these diseases later in life. However, the stage of growth during which the detrimental effects of growth restriction are established is not known. There is a paucity of information regarding the general effects of intrauterine growth restriction on adipose tissue distribution at birth, and changes in total and regional content during the first few months of postnatal growth.

Previous studies of body composition in AGA and GR infants have largely relied on indirect methodology to estimate adipose tissue content and are unable to provide information about specific adipose tissue depots, particularly intra-abdominal adipose tissue.

We, and others, have shown that MRI can be used in the direct assessment of adipose tissue content and distribution in neonates (2, 3). The technique has been validated in animals and cadavers and can be readily used in longitudinal measurements (4–7).

In the present study, we have compared the content and distribution of adipose tissue at birth in infants born at or near term, with an appropriate birth weight for gestational age, with that of infants with intrauterine growth restriction.

METHODS

Mothers undergoing investigation for fetal growth restriction were initially approached during the antenatal period to explain the study. Consent to recruit their infant into the study was formally obtained after delivery. Details of antenatal growth assessments were documented. Infants were classified as either AGA or GR if they had evidence of deceleration in growth *in utero*, together with clinical signs at birth suggestive of fetal malnutrition (loose thin skin with prominent ribs, a scaphoid abdomen, and muscle wasting over the cheeks, arms, buttocks, and thighs) and had a birth weight at or below the 9th centile

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(8). Mothers were asked whether they had any dietary restrictions and data were obtained on racial group (Caucasian, African, Asian, mixed) and infant gender. The study was approved by the Hammersmith Hospitals Trust and Imperial College Faculty of Medicine Research Ethics Committee. Written informed parental consent was obtained.

MRI

Subjects were imaged in a 1.5-T Marconi Medical System Eclipse scanner as previously described (3, 7). No sedation was used and infants were positioned supine during natural sleep. We used a T_1-weighted spin-echo image sequence, with repetition time (TR) = 600 ms, echo time (TE) = 16 ms, field of view = 24 cm, number of signal averages = 2, and a 256 × 256 matrix with phase conjugate symmetry. Images were analyzed as previously described (3, 7) using an image segmentation program that employs a threshold range and a contour-following algorithm with an interactive image-editing facility.

The initial analysis of MRI data results in the expression of adipose tissue content as volume of adipose tissue. This value may be converted to adipose tissue mass on the assumption that the density of adipose tissue is 0.9g/cm^3 (5). Therefore, adipose tissue volume (cm^3) × 0.9 = adipose tissue mass (g). Adipose tissue mass may be converted to lipid (fat) mass, assuming that 1 g of adipose tissue contains approximately 0.45g lipid (9). Therefore, adipose tissue mass (g) × 0.45 = lipid mass (g).

Data in this article are expressed as adipose tissue mass, because the adipose tissue lipid content may not be the same in GR and AGA infants, nor the same in infants as in adults. We have expressed our data as lipid (fat) mass only where necessary for comparison with other published data,

Total adipose tissue was separated into total subcutaneous adipose tissue and total internal adipose tissue. Total subcutaneous adipose tissue was subdivided into abdominal subcutaneous and nonabdominal subcutaneous adipose tissue. Total internal adipose tissue was subdivided into intra-abdominal internal and nonabdominal internal adipose tissue. Abdominal adipose tissue content was obtained by quantifying adipose tissue in the slices from the top of the sacrum to the slice containing the top of the liver or base of the lungs (3). Subcutaneous adipose tissue in this region of the body was termed abdominal subcutaneous adipose tissue. Internal adipose tissue in the region was termed intra-abdominal internal adipose tissue. All other internal adipose tissue was termed nonabdominal internal. This includes, for example, adipose tissue between muscle planes and in bone marrow.

ANTHROPOMETRY

Weight (g), length (cm), and head circumferences (cm) were recorded at each examination by a single trained observer (TAMH). Each infant was weighed naked on an electronic scale (Marsden Professional Baby Scale; precision ± 2 g). Crown–heel length was measured on a recumbent infant board with a sliding footboard (Rollametre: Raven Equipment Ltd., Dunmow, Essex, U.K.). Head circumference was measured using a plastic tape measure, taking the mean of three measurements (Child Growth Foundation tape measure). Ponderal index was calculated as [birth weight (g) ÷ length^3 (cm)] × 100.

STATISTICAL ANALYSIS

Results are presented as mean and SD. Multiple regression analysis was used to compare the AGA and GR groups after adjusting for gender, postmenstrual age at the time of imaging, racial group, and whether the mother had any dietary restrictions. In view of the relatively small number of infants, these variables were selected for inclusion in the multiple regression model based on knowledge of the relevant literature and not on internal study evidence. In addition, residuals from the multiple regression model were tested for normality. The level of significance was set at 5%. Data were analyzed using Stata (10).

RESULTS

Thirty-five singleton infants were recruited into this study. Infants were imaged as soon as possible after birth. There were 25 AGA infants (14 male, 11 female) and 10 GR infants (5 male, 5 female). The mean (SD) age at imaging was 1.9 (1.8) d.

There were 20 Caucasian, 8 African/Afro-Caribbean, 2 Asian, and 8 mixed race infants. Four mothers excluded eggs, fish, and meat, but not dairy products, from their diet.

Although the infants were all born at or near term, the gestational age at birth of the GR infants was slightly but significantly less than that of the AGA infants [GR, 37.79 (1.11) wk; AGA, 39.16 (1.39) wk; $p = 0.006$]. All subsequent multiple regression analyses were therefore performed allowing for postmenstrual age at time of imaging.

As anticipated, the length, weight, head circumference, and ponderal index of the GR infants were significantly smaller than the AGA infants (Table 1).

Absolute total adipose tissue mass and all subcutaneous compartments were significantly smaller in GR infants (Table 2) having allowed for postmenstrual age, gender, maternal dietary restriction, and race. There was no significant differ-

Table 1. *Anthropometric data (mean, SD) in GR and AGA infants*

	GR (n = 10)	AGA (n = 25)	Coefficient	95% CI	p Value
Birth weight (kg)	2.28 (0.25)	3.30 (0.44)	−0.884	−1.203 to −0.565	<0.001
Length (cm)	46.39 (1.56)	50.20 (1.48)	−3.281	−4.499 to −2.063	<0.001
Head circumference (cm)	32.08 (1.22)	34.36 (1.35)	−1.927	−3.021 to −0.832	0.001
Ponderal index (g/cm^3)	2.29 (0.25)	2.61 (0.31)	−0.281	−0.529 to −0.032	0.028

Coefficient and 95% confidence interval (CI) estimating mean difference (GR-AGA) from multiple regression analysis allowing for gestational age.

Table 2. *Mean (SD) absolute adipose tissue content (kg) in GR and AGA infants*

	GR (n = 10)	AGA (n = 25)	Coefficient	95% CI	p Value
Total	0.398 (0.071)	0.757 (0.219)	−0.315	−0.505 to −0.125	0.002
Total internal	0.035 (0.010)	0.064 (0.024)	−0.021	−0.04 to 0.0009	0.060
Total subcutaneous	0.363 (0.068)	0.693 (0.205)	−0.295	−0.473 to −0.116	0.002
Intra-abdominal	0.010 (0.005)	0.020 (0.011)	−0.006	−0.016 to 0.0035	0.194
Internal nonabdominal	0.026 (0.006)	0.044 (0.015)	−0.014	−0.028 to − 0.0006	0.042
Subcutaneous abdominal	0.045 (0.011)	0.100 (0.041)	−0.045	−0.081 to − 0.009	0.016
Subcutaneous nonabdominal	0.317 (0.059)	0.593 (0.168)	−0.250	−0.396 to −0.104	0.002

Coefficient and 95% confidence interval (CI) estimating mean difference (GR-AGA) from multiple regression analysis allowing for postmenstrual age, gender, race and maternal dietary restriction.

Table 3. *Mean (SD) adipose tissue mass expressed as a percentage of body weight in GR and AGA infants*

	GR (n = 10)	AGA (n = 25)	Coefficient	95% CI	p Value
Total	17.70 (2.17)	23.40 (3.85)	−5.529	−8.964 to −2.092	0.003
Total internal	1.56 (0.39)	1.96 (0.58)	−0.257	−0.805 to 0.291	0.344
Total subcutaneous	16.13 (2.20)	21.44 (3.81)	−5.272	−8.695 to −1.848	0.004
Intra-abdominal	0.42 (0.22)	0.61 (0.31)	−0.108	−0.397 to 0.181	0.449
Internal nonabdominal	1.14 (0.24)	1.35 (0.34)	−0.149	−0.476 to 0.179	0.360
Subcutaneous abdominal	2.03 (0.48)	3.07 (0.84)	−0.854	−1.623 to −0.085	0.031
Subcutaneous nonabdominal	14.10 (1.80)	18.37 (3.17)	−4.418	−7.221 to −1.615	0.003

Coefficient and 95% confidence interval (CI) estimating mean difference (GR-AGA) from multiple regression analysis allowing for postmenstrual age, gender, race and maternal dietary restriction.

ence between the groups in total internal and intra-abdominal internal adipose tissue content.

Similarly, when expressed as a percentage of body weight, total adipose tissue mass and all subcutaneous compartments were significantly smaller in the GR infants but there was no significant difference in internal adipose tissue compartments (Table 3).

Both Asian infants were GR. However, multiple regression analysis revealed no influence of race on adipose tissue compartments, nor was any influence of maternal dietary restriction identified. There were no significant differences in adipose tissue mass between male and female GR and AGA infants (total adipose tissue mass: GR male, 17.08% (2.90%); GR female, 18.31% (1.12%); AGA male, 22.76% (4.11%); AGA female, 24.22% (3.51%)].

DISCUSSION

In adults and children, obesity is strongly associated with increased morbidity. A growing body of largely epidemiologic evidence, such as that from the Dutch famine cohort (11), suggests that intrauterine malnutrition is a risk factor for later obesity but the pathophysiological basis for this association is unknown. Although the Dutch famine cohort was exposed to extremes of maternal malnutrition and, as such, is different from the infants in our study, evidence from studies in other groups of infants in whom the etiology of poor intrauterine growth was unlikely to be due to maternal malnutrition have reported similar associations with later obesity (1). The infants in our study had the characteristic phenotype of compromised nutrition, namely loose, thin skin with prominent ribs, a scaphoid abdomen, and muscle wasting over the cheeks, arms, buttocks and thighs, and fetal malnutrition was due to preeclampsia and placental insufficiency and not to maternal malnutrition.

There appear to be depot-specific differences in the risks of adiposity-associated morbidity (12, 13), in keeping with depot-specific differences in the expression of genes coding for adipocyte proteins (14). Insulin resistance appears more powerfully related to increased intra-abdominal adipose tissue mass than to subcutaneous tissue mass. Conversely, generalized obesity and increased subcutaneous adipose tissue mass are associated with raised leptin levels (13, 15). Nonobese adults who had fetal malnutrition have been shown to have low serum leptin levels but higher intra-abdominal adipose tissue mass and greater insulin resistance than control subjects (16). Although these and similar observations have largely been made in adults, evidence is beginning to emerge that the distribution of adipose tissue is also a marker for morbidity in younger age groups. Intra-abdominal obesity has been shown to be associated with raised triglyceride levels in children (17). Whether the distribution of adipose tissue in infancy is also a marker for later morbidity is a question that will need to be addressed.

Using whole body adipose tissue MRI, we have confirmed previous observations of a reduction in total adipose tissue mass in GR infants. However, we have additionally made the novel observation that this difference arises because of a difference in subcutaneous adipose tissue but not intra-abdominal adipose tissue. To ensure that we identified infants with evidence of intrauterine malnutrition as distinct from infants who were merely small at birth, we used a stringent set of criteria encompassing antenatal anthropometry and newborn phenotype together with a birth weight below the 10th centile.

Our findings support the conclusions of Gardeil *et al.* (18), who showed that reduced abdominal subcutaneous adipose tissue detected on antenatal ultrasonography is a predictor of growth restriction. In contrast to the highly significant difference in subcutaneous adipose tissue between the AGA and GR groups, we found no significant difference in intra-abdominal adipose tissue content, though we accept that our sample size was small. If confirmed in a larger sample size, this observation would suggest that subcutaneous adipose tissue and intra-abdominal adipose tissue may be under different regulatory control during intrauterine life. Our data have added to the limited information on body composition in infancy. Fomon and Nelson (19) have reviewed the available evidence in updating their characterization of the "reference infant." Much of the data relating to fat and/or adipose tissue content in infancy is derived from indirect methods, principally DEXA, skinfold thickness, and total body electrical conductivity. These methods are unable to distinguish discrete adipose tissue deposits. Fat mass can also be estimated indirectly from the determination of lean body mass by methods such as total body potassium, total body water, total body impedance, and total body electrical conductivity. These methods are sensitive to the hydration factor of lean body mass and, as the error of estimation of fat mass is inversely related to fat mass, are inappropriate for use in preterm infants. Although widely used, the error of estimation of fat mass by DEXA also increases as fat mass decreases and has been reported to be $\pm 30\%$ at a fat content of 100 g (20).

The evidence relating to gender-specific differences in adiposity at birth are conflicting. We found no significant difference in percentage adipose tissue mass for AGA male and female infants at birth. We have also presented these data as lipid (fat) mass for comparison with other published data. Fomon *et al.* (21), in constructing their "reference" male and female infants, drew on body composition data from a variety of sources. They made the assumption that the ratio of fat to body weight is the same as the ratio of truncal skinfold thickness to body weight. They derived values for fat mass at birth of 13.7% for males and 14.9% for females. We estimated percentage lipid (fat) mass in AGA infants to be 10.2% in male infants and 10.9% in females. This estimate is similar to that of Butte *et al.* (22) (11.4% males, 14.2% females), but less than the estimate of Rigo *et al.* (20) (15.7%), derived from DEXA analyses. These differences may relate to differences in deriving fat mass from adipose tissue mass, the wide range of techniques used, and poor discrimination between AGA and GR infants. We calculated fat mass from adipose tissue mass assuming that 1 g of adipose tissue contains 0.45 g fat. This figure was based on the results of direct analysis of adipose tissue in biopsy specimens from nine term neonates (9). However, Kabir and Forsum (23) suggest that 1 cm^3 of adipose tissue contains 0.66 g fat. They derived this estimate from direct analysis of adipose tissue in biopsy specimens from 38 infants. The use of these data would lead to an estimate of percentage lipid (fat) mass in the neonates in our study of AGA male 16.7% and AGA female 17.8%. Fomon *et al.* (21) based their calculations on the assumption that adipose tissue contains 40% fat. Butte *et al.* (22) studied a total of 76 male and female infants from 0.5 mo to 2 y. They measured total body water by deuterium oxide dilution and estimated total body potassium ^{40}K using a whole body counter. Using a multicomponent model based on a series of classical assumptions, they estimated percentage fat mass to be significantly higher in girls than boys at 6 and 9 mo of age but not at birth. The values that Butte *et al.* (22) derived for fat mass in male and female infants at birth are very similar to that of Owen *et al.* (24), who summarized data from whole body chemical analyses of six stillborn male infants, believed to have been born at term, and found that fat comprised 12% of body weight.

Genetic influences on adiposity are recognized. The Pima Indians, who have disproportionate levels of obesity and type-II diabetes in adult life, have been shown to gain excessive weight in infancy (25). Racial differences in adipose tissue distribution have been demonstrated in prepubertal children (26), but evidence to support the view that genetic differences are manifest at birth is lacking. Koo *et al.* (27) showed no effect of race on adipose tissue mass in normally grown or large for gestational age infants. We found no influence of race on adipose tissue distribution but we accept that our sample size was small. Equally, the power of our study was insufficient to detect any influence of differences in maternal dietary intake.

The postnatal growth trajectory appears to be a further influence on the risk of later obesity (28, 29). Observations in the ALSPAC (Avon Longitudinal Study of Pregnancy and Childhood) cohort show that children who experienced antenatal malnutrition and subsequent rapid catch-up growth were at greater risk of adiposity at the age of 5 y than infants who maintained stable postnatal weight centiles (28). Currently accepted pediatric practice is to promote catch-up growth in infants with intrauterine growth restriction. Improved understanding of the determinants of differential adiposity may lead this view to be questioned. Our novel observation suggesting that the reduction in subcutaneous adipose tissue in GR infants at birth is not accompanied by a reduction in intra-abdominal adipose tissue suggests that there may be differences in the fetal programming of specific adipose tissue depots. To our knowledge, there have been no studies examining depot-specific alterations in adipose tissue during early postnatal growth. These will be essential to pave the way for focused nutritional or other interventions aimed at ameliorating the long-term consequences of intrauterine growth restriction.

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