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Predictors of ratio of placental weight to fetal weight in multiethnic community

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Abstract

***Objective*—To determine whether placental ratio is influenced by maternal ethnic origin, obesity, hypertension, and haematological indices of iron deficiency anaemia.**

***Design*—Observational study.**

***Setting*—District general hospital in Birmingham.**

***Subjects*—692 healthy nulliparous pregnant women, of whom 367 were European, 213 Asian, 99 Afro-Caribbean, and 13 of other or undocumented ethnic origin.**

***Main outcome measures*—Placental ratio and maternal body mass index, blood pressure, and haematological indices.**

***Results*—Though birth weight and placental weight were lower in Asian women than in other groups, mean placental ratio was similar in Asian (19·5% (SD 3·3%)), European (20·0% (4·0%)), and Afro-Caribbean women (20·4% (5·3%)). Gestational age at birth was the main predictor of placental ratio in the univariate analysis ($r = -0·34$, $P < 0·001$) and multivariate analysis. The only other significant predictor of placental ratio in multivariate analysis was maternal body mass index, which was positively associated with placental ratio ($r = 0·1$, $P = 0·01$). Mean (SD) placental ratio was not significantly higher in women who developed gestational hypertension (20·4% (4·5%)) and pre-eclampsia (23·3% (7·3%)) than in normal women (19·8% (3·8%)). No evidence of a relation between placental ratio and first antenatal visit haemoglobin concentration or mean cell volume was detected, and placental ratio was not associated with change in mean cell volume during pregnancy or with third trimester serum ferritin concentration.**

***Conclusions*—These data do not support the proposed association between poor maternal nutrition and increased placental ratio. The association between high placental ratio and adult hypertension may be confounded by genetic and environmental factors associated with maternal obesity (and possibly maternal hypertension).**

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Introduction

Barker and colleagues have reported that the risk of essential hypertension in adult life falls with increasing birth weight and rises with increasing placental weight and that the people at highest risk are those with a high placental weight relative to birth weight—that is, a high placental ratio.[1] The group has stressed the likely dominant influence of maternal nutrition, and in particular maternal iron deficiency anaemia, on intrauterine growth.[2] Using data from the Oxford record linkage system, they described a specific association between low maternal haemoglobin combined with a fall in mean cell volume during pregnancy and a raised placental ratio.[3]

The maternal factors that lead to discordance between birth and placental weight are, however, poorly understood. The importance of maternal nutrition as a determinant of placental ratio is disputed.[4] The data from the Oxford record linkage system showed a strong positive association between maternal body mass index and placental ratio, an association which has received relatively little attention.[3] Obesity and hypertension are linked in pregnancy as in the non-pregnant state.[5] Though pre-eclampsia is known to be associated with increased placental ratio,[6] the relation between more common manifestations of hypertension in pregnancy, such as gestational hypertension, and placental ratio is unclear.

We studied maternal factors that influence the ratio of placental weight to birth weight (placental ratio) in women recruited for a study of early markers for pre-eclampsia. The women were from a multiethnic inner city community with a relatively high proportion of patients of Asian (mainly first generation from northern India, Pakistan, and Bangladesh) and Afro-Caribbean ethnic origin. There is evidence of poor maternal nutrition in pregnant Asian women relative to European women.[7,8] If nutrition is an important determinant of placental ratio the placental ratio would be expected to be higher in this group.

The aim of this study was to identify maternal factors which influence placental ratio in this population, with particular reference to the role of maternal ethnic origin, obesity, and hypertension and haematological indices of maternal iron deficiency anaemia.

Subjects and methods

We studied a group of 692 healthy nulliparous pregnant women referred for antenatal care to Dudley Road Hospital, Birmingham, before the 31st week of gestation. The median gestational age at referral was 16 weeks (interquartile range 13-18 weeks), and 85% of the women were referred at or before the 20th completed week of pregnancy. These women were recruited for a prospective study of early markers for pre-eclampsia.[9,10] Women with diabetes and twin births were excluded. Of the 692 women, 367 were European, 213 Asian, 99 Afro-Caribbean, and 13 of other or undocumented ethnic origin. This represents the usual ethnic distribution of nulliparous women referred to the hospital. Iron supplements were prescribed at the booking antenatal visit for women with a haemoglobin concentration below 11·0 g/l.

Proteinuric pre-eclampsia and gestational hypertension were defined according to current internationally agreed criteria.[11] Data on birth weight and

placental weight (untrimmed) and gestational age at birth (estimated from last menstrual period and ultrasound scan at first visit) were obtained from labour ward records. Additional data from the antenatal record included maternal ethnic origin, age, height, blood pressure at first antenatal visit, smoking status, and haemoglobin concentration, packed cell volume, and mean cell volume at first visit. Complete data from the antenatal and labour ward records were available for 80% of pregnancies. Third trimester blood samples (additional to those obtained routinely) were obtained for measurement of full blood count and serum ferritin from 407 and 318 women respectively. The availability of these data depended on whether women were asked and agreed to provide third trimester samples for research purposes. The women from whom third trimester samples were obtained were representative of the total study population with regard to ethnic origin, age, gestational age at booking, smoking status, and birth weight. Blood counts were determined on a Technicon H1 auto-analyser (Bayer, Basingstoke) and ferritin was measured by a quantitative immunoradiometric determination (Becton Dickinson, Oxford).[12] Maternal body mass index was calculated as weight at the first visit divided by the square of height (kg/m^2).

Factors influencing placental ratio, birth weight, and placental weight were studied in multiple regression analysis. Gestational hypertension and pre-eclampsia were fitted as separate indicator variables in regression analysis. Stepwise regression with the maximum F statistic criteria was used to identify significant predictors (at the 5% level) of placental ratio, birth weight, and placental weight.[13] The distribution of serum ferritin was markedly skewed, and these data were log transformed before analysis.

TABLE I—*Comparison of baseline characteristics and outcome variables by ethnic origin. Values are means (SD) unless stated otherwise*

	European (n=367)	Asian (n=213)	Afro-Caribbean (n=99)	P value**
Age (years)	23·4 (4·5)	22·5 (3·9)	22·3 (4·8)	0·03
Height (m)	1·63 (0·06)	1·58 (0·06)	1·63 (0·06)	<0·001
Body mass index†	23·5 (3·6)	22·0 (3·7)	23·3 (3·8)	<0·001
No (%) who smoked	121 (33)	5 (2)	23 (23)	<0·001
Haemoglobin‡ (g/l)	126 (11)	122 (13)	119 (11)	<0·001
Packed cell volume‡ (%)	36·0 (2·7)	35·7 (3·3)	34·7 (3·0)	<0·001
Mean cell volume‡ (fl)	87·0 (4·7)	80·5 (8·1)	83·9 (6·4)	<0·001
Systolic blood pressure‡ (mm Hg)	109 (11·3)	105 (11·4)	108 (12·3)	<0·001
No (%) with gestational hypertension	38 (10)	13 (6)	4 (4)	0·03
No (%) with pre-eclampsia	5 (1)	3 (1)	3 (3)	0·3
Gestational age at birth§ (weeks)	39·5 (1·9)	39·5 (2·0)	39·2 (2·6)	0·3
No (%) of preterm births (%)	43 (12)	21 (10)*	9 (9)	0·3
Birth weight (g)	3241 (522)	3036 (478)	3103 (609)	<0·001
Placental weight (g)	643 (142)	588 (128)	618 (136)	<0·001
Placental ratio (%)	20·0 (4·0)	19·5 (3·3)	20·4 (5·3)	0·2

*Data were missing for some variables: age (78 women), full blood count (42), height (21), gestational age at birth (9), placental ratio (8), and smoking status (2).
**Analysis of variance or χ^2 test. †Excluding 126 women booking after 20 weeks of gestation. ‡Obtained at first antenatal visit. §Median (interquartile range) 40 (39 to 41) weeks in each ethnic group.

TABLE II—*Significant predictors of placental ratio, placental weight, and birth weight in multiple regression analysis*

	Regression coefficient (slope)*	95% confidence interval	P value
Placental ratio (%):			
Gestational age at birth (weeks)	−0·7	−0·84 to −0·56	<0·001
Body mass index	0·1	0·02 to 0·18	0·007
Placental weight (g):			
Gestational age at birth (weeks)	20·8	15·7 to 25·9	<0·001
Body mass index	6·0	3·0 to 9·0	<0·001
First visit packed cell volume (%)	−3·8	−7·4 to −0·24	0·03
Asian ethnic origin (1/0)†	−48·4	−71·2 to −25·6	<0·001
Birth weight (g):			
Gestational age at birth (weeks)	153·1	137·3 to 168·9	<0·001
Body mass index	20·2	11·1 to 29·3	<0·001
First visit packed cell volume (%)	−11·0	−21·9 to −0·11	0·05
Asian ethnic origin (1/0)†	−168·2	−246·2 to −90·2	<0·001
Height (m)	12·8	7·3 to 18·3	<0·001
Cigarette smoking (0/1)	−212·0	−294 to −129	<0·001
Gestational hypertension	−170·4	−290·4 to −50·4	0·005
Pre-eclampsia	−339·6	−588·7 to −90·5	0·007

*The regression coefficient represents the change in placental ratio, placental weight, and birth weight for each unit increase of the relevant predictor variable.
†Coded Asian = 1, European plus Afro-Caribbean = 0.
Each model included gestational age at birth, maternal age, body mass index, height, smoking status, systolic blood pressure, ethnic origin, pregnancy hypertension, and first visit haemoglobin, packed cell volume, and mean cell volume.

Results

Table I shows the baseline characteristics and pregnancy outcome variables, including placental ratio, for the three ethnic groups. There were significant differences between the groups in age, height, body mass index, smoking prevalence, haemoglobin concentration, packed cell volume, mean cell volume, systolic blood pressure, and incidence of gestational hypertension. Gestational age at birth was similar in the three groups. Placental ratio was also similar in the three ethnic groups, but birth weight and placental weight were lower in Asian than in European and Afro-Caribbean women (table I). Gestational age at delivery was inversely associated with placental ratio ($r = -0·34$, $P < 0·001$). A positive linear association between body mass index and placental ratio was observed ($r = 0·1$, $P = 0·01$). Mean (SD) placental ratio was not significantly higher in smokers (n = 146) than in non-smokers n = 536) (20·4% (3·9%) *v* 19·8% (3·9%); 95% confidence interval for difference −1·3% to 0·1%).

Lower gestational age at birth and higher maternal body mass index were the only significant independent predictors of placental ratio in the multiple regression analysis (table II). Gestational age at delivery and body mass index were also both positively associated with placental weight and birth weight. Additional predictors of placental weight and birth weight are shown in table II. These findings were unaltered by exclusion of women booking after 20 weeks of gestation.

Of the 692 nulliparous women, 55 (8%) developed gestational hypertension and 12 (1·7%) pre-eclampsia. Placental ratio tended to be higher in women who developed gestational hypertension (20·4% (SD 4·5%)) and pre-eclampsia (22·3% (7·3%)) compared with those who remained normotensive (19·8% (3·8%)), though this trend, which largely reflects gestational age at delivery, was not significant.

Of 638 women with first antenatal haemoglobin data whose ethnic origin was documented, 33 (16%) of the Asian women had haemoglobin concentrations below 110 g/l, regarded as a cut off point for anaemia in pregnancy, compared with 43 (10%) of the combined European and Afro-Caribbean group (95% confidence interval for difference 0·6% to 11%, $P = 0·03$). Placental ratio was not significantly associated with first antenatal visit haemoglobin concentration or mean cell volume ($r = -0·02$ and $r = -0·02$). Among 643 women with data available, placental ratio did not differ between the 79 women with a haemoglobin concentration below 110 g/l and the 564 women with a concentration of 110 g/l or above (19·8% (3·9%) *v* 19·8% (3·4%)). Mean cell volume increased by a mean (SD) of 3·3 (4·9) fl between the first antenatal visit and the third trimester (range −14 to 20 fl). Among 396 women with data available, no significant difference in placental ratio was observed between the 90 women whose mean cell volume fell between the first visit and third trimester measurements and the 306 whose mean cell volume was unchanged or increased (19·3% (3·0%) *v* 19·7% (3·6%); 95% confidence interval for difference −1·1% to 0·4%). Similarly, no significant associations were observed between placental weight or birth weight and change in mean cell volume between the first and second antenatal visit.

Third trimester serum ferritin concentrations were higher in women whose mean cell volume was unchanged or increased between the first and second

antenatal visit (geometric mean (range) 13·9 (12·3-15·8) μg/l) than in women whose mean cell volume fell (8·9 (6·6-12·1) μg/l; P=0·009). However, no significant association between placental ratio and serum ferritin was observed ($r=0·02$). The univariate analysis showed weak inverse associations between birth weight and serum ferritin ($r=-0·12$, P=0·04) and between placental weight and serum ferritin ($r=-0·12$, P=0·04). When ethnic origin and gestational age at birth were adjusted for these associations were no longer significant. Of the 318 women for whom ferritin data were available, 113 had concentrations below the normal range (10 μg/l). Placental ratio was not significantly higher in these women than in those with normal ferritin concentrations (20·0% (3·5%) *v* 19·4% (3·7); −1·4% to 0·3%).

Discussion

In this study the ratio of placental weight to fetal weight, which has been linked to hypertension in adult life, was increased in the offspring of obese women. Placental ratio was similar in European, Asian, and Afro-Caribbean women despite the evidence of poor nutrition among pregnant Asian women in Birmingham.[7,8] Placental ratio was not significantly associated with first visit haemoglobin concentration, mean cell volume, change in mean cell volume between the first antenatal visit and the third trimester, or third trimester serum ferritin concentration.

These findings do not support the hypothesis that a raised placental ratio is a marker for poor maternal nutrition. Edwards *et al* have argued that maternal nutrition (excepting extreme malnutrition) has little importance in fetal and placental growth, instead suggesting that raised placental ratios reflect increased fetal exposure to maternal glucocorticoids.[4,14] Their work, taken with our results, suggests that maternal obesity rather than poor nutrition may be an important determinant of placental ratio through effects on maternal glucocorticoid concentrations. The link between raised placental ratio at birth and the subsequent development of essential hypertension may therefore be confounded by maternal obesity.

ROLES OF OBESITY AND ANAEMIA

Maternal hypertension, which is closely linked with obesity in pregnancy, is an additional potential confounding factor in studies of intrauterine growth and adult hypertension. Children born after hypertensive pregnancies have higher blood pressure than those born after a normotensive pregnancy.[15] Pregnancy hypertension was a significant predictor of low birth weight in this study, and there was a non-significant trend towards a higher placental ratio among women with gestational hypertension and pre-eclampsia compared with normotensive women. In this population almost 10% of women met current international criteria for either gestational hypertension or pre-eclampsia. Though no good data exist on the incidence of the pregnancy hypertensive syndromes over time, there is some evidence that current rates are lower than in the early 1970s,[16] and the incidence of the hypertensive disorders of pregnancy may have been considerably higher earlier this century.

Clearly, this study had less power to examine the relation between haemoglobin, mean cell volume, change in mean cell volume, and placental ratio than that of Godfrey *et al*,[13] which included data from 8684 pregnant women, over 700 of whom had a haemoglobin concentration of ≤99 g/l or lower. However, our data do not support an important role for iron deficiency anaemia as a cause of discordance between placental and fetal growth. The findings are consistent with those from the study of Whincup *et al*, in which no consistent relation was observed between change in mean cell volume between first and third trimesters and placental ratio (or blood pressure at 9-11 years) in a study of over 600 children.[17]

The association of higher packed cell volume at first antenatal visit with both lower birth weight and lower placental weight probably reflects a relative failure of the mid-trimester physiological haemodilution of pregnancy in women with retardation of fetal and placental growth. The Oxford record linkage study found a similar inverse association between minimum maternal haemoglobin and both birth weight and placental weight.[3] It is clear therefore that the value of haemoglobin as a marker for iron deficiency is constrained by the effects of haemodilution in pregnancy. In addition, evidence of iron deficiency is not necessarily synonymous with poor maternal nutrition.

The reported association between fetal and placental growth and cardiovascular disease in later life has raised intriguing scientific questions which have major health policy implications. The importance of adequate nutrition in pregnancy is not in dispute. We suggest, however, that the hypothesis linking poor maternal nutrition in pregnancy with discordance between fetal and placental growth and adult hypertension and cardiovascular disease is not supported by these data.

Key messages

- A high ratio of placental weight to fetal weight (placental ratio) at birth has been linked with essential hypertension in later life
- Poor maternal nutrition has been suggested as a determinant of placental ratio, and an association with haematological indices of iron deficiency anaemia has been described
- In this study of European, Asian, and Afro-Caribbean pregnant women placental ratio was increased in the offspring of obese women
- Placental ratio was not significantly associated with first antenatal visit haemoglobin concentrations or mean cell volume, change in mean cell volume between the first antenatal visit and the third trimester, or third trimester serum ferritin concentration
- These findings do not support the hypothesis that raised placental ratio is a marker for poor maternal nutrition

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The NHS and Community Care Act 1990: is it a success for elderly people?

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The NHS and Community Care Act 1990 signified a controversial change in the government's health and social policy.[1] Phase 3 of the act was implemented on 1 April 1993, transferring the responsibility for funding of patients in the voluntary sector and in private residential and nursing homes from the Department of Social Security to local authority social services departments. We studied the effects of this reform on hospital practice.

Methods and results

From 1 April 1992 to 31 March 1994 we observed all patients aged >65 in the general medical, geriatric, and orthopaedic wards of our hospital who had been inpatients for >28 days. We observed these 766 patients weekly until discharge and noted the date when the patients became "medically stable"—that is, no longer benefiting from acute hospital care or rehabilitation—where they were discharged to, and reasons for delay of discharge. We used χ^2 test with Yates's correction to compare results before 1 April 1993 with those after 1 April.

We enrolled 362 patients into the study during 1 April 1992 to 31 March 1993 and 404 patients during 1 April 1993 to 31 March 1994. In this preliminary report we present an analysis of 100 randomly selected patients discharged before and 100 such patients discharged after 1 April 1993; 142 were aged 80-90, and 143 were women. The 100 patients before 1 April and those after 1 April accounted for 8875 (median 63) and 7131 (35) patient bed days respectively—a reduction of 19·7% after 1 April.

The number of patients discharged within 10 days of becoming medically stable increased after 1 April—from 30 to 52 ($P<0·0001$). The patients remained in hospital after being designated medically stable for 3958 bed days before 1 April compared with 2796 after, a reduction of 29·4%.

The number of patients discharged home increased from 44 before 1 April to 68 after ($P=0·001$); the number discharged to nursing homes decreased from 38 to 13 ($P<0·0001$). No change occurred in the number of patients discharged to other destinations. The reasons for final delay before discharge varied before and after 1 April (table).

Reasons for last delay before discharge among 100 patients before and 100 patients after 1 April 1993

Delay	Before 1 April 1993	After 1 April 1993
Patient benefiting from acute care	2	4
Rehabilitation	16	38
Awaiting the arrangement of care package	0	3
Awaiting placements	48	22
Lack of input by occupational therapist	1	1
Awaiting aids and adaptations	6	8
Waiting for flat to be cleaned	4	0
Lack of cooperation from relatives	0	2
Lack of agreement from patient	3	2
Patient undergoing further investigations	1	6
Awaiting the outcome of a home visit	5	1
Awaiting rehousing	1	2
Discharge arrangements in progress	10	1
Other	3	10
Total	100	100

Comment

The transfer of funding under the NHS and Community Care Act provoked anxiety that discharging patients would become more difficult.[1] Early reports seemed to confirm these suspicions.[2,3] At our hospital the procedure for discharging patients needing social workers changed substantially after 1 April. The findings presented here are relevant to the Borough of Hammersmith and Fulham and may not be applicable nationally as other boroughs and counties have interpreted the legislation in different ways.

We tried to reduce the possibility of bias. Most hospital staff were unaware that we were monitoring the effect of the community care legislation as our study began 12 months before the legislation was implemented. Many end points were precise and not open to interpretation—for example, discharge date. When the end points were less precise, such as when a patient became medically stable, the decision to discharge was taken by ward staff and not by the investigators. VA collected the data throughout the study.

The reduction in the length of hospital stay, both overall and after the classification of medically stable, is consistent with the findings that after the legislation patients were more likely to be in hospital for rehabilitation than awaiting placement and fewer were having difficulty with discharge arrangements. Some factors, such as delays in aids and adaptations, showed no change between the 12 months before legislation and the 12 months after.

This study shows that the changes implemented on 1 April 1993 as a result of the new act enabled patients in the Borough of Hammersmith and Fulham to be discharged from hospital earlier and that they were more likely to be discharged home than elsewhere.

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