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Early growth and abdominal fatness in adult life

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Abstract

***Study objective*—The aim was to determine whether abdominal fatness in adult men is associated with retarded growth in fetal life and infancy.**

***Design*—This was a follow up study of (1) men born during 1920–30 whose birthweights and weights at one year were recorded at the time by health visitors; and (2) men born during 1935–43 whose size at birth was measured in detail. The main outcome measure was the ratio of waist circumference to hip girth.**

***Setting*—Hertfordshire and Preston, England.**

***Subjects*—Subjects were 845 men born in east Hertfordshire who still live there; and 239 men born in Preston who still live in or close to the city.**

***Main results*—After allowing for body mass index, mean waist to hip ratio fell with increasing birthweight and rose as the ratio of placental weight to birthweight increased. These trends were independent of duration of gestation and therefore reflected retarded fetal growth. Waist to hip ratio also fell with increasing weight at one year. All these trends were independent of adult height, alcohol consumption, smoking, social class, and age.**

***Conclusions*—The tendency to store fat abdominally, which is known to increase the risk of cardiovascular disease and diabetes independently of obesity, may be a persisting response to adverse conditions and growth failure in fetal life and infancy.**

People who are obese, being heavy in relation to their height, have an increased incidence of cardiovascular disease and diabetes.[1] However, high weight in relation to height, quantified by a high body mass index (weight divided by height squared), gives no indication of the relative distribution of fat on different parts of the body.[2] Abdominal fatness, as measured by the ratio of waist circumference to hip girth,[2] increases the risk of cardiovascular disease and diabetes independently of body mass index.[2–7] It is independently associated with high blood pressure,[2–4 6 7] serum cholesterol and triglycerides,[2–4 7] plasma glucose,[4 7] and fibrinogen.[2]

Excess intra-abdominal and extra-abdominal fat could have a direct pathogenic effect or it could simply be a marker of other processes. Mechanisms to support both possibilities have been suggested. Lipid mobilisation from the omental and mesenteric fat depots could lead to high concentrations of free fatty acids in the portal blood. This in turn could stimulate synthesis of very low density lipoprotein (VLDL) cholesterol and low density lipoprotein (LDL) cholesterol, increase glucose production and decrease insulin clearance.[8] Alternatively, adrenal overactivity may lead to abdominal fatness and, separately, to hypertension and impaired glucose tolerance, as occurs in Cushing's disease.[2 9] These hypotheses are not mutually exclusive and as yet there is little evidence bearing on either.

Cardiovascular disease and diabetes have recently been shown to be associated with retarded growth during fetal life and infancy.[10 11] These associations are thought to reflect the long term consequences of growth restraint of the developing vasculature, pancreas, liver, and other tissues. They suggest an explanation for the association between abdominal fatness and disease. A tendency to store fat centrally could be a persisting response to early growth restraint and a marker of it. We have examined the relation between the waist to hip ratio and fetal growth in two samples of adult men.

Methods

PRESTON

In the city of Preston, England, a standardised record form was kept for each woman admitted to the labour ward at Sharoe Green Hospital during 1935–43. The record included the date of the mother's last menstrual period, used to calculate duration of gestation, and the baby's birthweight and placental weight. Weights were recorded in pounds (one pound = 0·45 kg) and had often been rounded: we therefore preserved the original units. Of the singleton boys born during 1935–43, 252 still live in Lancashire; 239 agreed to be interviewed at home and were visited by one of four field workers. The field workers had not seen the recorded birth data. Height was measured with a portable stadiometer and weight with a portable Seca scale. The waist circumference and hip girth were measured. The man was asked about his social history, and about his smoking and drinking habits. Alcohol consumption was converted to the total number of units each week (1 unit = 10 ml ethanol). Current social class was derived from the man's occupation.[12] Before starting the study the procedures for the measurements were standardised and the field workers trained.

HERTFORDSHIRE

In the county of Hertfordshire, England, from 1911 onwards each birth was notified by the

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attending midwife. A health visitor saw the child at home periodically through infancy. She recorded birthweight and weight at one year. Weights were measured in pounds and were often rounded to the nearest half pound or pound. We therefore used the original units. We have traced singleton boys born in east Hertfordshire during 1920–1930 who had both birthweight and weight at one year recorded. Of these, 1157 still live in east Hertfordshire, and 845 agreed to be interviewed at home and were visited by one of four field workers. The field workers had not seen the infant data recorded for the men. Height, weight, waist circumference and hip girth, social history, smoking and alcohol consumption were recorded in the same way as in Preston.

Statistical analysis was by multiple linear regression. For clarity of presentation waist to hip ratio has been expressed as a percentage.

Results

The characteristics of the men by place are given in table I. Men in Hertfordshire weighed more at birth, were older, drank less alcohol, and smoked less. Placental weight was not available in Hertfordshire and weight at one year was not available in Preston.

In both places waist to hip ratio was positively related to weight and body mass index but not to height. In Preston the regression coefficients were 0·22 (SE 0·026) for weight in kg and 0·70 (0·083) for body mass index in kg/m^2. In Hertfordshire they were 0·22 (0·012) for weight and 0·82 (0·040) for body mass index. In both places waist to hip ratio was positively related to alcohol consumption, but this did not reach statistical significance. Waist to hip ratio was positively related to smoking in Preston and negatively related to smoking in Hertfordshire, but neither of these reached statistical significance.

Table I Characteristics of the men, by place of study

	Preston (n=239)	*Hertfordshire (n=845)*
	Mean (SD)	*Mean (SD)*
Birthweight (lb)	7·0 (1·2)	7·9 (1·3)
Placental weight (lb)	1·4 (0·3)	Not available
Weight at one year (lb)	Not available	22·6 (2·6)
Age (years)	51 (2)	64 (3)
Height (m)	1·72 (0·07)	1·72 (0·07)
Weight (kg)	79·8 (13·5)	78·9 (12·7)
BMI (kg/m^2)	26·9 (4·3)	26·8 (3·8)
Waist to hip ratio (%)	93·3 (6·2)	93·5 (5·3)
	Median (quartiles)	*Median (quartiles)*
Alcohol consumption (units)	11 (2,29)	3 (0,9)
	Number (%)	*Number (%)*
Never smoked cigarettes	51 (21)	129 (15)
Current smoker	94 (39)	243 (29)
Ex-smoker	94 (39)	468 (56)

BMI = body mass index

Table II Simultaneous effects of adult body mass index, and weights at birth and at one year, on waist to hip ratio (%)

		Preston Regression coefficient (95% CI)	*Hertfordshire Regression coefficient (95% CI)*
IIa:	Body mass index (kg/m^2)	0·70 (0·54 to 0·86)	0·83 (0·75 to 0·91)
	Birthweight (lb)	−0·56 (−1·12 to 0·00)	−0·29 (−0·52 to −0·05)
IIb:	Body mass index (kg/m^2)	0·70 (0·54 to 0·86)	
	Birthweight (lb)	−0·83 (−1·50 to −0·15)	
	Placental weight (lb)	1·76 (−0·73 to 4·24)	
IIc:	Body mass index (kg/m^2)		0·83 (0·75 to 0·91)
	Birthweight (lb)		−0·20 (−0·46 to 0·06)
	Weight at one year (lb)		−0·10 (−0·23 to 0·03)

CI = confidence interval

Waist to hip ratio was not related to birthweight in either place, nor to placental weight in Preston, nor to weight at one year in Hertfordshire. Body weight and mass, however, were related both to birthweight and to weight at one year. Therefore we allowed for body mass index in our analysis of the relations between waist to hip ratio and early weight. Waist to hip ratio was inversely related to birthweight (table IIa). In contrast it was positively related to placental weight, though this did not reach statistical significance (table IIb). In a simultaneous regression with body mass index, waist to hip ratio rose as the ratio of placental weight to birthweight increased ($p=0·05$). Although waist to hip ratio was inversely related to weight at one year, this relation was not statistically significant in a simultaneous analysis with birthweight (table IIc).

In Preston and Hertfordshire the association of birthweight and the waist to hip ratio was stronger in men with higher body mass indices. This interaction was statistically significant in Hertfordshire.

Duration of gestation, age, social class, smoking, and alcohol consumption were not related to waist to hip ratio after allowing for body mass index.

Discussion

We have shown that in adult men a higher waist to hip ratio, which is an indicator of abdominal fatness,[2] is associated with reduced growth during fetal life and infancy. Waist to hip ratio rose with increasing obesity, as measured by the body mass index, but at any level of obesity there was more abdominal fat in men who weighed less at birth and at one year (table II). This relation was most marked in those with the highest body mass indices. The associations between early growth and waist to hip ratio were independent of alcohol consumption, cigarette smoking, and social class.

The association between waist to hip ratio and lower birthweight was independent of the duration of gestation and must therefore reflect an association with reduced fetal growth. At any birthweight, waist to hip ratio increased with placental weight (table IIb) so that the highest waist to hip ratios were in men who had low birthweight in relation to their placental weight. Low birthweight for placental weight may be interpreted as a sign of fetal growth failure.[13] Other analyses of the Preston data have shown that it is also related to high systolic and diastolic blood pressure,[14] to high plasma fibrinogen concentrations,[15] and to impaired glucose tolerance (unpublished data). The processes which link reduced fetal growth with increased abdominal fat deposition are unknown. Sustained adrenal overactivity,[2] initiated by early growth restraint, is one possible explanation.

Differences in the waist to hip ratio have been shown to correspond to substantial differences in risk of ischaemic heart disease, even after adjustment for body mass index.[2] In a study of 54 year old men with a similar distribution of waist to hip ratio to that described here, those who were in the highest fifth of waist to hip ratio had a risk 2·5 times higher than those in the lowest fifth.[3]

It could be argued that people who experience an adverse early environment, which restrains fetal and infant growth, tend to remain in such an environment and that the association between lower early weight and higher adult waist to hip ratio reflects influences acting later in life. In our data, however, the mean waist to hip ratio was not different between the social classes, as it would be expected to be from this argument.

Our findings are also open to the interpretation that genetic influences determine both reduced early growth and later abdominal fatness. The similarity of fat distribution in siblings, especially identical twins, has led to the suggestion that, unlike body mass, abdominal fat distribution has an important genetic contribution.[16 17] These similarities could, however, reflect similar intrauterine and infant environments rather than genetic resemblance.

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