Should low birth-weight be considered a relevant risk factor for rise in pulse pressure among adult overweight-obese subjects?

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ABSTRACT

Low birth-weight (BW) is related to rise in blood pressure (BP) later in life. Aim of this study is investigating whether presence of overweight-obesity modifies this relationship, independently from any additional correlate of metabolic syndrome. We studied 535 (216 M/319 F) otherwise healthy overweight-obese people (body mass index≥25 kg/m^2), recording systolic, diastolic and pulse BP as well as plasma glucose and lipids, additionally interviewing them about BW and weight-change after age of 18 years. The reciprocal of BW was related only to pulse pressure (PP, r=0.14; P=0.04), uniquely in men and individuals with BW≤2500 g had a higher relative risk of having PP above upper quartile (>60 mmHg), independently of sex. After adjusting for confounders each 1 kg rise in BW was associated with 2.84±0.88 (standard error) mmHg decrease in PP; P=0.0042. Moreover, again only among males, the lower BW the higher was the risk of a PP>60 mmHg [odds ratio (95% confidence interval): 2.43 (1.39-4.24); P=0.0018]. In conclusion BW was inversely related only with PP in overweight-obese subjects, uniquely in men, being such effect independent from other correlates of metabolic syndrome. Since elevated PP can be considered a proxy of vascular damage, these findings further stress the importance of inquiring about BW to better stratify the risk of vascular damage, in adult overweight-obese individuals.

Introduction

Several studies, published during the last couple of decades, have clearly demonstrated that low birth-weight (BW) is a significant predictor of diabetes mellitus, hypertension or metabolic syndrome later in life.[1-8] Among correlates of metabolic syndrome obesity seems, however, to be an exception since the majority of studies has pointed out that there is a direct, instead of an inverse, relationship between BW and obesity in children, adolescents or even in adults.[9] The consequence of this, in the light of the strict link existing between obesity and arterial hypertension, is that in obese subjects an attenuated or even a reversed relation between a small BW and raised blood pressure (BP) in adult life may be expected.

In addition, all meta-analyses concerning either case control or population studies, have not been able to clearly ascertain what type of blood pressure (systolic, diastolic or pulse BP) was more related to low BW, as well as whether this is observed in overweight-obese subjects independently from the frequently associated correlates of hypertension or metabolic syndrome (altered glucose metabolism, rise in plasma lipids, increase in body weight during the adult life, *etc.*). Furthermore a poorly investigated point is the effect exerted by change in body weight in age beyond 18 years on raised levels of blood pressure, since the majority of previous studies regard only females.[10-12] Finally, a further question which has not yet been completely clarified is whether the relationship between low BW and rise in BP is equally present in men and in women.

This study is aimed at answering each of these questions, by retrospectively reviewing the database of overweight-obese patients who came to our hospital's outpatient clinic asking for dietetic advice.

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Contributions: AF, GS, FC, EA conceived and designed the study; GF, MS extracted records and controlled the quality of the dataset; GS, LP discussed and edited the manuscript; GS, guarantor of this paper, had full access to all the data in the study and takes the responsibility for the integrity of data and accuracy of data analysis.

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Materials and Methods

Patients

This study presents the results obtained by the retrospective analysis of a dataset concerning 535 overweight-obese people [body mass index (BMI) ≥25 kg/m^2], 216 males, and 319 females, otherwise healthy, who consecutively came to the dietetic outpatient clinic of our hospital to obtain dietetic advice, during the last four years, sent by their family practitioners.

Methods

In all patients we recorded BMI (body weight in kilograms divided by the square of the height in meters), waist circumference (recorded as centimetres at the upper hip bone by a measuring tape around the abdomen), and fasting plasma concentration of glucose or lipids, by standardized methods. BW was ascertained by means of a standardized interview as previously described,[13] additionally inquiring about body weight value at the approximate age of 18 years, recorded as the ratio of the difference between actual and recalled weight at age of 18 (Δ-weight at numerator), to actual age subtracting 18 at denominator [Δ weight/age-18; (kg/y)].

BW was considered as a continuous variable or categorized by two classes (≤2500 g or >2500 g).

In each patient office blood pressure was recorded as the mean of three measurements obtained in the sitting position, using suitable cuffs for obese people (12x40 cm).[14]

This study has been approved by the Ethical Committee of our Hospital.

Statistics

Univariate statistics included comparisons between male and female subjects or between those with BW ≤2500 g and those >2500 g by 2-tailed-Wilcoxon-rank-sums test. Spearman's coefficients after least-squares method were used to evaluate correlations among continuous variables, and, in this same analysis BW was transformed into its reciprocal to normalize the distribution.

Relative risks according to BW ≤ or >2500 g, separately evaluated by sex, were calculated with the Mantel-Haenszel method after Chi-square test.

Stepwise multivariate regression analysis, was used to evaluate the hierarchy of independent variables in building up the total BP variance (systolic, diastolic, and pulse pressure) by a model where BP was the dependent variable and age, BMI, waist circumference, Δ-weight/age, total cholesterol, triglycerides, fasting plasma glucose and high-density lipoprotein-cholesterol entered the model as covariates. The assumed significance level for variables entering into the model was 0.15.

A multiple logistic regression analysis was carried out to assess the adjusted relative risk of elevated BP values: according to this model the dependent variables (y) were, alternatively, systolic, diastolic or pulse pressure stratified as under or above their upper quartile and expressed as dummies 0 and 1. Independent variables were age, BMI, waist circumference, Δ-weight/age, and BW introduced as a continuous variable or alternatively categorized as ≤2500 (dummy=0) or >2500 g (dummy=1).

Significance of P-value was set at <0.05. All statistical analyses were carried out by means of SAS software for Windows, version 9.3 (SAS Institute Inc., Cary, NC, USA).

Results

The main characteristics of population stratified by sex are reported in Table 1. Prevalence of smokers was higher among males, who were moreover characterized by a greater Δ-weight/age, as well as by a larger waist circumference and increased prevalence of dyslipidemia. BW was, as expected, on average, higher among males while, conversely, the prevalence rate of small babies (BW≤2500 g) was lower in men than in women. With regard to BP values, only systolic BP, on average higher among males, was significantly different between genders (Table 1).

In univariate analysis the reciprocal value of BW was not related to any of the measured variables, except for a weak relation with pulse pressure, among males (r=0.14; P=0.04; Table 2).

While mean diastolic and systolic BP were not significantly different between those with BW above or under 2500 g, pulse pressure, defined as the difference between systolic and diastolic BP, was significantly higher in the group with BW≤2500 g in men (58±11 standard deviation mmHg *vs* 51±13 mmHg; P=0.012 by Wilcoxon rank sums test), not among the women (52±15 mmHg *vs* 48±14 mmHg; P=not significant). Pulse pressure resulted significantly higher in women with BW≤2000 g (n=18; 5.6%) compared to those above this cut-off (57±17 mmHg *vs* 49±14 mmHg; P=0.0192). In addition, the relative risk of having a pulse pressure exceeding its upper quartile (>60 mmHg) was significantly higher in those with BW ≤2500 g both in men and women, while, contrarily, a lower BW was not associated with the rise in relative risk of having a systolic or diastolic BP exceeding their upper quartile (>150 mmHg for systolic and >95 mmHg for diastolic BP) (Table 3).

Only pulse pressure elevation was significantly and independently associated with a low BW in men, independently from any of the main constituents clustering metabolic syndrome (waist circumference, increase in plasma glucose and lipids, BMI) (Table 4).

From this same model, among men, each 1 kg rise in BW, after adjusting for covariates, was associated with a decrease of 2.84±0.88 (standard error) mmHg in pulse pressure; P=0.0042.

The relative risk, expressed as odds ratio (OR), of having a BP in the upper quartile of systolic, diastolic and pulse pressure, according to each kg decrease in BW, after adjusting for BMI, age, past weight increase, blood glucose and plasma lipids is shown in Figure 1. While the adjusted relative risk was not significant for systolic and diastolic BP, the OR of having pulse pressure >60 mmHg was significantly higher only among men, rising by about 2.5 fold for each kg decrease in BW [OR (95% confidence interval, CI): 2.43 (139-4.24); P=0.0018]. Similar results were obtained replacing BW with its categorical measures ≤ or >2500 g (data not shown).

No significant associations were observed between small BW and the presence of arterial hypertension defined as systolic BP >140 mmHg and/or diastolic BP >90

Table 1. Characteristics of patients under study [mean±standard deviation or median (interquartile range)].

	Males	Females	P value
No.	216	319	-
Age (y)	42±15	43±14	ns
Menopause (%)	-	103 (19.2)	-
Smokers (%)	35	25	0.02
BMI (kg/m^2)	32±4.5	31.2±5.7	ns
BMI at 18 years (kg/m^2)	24.2±3.9	22.9±3.7	0.02
Δ-weight/age (kg/y)*	1.00 (1.08)	0.87 (0.78)	0.02
Waist circumference (cm)	108±11	98.6±12.9	0.0001
Systolic blood pressure (mmHg)	140±17	135±20	0.02
Diastolic blood pressure (mmHg)	89±10	86±11	ns
Pulse pressure (mmHg)°	51±13	49±14	ns
Fasting plasma glucose (mg/100mL)	97±17	94±16	ns
Plasma cholesterol (mg/100 mL)	213±40	212±43	ns
Plasma triglycerides (mg/100 mL)	183±110	124±67	0.0001
HDL cholesterol (mg/100 mL)	43±11	55±13	0.0001
Birth-weight (g)	3757±866	3343±793	0.0001
No. (%) of small babies (≤2500 g)	14 (6.5)	40 (12.5)	0.02

ns, not significant; BMI, body mass index; HDL, high-density lipoprotein. *(Actual weight-weight at 18 year)/age-18, expressed as median value [interquartile range]; °difference between systolic and diastolic blood pressure.

Table 2. Univariate correlations with the reciprocal of birth-weight, in males and females.

	Males		Females	
	r	P value	r	P value
BMI	–0.09	ns	–0.004	ns
Weight circumference	–0.08	ns	0.01	ns
Δ-weight*	–0.009	ns	0.004	ns
Systolic blood pressure	0.04	ns	0.06	ns
Diastolic blood pressure	–0.09	ns	–0.006	ns
Pulse pressure	0.14	0.04	0.10	ns
Plasma fasting glucose	0.11	ns	0.05	ns
Triglycerides	–0.08	ns	0.06	ns
Cholesterol	0.05	ns	0.13	0.02
HDL cholesterol	0.05	ns	0.03	ns

BMI, body mass index; ns, not significant; HDL, high-density lipoprotein. *Weight increase since age of 18 years.

mmHg, both in univariate and multivariate analyses.

Finally no differences, in systolic, diastolic or pulse pressure were observed after stratifying BW as < or ≥4000 g (data not shown).

Discussion and Conclusions

A small BW is a major risk factor for development of increased BP values later in adult life[15-19] as stressed by a recently published meta-analysis, which has moreover evidenced that BW is inversely related to systolic and to a lesser extent to diastolic BP in a linear way estimating that 1 kg increase in BW is associated with 2-4 mmHg reduction in systolic BP.[7] Moreover several evidences have been accrued in the past, unanimously highlighting that a small BW, as observed in preterm births, is associated with the development of arterial hypertension later, even in children or in adolescents.[1,20]

The reason of this inverse relation between small BW and increased BP has yet not been fully clarified, suggesting that a growth retardation *in utero* may lead to a significantly greater risk for the development of the cluster of metabolic syndrome's correlates (diabetes, hypertension, dyslipidemia, obesity) and, consequently, to the risk of any among the associated cardiovascular events, later in the adult life.[21]

Furthermore, since hypertension and obesity may frequently occur together, we guessed being of interest to study whether the increased risk conferred by a low BW is being equally present in overweight-obese people. This latter aspect has not fully been addressed by previous studies since the majority of them have calculated the risks simply after adjusting for body weight, and the issue is moreover more complicated by the fact that, contrarily, a high BW seems to predict a raised risk for development of obesity later in adult life,[9] so partially dampening the expected inverse relation of BW with high blood pressure. A second point which has, till now, scarcely been investigated is whether sex may modify the relation between small BW with risk and increased BP in the adult life.

Table 3. Unadjusted relative risks (95% confidence interval) by Mantel-Haenszel method after Chi-square test of having systolic, diastolic or pulse pressure in the upper quartile according to birth-weight class ≤ or >2500 g, in males and in females.

	Males				Females			
	Birth-weight		Relative risk	P value	Birth-weight		Relative risk	P value
	≤2500 g	>2500 g	(95% CI)		≤2500 g	>2500 g	(95% CI)	
Subjects in upper PP quartile (>60 mmHg)	9/63 (14.3)	5/153 (3.3)	2.1 (1.1-4.2)	0.002	17/83 (20.5)	23/236 (9.7)	1.33 (1.1-1.7)	0.01
Subjects in upper DBP quartile (>95 mmHg)	2/52 (3.8)	12/164 (7.3)	0.9 (0.7-1.1)	ns	8/54 (14.8)	32/265 (12.1)	1.1 (0.9-1.3)	ns
Subjects in upper SBP quartile (>150 mmHg)	5/72 (6.9)	9/144 (6.2)	1.1 (0.7-1.5)	ns	11/84 (13.1)	29/235 (12.3)	1.1 (0.8-1.2)	ns

CI, confidence interval; PP, pulse pressure; ns, not significant; DBP, diastolic pressure; SBP, systolic pressure.

Table 4. Stepwise regression analysis relating pulse, systolic, and diastolic blood pressure as dependent variables with reciprocal of birth-weight, body mass index, age, waist circumference, past weight increase, blood glucose, and plasma lipids as independent variables. The assumed significance level for variables entering into the model was 0.15.

Variables	Males		Females	
	Model R^2	P value	Model R^2	P value
Pulse pressure				
Age	0.054	0.0056	0.127	0.0001
BMI	0.136	0.038	0.171	0.0023
Birth weight	0.109	0.0042		
Systolic blood pressure				
Age	0.080	0.0007	0.177	0.0001
BMI	0.162	0.0003	0.255	0.0001
Triglycerides	0.229	0.0008		
Birth weight	0.2511	0.0471		
Diastolic blood pressure				
Age	0.211	0.0252	0.101	0.0001
BMI	0.181	0.0007	0.164	0.0003
Triglycerides	0.110	0.0001		

BMI, body mass index.

As to the first point our findings suggest that only pulse pressure, uniquely among men, is linearly related to small BW in overweight-obese individuals. From a recent meta-analysis, evaluating a wide panel of studies, the mean difference in systolic BP between subjects with low BW (<2500 g) compared with subjects with BW >2500 g was 2.58 mmHg (95% CI 1.51-3.64), being the difference in diastolic BP, on average, much lower [1.01 mmHg (0.19-1.83)], while no data have been given about pulse pressure.[7]

In this context a previous study by Lurbe *et al.*[22] showed that obesity magnifies the inverse relation between BW and BP in adolescents (mean age 13 year), even if in multivariate analysis this relation lost any significance for office PP (P=0.165).

Since pulse pressure is the difference between systolic and diastolic BP it is not surprising that it may have a significant negative association with BW. What is at variance with previous studies, however, is the lack of any inverse relation between systolic BP and BW, and it can be hypothesized that this could represent a specific feature differentiating obese from non-obese individuals, even considering that comparisons with past studies are made difficult by the not homogeneous prevalence of overweight-obese individuals in each of them.

A further observation from our findings is that low BW was not associated with the risk of arterial hypertension defined as a BP>140/90 mmHg, at variance with what observed by previous population studies.[23] This is probably due to the fact that cut-off values for hypertension may be more elevated in obese people even if, as also suggested by multivariate analysis; males have a weak association between low BW and upper systolic BP quartile (Table 4).

An additional support to the relation between pulse pressure and small BW is given by the fact that, in the elderly pulse pressure increases as the result of increased arterial stiffness[24,25] and arterial rigidity, by whichever way measured, *i.e.*, as stiffness index or arterial pulsatile function, has been demonstrated as being significantly related to small BW, even at a much higher extent than systolic or diastolic BP.[26,27]

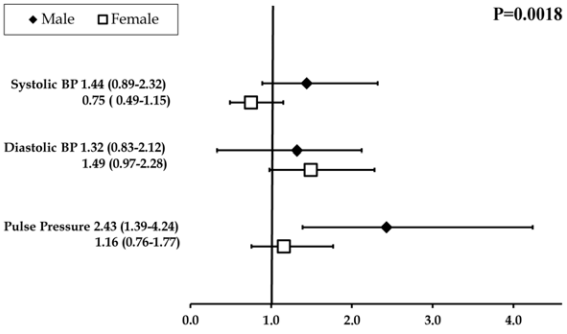

Figure 1. Risk of being in the upper quartile of systolic, diastolic or pulse blood pressure (BP), expressed as odds ratio (95% confidence interval) according to each kg decrease in birth-weight, adjusted for body mass index, age, waist circumference, past weight increase, blood glucose, and plasma lipids in men and in women.

The reason of sex dimorphism in the association low BW-pulse pressure is not clarified by our study. What appears from meta-analyses is that the inverse relation between BP and BW seems more evident in males[7] even if this is yet widely controversial. To the best of our knowledge neither previous studies, nor any meta-analysis, have been carried out with the primary purpose of answering this question. What is suggested by our data is that defining the lower cut-off threshold to categorize a small BW may be crucial also in the light of the observation that the rate of individuals with BW≤2500 g is significantly higher among women. In this regard, it is interesting to note that, from our data, mean pulse pressure resulted significantly higher in women with BW≤2000 g, obtaining values close to what observed in men. A further issue to be considered is that in the age range of our study, centred on a mean value of about 43 years for both genders, the risk of arterial hypertension is greater among males[28] and such an excess risk could contribute to the gender difference, which we observed.

A main point suggested by the present study is that among the clinical correlates of metabolic syndrome, none has, hierarchically, the same relevance presented by low BW in predicting raised pulse pressure in adult life. In other words, even in overweight-obese people, it seems opportune adding a low BW, when available, to the list of the risk factors able to eventually predict high pulse pressure, especially among men. The value expressed by mean change in body weight after age 18, is, nonetheless, negligible in explaining the variance of adult BP.

This study has some limitations. A first limit is the way BW was recalled, namely by interview. This is, however a bias that is present in many of previous studies, and even if recording BW by a simple interview is obviously less sensitive than doing it by obstetrical records, it seems to be even more accurate than using a structured questionnaire,[7] having, in addition, the advantage of being simple and immediate. A further related limitation is that information about gestational age as well as about more detailed measures of birth size is lacking.

Conclusions

Even if with the above mentioned limitations, this study confirms that also in overweight-obese subjects there is an inverse relationship between BW and increase in BP (pulse pressure), either considering BW

as a continuous variable or after categorizing it as ≤ or >2500 g. The entire effect of BW on later increment in BP is, however, modest since 1 kg increase in BW is associated with a mean of 2.84 mmHg reduction in pulse pressure, even if it seems hierarchically more important in predicting raised arterial stiffness than any other correlate of the metabolic syndrome.

The second conclusion to be drawn from our findings is that the relation between small BW and rise in pulse pressure is more evident among men, and that such sexual dimorphism is for the most part reversed after introducing for women a lower BW cut-off threshold (2000 g).

The third conclusion is that inquiring about weight change after adolescence is of poor significance in determining the risk of elevated BP values in our population of overweight-obese patients.

Since elevated pulse pressure can be considered a proxy of vascular damage, these findings further stress the importance of inquiring about BW to better stratify the risk of vascular damage, in adult life among overweight-obese individuals, especially in men.

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