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PAPER

Neonatal anthropometry: the thin–fat Indian baby. The Pune Maternal Nutrition Study

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OBJECTIVE: To examine body size and fat measurements of babies born in rural India and compare them with white Caucasian babies born in an industrialised country.
DESIGN: Community-based observational study in rural India, and comparison with data from an earlier study in the UK, measured using similar methods.
SUBJECTS: A total of 631 term babies born in six rural villages, near the city of Pune, Maharashtra, India, and 338 term babies born in the Princess Anne Hospital, Southampton, UK.
MEASUREMENTS: Maternal weight and height, and neonatal weight, length, head, mid-upper-arm and abdominal circumferences, subscapular and triceps skinfold thicknesses, and placental weight.
RESULTS: The Indian mothers were younger, lighter, shorter and had a lower mean body mass index (BMI) (mean age, weight, height and BMI: 21.4 y, 44.6 kg, 1.52 m, and 18.2 kg/m^2) than Southampton mothers (26.8 y, 63.6 kg, 1.63 m and 23.4 kg/m^2). They gave birth to lighter babies (mean birthweight: 2.7 kg compared with 3.5 kg). Compared to Southampton babies, the Indian babies were small in all body measurements, the smallest being abdominal circumference (s.d. score: −2.38; 95% CI: −2.48 to −2.29) and mid-arm circumference (s.d. score: −1.82; 95% CI: −1.89 to −1.75), while the most preserved measurement was the subscapular skinfold thickness (s.d. score: −0.53; 95% CI: −0.61 to −0.46). Skinfolds were relatively preserved in the lightest babies (below the 10th percentile of birthweight) in both populations.
CONCLUSIONS: Small Indian babies have small abdominal viscera and low muscle mass, but preserve body fat during their intrauterine development. This body composition may persist postnatally and predispose to an insulin-resistant state.
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Keywords: Indian babies; body size; muscle; body fat; insulin resistance syndrome; thrifty phenotype

Introduction

India is experiencing a rapidly escalating 'epidemic' of Type II diabetes[1] and coronary heart disease (CHD).[2–4] In a recent survey of adults in six cities, 12% were found to be diabetic and 14% had impaired glucose tolerance (IGT).[5] This represents a more than five-fold increase over the last 30 y. Today, India has more diabetic patients (~25 million) than any other single country, and the number is predicted to rise to 57 million by the year 2025.[6] It is also predicted that CHD will become the leading cause of death by the year 2015.[4] Indians as a group are insulin resistant compared to many other populations[7] and manifest features of the 'insulin resistance syndrome (IRS).[8,9] This occurs on the background of a characteristic body composition: Indians are thin by conventional criteria (low body mass index (BMI)) but are centrally obese.[10] Recent research suggests that adult Indians have more body fat and lower muscle volumes than white Caucasians, African Americans and other ethnic groups of comparable BMI.[11–14] Thus, the Indian body composition could be described as adipose but muscle thin.

The current diabetes epidemic in India is traditionally attributed to 'thrifty' genes, which helped survival in the past when food supply was intermittent but have become detrimental in the modern context of plentiful food and reduced physical work.[15] A recently proposed alternative explanation is the 'thrifty phenotype' (fetal origins) hypothesis,[16,17] which proposes that persistent metabolic and structural changes caused by fetal under-nutrition increase

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the risk of type II diabetes and CHD. In the majority of studies, low birthweight is used as an indicator of fetal undernutrition. Indian babies are among the lightest in the world and might be expected to be at increased risk of type II diabetes and CHD.[18] However, birthweight is a poor indicator of the complexities of intrauterine growth and does not provide information about body composition that may be relevant to subsequent morbidity and mortality. A better characterisation of the body composition at birth of Indian babies may improve our understanding of the 'thrifty phenotype' and fetal origins of adult disease. We, therefore, studied in detail the birth measurements of Indian babies and compared them with those of white Caucasian babies born in Southampton, UK, who were measured using similar methods. The babies in the UK belong to a population that is relatively well nourished, providing a 'control' population.

Methods

Details of the Pune Maternal Nutrition Study have been reported.[19,20] In brief, all married women ($n=2675$) of childbearing age living in six villages near the city of Pune (Maharashtra, India) who agreed to be studied were followed up every month to record their menstrual dates, and every 3 months for detailed anthropometry. A total of 797 women who became pregnant were studied twice during pregnancy for their nutritional status, biochemical parameters and fetal growth. Enrolment began in June 1994 and ended in April 1996. The babies in Southampton were measured as part of a student research project in 1987 that aimed to recruit all liveborn singleton babies without major congenital malformations born on weekdays between 19 January and 25 April 1987[21] ($n=668$) in the Princess Anne Hospital, the main maternity hospital in the city. In both populations, gestational age was derived from the LMP, unless it differed from that derived from an early ultrasound scan (<20 weeks gestation) by more than 2 weeks, in which case the latter was used. The analysis is limited to singleton, liveborn, full-term (gestational age $\geq$ 37 weeks) babies.

Measurement of the babies

In Pune, the babies were measured at birth by one of five trained fieldworkers. Birthweight was measured using a Salter spring balance; crown–heel length using a portable Pedobaby Babymeter (ETS JMB, Brussels, Belgium); occipitofrontal head circumference, mid-upper-arm circumference (MUAC) and abdominal circumference using a fibre glass tape (CMS Instruments, London, UK) and subscapular and triceps skinfold thicknesses using Harpenden skinfold callipers (CMS Instruments, London, UK). Abdominal circumference was measured immediately above the umbilical cord insertion, in expiration. In Southampton, birthweight was measured using a digital electronic weighing scale in the labour ward. Other measurements were made by one of two trained observers. The crown–heel length was measured using a Harpenden infant stadiometer (CMS instruments, London, UK), head circumference, MUAC and abdominal circumference using blank paper tapes that were marked and measured against a steel rule. Unlike in Pune, the abdominal circumference was measured at the level of the xiphisternum. Subscapular skinfold thickness was measured using Harpenden skinfold callipers (CMS instruments, London, UK). In both centres, a time limit of 72 h postdelivery was put on these measurements.

Ethical permission for the study was given by the respective Ethical Committees of both institutes and by village authorities in Pune.

Statistical methods

Data are presented as means (s.d.) for normally distributed variables; skinfold thickness measurements and maternal weight and BMI were log transformed to satisfy assumptions of normality and are shown as the median and interquartile range. We compared Pune and Southampton babies using sex- and gestation-specific s.d. scores: Pune s.d. score = (Pune observation–Southampton mean)/Southampton s.d. Because abdominal circumference was measured at different levels in the two places, xiphisternum in Southampton and umbilicus in Pune, we subsequently measured 50 full-term Pune babies, at both levels, allowing us to calculate comparable regression-adjusted xiphisternum values for the Pune data. Both unadjusted and adjusted values are reported. We also examined within-population s.d. scores, (individual observation−whole cohort mean)/whole cohort s.d., to examine how different anthropometric measurements varied with birthweight within each population. Paired *t*-tests were used to compare s.d. scores for different measurements at different levels of birthweight (<10th centile, 10–90th centile and >90th centile) within each population. The data were analysed using the software package SPSS/PC v 5.0.

Results

Of the 797 pregnant women enrolled in the Pune study, 12 had spontaneous abortions, 14 had late terminations and one died of pregnancy-induced hypertension. In all, 770 infants were delivered, of whom 71 were premature, eight were stillborn, nine had major anomalies and 51 did not have birth measurements within 72 h. Babies born to one woman with diabetes, and one with pregnancy-induced hypertension were excluded. Our analysis relates to 631 babies. Of 668 normal liveborn singleton babies born in Southampton, 235 were not measured within 72 h, a further 13 were premature, four were born to diabetic mothers and 35 were born to mothers with hypertension. Our analysis relates to 338 babies.

Comparison between Pune and Southampton mothers and babies

The characteristics of the mothers and their babies are shown in Table 1. Pune mothers were younger and smaller in all

Table 1 Characteristics of mothers and babies

	Pune (n = *631*)		*Southampton* (n = *338*)	
	Mean	*(s.d.)*	*Mean*	*(s.d.)*
Mothers				
Age (y)	21.4	(3.6)	26.8	(5.1)
Pre-pregnant weight (kg)	41.7	(5.1)	Not measured	
Weight at 20 weeks gestation (kg)	44.6	(41.1, 48.2)[a]	63.6	(55.9, 71.3)[a]
Height (m)	1.52	(0.05)	1.63	(0.06)
BMI at 20 weeks gestation (kg/m^2)	18.2	(19.2, 20.5)[a]	23.4	(21.5, 26.4)[a]
Babies				
Gestational age (days)	275.9	(8.2)	280.6	(8.1)
Birthweight (g)	2666	(355)	3494	(483)
Crown–heel length (cm)	47.7	(2.0)	49.8	(1.9)
Ponderal index (kg/cm^3)	24.5	(2.5)	28.2	(2.3)
Head circumference (cm)	33.1	(1.2)	35.2	(1.3)
Skinfold thickness				
Triceps (mm)	4.2	(3.6, 4.6)[a]	Not measured	
Subscapular (mm)	4.2	(3.6, 4.6)[a]	4.6	(4.1, 5.5)[a]
MUAC (cm)	9.7	(0.9)	11.5	(1.0)
Abdominal circumference at the level of xiphisternum (cm)	29.6	(1.9)[b]	33.6	(1.7)
Abdominal circumference at the level of umbilicus (cm)	28.6	(1.9)	Not measured	
Placental weight (g)	360	(76)	520	(112)

[a]Median and interquartile range.
[b]Derived from umbilical measurement (see statistical methods).

respects than the UK mothers. Maternal weight and height were lower by 2.09 (95% CI: −2.15 to −2.03) and 1.68 (95% CI: −1.74 to −1.62) standard deviations, respectively (Figure 1a). Gestational age at delivery was lower in Pune by an average of 4.7 days. Even after allowing for their smaller gestational age, the Indian babies were smaller in all measurements than the babies born in Southampton (Figure 1a). Birthweight, placental weight, and neonatal head and mid-arm circumferences were reduced to a degree comparable to maternal measurements (s.d. scores: −1.74 (95% CI: −1.81 to −1.68); −1.43 (95% CI: −1.50 to −1.37), −1.68 (95% CI: −1.76 to −1.61) and −1.82 (95% CI: −1.89 to −1.75), respectively). There was a larger deficit in abdominal circumference (s.d. score: −2.99; 95% CI: −3.09 to −2.89), even after adjustment for the different measurement technique in Southampton (s.d. score: −2.38; 95% CI: −2.48 to −2.29). In contrast, neonatal length was relatively spared (s.d. score: −1.01; 95% CI: −1.09 to −0.93), and subscapular skinfold thickness markedly so (s.d. score −0.53; 95% CI: −0.61 to −0.46). Low birthweight Pune babies (<2500 g) also demonstrated fat preservation relative to birthweight when compared to the Southampton babies (Figure 1b). When we compared neonatal measurements of babies with comparable birthweight (2800–3300 g) in Pune and Southampton, Pune babies were longer and more adipose (subscapular skinfold), although Southampton babies were larger in other measurements (Table 2). A conventional measure of neonatal 'thinness' is the ponderal index (PI). A comparison of the relationship between subscapular skinfold and PI in the two populations revealed that at any subscapular skinfold thickness, the Indian babies have a lower PI (thinner) than that of the white Caucasian babies (Figure 2).

Within-population analysis

Figure 3 shows s.d. scores for the anthropometric measurements, with the babies divided into three groups: <10th percentile, 10th–90th percentile and >90th percentile, in this case calculated within each of the two populations. A similar pattern was seen in both places, such that at low birthweights (<10th percentile) there was relative sparing of body fat (Pune subscapular s.d. score: −1.00, 95% CI: −1.23 to −0.78; Southampton subscapular s.d. score: −0.92, 95% CI: −1.17 to −0.68) compared with birthweight (Pune birthweight s.d. score: −1.57, 95% CI: −1.71 to −1.43; Southampton birthweight s.d. score: −1.39, 95% CI: −1.53 to −1.25). The difference between subscapular s.d. score and birthweight s.d. score was statistically significant ($P<0.001$ in both centres). In both Pune and Southampton, the only

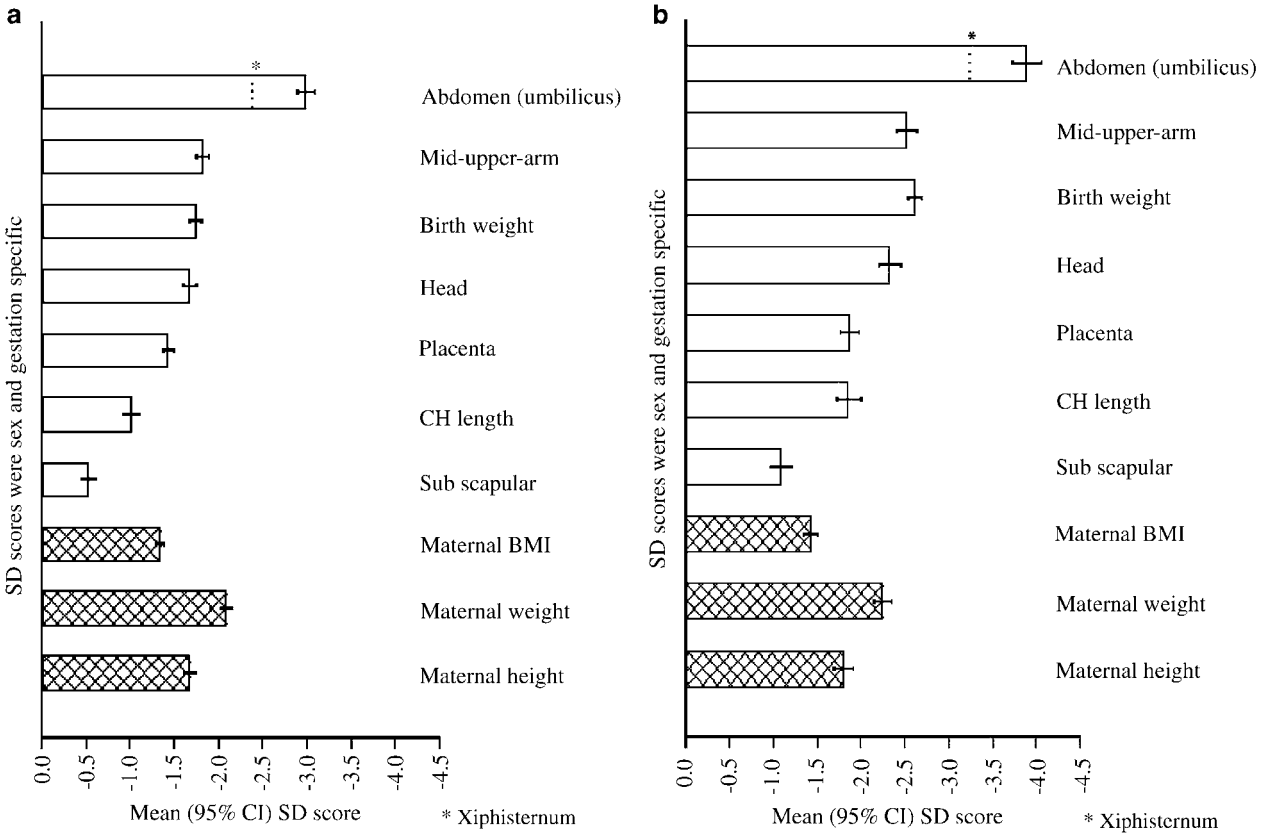

Figure 1 (a) s.d. Scores for mothers and neonates in Pune compared to Southampton. (b) s.d. scores for mothers and neonates in Pune weighing <2500 g compared to Southampton.

Table 2 Comparison of body composition in babies of comparable birthweight (2800–3300 g) in Pune and Southampton

	Pune (n = *162*)		*Southampton* (n = *114*)		
	Mean	*(s.d.)*	*Mean*	*(s.d.)*	P
Gestational age (days)	279	(7.2)	278	(8.0)	0.3
Birthweight (g)	3008	(116)	3066	(139)	<0.001
Crown–heel length (cm)	49.1	(1.5)	48.4	(1.2)	<0.001
Ponderal index (kg/cm^3)	25.6	(2.3)	27.0	(1.8)	<0.001
Head circumference (cm)	33.9	(0.9)	34.4	(0.8)	<0.001
Skinfold thickness					
Triceps (mm)	4.6	(4.0, 5.2)[a]	Not measured		
Subscapular (mm)	4.6	(4.2, 5.2)[a]	4.1	(3.7, 4.5)[a]	<0.001
MUAC (cm)	10.3	(0.6)	10.8	(0.5)	<0.001
Abdominal circumference at the level of xiphisternum (cm)	30.9	(1.2)[b]	32.3	(0.9)	<0.001
Abdominal circumference at the level of umbilicus (cm)	30.0	(1.2)	Not measured		
Placental weight (g)	399	(72)	460	(85)	<0.001

[a]Median and interquartile range.
[b]Derived from umbilical measurement (see Statistical methods).

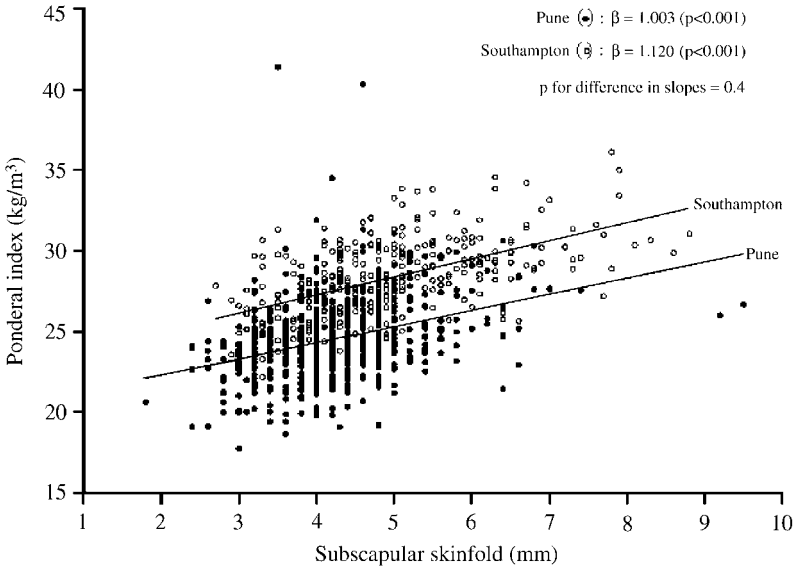

Figure 2 Neonatal ponderal index according to subscapular skinfold thickness.

other body measurement similarly preserved was the head circumference (Figure 3, Pune head circumference s.d. score: −0.94, 95% CI: −1.18 to −0.70; Southampton head circumference s.d. score: −0.96, 95% CI: −1.22 to −0.70).

Discussion

To our knowledge, this is the first population-based study in which detailed anthropometric measurements have been made in a large number of rural Indian women and their newborn babies. Consistent with other Indian data,[18,22] the babies had low birthweights. We have gone beyond birthweight, however, to define the detailed phenotype of Indian babies. There was a pattern in their smallness (Figure 1), with relative sparing of subcutaneous fat. In contrast, abdominal circumference, which reflects visceral size, was markedly reduced, as was MUAC, which reflects muscle bulk.

We have compared Indian babies with those born in Southampton 10 y ago and, importantly, measured using similar anthropometric techniques. There are few other data sets with such detailed measurements of the baby. The UK represents an affluent industrialised country where mean birthweight has changed only modestly over many decades: there has been an increase in percentage of heavier birthweights as well as very low birthweights, the distribution shifting towards heavier birthweights.[23] Analysis in Scotland suggested that the increase in birthweight is contributed by increased maternal height and age, by a decrease in the proportion of induced births and also by a reduction in maternal smoking.[24]

Our findings suggest that in these underweight Indian babies, fat deposition continues, while abdominal viscera and muscle are 'sacrificed'. Although apparently 'thin', these babies are relatively adipose. This is clearly seen in Table 2, which shows that at comparable birthweights Indian babies have higher subscapular fat compared to the white babies and Figure 2, which shows that for a given subcutaneous fat, Indian babies are thinner as measured by PI. Our findings are consistent with studies of aborted fetuses from undernourished Indian mothers, who were deficient in protein, calcium and iron, but had more fat than UK fetuses.[25] The smallest Indian babies (<10th centile of birthweight for the population) also showed a fat-preserving tendency compared to normal weight (10–90th centile) babies, in addition to the well-known brain-preserving tendency. This was true even for Southampton babies. Fat preservation in growth-retarded fetuses has been shown in studies of animals. Widdowson[26] reported 20 y ago that growth-retarded newborn guinea pigs had a higher percentage of body fat compared with controls. Relative fat preservation has also been described in small for gestational age (SGA) babies in the USA[27] and in the smaller of twins in later life.[28] A comparison of birthweights in Australian Aboriginal and non-Aboriginal neonates showed the former to be 450 g lighter and had smaller triceps skinfold thickness, subscapular skinfolds were not measured.[29] Late gestation is a period of rapid fat gain for the fetus because of differentiation and hyperplasia of adipocytes.[30] We have reported that maternal pre-pregnant size, weight gain during pregnancy and intake of fats and micronutrient-rich foods (green leafy vegetables, fruits and milk) are significant determinants of neonatal size.[19,31] Neonatal skinfold thicknesses are specifically predicted by maternal pre-pregnant fat mass, head circumference, and her fat intake and the frequency of consumption of green leafy vegetables during pregnancy. Thus, the nutritional his-tory of the mother in the distant past as well as during preg-nancy may influence fetal adiposity and body composition. Genetic polymorphisms (eg PPAR-γ)

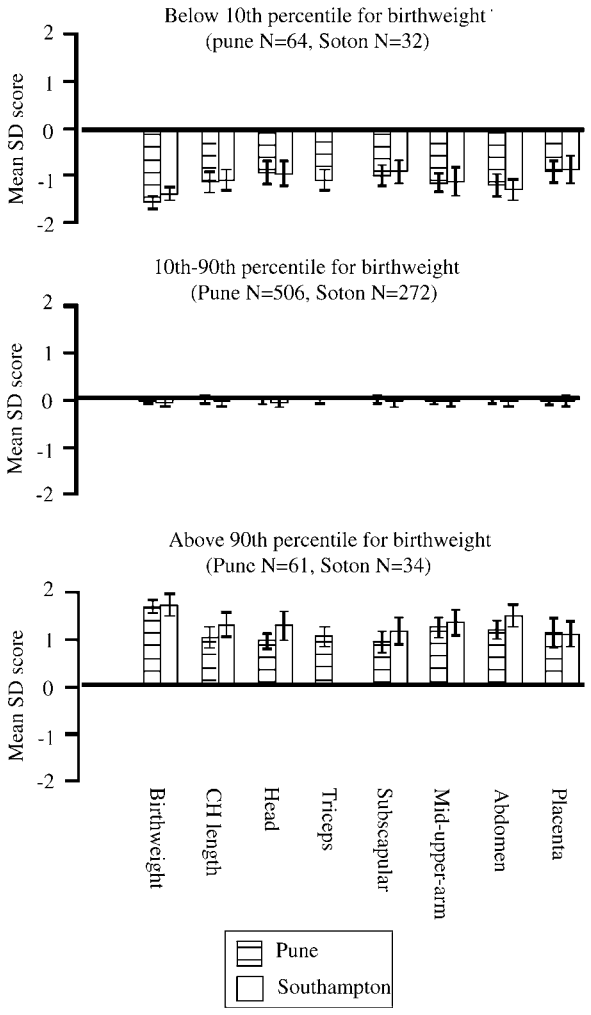

Figure 3 Within-population s.d. scores for neonatal measurements at three levels of birthweight.

may also influence adipogenesis through nutrient–gene interactions.[32,33]

Fat stores have an advantage for neonatal survival, acting as a store of energy, insulation to maintain body temperature and a depot of precursors for the rapidly developing brain. Sociobiologists believe that fat also makes the baby more attractive to the mother and thus promotes feeding.[34] A tendency to lay down fat may continue into adult life and lead to obesity, as seen in offspring of diabetic mothers.[35,36] Birthweight is a strong predictor of body size in Indian children.[37] The only Indian study to report on the association between birth measurements and adult diabetes showed that higher PI ('obesity') at birth predicted later diabetes.[38]

Neonatal triceps skinfold measurements were not done in Southampton; therefore, we do not know if the 'fat-sparing' tendency of the Indian babies is generalised or specific to the subscapular region. We have shown, however, that the ratio of subscapular to triceps skinfold thickness is increased in 8-year-old Indian children who were born with low birthweight.[37] Subscapular fat, is a well-recognised depot of 'central' fat, which is associated with an increased risk of insulin resistance and cardiovascular disease. Adult Indians show a tendency to excess central fat deposition, which is linked to the IRS, diabetes and CHD.[7–10,13] We propose that this phenotype results from persistence of central fat laid down *in utero*. Adiposity, especially with central distribution, is also associated with elevated circulating levels of glucocorticoids, cytokines IL-6 and TNF-α, which contribute to insulin resistance.[39–41] A reduced muscle mass could compound insulin resistance by a tendency to poor physical activity and promoting obesity. Compromised abdominal visceral development (hepatic, pancreatic and renal) could predispose to lipid and coagulation abnormalities, disturbances of insulin secretion and abnormalities of salt handling, thus increasing cardiovascular risk.

A number of other physiologic peculiarities observed in Indians may be explained by intrauterine programming of metabolism and structure. Thus, a tendency to synthesise fat even in adverse conditions may explain the phenomenon of ketosis resistance in varieties of diabetes peculiar to developing countries.[42] Poor muscle mass in Indians reflects in poor work capacity and in reduced energy expenditure.[43,44]

In summary, we have studied neonatal size and body measurements in a rural Indian population. Compared with UK babies, Indian babies were small. There was a substantial deficit in nonfat soft tissues, while subcutaneous fat was preserved. Thus, abdominal viscera and muscle suffered the most, while subscapular fat was the most preserved. Genetic factors undoubtedly influence body size and composition of a developing fetus.[45,46] We have shown that maternal nutrition before and during pregnancy also plays an important role in determining fetal body composition. The adult Indian phenotype of excess total and central body fat in a relatively 'thin' individual may originate *in utero* and predispose Indian men and women to IRS. Serial follow-up of our children will indicate the importance of body fat at birth in relation to adult disease. Further research will clarify whether fetal body composition is modifiable by nutritional interventions in the mother and thus influence the risk of disease in the offspring.

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