Influence of birthweight and current body mass on cardiovascular risk factors in young adults

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Abstract: Introduction. Results of other studies indicate at increased predisposition of metabolic diseases in the adulthood in subjects born with low birthweight. **Objectives.** To estimate the prevalence of cardiovascular risk factors in adults in relation to birthweight and current body mass. **Patients and methods.** The study was performed in 498 subjects aged 24–29 years, born in Warsaw in 1974–1977, whose mothers during pregnancy participated in a prospective study of risk factors of low birthweight. Basic anthropometric and blood pressure measurements were performed; total cholesterol, triglycerides, glucose, insulin, fibrinogen and glycosylated hemoglobin were determined in the blood. **Results.** 1) In males body mass index (BMI) and indices of abdominal fat distribution (WHR, waist circumference) correlated positively with the insulin resistance index, blood insulin level, glycated hemoglobin, glucose, triglycerides, total cholesterol, LDL-cholesterol, fibrinogen and also blood pressure, and negativelely with HDL-cholesterol. In females BMI, WHR and waist circumference correlated significantly only with the insulin resistance index, and blood levels of insulin, triglycerides, LDL-cholesterol, HDL-cholesterol and fibrinogen. 2) In males birthweight correlated negatively only with the insulin resistance index and serum insulin level. In females such correlations were not observed. 3) Logistic regression analysis revealed that obesity, particularly abdominal, was a stronger predictor of increased insulin resistance than birthweight. **Conclusions.** In young males abdominal obesity is a much stronger determinant of coronary risk factors than birthweight.

Key words: birthweight, body mass index, cardiovascular risk factors, insulin resistance, young adults

INTRODUCTION

Since the 1980s a number of papers indicating increased predisposition to arterial hypertension, dyslipidemia, diabetes and cardiovascular diseases in adulthood of subjects born with low birthweight have been published [1,2,3]. Intrauterine malnutrition, resulting in low birthweight, seems to predispose to future metabolic abnormalities by aberration in the development of blood vessels, muscles, and pancreatic ß-cells, insulin resistance, and abnormal liver, kidney and other organ functions [4]. Environmental factors that act during extrauterine life may play an important role in aggravating disturbances of fetal origin [5]. Low physical activity and overnutrition that lead to obesity appear to be of particular importance.

This described hypothesis is still under investigation. It should be stressed that in the developed countries, low birthweight occurs in a relatively low percentage (4–9%) [6] of live births. However, metabolic cardiovascular risk factors, particularly obesity, are prevalent. Their relation to nutrition and physical activity, as well as the cause-and-effect relationship with atherosclerosis development are well documented. Therefore, before the suggested relationship could be validated as important for practice of public health protection, further studies on links between intrauterine development retardation and chronic noncommunicable diseases are necessary.

The available data suggest that the association between metabolic abnormalities and/or artherosclerotic vascular disease and birthweight can be shown mostly in adults aged 40-60 years [7,8]. There are only few studies performed in young subjects [9].

The aim of our study was to estimate the prevalence of cardiovascular risk factors in adults aged 24–29 years in relation to birthweight and current body mass.

PATIENTS AND METHODS

In 2000–2004, this study was performed in subjects aged 24–29 years, born in Warsaw in 1974–1977, whose mothers during pregnancy participated in the prospective study of risk factors of low birthweight that was performed by the National Research Institute of Mother and Child. we invited

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1899 subjects to participate in our trial. The invitations were sent at addresses of mothers who were registered in 1974–77. In the case of lack of reply, invitations were sent twice again. Overall, 498 subjects, i.e. 26.2% of the invited subjects, took part in our study.

Data concerning education, profession, current and past diseases, cardiovascular diseases in the family, smoking, physical activity, alcohol consumption, selected features of nutrition and current medication were collected.

Sedentary work and less than 2 hours of physical exercise in leisure per week was considered as low physical activity.

Physical examination included: body height and mass, waist and hip circumference and arterial blood pressure. Height was measured to the nearest 0.5 cm after taking off shoes. Body mass was measured using the medical balance after taking off shoes and all clothes, except of undergarments, to the nearest 0.1 kg. Waist and hip circumferences were measured by means of the anthropometric tape after rolling down undergarments. Waist was measured midway between the iliac crest and the lower rib margin, and hip at the widest circumference over the buttocks, both to the nearest 0.5 cm.

Body mass index was calculated using the formula: BMI = body mass (kg)/ height $(m)^2$. WHR (waist/hip ratio) was also calculated. Overweight was diagnosed when BMI was 25–29.9 kg/m^2, and obesity when BMI ≥30 kg/m^2, abdominal fat distribution when WHR in males was >1.0 and in females >0.85. Data concerning birthweight (BW) and birth body length (BBL) were taken from the archives. Birth Ponderal Index (PI) was calculated using the formula: PI = $BW/(BBL)^3$ (kg/m^3).

Arterial blood pressure was measured using a mercury sphyngomanometer, at the right arm, in the sedentary position, after at least 5 minutes rest, to the nearest 2 mmHg.

Laboratory tests included fasting venous blood concentrations of total cholesterol (TC), triglycerides (TG), glucose (Glu), insulin (Ins), fibrinogen (Fib), and glycosylated hemoglobin (HbA_{1c}). Fasting serum insulin and glucose levels were used for calculation of the insulin resistance index HOMA-IR using a formula: HOMA-IR = (fasting insulin (μU/ml) × fasting glucose (mg/dl) x 0.0555/ 22.5. HOMA-IR <3.0 was recognized as normal [10].

Cholesterol, triglycerides and glucose levels were determined according to the colorimetric method using Ektachem Clinical Chemistry Slides, and HDL-cholesterol to the magnetic method, at the Vitros-250 device produced by Johnson and Johnson Poland ltd. LDL-cholesterol level was calculated using the Friedewald formula (if triglyceride level did not exceed 400 mg/dl [4.5 mmol/l]). Insulin concentration was determined according to the immunochemistry method using IMX device (Abbott), fibrinogen using the Clauss method using Biomerieux kit and Option 2 Plus device, blood glycosylated hemoglobin according to the ion capture assay using IMX device (Abbott).

We admitted cut-off values of risk factors indicated in the European Guidelines on Cardiovascular Disease Prevention in Clinical Practice [11], except for HbA_{1c} and insulin, that corresponded with the laboratory reference values. A value of HbA_{1c} >6,4% and insulin >17 μU/ml were considered as elevated.

Laboratory tests were performed In the Biochemical Diagnostics Laboratory of the Outpatient Clinic of Metabolic Diseases of the National Food and Nutrition Institute, that is under the RIQAS control and also participates in the interlaboratory control organized by the Center of the Laboratory Quality Control.

The Spearman's coefficient, t Student test and nonparametric Kruskall-Wallis test, and also logistic regression were used for statistical analysis of the results. A p-value of 0.05 was considered statistically significant.

Estimation of the representativity of the study group for the general population was performed by comparison of their education, somatic features and smoking with those of the random sample from the Praga-Południe district of Warsaw that took part in the Pol-MONICA BIS Programme [12].

RESULTS

Out of 498 subjects that participated in the study, we excluded 8 males and 76 females. Among them, 4 males and 2 females were excluded because of antihypotensive medication. None of these subjects was born with low birthweight. Reasons for excluding other subjects were: hypo- or hyperthyroidism (n = 5), use of steroid hormones (n = 1), use of hormonal contraception (n = 69), or other medication that could affect lipid or glucose metabolism. The final analysed group consisted of 209 males and 205 females. Thirty-two subjects, i.e. 7.7% of the whole group of 414 participants, had birthweight lower than 2500g. According to the Central Statistical Office data, in 1975–1977 the percentage of low birthweight was 7.3–7.5% in Poland as a whole, and 8.0–8.2% in the cities. It has shown that exclusion of 84 subjects from the study group did not influence the characteristics of the final population. As our population was not a random sample from the general population we compared selected features of our population with the features of the Warsaw population that participated in 2001 in the Pol-MONICA Programme. The majority of subjects that participated in our study had higher education, and their percentage was higher than in the Warsaw general population. Most of females and almost half of males were sedentary. The comparison of this parameter with the population studied in the Pol-MONICA Programme was not possible due to methodological differences in the physical activity assessment. Differences in smoking, body height, body mass, BMI and WHR were not significant.

The analysis of correlations between the cardiovascular risk factors under study revealed the strongest correlations of BMI and abdominal fat distribution with other risk factors. As shown in the table 1, a particularly strong correlation was observed between BMI, WHR and waist circumference on the one side and serum insulin on the other. Also relationship with

Table 1. Spearman's correlation coefficients of coronary risk factors with BMI, WHR, waist circumference, birthweight (BW), and birth ponderal index (PI).

	SBP	DBP	TC	LDL-C	HDL-C	TG	Fib	Glu	HbA_{1c}	Ins	HOMA-IR
Males (n = 209)											
BMI	0.386	0.302	0.306	0.301	– 0.344	0.458	0.396	0.165	0.192	0.564	0.561
p	<0.001	<0.001	<0.001	<0.001	<0.001	<0.001	<0.001	<0.05	0.005	<0.001	<0.001
WHR	0.343	0.325	0.271	0.257	– 0.330	0.435	0.349	0.179	0.170	0.467	0.480
p	<0.001	<0.001	<0.001	0.001	<0.001	<0.001	<0.001	0.009	<0.05	<0.001	<0.001
Waist	0.429	0.350	0.301	0.293	– 0.366	0.479	0.404	0.180	0.183	0.576	0.576
p	<0.001	<0.001	<0.001	<0.001	<0.001	<0.001	<0.001	0.009	0.008	<0.001	<0.001
BW										– 0.158	– 0.164
p	NS	NS	NS	NS	NS	NS	NS	NS	NS	<0.05	<0.05
PI	– 0.138						– 0.164		– 0.165	– 0.171	– 0.179
p	<0.05	NS	NS	NS	NS	NS	<0.05	NS	<0.05	<0.05	<0.05
Females (n = 205)											
BMI			0.156	0.225	– 0.293	0.288	0.342	0.163		0.395	0.393
p	NS	NS	<0.05	0.001	<0.001	<0.001	<0.001	<0.05	NS	<0.001	<0.001
WHR				0.181	– 0.282	0.194	0.181			0.288	0.272
p	NS	NS	NS	0.009	<0.001	0.005	<0.05	NS	NS	<0.001	<0.001
Waist				0.217	– 0.330	0.259	0.324			0.407	0.391
p	NS	NS	NS	0.002	<0.001	<0.001	<0.001	NS	NS	<0.001	<0.001
BW					– 0.222						
p	NS	NS	NS	NS	<0.01	NS	NS	NS	NS	NS	NS
PI											
p	NS	NS	NS	NS	NS	NS	NS	NS	NS	NS	NS

BMI – body mass index, Fib – fibrinogen, Glu – glucose, HbA_{1c} – glycosylated hemoglobin, HDL-C – high-density lipoprotein cholesterol, HOMA-IR – insulin resistance index, Ins – insulin, LDL-C – low-density lipoprotein cholesterol, BW – birthweight, NS – statistically non significant, PI – ponderal index, SBP – systolic blood pressure, DBP – diastolic blood pressure, TC – total cholesterol, TG – triglycerides, WHR – waist to hip ratio

HOMA-IR was strong. It was more evident in males than in females.

In males we observed weak negative correlations of birthweight and PI with only serum insulin and HOMA-IR, and in the case of PI also with the systolic blood pressure (SBP), fibrinogen and HbA_{1c}. We did not show these associations in females. It should be stressed that BW and PI did not correlate with BMI, WHR and waist circumference. It may indicate that the indices of the intrauterine development are independent determinants at least of insulin resistance.

For more accurate estimation of the relationship between coronary risk factors and indices of body fatness, mean values of BMI, WHR and waist circumference were calculated after dividing the values of SBP, DBP, TC, LDL-C, HDL-C, TG, Fib, Glu, HbA_{1c}, Ins, and HOMA-IR into normal and abnormal. As it is shown in the table 2 and 3, the increased values of risk factors were related to increased indices of body fatness. In the most of the parameters the differences were statistically significant. However, it did not concern birthweight, that did not relate unequivocally to the intensity of the risk factors. Only in males higher values of HOMA-IR was significantly related to lower birthweight.

To better differentiate the effect of a number of independent parameters (such as the amount of body fat, type of fat distribution, and birthweight) on insulin resistance, models of logistic regression of the dependent parameter HOMA-IR ≥3.00 were used (tab. 4). In males, after excluding the influence of WHR, the 100 g increase in birthweight was associated with about 10% decrease in the insulin resistance ($p = 0.004$) and about 13% after excluding the effect of BMI ($p = 0.001$) or waist circumference ($p = 0.001$). In females such a statistically significant relationship was not observed. Current body fatness influenced insulin resistance to a greater extent than birthweight. An increase in BMI by 1 unit was associated with a higher risk of insulin resistance by 51% in males ($p < 0.001$) and by 42% in females ($p < 0.001$); the increase in waist circumference by 1 cm led to a rise in this risk by 17.5% in males ($p < 0.001$) and by 12% in females ($p < 0.001$). WHR influenced the risk of insulin resistance to the greatest extent. An increase in WHR by 0.1 increased the insulin resistance risk 10.9 times in males ($p < 0.001$), and 3.6 times in females ($p < 0.001$).

Table 2. Body fat indices and birthweight in relation to coronary risk factors in males

Risk factors	N	BMI (mean ±SD)	WHR (mean ±SD))	Waist – cm (mean ±SD)	Birthweight – g (mean ±SD)
SBP <140 mm Hg	125	24.6 ±3.7	0.89 ±0.06	88.8 ±10.2	3370 ±583
≥140 mm Hg	84	27.0 ±4.3	0.93 ±0.06	97.0 ±11.7	3478 ±548
p*		<0.001	<0.001	<0.001	NS
DBP <90 mm Hg	129	24.5 ±3.5	0.89 ±0.05	88.7 ±9.5	3424 ±568
≥90 mm Hg	80	27.2 ±4.5	0.94 ±0.06	97.6 ±12.5	3396 ±577
p*		<0.001	<0.001	<0.001	NS
TC <5 mmol/l	120	24.7 ±3.8	0.90 ±0.06	89.9 ±11.3	3443 ±549
≥5 mmol/l	89	26.6 ±4.3	0.93 ±0.06	95.2 ±11.2	3400 ±601
p*		0.001	<0.001	0.001	NS
LDL-C <3 mmol/l	116	24.5 ±3.9	0.90 ±0.06	89.5 ±11.7	3438 ±545
≥3 mmol/l	92	26.7 ±4.0	0.92 ±0.06	95.2 ±10.5	3379 ±606
p*		<0.001	0.003	<0.001	NS
HDL-C ≥1 mmol/l	175	25.1 ±3.9	0.91 ±0.06	91.1 ±11.3	3437 ±584
<1 mmol/l	34	27.6 ±4.4	0.94 ±0.06	97.6 ±11.1	3291 ±485
p*		0.002	0.009	0.002	NS
TG <1.7 mmol/l	161	24.8 ±3.7	0.90 ±0.06	90.0 ±10.3	3409 ±594
≥1.7 mmol/l	48	28.0 ±4.5	0.95 ±0.07	99.4 ±12.6	3427 ±490
p*		<0.001	<0.001	<0.001	NS
Fib ≤3 g/l	199	25.4 ±4.0	0.91 ±0.06	91.8 ±11.4	3428 ±569
>3 g/l	9	28.1 ±5.7	0.94 ±0.07	98.3 ±14.1	3144 ±573
p*		NS	NS	NS	NS
Glu <6.0 mmol/l	201	25.4 ±4.0	0.91 ±0.06	91.9 ±11.3	3415 ±564
≥6.0 mmol/l	8	27.8 ±6.0	0.95 ±0.07	97.8 ±16.6	3375 ±750
p*		NS	NS	NS	NS
HbA_{1c} ≤6.4%	201	25.4 ±3.9	0.91 ±0.06	91.8 ±11.0	3421 ±565
>6.4%	7	27.5 ±7.9	0.94 ±0.11	99.0 ±21.8	3257 ±741
p*		NS	NS	NS	NS
Ins ≤17 μU/ml	198	25.1 ±3.7	0.90 ±0.06	91.0 ±10.3	3412 ±578
>17 μU/ml	11	32.8 ±4.3	1.01 ±0.07	113.0 ±12.7	3427 ±434
p*		<0.001	<0.001	<0.001	NS
HOMA-IR <3.00	175	24.6 ±3.4	0.90 ±0.05	89.6 ±9.5	3457 ±572
≥3.00	34	30.1 ±4.4	0.97 ±0.06	105.3 ±12.2	3184 ±511
p*		<0.001	<0.001	<0.001	0.012

Cut-off values for risk factors in traditional units: Fib 300 mg/dl, Glu 110 mg/dl, HDL-C 46 mg/dl, LDL-C 115 mg/dl, TC 190 mg/dl,TG 150 mg/dl.
p* – t Student test. Abbreviations as in the table 1

DISCUSSION

Cardiovascular disease prevention is based on the use of risk factors. Studies indicating a possible influence of birthweight on the later predisposition to these diseases may suggest its recognition as a risk factor to be useful. This would give the rationale for the much earlier implementation of the prevention than at present. However, more evidence of cause and effect relationship between birthweight and diseases of atheromatous origin, and also better understanding of its pathologic mechanisms is necessary to establish such indications. As already mentioned, better understanding of links be-

Table 3. Body fat indices and birthweight in relation to coronary risk factors in females

Rrisk factors	N	BMI (mean ±SD)	WHR (mean ±SD))	Waist – cm (mean ±SD)	Bbirthweight – g (mean ±SD)
SBP <140 mm Hg	180	22.3 ±3.8	0.75 ±0.06	74.0 ±9.4	3266 ±550
≥140 mm Hg	25	24.6 ±6.6	0.78 ±0.11	80.8 ±19.4	3344 ±444
p*		0,011	0.024	0.004	NS
DBP <90 mm Hg	164	22.4 ±3.8	0.75 ±0.06	73.9 ±9.4	3263 ±523
≥90 mm Hg	41	23.4 ±5.8	0.78 ±0.09	78.4 ±16.4	3324 ±597
p*		NS	0.015	0.021	NS
TC <5 mmol/l	147	22.4 ±4.0	0.75 ±0.06	74.2 ±9.6	3259 ±564
≥5 mmol/l	58	23.1 ±4.8	0.76 ±0.09	76.3 ±14.5	3319 ±466
p*		NS	NS	NS	NS
LDL-C <3 mmol/l	162	22.2 ±3.9	0.75 ±0.06	73.5 ±9.3	3236 ±559
≥3 mmol/l	43	24.2 ±5.2	0.78 ±0.10	79.8 ±15.9	3423 ±421
p*		0.004	0.005	0.001	0.043
HDL-C ≥1 mmol/l	190	22.3 ±4.0	0.75 ±0.07	74.0 ±10.7	3255 ±542
<1 mmol/l	15	25.9 ±6.1	0.80 ±0.07	84.2 ±14.0	3533 ±415
p*		0.002	0.003	0.001	NS
TG <1.7 mmol/l	199	22.3 ±3.8	0.75 ±0.06	74.0 ±9.4	3280 ±529
≥1.7 mmol/l	6	30.9 ±8.5	0.90 ±0.18	100.2 ±29.8	3133 ±838
p*		<0.001	<0.001	<0.001	NS
Fib ≤3 g/l	181	22.2 ±3.6	0.75 ±0.06	73.7 ±9.0	3246 ±532
>3 g/l	23	26.0 ±6.9	0.79 ±0.11	83.2 ±20.7	3496 ±553
p*		<0.001	0.005	<0.001	0.036
Glu <6.0 mmol/l	202	22.5 ±4.0	0.75 ±0.06	74.4 ±9.9	3275 ±540
≥6.0 mmol/l	3	30.3 ±13.6	0.91 ±0.26	100.7 ±44.1	3333 ±416
p*		0.001	<0.001	<0.001	NS
HbA_{1c} ≤6.4%	201	22.5 ±4.1	0.75 ±0.07	74.6 ±10.8	3267 ±540
>6.4%	3	30.5 ±10.1	0.82 ±0.13	91.7 ±29.3	3733 ±58
p*		0.001	NS	0.009	NS
Ins ≤17 μU/ml	199	22.2 ±3.6	0.75 ±0.06	73.8 ±8.8	3285 ±537
>17 μU/ml	6	34.5 ±7.8	0.92 ±0.17	107.3 ±27.7	2950 ±497
p*		<0.001	<0.001	<0.001	NS
HOMA-IR <3.00	189	22.0 ±3.3	0.75 ±0.05	73.3 ±8.2	3284 ±539
≥3.00	16	29.8 ±7.1	0.83 ±0.14	92.3 ±22.9	3175 ±532
p*		<0.001	<0.001	<0.001	NS

Cut-off values for risk factors in traditional units: Fib 300 mg/dl, Glu 110 mg/dl, HDL-C 46 mg/dl, LDL-C 115 mg/dl, TC 190 mg/dl,TG 150 mg/dl.
p* – t Student test. Abbreviations as in the table 1

tween birthweight and cardiovascular diseases is considerably hindered by the influence of environmental factors that act during extrauterine life, especially lifestyle, on the risk factors. Therefore, there is a need for the estimation of the influence of birthweight on conventional coronary risk factors after taking lifestyle into account. It is particularly reasonable to perform it in young subjects with signs or symptoms of atherosclerotic vascular disease.

In males we observed a significant correlation of BMI, WHR and waist circumference with blood pressure and metabolic coronary risk factors. The correlation coefficient was particularly strong for serum insulin level and HOMA-IR. It is un-

Table 4. Influence of birthweight and current body fatness on HOMA-IR ≥3.00 in the estimated models of logistic regression

		Males			Females		
		Exp(β)	95% CI for Exp(β)	p value	Exp(β)	95% CI for Exp(β)	p value
Model 1	birthweight (100 g)	0.922	0.863–0.983	0.014	0.964	0.880–1.057	NS
Model 2	BMI	1.510	1.316–1.734	<0.001	1.417	1.231–1.631	<0.001
	birthweight (100 g)	0.866	0.796–0.941	0.001	0.930	0.839–1.031	NS
Model 3	WHR (0.1)	10.953	4.693–25.564	<0.001	3.586	1.745–7.372	0.001
	birthweight (100 g)	0.894	0.829–0.964	0.004	0.964	0.882–1.055	NS
Model 4	waist (cm)	1.175	1.111–1.243	<0.001	1.120	1.065–1.177	<0.001
	birthweight (100 g)	0.864	0.793–0.940	0.001	0.938	0.853–1.031	NS

BMI – body mass index, Exp(β) – odds ratio, HOMA-IR – insulin resistance index, WHR – waist to hip ratio

derstandable in the light of numerous data concerning higher levels of metabolic coronary risk factors in obesity [13] and also hyperinsulinemia and insulin resistance in obese subjects [14]. However, there were no correlations of birthweight with blood pressure and blood lipoproteins, fibrinogen and glucose level. Correlations with serum insulin level and HOMA-IR were significant, but much weaker than that of current body fatness. The relationship between PI and the risk factors was slightly more pronounced. However, correlation coefficients concerned only some parameters and were much weaker than that of BMI, WHR and waist circumference. In females the discussed relationships were weaker. However, similarly as in males, correlations of body fat indices with serum insulin level and HOMA-IR were the most pronounced. In females birthweight and PI did not correlate with the coronary risk factors. Our results indicate that in the study group body fat and abdominal fat distribution were strong determinants of insulin resistance, hyperinsulinemia, arterial hypertension, and metabolic risk factors, whereas birthweight was much less important.

The results presented in tables 2 and 3 confirm this conclusion. Abnormal values of the investigated risk factors related to significantly higher values of BMI, WHR and waist circumference. Such relationship was not observed in the case of birthweight. High values of HOMA-IR related to lower birthweight only in males. However, the level of statistical significance was lower than in the case of BMI, WHR and waist circumference.

Results presented in the table 4 are the additional confirmation of the discussed observations. In the logistic regression models, both in males and in females, higher values of BMI, WHR and waist circumference related to higher risk of insulin resistance. Influence of birthweight was much weaker, and statistically significant only in males. Increase in birthweight decreased the risk of insulin resistance in males. However, the effect of BMI, WHR and waist circumference on HOMA-IR was much more pronounced than that of the birthweight.

In conclusion, we have demonstrated that the abdominal obesity is a much stronger determinant of metabolic coronary risk factors than birthweight. However, this conclusion refers to young subjects that were enrolled in our current study. In older subjects, as described by other authors, the effect of birthweight on metabolic risk factors may be more potent, which may result from the earlier depletion of Langerhans' islets in subjects born with low birthweight. Indices of body fatness correlated with all coronary risk factors in males, and only with some of them in females. In m ales, birthweight correlated negatively with insulin resistance and serum insulin level. In females such relationship was not observed. In males abdominal obesity was a much stronger determinant of coronary risk factors than birthweight.

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