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Intergenerational Study of Trends in Human Birth Weight across Two Successive Generations

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Abstract

Objective—To determine the correlation between parental and offspring birthweight (BW) in a developing country like India.

Methods—The study involved two birth cohorts of successive generations. The parental cohort comprised of 472 fathers and 422 mothers from an earlier study. Details of their anthropometry at birth and in adulthood were available. 1525 children born to them comprised the offspring cohort. BW was obtained from hospital records for the offspring cohort. Odds ratios and regression coefficients were calculated to estimate the risks of a low birth weight (LBW) parent producing a LBW baby and quantitate the effects after adjusting for confounders.

Results—A LBW mother had a 2.8 times risk (95%CI 1.2 - 6.4) of delivering a LBW baby (p=0.02) and a LBW father was twice as likely to produce a LBW baby (OR 2.2; 95%CI 1.0 - 4.8; p=0.05). Every 100g increase in maternal BW was associated with an increase in offspring BW of 14g; the equivalent figure for paternal BW was 18.1g (p<0.001 for both). Between the generations, the incidence of LBW decreased from 19.7% to 17.2% (p=0.1). Mean BW increased in males (2846 g v 2861 g; p=0.59) but not in females (2790 g v 2743 g; p=0.08).

Conclusion—Both maternal and paternal BW are strong determinants of offspring BW. The effect of mothers' BW on offspring BW is weaker than that seen in developed nations. Stronger intrauterine constraint exhibited by Indian women secondary to a higher prevalence of growth restriction in utero may be responsible. Paternal effects may be governed by paternal genes inherited by the offspring.

Keywords

Intergenerational study; low birth weight; birth cohort

INTRODUCTION

LBW is a major health problem in India, affecting 30% of births. Though not a disease by itself, yet its ramifications are vast, with major influence on neonatal and infant survival as well as under-five and long term morbidity. In India, intrauterine growth restriction is considered the main causal factor1 and is attributed to chronic maternal malnutrition and stunting2. In the post-independence era, improvements in obstetric practices, health care,

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maternal nutrition and socio-economic conditions may have a role in bringing down the incidence of LBW babies. Still, much has to be done to improve BW in India. The first step is to examine the determinants of LBW, so that new factors can be identified and addressed accordingly.

Intergenerational studies, mainly carried out in western populations, have investigated relationships between parental (mainly maternal) BW and that of the offspring.3-21 Only one such study has been published from an Indian population.22 A high correlation was found between maternal and offspring BW to the tune of an increment in birth weight of 15 - 30 g for every 100 g increase in maternal BW. This was explained in terms of heritable maternal genes, similar intrauterine experiences from grandmother to granddaughter through the mother and external environmental factors affecting general health and nutrition. Paternal BW as a determinant of offspring BW has been less well studied.6-11 Paternal and fetal genes regulating intrauterine growth have been described.23-27 Therefore a father could also regulate fetal growth, depending on the regulation of genetic expression by intrauterine environment. This study was undertaken to assess the influence of maternal and paternal BW on offspring BW in India.

MATERIAL AND METHODS

Study design

Intergenerational study involving two birth cohorts.

Period of study

2002 - 2004.

Subjects

The present study involved two cohorts of successive generations - a parental cohort and an offspring cohort. The parents were drawn from the original cohort which formed the core population for ‘longitudinal studies in human reproduction’, reported in earlier papers.28-30 A sample of 5 localities from a total of 15 in Vellore town representing low, middle and high socio-economic strata were randomly chosen. 25 geographically contiguous villages were selected from a total of 41 villages representing the rural area in the K.V. Kuppam development block in the North Arcot (now Vellore) district of Tamilnadu. All the women in the reproductive age group were recruited and all the babies born to these women during 1969-73 were included for the earlier research studies. Figure 1 provides the details of the parental cohort. 4,092 subjects from this original birth cohort who had complete parental measurements, anthropometry at birth, and longitudinal growth assessment till adolescence recorded in their homes by trained health workers were traced for another research study undertaken during 1998-2002 (“Maternal nutrition, fetal growth and cardiovascular risk factors in young adults” - submitted for publication”). 1,837 were currently married. None had married within the cohort. Hence early life data for both members of a married couple were unavailable and either fathers or mothers with birth data, now having children were studied. Current height and weight of the spouses were recorded prospectively. There were two groups of children, one in whom only maternal data was available and one with paternal data alone (Figure 2).

Detailed information was available for the parental cohort from the previous study with regards to anthropometry at birth as well as current anthropometry, SES and parity. SES score was calculated based on 11 factors (education, occupation, type of housing, number of houses owned, number of rooms, number of people per room, water and sanitation, material

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possessions, farm, land and animals owned, agricultural land ownership, farm equipment ownership).

During the present study, for the offspring data, details of the children such as date of birth, antenatal medical problems, birth order, gestational age and birth weight were obtained from the medical records of hospitals where the equipments are precise, staff are trained and supervised, good records are maintained and available and where a reasonable standard of care is practised. A reliable gestational age was available for only 948 offspring (62%). Length at birth and head circumference were scarcely available for the offspring cohort, and therefore these data could not be used for analysis. Current anthropometry of the children was collected prospectively.

Definitions—LBW was defined as a BW less than 2500 g.

Prematurity was defined as a gestation of less than 37 completed weeks.

Statistical methods—Statistical analysis was carried out using statistical package for social sciences (SPSS) - Version 11.5 and STATA version 8.0. All birth and adult measurements were normally distributed and were used after categorization and also as continuous variables where indicated. Parental BW groups were related to offspring birth size groups using chi square tests. Associations between parent and offspring BWs were examined using linear and logistic regression with and without adjustment for parental adult height, body mass index (BMI), SES score, parity, sex of offspring and age. Random effects generalized least square (GLS) model was used to address the situation where a family had more than one child. This allowed us to include all the offspring born to a parent in the analysis (41.4% of the families had more than one offspring).

RESULTS

Birth weights of parents who were studied (894) was not significantly different from those of the parents who were not studied (3198) - mean birth weights of mothers studied (n=422) and not studied (n=1592) were 2790 (SD 444)g and 2749 (SD 528)g respectively (p=0.14); mean birth weights of fathers studied (n=472) and not studied (1606) were 2846 (SD 489)g and 2824 (SD 565)g respectively (p=0.44).

Two thirds of the families studied hailed from rural areas. In both generations, girls weighed less than boys at birth (Table 1). The mean BW remained unchanged between the two generations (parental cohort 2820 g, offspring cohort 2803 g). The incidence of LBW babies had decreased from 19.7% to 17.2% (p=0.1) between the two generations and the trend was more evident among boys (Table 1). As compared to their fathers or mothers, the sons had a higher mean BW and were less likely to be of LBW. On the other hand daughters were likely to be lighter than their mothers at birth though they were less likely to be of LBW. Parents of higher birth weight were taller as adults. For every Kg increase in birth weight, the adult height of the father increased by 2.45 cm [95% CI: 1.5-3.4] and a mother's height by 2.04 cm [95%CI: 1.2-2.9] (p <0.001 for both).

Influence of maternal characteristics on offspring BW

The mean BW of mothers producing LBW offspring was 186 g lower than that of the mothers who produced babies of normal BW (p<0.001). The unadjusted odds ratio revealed that a LBW mother was 3.5 [95%CI: 1.5-8.0] times more likely to produce a LBW baby compared to a woman weighing >3000 g at birth (p <0.001). After adjusting for adult BMI, adult height, SES score, parity and sex of the infant, it was found that a LBW mother had a 2.8 times risk of bearing a LBW baby as compared to a mother who weighed >3000 g at

birth (p=0.02; Table 2). In this model, low maternal height was also found to be an independent risk factors for producing a LBW baby (p=0.02) while maternal age, parity, sex of the infant and SES score had no significant association with offspring BW. When birth weight was used as a continuous dependent variable, maternal BW was positively related to offspring BW (20.5g per 100g increment in maternal BW, p<0.001; Fig 2). This effect was slightly blunted after adjusting for maternal adult BMI, height, parity, sex of the infant and SES score where every 100 g increase in maternal BW was associated with an increase in offspring BW of 14.0 g (p<0.001; Table 3). Adult height (p=0.002) and BMI (p<0.001) were also positively related to the BW of the offspring.

Influence of paternal characteristics on offspring birth weight

A similar relationship was found between the BW of fathers and their offspring. LBW fathers went on to become shorter and lighter as adults (p<0.001). The mean BW of fathers producing LBW babies was 238 g less than that of fathers producing normal BW babies (p=0.001). The unadjusted odds ratio showed that a LBW father had a 2.6 [95%CI: 1.4-5.5] times risk of producing a LBW infant as compared to a father weighing >3000 g at birth (p=0.01). After adjusting for sex of the infant, paternal age, SES, adult height and BMI, the odds ratio was 2.2 (p=0.05; Table 2). Paternal BW was positively related to offspring BW (23.1g per 100g increment in paternal BW, p<0.001; Fig 3). After adjusting for paternal adult BMI, height, age, parity of spouse, sex of the infant and SES score, a 100 g increment in paternal BW was associated with an 18 g increment in the offspring BW (p<0.001; Table 3). Paternal SES score and age were unrelated to offspring BW. In this model paternal height (p=0.01) and BMI (p=0.01) were also positively related to offspring BW.

Accurate gestational age at birth was not available for 38% of the offspring hence the BWs could not be adjusted for this in the analysis. When a sub analysis was done after adjusting for gestational age for the 62% of the offspring for whom gestational age was known, the parental effects on offspring BW were similar.

DISCUSSION

While intergenerational effects on human birth weight have been extensively studied in developed nations3-16,18-21, there is a paucity of data from developing countries17,22. Strengths of our study were a large sample size, birth data were collected prospectively for the parental cohort (for whom birth measurements were made by trained research staff), birth data of the offspring were obtained only from reliable hospital records (rather than recall) and we had good data on potential confounding factors such as parental age, height, weight, sex of infant, birth order and SES. We were able to examine paternal effects while most previous studies did not address the issue. Weaknesses of the study were a lack of gestational age data, and the fact that we did not have birth measurements for both members of the married couple in the parent's generation. Hence it was not possible to compare both paternal and maternal effects in determining a given offspring's BW, or to adjust for assortative mating (people tending to marry people of similar size and from the same social background).

The incidence of LBW among the rural population near Vellore lies between 16-18%. In our own study extending over the past 30 years, the prevalence of LBW has decreased from 19.7% to 17.2% between the generations. This is a welcome change though a disappointingly small improvement. It was, however, interesting to note that mean BW did not improve as expected (parental cohort 2820 g and offspring cohort 2803 g). Among girls the mean BW had actually decreased. A similar effect was seen by Ramakrishnan et al in Guatemala where LBW rates increased from 5.1% to 13.5% and mean BW decreased by 90g between generations.17 In contrast a small increment in mean BW of 121 g was

observed in the Mysore study.22 Most of the studies from the western world have shown an increment in mean BW between generations.3, 4, 8-10, 12 This phenomenon of decreasing mean BW may be explained by the fact that, a) of late, in developing countries, more LBW babies are surviving and/or b) that birth weights in the parent's generation were falsely high because only the heavier babies at that time survived to reach reproductive age. A similar analogy can be provided for the static mean BWs.

In our study maternal BW emerged as a significant predictor of offspring BW. After adjusting for confounders, a LBW mother was at a 2.8 times greater risk of giving birth to a LBW baby than a mother who weighed more than 3000g at birth. An increase of 100g in maternal BW in one generation was associated with an increase in offspring BW of 14g in the next generation. Previous western studies and the study in Mysore have shown an increment of 15-30g.3-22 The unimpressive maternal effects on offspring BW in Vellore may be explained by the high prevalence of intrauterine growth restriction in our population. There is an intergenerational cycle of growth failure such that young girls who grow poorly become stunted women and are more likely to give birth to LBW babies.17 The ultimate determinant of intrauterine growth in these settings is probably maternal intrauterine constraint. The effect of a better extra uterine environment and good paternal and fetal genes can only improve the intrauterine growth if the intrauterine constraint is removed.

Until recently it was believed that genetic factors had little influence on BW and that the maternal intrauterine environment had the predominant role in governing offspring BW. This was based on findings that intergenerational effects on BW seem to pass through the maternal and not the paternal line.7, 31-33 A mother can theoretically influence her baby's BW both through the genes she passes to her fetus and the intrauterine environment that she provides. The effect of maternal genetic influence was questioned by observations that babies born after ovum donation correlated strongly with the ovum recipients rather than the donors with respect to BW.34 Experiments on horses by Walton and Hammond35 also had similar observations and so did others who experimented on domestic animals.35, 36 They opined that somatic growth was regulated chiefly by uterine size and the intrauterine milieu.

Our study showed that paternal BW is a strong predictor of offspring BW. The father's contribution is mainly genetic6-11,22 ,although it must be remembered that the father also contributes to the environment of the mother, economically and materially, in ways that may not be captured by relatively crude measures of SES. After adjusting for confounding factors, it was found that a LBW father was at a 2.2 times greater risk of begetting a LBW offspring than a father weighing >3000 g at birth. Every 100g increase in the BW of a father was associated with an increase in the BW of his progeny by 18 g. This effect is on a par with western populations despite the higher maternal intrauterine constraint prevalent in India. In the Mysore study this increment was found to be 25 g.

Thus, both parental and fetal genes as well as the intrauterine environment probably influence BW, but the proportion of influence that each factor exerts is debatable. The non-genetic effect probably works in one of the following three ways:

- **i.** Fetal undernutrition among female fetuses may lead to poor growth and vascularity of reproductive organs in utero and impaired placentation subsequently when they reproduce as adults. This in turn leads to another generation of growth failure. 26
- **ii.** Mother who has a good environment in utero is well nourished and grows taller and heavier as adults and this improves the BW of her own offspring.
- **iii.** A grandmother may have belief systems (e.g. restricting weight during pregnancy), which she passes on to her daughter thus causing generations of growth failure.

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Any genetic influence on BW is probably not amenable to public health intervention but improving maternal nutrition, socioeconomic status and education may positively influence the 'non genetic' heritable factors (intrauterine nutrition and environment) and this may improve BWs over subsequent generations.

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Abbreviations

LBW	low birth weight
BW	birth weight
SES	socio economic status

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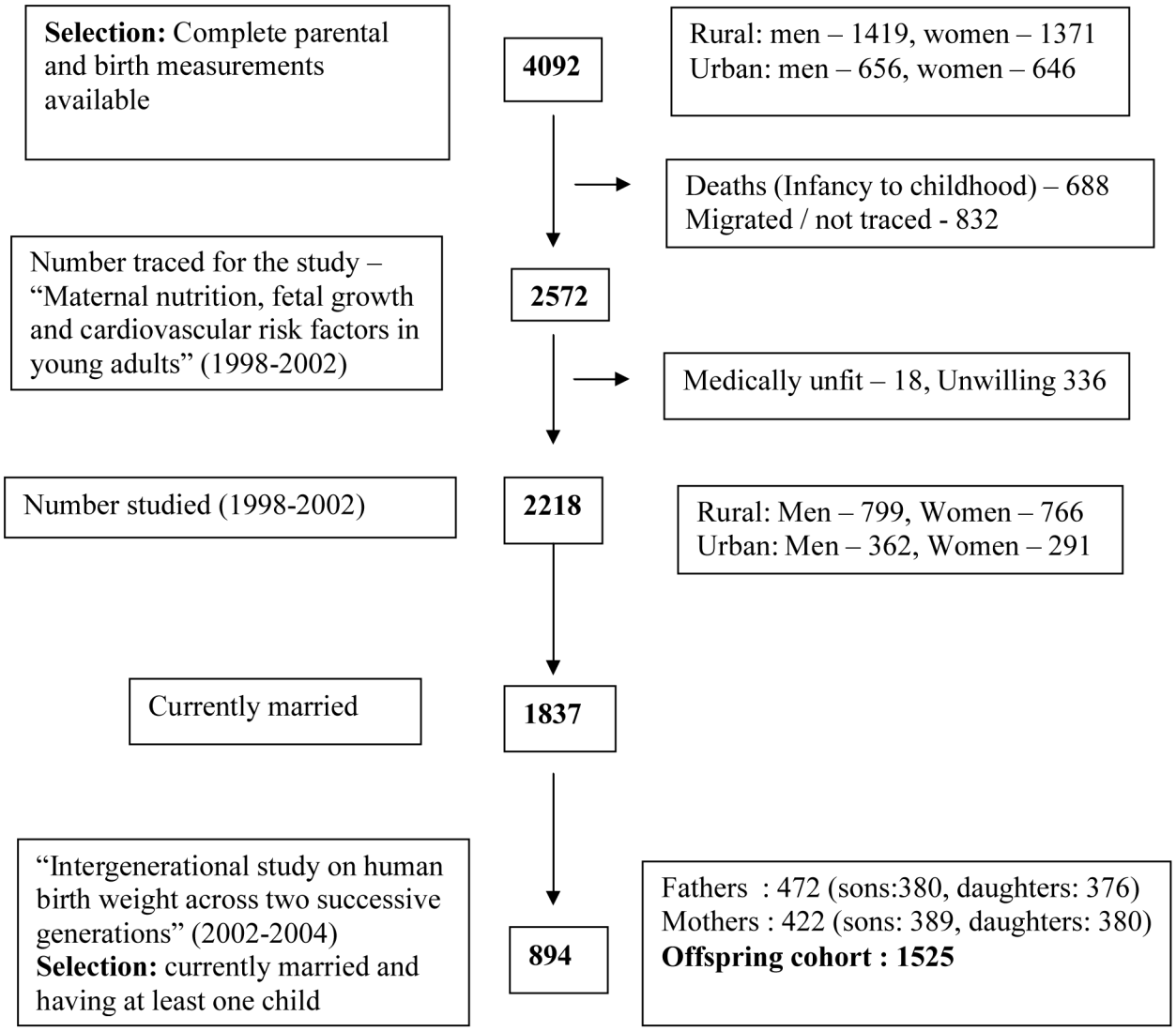

Figure 1. Flow chart showing the parental cohort derived from earlier studies

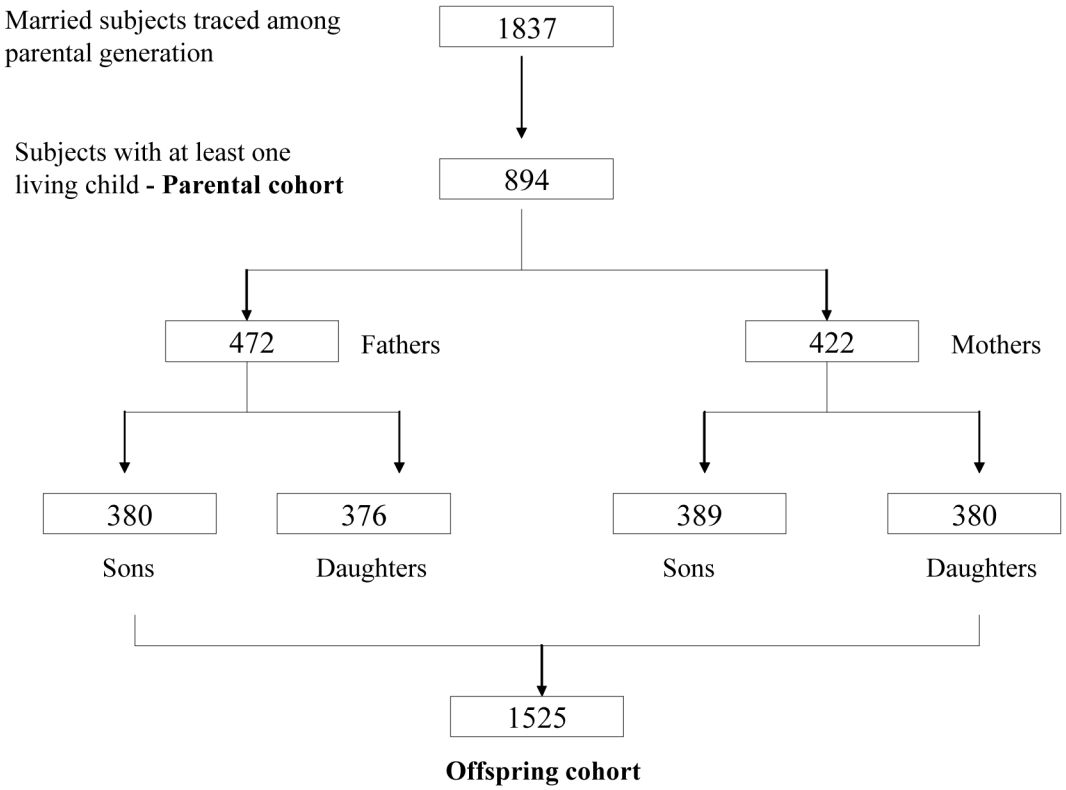

Fig.2. Flow chart showing recruitment of subjects
Multifetal pregnancy, stillbirth, early infant death, adoptions were excluded

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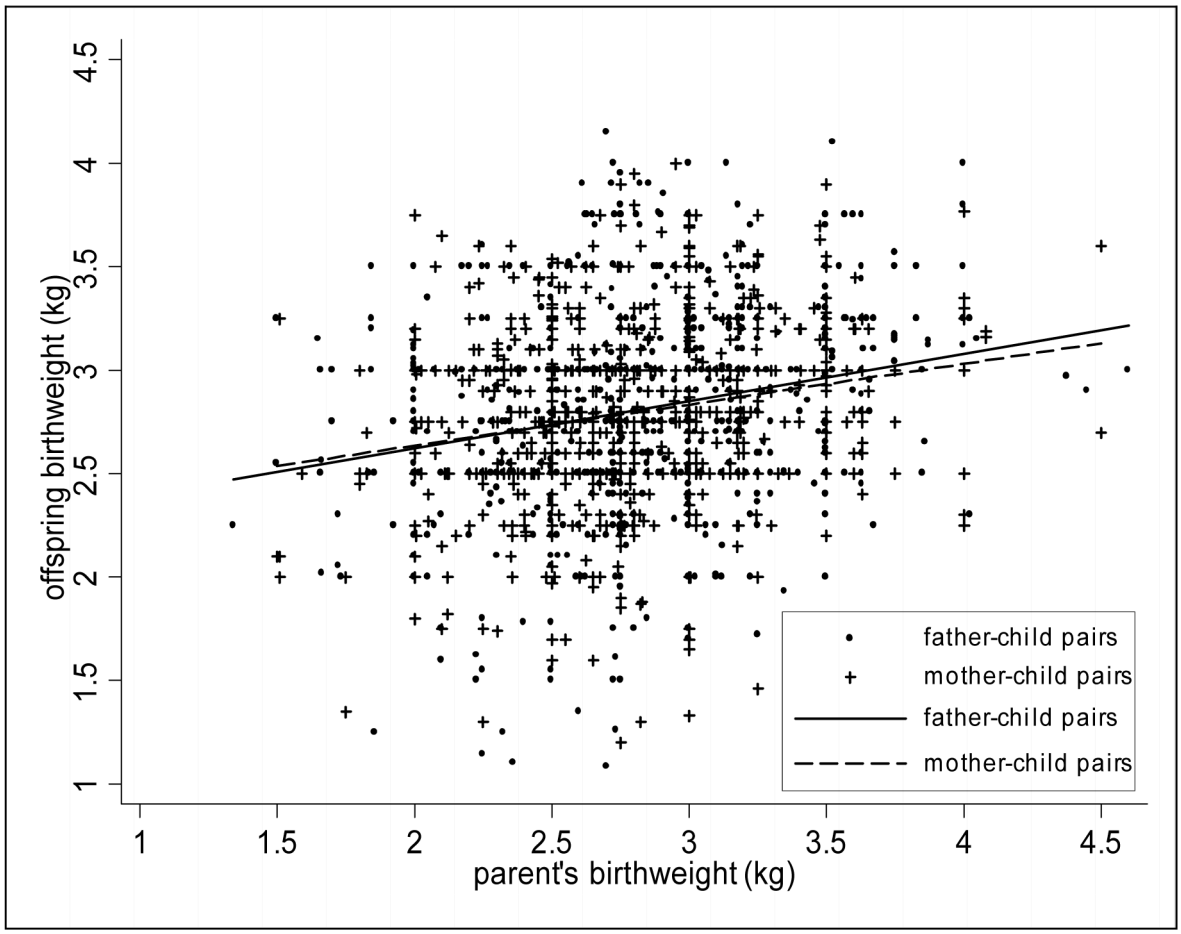

Father – child pairs ($r = 0.23$, $p < 0.001$) Mother – child pairs ($r = 0.20$, $p < 0.001$)

Fig. 3. Correlation between parental and offspring birthweight

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Table 1

Baseline characteristics of parents and offspring

	Mothers		Daughters		Sons	
	n	**mean(SD)**	**n**	**mean (SD)**	**n**	**mean (SD)**
Gestational age(wks)	---	---	230	39.1(2.24)	232	39.3(1.65)
Birth weight (g)	422	2790(444)	380	2716(438)	389	2865(438)
Adult height (cm)	422	153.9(5.47)				
Adult weight (kg)	422	50.3(10.49)				
BMI (kg/m^2)	422	21.2(4.26)				
Low birth weight [%]	89 [21.1%]		73 [19.2%]		58 [14.9%]	

	Fathers		Daughters		Sons	
	n	**mean(SD)**	**n**	**mean (SD)**	**n**	**mean (SD)**
Gestational age(wks)	---	---	238	39.4(2.17)	248	38.8(2.18)
Birth weight (g)	472	2846(489)	376	2771(479)	380	2857(498)
Adult height (cm)	472	166.5(6.41)				
Adult weight (kg)	472	58.5(11.91)				
BMI (kg/m^2)	472	21.0(3.64)				
Low birth weight [%]	88 [18.6%]		76 [20.2%]		56 [14.7%]	

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Table 2

Random effects logistic regression showing risk of producing a low birth weight offspring

Parental * Birth weight (g)	Effects of father (n = 472)			Effects of mother (n = 422)		
	Odds ratio	95%CI	p value	Odds ratio	95%CI	p value
< 2500	2.19	[1.0-4.8]	0.05	2.76	[1.2-6.4]	0.02
2500 - 3000	1.75	[0.9-3.3]	0.09	2.19	[1.1-4.6]	0.03

Adjusted for sex of the offspring, parity of the mother, BMI, adult height and SES score of parents

*Reference parental birth weight >3000 g

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Table 3

Random effects generalized least square regression (regression coefficient β) giving a measure of parental effects on offspring birth weight

Parental characteristics	Effects of father (n = 472)			Effects of mother (n = 422)		
	Offspring BW (g)	95%CI	p value	Offspring BW(g)	95%CI	p value
Birth weight (kg)	181.4	[100.7-262.0]	<0.001	139.6	[64.2-214.9]	<0.001
Adult height (cm)	9.0	[2.7-15.4]	<0.001	9.8	[3.5-16.0]	0.002
BMI (kg/m^2)	14.2	[3.5-24.9]	0.001	16.8	[8.9-24.5]	<0.001

Adjusted for sex of the offspring, parity of the mother and SES

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