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ORIGINAL COMMUNICATION

Association between size at birth, truncal fat and obesity in adult life and its contribution to blood pressure and coronary heart disease; study in a high birth weight population

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Objective: The aim of the study was to assess the relationship between size at birth and obesity as well as truncal fat, and its contribution to cardiovascular risk in a high birth weight population.
Design: Cohort-study with retrospectively collected data on size at birth.
Setting: Reykjavik, Iceland.
Subjects: A total of 1874 men and 1833 women born in Reykjavik during 1914–1935.
Main outcome measures: Size at birth. Adult weight, height and skinfold thickness measurements, systolic and diastolic blood pressure, fatal and nonfatal coronary heart disease (CHD).
Results: Birth weight was positively related to adult body mass index (BMI) in both genders (B=0.35±0.14 kg/m^2, adj. R^2=0.015, P=0.012 and B=0.34±0.17 kg/m^2, adj. R^2=0.055, P=0.043 in men and women, respectively). However, high birth weight was not a risk factor for adult obesity (BMI≥30 kg/m^2). In the highest birth weight quartile, the odds ratio (95% CI) for being above the 90th percentile of truncal fat was 0.7 (0.6–1.0, P=0.021) for men and 0.4 (0.3–0.8, P=0.002) for women, compared with the lowest birth weight quartile. Truncal fat and BMI were positively related to blood pressure in both genders ($P<0.05$), but not to CHD. The regression coefficient for the inverse association between birth weight and blood pressure hardly changed when adding truncal fat to the model.
Conclusion: In this high birth weight population, high birth weight was related to higher BMI in adulthood without being a risk factor for adult obesity. The inverse association between birth weight and truncal fat in adulthood suggests a role for foetal development in determining adult fat distribution. The inverse relationship of birth weight to blood pressure seems not to be mediated through the same pathway as to truncal fat.
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Keywords: birth weight; anthropometry; obesity; foetal growth retardation; nutrition; adult; infant

Introduction

Birth weight has been shown to be positively associated with body mass index (BMI) in adulthood (Curhan *et al*, 1996a, b; Eriksson *et al*, 2001). In Iceland, the mean birth weight is among the highest in the world (Meeuwisse & Olausson, 1998; Thorsdottir & Birgisdottir, 1998; Atladottir & Thorsdottir, 2000). Also, the prevalence of obesity, measured as BMI ≥30 kg/m^2, is higher than in many related nations (Thorgeirsdottir *et al*, 2001). It is important to assess whether the high birth weight of the Icelandic population could contribute to the high prevalence of obesity. Recently,

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however, it has been suggested that greater fat-free mass rather than increased fat mass explains the positive relationship between birth weight and BMI in adulthood (Singhal *et al*, 2003).

Although birth weight has been shown as positively related to adult BMI, studies have shown birth weight to be inversely related to truncal and abdominal fatness in both adolescence and adult life (Law *et al*, 1992; Valdez *et al*, 1994; Barker *et al*, 1997; Byberg *et al*, 2000). It has therefore been postulated that the tendency to store fat abdominally may be a persistent response to adverse conditions and growth in foetal life (Law *et al*, 1992). Small size at birth has previously been related to both hypertension and coronary heart disease (CHD) in the Icelandic population (Gunnarsdottir *et al*, 2002a, b). It is of high importance to assess thoroughly the relationship between size at birth and adult BMI as well as adult truncal fat in the high birth weight population in Iceland. The ability to assess whether the association between size at birth and both blood pressure and CHD could be mediated through increased truncal fat or BMI will add to the understanding of adult disease programming.

The aim of the present study was to assess the relationship between size at birth and obesity as well as truncal fat and the contribution of the relationship to the risk of developing CHD and hypertension in a high birth weight population.

Materials and methods

Study population

The study population was a subcohort from randomly selected participants of the ongoing prospective Reykjavik Study, which started in 1967 (Sigurdsson *et al*, 1993). The present study included those born in the greater Reykjavik area during 1914–1935, who were still living in Reykjavik in 1967 (N=6120). Information on weight and length at birth was found in original midwives' birth records obtained from the National Archives of Iceland and matched for 4828 individuals. The criteria for participation in the present study were a complete midwife's birth record, single birth, measured weight, height and skinfold thickness, and being between the ages 33 and 65 y when examined at the Icelandic Heart Association Heart Preventive Clinic. The mean age at examination was 50 ± 8 y (mean$\pm$s.d.). Twins were excluded (56 individuals). A total of 3708 men and women, or 60.5% of the original study population had complete data sets available and were included in the current analysis.

Approval of the study protocol was granted by the Icelandic National Bioethics Committee and the Data Protection Commission, which provided the birth-size information. Written informed consent was obtained from the subcohort of participants in the Reykjavik Study.

Collection of birth data

Midwives' birth records included data on gender, birth weight to the nearest 0.1 kg, and length in centimetres from crown to heel as well as information on singleton *vs* multiple births. From the information in midwifes' birth records, the ponderal index was calculated (kg/m^3) (Gunnarsdottir *et al*, 2002a). Preterm births were not identified clearly in the midwives' records, but at the time of the births, the method used to define preterm newborns was a birth length less than 48 cm, which occurred in only 2.5% of cases. All calculations were tested with and without these subjects, and their inclusion or exclusion made no difference in the results.

Collection of adult data

Information about the health and anthropometric measurements of the subjects was collected at the Heart Association Heart Preventive Clinic in the prospective longitudinal Reykjavik Study.

The subjects' weight was measured to the nearest 100 g, without shoes, in light undergarments. Height was measured to the nearest 0.5 cm. Subcutaneous skinfolds were measured with calibrated callipers to the nearest 1.0 mm (Lange, Cambridge Scientific Industries, Inc., Cambridge, MD, USA), having a pressure of 10 g per square mm. The triceps skinfold was measured on the back of the pendant right arm at a level midway between the tip of the acrominon and the tip of the olecranon. The subscapular skinfold was measured with the participant in the erect position. The skinfold was pinched up in a transverse axis in the dedio-axillar-line. The ratio of subscapular to triceps skinfold was used as an index of truncal fatness (Valdez *et al*, 1994; Byberg *et al*, 2000).

Systolic and diastolic blood pressures were measured to the nearest 2 mmHg, with a mercury sphygmomanometer of the type 'Erkameter' wall-model (Erka, Germany), using a $12\,\text{cm}\times23\,\text{cm}$ cuff, after 5 min rest. Hypertension was defined as pharmacological treatment for high blood pressure and/or systolic blood pressure >140 mmHg and/or diastolic blood pressure >90 mmHg.

Since 1981, data regarding the incidence of myocardial infarction have been collected as part of the World Health Organization MONItoring trends and determinants in CArdiovascular disease (MONICA) Project (WHO MONICA, 1988). Hospital records for those having a myocardial infarction before 1981 were reviewed and evaluated according to the criteria used in the MONICA study. For a nonfatal event to be definite, there had to be either a progression of Minnesota codes on serial ECGs or cardiac enzyme levels twice the limit of normal, either with typical symptoms and an ECG that was not normal or with an ECG progression labelled 'probable' and lesser symptoms (Tunstall-Pedoe *et al*, 1994). Information on death due to CHD was obtained from death certificates from 1967 to 1999 on file in the Statistics Iceland by using the following codes from the International Classification of Diseases: 1967–1970, ICD (seventh revision) code 420; 1971–1980, ICD (eighth revision) codes 410–413;

1981–1999, ICD (ninth revision) codes 410–414. All death certificates were reviewed and coded by an official government pathologist.

Using a questionnaire, information was collected on smoking habits, educational status and physical activity.

Statistical analysis

Mean and standard deviation (s.d.) were used to describe the data. Linear regression was used to assess the relationship between birth measurements and adult anthropometric measurements. Logistic regression was used for calculating the odds ratio for hypertension, CHD, obesity (BMI $\geq 30\,kg/m^2$) and being above the 90% percentile in truncal fatness, and 95% confidence intervals for the birth size categories (Altman, 1991). For simplicity, means and s.d. on untransformed data are shown in tables, but as some variables were naturally skewed (BMI and skinfold thickness), parametric tests and *P*-values are based on the transformed data.

Analysis was performed adjusting for gender, but men and women were also analysed separately. Birth weight, birth length and figures for the ponderal index were categorised into quartiles for comparison. The quartiles were as follows: $\leq 3.45\,kg$, >3.45–$3.75\,kg$, >3.75–$4.0\,kg$ and $>4.0\,kg$ for birth weight, $\leq 50.5\,cm$, >50.5–$52.5\,cm$, >52.5–$54.0\,cm$ and $>54.0\,cm$ for birth length, and $\leq 24.0\,kg/m^3$, >24.0–$26.2\,kg/m^3$, >26.2–$28.3\,kg/m^3$ and $>28.3\,kg/m^3$ for ponderal index. When the genders were analysed separately, the birth weight categories were different due to higher birth weight among men (see Table 2). Trend tests for birth weight, birth length and ponderal index were calculated on the basis of the continuous variable. Multiple logistic regressions were used to assess the combined effect of size at birth and potential confounding variables, such as educational status, smoking habits and physical activity. Correlations of the year of birth with birth weight ($r=-0.044$; $P<0.001$) and birth length ($r=0.130$; $P<0.001$) were adjusted for. Adjustments were also made for age at examination. The *t*-test was used to compare birth size and adult anthropometric data between men and women. Significance was defined as $P<0.05$. The statistical software used was SPSS for Windows, version 11 (SPSS Inc., Chicago, USA).

Results

Anthropometric data for subjects at birth and in adulthood are shown in Table 1. Men were heavier and taller than women, both at birth and in adulthood, and had a higher adult BMI ($P<0.001$), but the ponderal index at birth was not different.

Table 2 shows the BMI, skinfold thickness measurements and truncal fat, measured as the subscapular-to-triceps ratio, by quartiles of birth weight. Birth weight was positively related to adult BMI, $B=0.36\pm0.11$, $P<0.001$, adjusted for gender, age and year of birth. Analysing the genders separately showed a similar pattern in both genders with a $0.35\pm0.14\,kg/m^2$ (adj. $R^2=0.015$, $P=0.007$) and $0.34\pm0.17\,kg/m^2$ (adj. $R^2=0.055$, $P=0.023$) increase in adult BMI in men and women, respectively, for each 1-kg increase in birth weight. Birth weight was inversely related to truncal fat ($B=-0.05\pm0.02$, $P=0.002$, adjusted for gender, age and year of birth). Analysing the genders separately showed that birth weight was related to truncal fat only in women (Table 2), but not in men. In the regression model used, birth weight was found to explain 5.5% ($P=0.035$) of the variance in truncal fat among women. Adjustments for current BMI strengthen the relationship. However, in men the relationship between birth weight and truncal fat was not significant (Table 2). When adjusting for adult BMI, an inverse relationship was seen between birth weight and adult subscapular skinfold thickness in women ($B=-0.701\pm0.366$, $P<0.001$), but the relationship was of borderline significance in men ($P=0.075$).

Table 1 Characteristics of the subjects[a]

	Men n=*1874*	*Women* n=*1833*
Birth weight (kg)	3.8 ± 0.6	3.7 ± 0.5[b]
Birth length (cm)	52.6 ± 2.4	52.1 ± 2.3[b]
Ponderal index (kg/m^3)	26.2 ± 3.4	26.1 ± 3.3
Age at examination (y)	49 ± 7	50 ± 7
Body weight (kg)	82.2 ± 12.5	67.8 ± 11.4[b]
Height (cm)	177.6 ± 6.5	164.7 ± 5.7[b]
BMI (kg/m^2)	26.0 ± 3.5	25.0 ± 4.0[b]

[a]Mean $\pm$ s.d.
[b]Significantly different from men, $P<0.001$.

Table 3 shows the mean BMI, skinfold thickness and subscapular-to-triceps ratio of men and women by quartiles of ponderal index. Ponderal index at birth was positively related with adult BMI in both men and women (adj. $R^2=0.016$, $P=0.010$ and adj. $R^2=0.056$, $P=0.013$ for men and women, respectively). Each unit in ponderal index at birth (kg/m^3) increased BMI in adulthood by about $0.07\pm0.01\,kg/m^2$ ($P<0.001$) for the total cohort, adjusted for gender. Ponderal index at birth was inversely related to truncal fat ($B=-0.01\pm0.004$, $P=0.019$, adjusted for gender, age and year of birth). When analysing the genders separately the relationship was only seen in men but not in women. A positive relationship between ponderal index and triceps skinfold thickness was seen in men ($B=0.09\pm0.04$, $P=0.002$). Length at birth was not related to BMI or skinfold thickness in adulthood.

The prevalence of obesity (BMI $\geq 30\,kg/m^2$) was 11.6 and 10.5% in men and women, respectively. The odds ratio for obesity was not different between the birth weight, birth length and ponderal index categories for either men or women.

Using logistic regression, the odds ratios for truncal fat above the 90th percentile by quartiles of birth size were assessed. Figures 1 and 2 show that for both birth weight and ponderal index, those in the highest quartile were

Table 2 BMI, skinfold measurements and truncal fat of adult men (n=1874) and women (n=1833) by quartiles of birth weight[a]

Quartiles of birth weight	n	*Adult BMI (kg/m^2)*	*Triceps skinfold (mm)*	*Subscapular skinfold (mm)*	*Adult truncal fat*
Men					
<3500 g	406	25.8±3.6	13.0±7.0	18.6±9.0	1.64±1.05
−3750 g	535	26.0±3.6	12.5±6.0	18.3±9.0	1.66±1.20
−4250 g	451	26. 0±3.5	12.4±6.1	17.8±9.1	1.55±0.93
>4250 g	482	26.3±3.4	13.4±6.8	18.5±9.1	1.55±1.0
P for trend[b]		0.007	0.148	0.713	0.218
P for trend[c]			0.800	0.075	0.084
Women					
≤3250 g	473	24.7±4.1	21.0±8.5	18.4±9.6	0.91±0.42
−3750 g	656	24.9±4.0	20.8±8.1	17.3±8.2	0.85±0.36
−4000 g	375	24.8±4.0	20.9±8.3	18.0±10.2	0.88±0.50
>4000 g	329	25.5±3.8	22.3±8.1	17.7±9.2	0.82±0.56
P for trend[b]		0.023	0.158	0.125	0.001
P for trend[c]			0.609	<0.001	<0.001

[a]Mean±s.d.
[b]Adjusted for age and year of birth.
[c]Adjusted for age, year of birth and BMI.

Table 3 BMI, skinfold measurements and truncal fat of adult men (n=1874) and women by quartiles of ponderal index (PI)[a]

Quartiles of PI	n	*Adult BMI (kg/m^2)*	*Triceps skinfold (mm)*	*Subscapular skinfold (mm)*	*Adult truncal fat*
Men					
≤24 kg/m^3	453	25.7±3.7	12.6±6.5	18.2±9.2	1.65±1.20
−26 kg/m^3	527	25.9±3.4	12.6±7.2	17.8±8.9	1.62±1.13
−28.3 kg/m^3	439	26.3±3.7	13.0±5.8	18.6±9.2	1.56±0.82
>28.3 kg/m^3	455	26.2±3.4	13.2±6.3	18.7±9.0	1.57±0.98
P for trend[b]		0.010	0.002	0.223	0.031
P for trend[c]			0.031	0.541	0.008
Women					
≤24 kg/m^3	468	24.6±4.0	20.5±8.5	17.5±9.8	0.87±0.42
−26 kg/m^3	476	24.8±4.1	21.1±8.3	17.8±10.3	0.86±0.45
−28.3 kg/m^3	481	24.9±4.02	21.2±8.1	17.8±9.3	0.87±0.52
>28.3 kg/m^3	408	25.2±3.9	21.7±8.4	18.1±9.0	0.85±0.35
P for trend[b]		0.013	0.157	0.359	0.772
P for trend[c]			0.793	0.420	0.330

[a]Mean±s.d.
[b]Adjusted for age and year of birth.
[c]Adjusted for age, year of birth and BMI.

significantly less likely to have truncal fat above the 90th percentile compared with the lowest birth weight quartile. Confining the analysis to value above the 75th percentile gave similar results for both the genders. Adjusting for educational status, smoking and physical activity did not affect the results of the analysis.

In this cohort, a positive linear relationship was found between truncal fatness and both systolic and diastolic blood pressure and risk of hypertension in both the genders (P<0.005). Truncal fatness was not found to be related to CHD in either men or women. The positive association between BMI and hypertension as well as the lack of relationship between BMI and CHD in this cohort has previously been described (Gunnarsdottir *et al*, 2002a, b). Previously, birth weight was found to be inversely related to blood pressure in this population (Gunnarsdottir *et al*, 2002a). To assess whether the inverse association between birth weight and blood pressure was mediated through BMI or truncal fat, several regression models were analysed (Table 4). As can be seen in Table 4, controlling for truncal fat hardly changes the birth weight coefficients.

Discussion

In the present study, high birth weight was positively related to BMI in adulthood, but high birth weight infants were not at increased risk of adult obesity. The increase in BMI with increasing birth weight in our study was around 1 kg/m^2

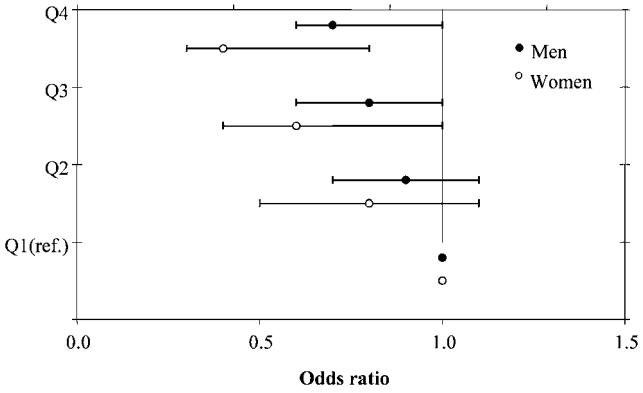

Figure 1 Odds ratios and 95% CI for adult truncal fat above the 90th percentile in men and women by quartiles (Q) of birth weight, adjusted for age and year of birth (Q1 is lowest and Q4 is highest). *P* for trend was 0.115 and 0.009 for men and women, respectively. Adjusting for adult BMI resulted in significant trend among men (*P* for trend=0.044), and (*P* for trend=0.029) among women.

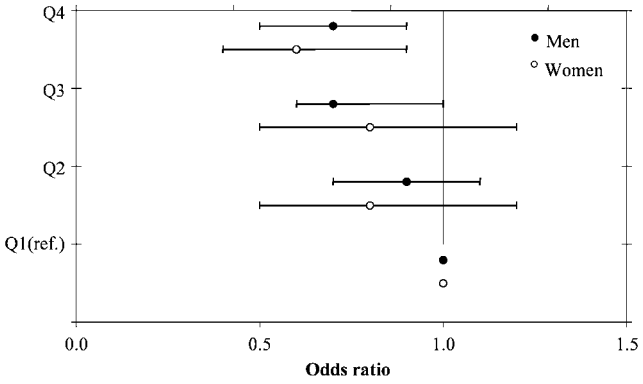

Figure 2 Odds ratios and 95% CI for adult truncal fat above the 90th percentile in men and women by quartiles (Q) of ponderal index at birth, adjusted for age and year of birth (Q1 is lowest and Q4 is highest). *P* for trend was 0.002 and 0.025 for men and women, respectively, and >0.001 and 0.037 when adjusting for adult BMI.

from the lowest to the highest quartile, which is similar to that found in other studies (Curhan *et al*, 1996a, b). An inverse relationship between birth weight and subscapular skinfold thickness was found in women, but there was a slight positive relationship between ponderal index and triceps skinfold thickness in men. Certain associations suggest a 'U'-type of curve, in particular, subscapular skinfolds and birth weight and ponderal index in men. This might indicate an association at both distribution extremes. High birth weight was inversely related to truncal fat in adulthood.

Previous work on the same cohort has shown that big size at birth is protective against high blood pressure, coronary artery disease as well as dysglycaemia later in life (Gunnarsdottir *et al*, 2002a, b; Birgisdottir *et al*, 2003). Iceland has lower event and mortality rates from CHD (WHO MONICA, 1994) and lower systolic blood pressure (Wolf *et al*, 1997) than many related populations. It has been suggested that the high birth weight of Icelandic infants, which is among the highest in the world (Meeuwisse & Olausson 1998; Thorsdottir & Birgisdottir 1998; Atladottir & Thorsdottir 2000), might contribute to protection against these diseases (Birgisdottir *et al*, 2003). Now we have shown that the high birth weight is positively related to higher adult BMI but not to obesity, defined as BMI $\geq 30\,kg/m^2$.

The body composition of Icelandic newborns has never been investigated. Foetal programming of lean body mass has been suggested in recent studies (Weyer *et al*, 2000; Eriksson *et al*, 2002; Singhal *et al*, 2003). It would have been desirable to include a measure of body composition at birth, such as arm circumference and skinfolds (Koletzko *et al*, 2002). There is a critical period for muscle growth around 30 weeks *in utero*, and fat-free mass may be determined during critical periods of muscle growth *in utero* as well as during childhood (Eriksson *et al*, 2002). The mean BMI of women of childbearing age was $24.4\,kg/m^2$ in Iceland at the time of the study (Statistics Iceland, 1997). This suggests that our subjects were not born during any crisis or period of possible undernutrition. It is possible that Icelandic infants have greater lean mass at birth than infants in other countries, and it might be assumed that the higher adult BMI of high birth weight infants in the present study is mostly due to higher fat-free mass, but not higher fat mass than in individuals of low birth weight. In a study by Erikson *et al* (2002), a positive correlation was found between birth weight and fat-free mass at any adult BMI, in both the genders. Another study by Weyer *et al* (2000) showed that high birth weight was associated with increased lean body mass, but not with increased adiposity later in life. A more recent study supports the hypothesis that foetal growth, measured by birth weight, programmes lean mass later in life, but in that study an increase in birth weight of 1s.d. was associated with a 2–3% increase in fat-free mass in adolescents but not with an increase in fat mass (Singhal *et al*, 2003). In the present study, an inverse relationship between subcutaneous adiposity (subscapular skinfold thickness) and birth weight was seen in women. Low birth weight has also been related to more raised leptin concentrations in adult life than would be expected from their BMI, which may be explained by a relatively greater fat mass in people who are small at birth (Phillips *et al*, 1999). BMI has frequently been shown to be associated with CHD in other populations (Kannel, 2002; Pi-Sunyer, 2002). The possibility cannot be excluded that the greater fat-free mass of Icelandic people with high BMI could explain the lack of association between BMI and CHD in the population studied. It is also possible that Icelanders benefit from muscularity, having lower systolic blood pressure and lower event and mortality rates from CHD than in many other nations of Caucasian origin.

An inverse linear relationship between birth weight and truncal fat later in life was seen in women, and the protective effect of high birth weight against very high truncal fat was

Table 4 Regression coefficients ($B \pm$s.e) for the relationship between birth weight and blood pressure in men (n=1874) and women (n=1833)[a]

	Systola	P	*adj.*R^2	*Diastola*	P	*adj.*R^2
Men						
Unadjusted[a]	-1.3 ± 0.8	0.118	0.07	-0.9 ± 0.5	0.058	0.06
Adjusted for adult BMI	-1.9 ± 0.8	0.017	0.16	-1.3 ± 0.4	0.003	0.19
Adjusted for TF[b]	-1.2 ± 0.8	0.141	0.07	-0.8 ± 0.5	0.072	0.07
Adjusted for TF[b] and BMI	-1.8 ± 0.8	0.021	0.16	-1.3 ± 0.4	0.004	0.19
Women						
Unadjusted[a]	-1.8 ± 0.8	0.027	0.16	-0.3 ± 0.5	0.535	0.11
Adjusted for adult BMI	-2.1 ± 0.8	0.007	0.20	-0.5 ± 0.5	0.295	0.15
Adjusted for TF[b]	-1.7 ± 0.8	0.040	0.16	-0.2 ± 0.5	0.669	0.11
Adjusted for TF[b] and BMI	-2.1 ± 0.8	0.009	0.20	-0.4 ± 0.5	0.358	0.15

[a]Adjusments for year of birth and age at examination are made in all regression models.
[b]TF=truncal fat (subscapular/triceps skinfold).

seen in both the genders. Having high ponderal index at birth was also beneficial for men. The findings of the present study are in accordance with some earlier studies (Barker *et al*, 1997; Byberg *et al*, 2000). Abdominal fat can be estimated as measurements of waist : hip ratio, while the subscapular-to-triceps ratio has been used to estimate truncal fat (Haffner *et al*, 1987; Barker *et al*, 1997). The measurements are related to each other and both have been related to metabolic factors (Haffner *et al*, 1987). However, the two measures are different concepts, and the two measurements possibly represent two different hormonal and metabolic situations (Haffner *et al*, 1987). In the present study, truncal fat was related to hypertension in both men and women, but not to CHD. Measurements of waist and hip circumference were not available, but the waist-to-hip ratio might have been related to CHD in the population studied. Results of the present study suggest that the previously shown relationship between birth weight and blood pressure (Gunnarsdottir *et al*, 2002a) is not mediated through truncal fat. If the birth weight effect on blood pressure arose through truncal fat, then it would be expected that the birth weight regression coefficient would approach zero. Adding truncal fat to the regression model hardly changed the birth weight coefficient.

In this study, we have added truncal fat to a cluster of variables previously investigated, including hypertension and glucose intolerance associated to size at birth in the Icelandic population. In previous work on the same cohort, birth length was associated with CHD in men, but birth weight or ponderal index at birth was not (Gunnarsdottir *et al*, 2002a, b). Weight at birth is related to hypertension, glucose intolerance and truncal fat in the population studied. The reason why different size measurements at birth are related to different end points in adult life might be difference in the mechanism underlying the 'programming' of the diseases. Vascular endothelial dysfunction is a key event early in atherosclerosis and is important in the development of cardiovascular diseases. A recent study has shown a relationship between low birth weight and endothelial dysfunction in young adults that was most marked in individuals with lower risk factor profiles. This suggests that the association does not operate via increased risk factors, such as cholesterol and blood pressure, and that early growth may directly influence the blood vessel wall (Leeson *et al*, 2001). Further studies are needed to understand fully the different pathways in programming of adult diseases.

The gender difference of the relationship between size at birth and later fatness and diseases seen in the present study, and in the previous studies on the same cohort might be caused by the different growth patterns of girls and boys *in utero* (Godfrey & Barker, 2000) and by endocrinological differences between men and women later in life (Forsén *et al*, 2000). Difference in body composition between genders has been seen in early infancy, where female infants had less lean mass and more fat mass from birth and throughout infancy (Koo *et al*, 2000). A new study of men and women from the Reykjavik Study shows that the major risk factors of CHD do not all confer an equal risk of myocardial infarction in men and women (Jonsdottir *et al*, 2002). All these studies indicate the importance of gender-specificity in cardiovascular research. However, it cannot be excluded that the gender difference seen in the present study is due to power effects.

In conclusion, in this high birth weight population, high birth weight and ponderal index were related to higher BMI in adulthood without being risk factors for adult obesity (BMI $\geq 30\,kg/m^2$). High birth weight is protective against truncal fat in adulthood, which suggests a role for foetal development in determining adult fat distribution. The relationship of birth weight to blood pressure seems not to be mediated through the same pathway as truncal fat.

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