

Impact of parity on anthropometric measures of obesity controlling by multiple confounders: a cross-sectional study in Chilean women

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ABSTRACT

Aim: To find out whether there is an association between parity and obesity, evaluated through body mass index (BMI), waist circumference (WC), waist-to-hip ratio (WHR) and waist-to-height ratio (WHtR) in Chilean women after controlling for sociodemographic characteristics, health risk and gynaeco-obstetric factors.

Design: Cross-sectional study, using baseline data of the San Francisco Project.

Setting: San Francisco de Mostazal, located in the central region of Chile, 6512 Chilean-Hispanic women (Spanish heritage with a variable indigenous component).

Methods: A weighted random sample of 508 women who had their first pregnancy inside the primary child-bearing ages. Data were collected between 1997 and 1999. Statistical associations between parity and different anthropometric measurements of adiposity in multiple linear (MLnR) and logistic regression models (MLtR) were evaluated.

Results: In MLnR a modest parity-related increment in BMI and practically null increment in WC, WHR and WHtR was observed. Covariates that showed a statistically significant association with anthropometric measures of adiposity were age, low education, marital status, employment, smoking, smoking cessation, hypertension, diabetes, dyslipidaemia, parent's obesity, menarche and fetal macrosomia. Crude odds ratio (OR) showed a strong association between parity and anthropometric markers of obesity. Nevertheless, after adjustments in MLtR models, the association remained only for BMI. All the measures of abdominal obesity related to parous women showed OR smaller than 1 (95% confidence intervals 0.57 to 0.96).

Conclusions: Parity modestly influences BMI, but does not seem to be related to WC, WHR and WHtR after controlling by confounders. Parity can increase adiposity but not necessarily following an abdominal pattern.

Obesity is a major risk factor for numerous non-communicable chronic diseases and mortality¹ and its prevalence, especially in women, is reaching epidemic proportions worldwide.²⁻⁵ Body mass index (BMI) is commonly used to diagnose obesity,² whereas other anthropometric measurements such as waist circumference (WC) and waist-to-hip ratio (WHR) are rarely utilised to measure abdominal adipose tissue distribution.^{6,7} Nowadays, it is accepted that the measurements of abdominal adipose tissue correlate better with cardiovascular risk factors than BMI.⁶⁻⁹ Moreover, recent epidemiological studies suggest that another abdominal adiposity marker, the waist-to-height

ratio (WHtR) is a better predictor of metabolic and cardiovascular risk than BMI, WC and WHR.^{9,10}

Women frequently perceive that pregnancy triggers their weight gain and obesity. The association between reproductive factors such as parity with weight gain and obesity prevalence in women has been intensely investigated with controversial results.¹¹⁻¹⁷ Biological explanations mainly refer to weight gain and/or weight retention as a result of hormonal changes during pregnancy, increased dietary intake, changes in the energy balance, heritable characteristics, adverse lifestyle risk factors associated with child-rearing and other postpartum behaviours.^{3,11,16-18} Although many studies describe an association between pregnancy and the increase of BMI after childbirth, its real impact would be modest and intertwined in a complex pattern which includes ethnic, social and demographic factors and other health risk factors. Furthermore, some prospective studies only found an association between BMI and the first pregnancy,^{5,14} whereas others suggest a positive gradient with consecutive pregnancies.^{15,16}

At the present time, it is recognised that abdominal adiposity and insulin resistance are linked in a cycle of recursive causality including reproductive problems, such as hyperandrogenism, polycystic ovarian syndrome (PCOS) and decreased fertility.¹⁹⁻²² However, it is not clear if biological changes that occur during pregnancy, including hormonal adaptations and postpartum behaviour, influence the regional distribution of adiposity, by promoting an abdominal or peripheral pattern. In fact, the relation between parity and regional adiposity accumulation has barely been investigated.

Recent cross-sectional studies suggest a complex parity-weight relation for women with a range of confounding factors interacting throughout their life. This association may not be the same in women outside of industrial developed countries so their parity-related overweight needs to be studied in their specific communities.^{23,24} We here report a cross-sectional study conducted in Chile, a middle income developing country, in order to establish if parity is associated with BMI and, especially, with abdominal adiposity anthropometric measures in women after controlling for potential confounders. The survey was performed during a time of dramatic increases in obesity and higher rates of fertility in Chilean women,²⁵ providing a rich and meaningful source of data on the association between obesity and parity.

METHODS

Data for this cross-sectional analysis were obtained from the baseline of a longitudinal study being conducted in San Francisco de Mostazal (San Francisco Project, SFP), located in the central region of Chile, in a population of 13 055 residents over 20 years of age, with 98.7% being Chilean-Hispanics (Spanish heritage with a variable indigenous component). The aim of this cohort study is to analyse different predictors of all-cause mortality and cardiovascular diseases. Parity, reproductive factors, metabolic variables and anthropometric measures were collected primarily for this purpose.²⁶ The cohort is conformed by a weighted random sample of 920 residents of an urban area previously delimited through a geographic information system. All the study participants were examined during the period between 1997 and 1999. The details of the baseline sampling method have been described elsewhere.²⁶⁻²⁷ For purpose of this study we excluded women who had their first pregnancy outside the primary child-bearing ages (<20 or >45 years; n = 12) and men (n = 395). Five women were excluded because they had missing or non-interpretable values for the covariates used in this study. Thus, the total sample was 508 women.

Sociodemographic characteristics were obtained through a home-applied questionnaire. Educational level was evaluated by self-report using the years of formal education reached. For subsequent statistical analysis low education level was defined as less than eight years of full education. Socioeconomic status (SES) was assessed using the scale of minimum income defined by the Chilean Ministry of Planning (MIDEPLAN) expressed in US dollars. An annual income below \$3000 was considered as low SES. In addition, we considered the marital status and employment as dichotomous variables (married vs unmarried and employed vs unemployed respectively).

Health risk conditions were evaluated through medical examination. Three serial measurements of systolic and diastolic pressure were performed to diagnose arterial hypertension (AHT) according to the criteria proposed by the Seventh Joint National Committee (JNC VII).²⁸ Fasting blood samples were obtained to determine blood glucose and lipid profile. Dyslipidaemia was defined accordingly to the cut-off values proposed by the National Cholesterol Education Program (NCEP).²⁹ Type 2 diabetes (T2DM) was diagnosed using a glucose tolerance test in subjects with plasma glucose level ≥ 110 mg/dl.³⁰ Smoking was measured using the number of cigarettes smoked per day, and alcohol consumption was assessed with a questionnaire in Spanish "Escala breve del bebedor adulto (EBBA)" ("Guidelines to assess the adult drinker") validated in Chile to identify heavy drinkers.³¹⁻³² The information about parent's obesity was self-reported.

Gynaeco-obstetric background was obtained by trained health professionals. The variables compiled were number of pregnancies, obstetric deliveries, miscarriages, use of birth control pill (BCP), menopause status, use of postmenopausal hormone replacement therapy (HRT) and birth weight of the biggest child. Fetal macrosomia was defined as birth weight greater than 4000 g. In addition, history of gestational diabetes and hypertension during pregnancy was assessed. Parity was classified as 0 through >6 based on self-reported number of live births. Few women reported parity of >6 (88th percentile; 61 women, with a range of parity of 7 through 18); therefore, women with parity >6 were recoded as having parity of six. Parity was treated as a continuous variable in multiple lineal regression models, whereas in logistic regression models it was treated as binary (parous vs nulliparous) and categorical (parity = 0 through parity ≥ 6).

All anthropometric measurements, including weight, height, waist and hip circumference, were carried out according to a standard protocol by previously trained medical staff at the local health centre of San Francisco de Mostazal. Study participants were evaluated in underwear and barefoot in the standing position. Waist circumference was measured halfway between the lowest costal edge and the ipsilateral iliac crest. Hip circumference at the level of maximum prominence of the buttocks in the lateral view in the standing position. Weight and height were measured using a calibrated physician scale to the nearest 0.1 kg and height-rod to the nearest 0.2 cm respectively. All the measurements were assessed twice and an average of these two measures was used.

It has been previously shown that the sample of the SFP presents a similar demographic composition to the distribution of the San Francisco de Mostazal population and a comparable risk profile with the participants of the National Health Survey.²⁷⁻³³ Thus, for statistical analysis purpose, the sample was weighted by age and sex based on local census data. Differences in prevalence rates were analysed with the Z-test. Means of anthropometric measurements were analysed through ANOVA with a post hoc Bonferroni test to assess differences between groups of parity. Multiple regression models were constructed for continuous values of BMI, WC, WHR and WHtR, estimating β -coefficients in five blocks of additive covariates: unadjusted (model 1), and adjusted by age in years (model 2), sociodemographic characteristics (model 3), health risk conditions (model 4) and gynaeco-obstetric factors (model 5 or full model). To explain the variance of each model the change in the multiple coefficient correlation (R) and the coefficient of determination (R²) was evaluated. Multicollinearity diagnostic tests were carried out by variance inflation factor (VIF) using SPSS v13.0. In general, it is considered that a VIF greater than 10 roughly indicates statistically significant problem of multicollinearity.³⁴⁻³⁵

Since populations may differ in the level of risk associated with a particular anthropometric marker, it is not advisable to identify universally applicable risk thresholds.³⁶⁻³⁸ Therefore, we used anthropometric measures according to specific cut-off points based on optimal sensitivity and specificity for detecting one or more cardiovascular and metabolic risk factors in the population under study.³⁹ These values were: BMI ≥ 28.4 kg/m²; WC ≥ 87.7 cm; WHR ≥ 0.84 and WHtR ≥ 0.55 (see table S1 on the *JECH* website). From these cut-offs points, we investigated the association between parity and different anthropometric measures of obesity through odds ratio (OR) computed by non-conditional logistic regression models.

RESULTS

The descriptive characteristics of the population under study are presented in table 1. Nulliparous women were younger than parous women, showing similar frequencies of low SES, smoking and prevalence of T2DM. Parous women showed greater prevalence of low education, unemployment, married status, smoking cessation, AHT, dyslipidaemia, obese parents, menopause status and use of BCP and HRT. Frequency of alcohol consumption was greater in nulliparous women. Mean values of BMI, WC and WHtR but not WHR showed a trend to be higher with increasing parity (fig 1). The ANOVA test showed that the statistic means of BMI, WC, WHR and WHtR were different between groups (p<0.001). The post hoc Bonferroni test showed that nulliparous women exhibited smaller values in all the anthropometric measures compared with parous women.

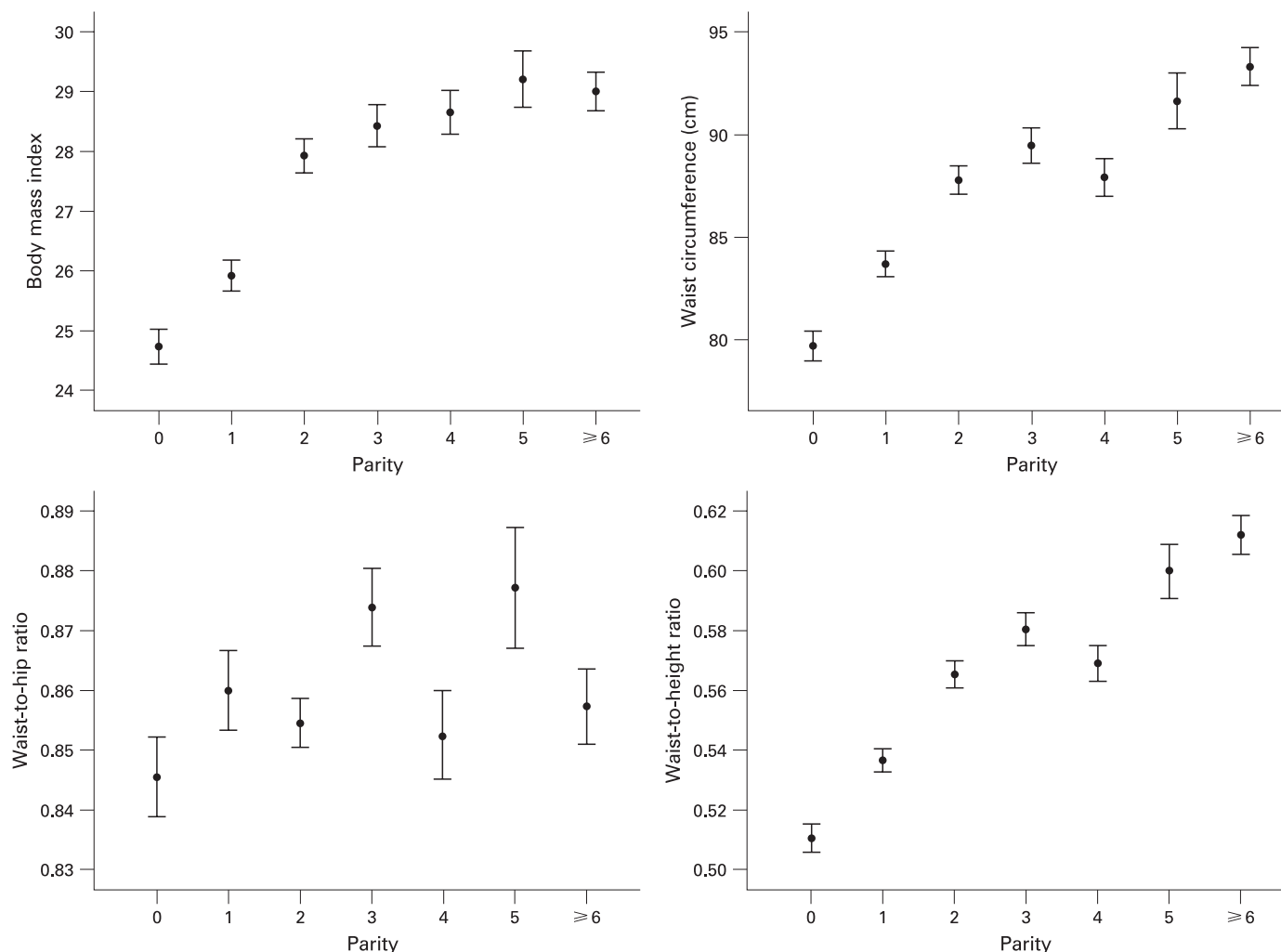


Figure 1 Means values of different anthropometric measures of adiposity with parity increase in a cross-sectional sample of Chilean-Hispanic women. Error bars represents 95% confidence intervals.

Table 2 presents the results of the multiple regression analysis from models 1 to 5 which considered parity as a continuous variable. There was a statistically significant gain in each model of additive covariates. Model 5, which included parity and 19 covariates, explained 20% of BMI variance, 21% of WC, 4% of WHR and 27% of WHtR variance. Age showed the greatest VIF in the final model reaching a maximum value of 3.27, because it was correlated with most of the covariates. The remaining variables (parity included) showed values around 1.03 to 2.19. When the covariates were added and then removed one-to-one in the full model, the β -coefficients and p values did not change significantly, which corroborates a non-statistical effect of multicollinearity. The change in β -coefficient values for BMI associated with parity decreased from 0.74 to 0.47 when age was incorporated and additionally decreased to 0.19 in the model adjusted for all the covariates. Thus, for a woman with an average stature of 155 cm, a weight gain of 0.46 kg per each child was estimated. The unadjusted β -coefficient for WC was 2.09 cm decreasing as far as 0.34 cm for each child in model 5. The β -coefficients for WHR and WHtR were amplified by 100 in order to make values more easily interpretable. An inverse correlation was observed for WHR, which was not statistically significant ($\beta = -0.17$; $p = 0.07$). WHtR and parity showed a positive correlation ($\beta = 0.15$; $p < 0.05$). An increase of 0.25 cm per each child was estimated for WC, expressed as a percentage

of the stature for a woman of 155 cm. The exclusion of outliers for parity, BMI, WC, WHR and WHtR did not change these results.

Table 3 shows the comparison of the β -coefficients of parity with the values of 19 covariates and their relation with anthropometric measurements mutually adjusted (that is, model 5). A direct correlation between BMI, WC and WHtR with age, low education, employment, marital status, smoking cessation, AHT, T2DM, dyslipidaemia, parent's obesity, fetal macrosomia and hypertension during pregnancy was observed. An inverse correlation was also found between age of menarche and BMI, WC and WHtR. WHR showed a direct correlation with age, low education, daily smoking, smoking cessation, parental obesity and use of BCP. In contrast to the other anthropometric measurements, the WHR did not show any statistically significant correlations with AHT, T2DM, dyslipidaemia, fetal macrosomia and hypertension during pregnancy. The β -coefficients observed for the explanatory variables mentioned above were of greater magnitude than the parity coefficients.

Table 4 shows crude and adjusted OR with 95% confidence intervals (CI) for parous vs nulliparous women and the 19 covariates considering population-specific cut-offs of BMI, WC, WHR and WHtR. Crude ORs showed a strong association between all obesity anthropometric markers and parity.

Table 1 Descriptive characteristics of Chilean-Hispanic women from a weighted random sample of 508 women (weighted sample size of 6512 women) of the San Francisco Project study

Variable	All 508 (6512)	Nulliparous 92 (1172)	Parous 416 (5340)
General characteristics			
Age (years)†	39.4 (16.4)	29.9 (14.7)	40.1 (15.1)*
Education (years)†	8.1 (4.1)	10.9 (3.5)	7.7 (4.1)*
Education <8 years (%)	42.3	13.8	48.8*
Annual income <\$3000 (%)	43.8	45.1	43.5
Employed (%)	22.9	35.8	20.1*
Married (%)	66.9	13.7	78.5*
Habits			
Cigarettes/day (only smokers)†	9.7 (8.9)	12.6 (12.2)	8.9 (7.8)
Smokers (%)	25.3	25.4	25.3
Never smoked (%)	33.1	42.5	30.5*
Smoking cessation (%)	41.6	31.0	43.9*
Alcohol consumption (%)‡	8.5	12.7	7.6*
Cardiovascular profile			
Heart rate (beats/min)†	75.7 (10.9)	76.9 (12.1)	75.5 (10.6)
Systolic pressure (mm Hg)†	126.0 (21.2)	120.0 (22.2)	127.3 (20.7)
Diastolic pressure (mm Hg)†	79.0 (12.1)	74.9 (11.9)	79.9 (11.9)
Hypertension (%)	29.7	17.0	32.4*
Metabolic profile			
Fasting blood glucose (mg/dl)†	95.7 (15.9)	88.7 (12.0)	97.4 (19.1)
Type 2 diabetes (%)	5.1	6.7	4.4
Dyslipidaemia (%)¶	18.8	9.0	20.9*
Hereditary factors			
Obese parents (%)	30.2	25.6	31.1*
Gynaeco-obstetric profile			
Menarche (age)†	12.73 (2.24)	11.7 (3.7)	12.9 (1.6)*
Use of birth control pills (%)	9.3	2.3	10.8*
Nulliparous (%)	17.8	–	–
Pregnancies§	3.13 (0.04)	0.11 (0.01)	3.79 (0.04)*
Parity§	2.84 (0.04)	–	–
Miscarriages§	0.50 (0.01)	1.18 (0.12)	0.44 (0.01)*
Newborn weight (g)†	3605 (612)	–	–
Fetal macrosomia (%)††	20.2	–	–
Gestational diabetes (%)	2.9	1.2	3.3
Hypertension during pregnancy (%)	22.6	1.2	27.3*
Menopause status (%)	21.3	8.4	24.3*
Use of HRT (%)	6.8	0.1	8.3*
Anthropometry			
WC (cm)†	86.8 (13.1)	79.7 (12.1)	88.3 (12.8)*
HC (cm) †	101.3 (11.9)	94.7 (11.9)	102.8 (11.3)*
WHR†	0.85 (0.09)	0.85 (0.11)	0.86 (0.10)
WHtR†	0.56 (0.09)	0.51 (0.08)	0.57 (0.09)*
Weight (kg) †	65.7 (12.7)	60.5 (12.4)	66.8 (12.5)*
Height (cm) †	154.9 (6.2)	156.4 (6.2)	154.6 (6.2)
BMI (kg/m ²) †	27.4 (5.2)	24.7 (5.0)	27.9 (5.1)*
Cut-off points of obesity			
BMI ≥28.4 kg/m ² (%)	40.3	20.7	44.6*
WC ≥87.7 cm (%)	46.6	30.3	50.1*
WHR ≥0.84 (%)	52.1	42.7	54.1*
WHtR ≥0.55 (%)	49.3	30.0	53.4*

WC, waist circumference; HC, hip circumference; WHR, waist-to-hip ratio; WHtR, waist-to-height ratio; BMI, body mass index; HRT, postmenopausal hormone replacement therapy.

*p<0.001 for difference between parous vs nulliparous women.

†Values are means (SD).

‡Heavy drinker.

¶NCEP criteria for lipid profile.²⁹

§Values are means (SE).

††Birth weight greater than 4000 g.

Nevertheless, after adjusting for age, sociodemographic factors, health risk conditions and gynaeco-obstetric background the association remained only for BMI. All the measures of

abdominal obesity associated with parous women showed ORs between 0.57 and 0.96 (values refer to the smallest and largest confidence interval observed for WC and WHtR,

Table 2 Multiple regression models for the association between parity and anthropometric measures of adiposity

Model	R	R ²	β for parity	SE for β	VIF	p Value
BMI						
1	0.28	0.08	0.74	0.03	1.00	0.001
2	0.30	0.09	0.47	0.04	1.63	0.001
3	0.33	0.11	0.31	0.05	1.98	0.001
4	0.39	0.15	0.34	0.05	2.05	0.001
5	0.45	0.20	0.19	0.05	2.19	0.001
WC						
1	0.31	0.10	2.09	0.08	1.00	0.001
2	0.37	0.14	0.97	0.10	1.63	0.001
3	0.40	0.16	0.47	0.11	1.98	0.001
4	0.44	0.19	0.52	0.11	2.05	0.001
5	0.46	0.21	0.34	0.11	2.19	0.001
WHR						
1	0.04	0.00	0.22	0.06	1.00	0.001
2	0.08	0.01	-0.03	0.08	1.63	0.001
3	0.15	0.02	-0.25	0.09	1.98	0.001
4	0.18	0.03	-0.20	0.09	2.05	0.001
5	0.19	0.04	-0.17	0.09	2.19	0.001
WHtR						
1	0.35	0.12	1.59	0.05	1.00	0.001
2	0.45	0.20	0.57	0.07	1.63	0.001
3	0.47	0.22	0.23	0.07	1.98	0.001
4	0.50	0.25	0.27	0.07	2.05	0.001
5	0.52	0.27	0.15	0.07	2.19	0.001

BMI, body mass index; WC, waist circumference; WHR, waist-to-hip ratio; WHtR, waist-to-height ratio.

Covariates model 1: None.

Covariates model 2: Age.

Covariates model 3: Age, education <8 years, income <\$3000, employed, marital status.

Covariates model 4: Age, education <8 years, income <\$3000, employed, marital status, daily smoker, smoking cessation, heavy drinker, hypertension, type 2 diabetes, dyslipidaemia, parent's obesity.

Covariates model 5: Age, education <8 years, income <\$3000, employed, marital status, daily smoker, smoking cessation, heavy drinker, hypertension, type 2 diabetes, dyslipidaemia, parent's obesity, menarche, use of birth control pill, fetal macrosomia, hypertension during pregnancy, gestational diabetes, menopause status, use of postmenopausal hormone replacement therapy.

Table 3 Beta-coefficients for anthropometric measures of adiposity on parity, socioeconomic variables, health behaviour and metabolic risk factors

	BMI (kg/m²)			WC (cm)			WHR*			WHtR*		
	β	SE	p Value	β	SE	p Value	β	SE	p Value	β	SE	p Value
Constant	27.01	0.38	0.001	80.92	0.96	0.001	83.06	0.80	0.001	49.87	0.62	0.001
Parity	0.19	0.05	0.001	0.34	0.11	0.002	-0.17	0.09	0.071	0.15	0.07	0.041
Age (years)	0.02	0.01	0.001	0.15	0.02	0.001	0.06	0.01	0.001	0.15	0.01	0.001
Education <8 years	0.69	0.15	0.001	2.94	0.37	0.001	3.15	0.31	0.001	2.03	0.24	0.001
Income <\$3000	0.35	0.13	0.006	0.30	0.31	0.341	-0.01	0.26	0.969	0.75	0.20	0.001
Employed	1.05	0.14	0.001	1.87	0.36	0.001	0.09	0.30	0.760	1.37	0.23	0.001
Married	1.38	0.16	0.001	3.05	0.39	0.001	-1.01	0.33	0.002	2.06	0.25	0.001
Daily smoker	0.29	0.16	0.071	0.70	0.40	0.079	1.63	0.33	0.001	0.20	0.26	0.433
Smoking cessation	0.47	0.14	0.001	1.28	0.35	0.001	0.93	0.29	0.001	0.59	0.23	0.009
Heavy drinker	0.19	0.22	0.373	-0.99	0.54	0.066	-1.60	0.45	0.001	-0.23	0.35	0.519
Hypertension	0.58	0.15	0.001	2.49	0.38	0.001	-0.35	0.32	0.277	0.63	0.25	0.010
Diabetes	2.16	0.28	0.001	4.75	0.69	0.001	-0.71	0.58	0.218	3.20	0.45	0.001
Dyslipidaemia	1.69	0.19	0.001	3.08	0.47	0.001	0.65	0.39	0.094	2.19	0.30	0.001
Parent's obesity	1.03	0.13	0.001	2.51	0.33	0.001	1.49	0.28	0.001	1.29	0.21	0.001
Menarche	-0.34	0.03	0.001	-0.63	0.07	0.001	-0.05	0.06	0.354	-0.39	0.04	0.001
Use of birth control pill	-0.76	0.21	0.001	-0.20	0.53	0.708	2.14	0.45	0.001	-0.24	0.34	0.488
Fetal macrosomia†	2.45	0.17	0.001	3.58	0.42	0.001	-0.05	0.36	0.898	2.05	0.27	0.001
Hypertension during pregnancy	0.77	0.15	0.001	0.92	0.37	0.013	-0.54	0.31	0.081	0.76	0.24	0.001
Gestational diabetes	0.54	0.36	0.136	1.49	0.91	0.100	-0.94	0.76	0.215	1.53	0.59	0.009
Menopause status	-0.13	0.21	0.541	-1.25	0.53	0.017	-1.46	0.44	0.001	-0.41	0.34	0.233
Use of HRT	0.58	0.24	0.015	0.28	0.60	0.639	-0.03	0.50	0.955	0.56	0.39	0.150

BMI, body mass index; WC, waist circumference; WHR, waist-to-hip ratio; WHtR, waist-to-height ratio; HRT, postmenopausal hormone replacement therapy.

*β-coefficient amplified by 100.

†Birth weight greater than 4000 g.

respectively). In the multivariate logistic model, covariates that showed statistically significant associations with all anthropometric measures of adiposity were age, low education, marital status (married), daily smoker, AHT, T2DM, dyslipidaemia and fetal macrosomia. Smoking cessation and hypertension during pregnancy showed an association with BMI, WC and WHtR, but not with WHR. The greater ORs observed were for low education, T2DM, dyslipidaemia and fetal macrosomia. Finally, parity was analysed as a categorical variable to assess the presence of a dose-response gradient in the cross-sectional association between parity and anthropometric obesity markers (table 5). After multivariate adjustments a positive association gradient was observed with BMI, but not with WC, WHR and WHtR. The exclusion of outliers of parity and/or anthropometric measurements did not modify these results.

DISCUSSION

This study found that parity shows a lineal relation with BMI after controlling for sociodemographic characteristics, health risk conditions and gynaeco-obstetric factors in Chilean-Hispanic women. Nevertheless, this impact is modest, estimating an increment of 0.46 kg per each child. These findings are not surprising and are in agreement with previous publications.¹¹⁻¹⁵ In the Stockholm Pregnancy and Weight Development Study (SPAWN), after 15 years of follow-up, an increase of 0.5 kg per each pregnancy was found.¹²⁻¹⁵ We estimated that parous women have higher BMI than nulliparous women after controlling for age differences and 18 potential confounders. Moreover, we observed a dose-response gradient in the cross-sectional association between parity and BMI, which suggests that weight gain would not be restricted only to the first pregnancy, as some studies have indicated.⁵⁻¹⁴

Only a limited number of studies have evaluated the relation between parity and abdominal adiposity measures.¹⁴⁻⁴⁰⁻⁴¹ The Coronary Artery Risk Development in Young Adults (CARDIA) study established that parous women present greater values of WC and WHR compared to nulliparous women.¹⁴⁻⁴⁰ Recently, in the Third National Health and Nutrition Examination Survey (NHANES III) an increasing parity in women was associated with a relative decrease in hip circumference and an increase in WC after controlling for age and BMI.⁴¹ Virtually no studies assessing the association of parity with WHtR have been reported. In the Chilean context, only a recent prospective study reports that WHtR is a better predictor of cardiovascular risk than BMI, WC and WHR.⁴² In the present study, we expected to find a cross-sectional association between parity and all the anthropometric measurements of abdominal obesity; however, this hypothesis was not corroborated. Although we observed a statistically significant relation of parity with WC and WHtR after adjusting for age and potential confounders, its real impact was so small that it can be considered negligible. An inverse association with WHR was observed, but it was not statistically significant, suggesting that anthropometric measurements are not interchangeable; in fact, when we used dichotomous population-specific cut-offs of WC, WHR and WHtR, parous women exhibited a smaller probability of presenting with abdominal obesity than nulliparous women. In contrast with BMI, anthropometric measures of abdominal obesity did not show a dose-response gradient. This suggests that parity can increase adiposity in women but not necessarily following an abdominal pattern. These findings are important because regional obesity has been associated with the majority of obesity-related metabolic complications.⁴³ Whether parity exerts any protective role in the distribution of adipose tissue in

women is a conjecture that requires further investigation using a prospective design. Even though multiparity has been related to a slightly higher risk of general and cardiovascular mortality⁴⁴⁻⁴⁵ it is possible that this association is not mediated by parity itself. On the other hand, potential confounders could be playing an important role in the relation between parity, abdominal obesity, metabolic complications and mortality.

Recent reviews show that the correlation between parity and weight gain is intertwined with numerous factors.¹¹⁻¹² More than 30 confounders have been identified,¹⁷ but a lack of uniformity in including them has characterised many cross-sectional studies³⁻⁵⁻⁴¹⁻⁴⁶⁻⁶⁵ (see table S2 on *JECH* website). In fact, in this study we observed a significant association of BMI with 15 covariates of a total of 19 potential confounders. Factors such as ethnicity, education level, economic status, marital status, employment, age of menarche, smoking, smoking cessation, alcohol consumption, use of HRT, physical activity, dietary intake and other postpartum behaviours have been identified as predictors of greater weight gain after pregnancy.³⁻⁵⁻¹¹⁻¹³⁻¹⁷ Nevertheless, the real impact of these factors and other health risk conditions in the relation of parity with anthropometric measurements of abdominal adiposity has not been investigated. In our study, practically the totality of the cross-sectional association of parity with WC, WHR and WHtR was explained by age, low education, marital status, smoking, smoking cessation, AHT, T2DM, dyslipidaemia and especially, by fetal macrosomia, a strong indicator for the pre-existence of overweight and abdominal obesity.⁶⁶⁻⁶⁹ Moreover, all of these factors had a higher impact over the anthropometric measures than parity. On the other hand, some of these associations can be highly population-specific. For example, in the NHANES III study, unmarried women exhibited greater risk of obesity than married women.⁵ In our study, married women had greater probability of overweight and abdominal obesity.

A recent study that analysed data from many countries concluded that the relation between parity and overweight is influenced by household wealth and national development.²³⁻²⁴ From this perspective, Chile is a middle income developing country (GNI per capita of \$5000) with persisting important social and health inequalities.⁷⁰⁻⁷¹ In addition, in the last decades Chile has experienced a dramatic decrease in fertility rates (present value of 2.1) and parity has diminished as a result of an intensive family planning programme characterised by broad access to contraceptive methods.⁷² However, obesity is running in an opposite direction with a dramatic increase in prevalence (present value of 27% for BMI ≥ 30 kg/m²), particularly of abdominal obesity in women with low SES and/or low education level.³³ Therefore, it is conceivable that parity has little or no influence on the present epidemic of obesity in Chilean women. Furthermore, from the biological perspective of recursive causality⁷³ it is tempting to propose an opposite conjecture. Numerous scientific studies corroborate the observation that abdominal obesity is associated with several endocrine alterations in a "vicious circle", including reverse causality between abdominal adiposity, insulin resistance and reproductive disorders, such as hyperandrogenism, PCOS and decreased fertility.¹⁹⁻²² Abdominal obesity is an important abnormality in patients with hypersensitivity and/or hyperactivity of the hypothalamo-pituitary-adrenal (HPA) axis.⁷⁴ Other endocrine abnormalities associated with visceral obesity such as diminished production of sex steroids and growth hormones may be derived from malfunction of the HPA axis, causing an excessive release of corticotrophin-releasing hormone and cortisol, which favours abdominal adipose tissue

Table 4 Crude and multivariate odds ratio for parity, age and 18 dichotomous covariates using population specific cut-offs for body mass index (BMI), waist circumference (WC), waist-to-hip ratio (WHR) and waist-to-height ratio (WHtR)

	Odds ratio (95% CI)							
	BMI ≥ 28.4 kg/m ²		WC ≥ 87.7 cm		WHR ≥ 0.84		WHR ≥ 0.55	
	Crude	Multivariate	Crude	Multivariate	Crude	Multivariate	Crude	Multivariate
Parous vs nulliparous	3.08 (2.64 to 3.59)*	1.42 (1.18 to 1.72)*	2.32 (2.02 to 2.66)*	0.69 (0.57 to 0.84)*	1.59 (1.39 to 1.81)*	0.78 (0.65 to 0.94)*	3.27 (2.86 to 3.75)*	0.79 (0.65 to 0.96)*
Age (years)	1.03 (1.03 to 1.04)*	1.02 (1.01 to 1.02)*	1.04 (1.04 to 1.05)*	1.03 (1.02 to 1.03)*	1.03 (1.03 to 1.03)*	1.02 (1.02 to 1.03)*	1.06 (1.06 to 1.07)*	1.03 (1.02 to 1.03)*
Low education† (yes/no)	2.25 (2.03 to 2.49)*	1.47 (1.30 to 1.67)*	3.68 (3.32 to 4.10)*	2.46 (2.17 to 2.80)*	2.28 (2.06 to 2.53)*	1.53 (1.35 to 1.74)*	4.08 (3.65 to 4.56)*	2.48 (2.18 to 2.82)*
Low income‡ (yes/no)	1.20 (1.09 to 1.33)*	1.08 (0.97 to 1.22)	1.10 (1.00 to 1.22)	0.88 (0.79 to 1.00)	1.22 (1.10 to 1.35)*	1.09 (0.97 to 1.22)	1.23 (1.11 to 1.36)*	1.06 (0.94 to 1.19)
Employed (yes/no)	0.88 (0.78 to 0.99)*	1.24 (1.09 to 1.42)*	0.80 (0.71 to 0.90)*	1.17 (1.02 to 1.33)*	0.75 (0.67 to 0.84)*	0.92 (0.81 to 1.04)	0.82 (0.73 to 0.92)*	1.14 (0.99 to 1.30)
Married (yes/no)	2.15 (1.92 to 2.41)*	1.28 (1.11 to 1.47)*	2.39 (2.14 to 2.67)*	1.43 (1.23 to 1.65)*	1.73 (1.56 to 1.92)*	1.20 (1.04 to 1.38)*	2.82 (2.53 to 3.14)*	1.49 (1.29 to 1.73)*
Daily smoker (yes/no)	0.93 (0.83 to 1.04)	1.16 (1.00 to 1.35)	0.88 (0.79 to 0.99)*	1.23 (1.06 to 1.42)*	1.21 (1.08 to 1.35)*	1.48 (1.28 to 1.70)*	0.81 (0.73 to 0.91)*	1.29 (1.11 to 1.49)*
Smoking cessation (yes/no)	1.11 (1.00 to 1.22)	1.16 (1.02 to 1.32)*	1.15 (1.04 to 1.27)*	1.25 (1.10 to 1.43)*	0.89 (0.81 to 0.99)	1.03 (0.91 to 1.16)	1.25 (1.13 to 1.39)*	1.47 (1.28 to 1.68)*
Heavy drinker (yes/no)	1.07 (0.89 to 1.28)*	1.23 (1.01 to 1.50)*	1.03 (0.86 to 1.23)	1.18 (0.96 to 1.44)	1.09 (0.91 to 1.30)	1.07 (0.89 to 1.30)	0.98 (0.82 to 1.17)	1.10 (0.90 to 1.35)
Hypertension (yes/no)	2.04 (1.83 to 2.28)*	1.16 (1.01 to 1.33)*	2.78 (2.49 to 3.11)*	1.38 (1.20 to 1.58)*	2.11 (1.89 to 2.36)*	1.49 (1.30 to 1.70)*	3.87 (3.41 to 4.39)*	1.26 (1.09 to 1.44)*
Diabetes (yes/no)	2.84 (2.25 to 3.59)*	2.33 (1.80 to 3.01)*	3.10 (2.42 to 3.97)*	2.20 (1.66 to 2.91)*	2.42 (1.89 to 3.10)*	1.73 (1.32 to 2.26)*	2.99 (2.27 to 3.95)*	1.90 (1.43 to 2.53)*
Dyslipidaemia (yes/no)	2.42 (2.07 to 2.82)*	1.84 (1.56 to 2.17)*	2.10 (1.80 to 2.45)*	1.39 (1.16 to 1.65)*	2.18 (1.86 to 2.56)*	1.78 (1.50 to 2.12)*	3.09 (2.58 to 3.71)*	1.46 (1.22 to 1.75)*
Parents obesity (yes/no)	1.29 (1.16 to 1.44)*	1.28 (1.14 to 1.44)*	1.12 (1.01 to 1.25)*	1.10 (0.97 to 1.24)	1.30 (1.17 to 1.45)*	1.37 (1.22 to 1.54)*	1.13 (1.02 to 1.26)*	1.01 (0.90 to 1.15)
Menarche¶ (yes/no)	1.19 (1.03 to 1.36)*	1.16 (1.00 to 1.34)	1.08 (0.94 to 1.24)	1.10 (0.95 to 1.29)	0.73 (0.64 to 0.84)*	0.77 (0.66 to 0.89)*	1.25 (1.08 to 1.44)*	1.01 (0.86 to 1.19)
Birth control pill (yes/no)	0.76 (0.64 to 0.91)	0.80 (0.66 to 0.98)*	0.73 (0.62 to 0.87)*	0.82 (0.68 to 1.00)	1.02 (0.86 to 1.21)	1.11 (0.92 to 1.33)	0.76 (0.64 to 0.90)*	0.64 (0.53 to 0.78)*
Fetal macrosomia§ (yes/no)	2.26 (1.97 to 2.60)*	1.92 (1.66 to 2.23)*	2.31 (2.00 to 2.66)*	1.71 (1.46 to 2.00)*	1.56 (1.36 to 1.80)*	1.22 (1.10 to 1.42)*	2.44 (2.08 to 2.86)*	1.69 (1.44 to 1.99)*
AHT during pregnancy (yes/no)	1.62 (1.44 to 1.83)*	1.62 (1.42 to 1.86)*	1.56 (1.38 to 1.76)*	1.63 (1.42 to 1.88)*	0.78 (0.69 to 0.88)*	0.72 (0.63 to 0.82)*	1.22 (1.07 to 1.38)*	1.55 (1.35 to 1.79)*
Gestational diabetes (yes/no)	0.69 (0.51 to 0.93)*	0.58 (0.42 to 0.81)*	1.31 (0.98 to 1.76)	1.26 (0.91 to 1.75)	1.14 (0.85 to 1.53)	1.28 (0.93 to 1.76)	1.45 (1.05 to 2.01)*	0.92 (0.66 to 1.27)
Menopause status (yes/no)	1.97 (1.75 to 2.23)*	0.78 (0.64 to 0.94)*	2.85 (2.51 to 3.23)*	0.78 (0.64 to 0.95)*	2.03 (1.80 to 2.30)*	0.76 (0.62 to 0.92)*	4.26 (3.67 to 4.95)*	0.93 (0.76 to 1.14)
HRT (yes/no)	1.50 (1.24 to 1.83)*	1.04 (0.85 to 1.29)	1.69 (1.38 to 2.05)*	1.04 (0.84 to 1.29)	1.00 (0.83 to 1.22)	0.71 (0.58 to 0.88)*	1.47 (1.20 to 1.81)*	1.02 (0.82 to 1.27)

AHT, arterial hypertension; HRT, postmenopausal hormone replacement therapy.

*p < 0.05.

†Education < 8 years.

‡Income < \$3000.

¶ < 12 years.

§Fetal macrosomia (birth weight greater than 4000 g).

Table 5 Crude, age-adjusted and multivariate-adjusted odds ratio for parity and different anthropometric measures of obesity

	Odds ratio (95% CI)		
	Crude	Age adjusted	Multivariate*
BMI ≥ 28			
Parity = 0	1.00	1.00	1.00
Parity = 1	1.72 (1.42 to 2.09)	1.46 (1.20 to 1.78)	1.11 (0.89 to 1.37)
Parity = 2	2.60 (2.17 to 3.11)	2.11 (1.75 to 2.54)	1.53 (1.22 to 1.91)
Parity = 3	3.11 (2.56 to 3.78)	2.37 (1.94 to 2.90)	1.65 (1.31 to 2.09)
Parity = 4	4.57 (3.70 to 5.64)	3.10 (2.48 to 3.88)	2.01 (1.55 to 2.61)
Parity = 5	5.78 (4.50 to 7.43)	3.72 (2.85 to 4.85)	2.90 (2.15 to 3.92)
Parity ≥ 6	4.60 (3.76 to 5.62)	2.32 (1.82 to 2.95)	1.37 (1.03 to 1.82)
WC ≥ 87			
Parity = 0	1.00	1.00	1.00
Parity = 1	1.22 (1.02 to 1.46)	0.93 (0.77 to 1.12)	0.64 (0.52 to 0.79)
Parity = 2	1.48 (1.26 to 1.75)	1.04 (0.87 to 1.24)	0.56 (0.45 to 0.70)
Parity = 3	3.11 (2.59 to 3.73)	2.00 (1.65 to 2.43)	1.06 (0.84 to 1.34)
Parity = 4	2.43 (2.00 to 2.97)	1.28 (1.03 to 1.60)	0.52 (0.40 to 0.68)
Parity = 5	4.57 (3.57 to 5.86)	2.24 (1.72 to 2.93)	1.07 (0.78 to 1.45)
Parity ≥ 6	6.30 (5.15 to 7.72)	2.13 (1.66 to 2.73)	0.85 (0.63 to 1.14)
WHR ≥ 0.84			
Parity = 0	1.00	1.00	1.00
Parity = 1	0.89 (0.75 to 1.06)	0.70 (0.59 to 0.84)	0.68 (0.56 to 0.83)
Parity = 2	1.38 (1.18 to 1.61)	1.01 (0.86 to 1.20)	0.80 (0.65 to 0.99)
Parity = 3	1.98 (1.66 to 2.36)	1.34 (1.11 to 1.62)	1.06 (0.85 to 1.33)
Parity = 4	1.69 (1.40 to 2.06)	0.98 (0.79 to 1.21)	0.74 (0.57 to 0.96)
Parity = 5	2.35 (1.85 to 2.99)	1.27 (0.98 to 1.65)	1.13 (0.84 to 1.53)
Parity ≥ 6	2.85 (2.35 to 3.44)	1.12 (0.88 to 1.43)	0.88 (0.66 to 1.17)
WHtR ≥ 0.55			
Parity = 0	1.00	1.00	1.00
Parity = 1	1.59 (1.33 to 1.89)	1.16 (0.96 to 1.39)	0.80 (0.65 to 0.99)
Parity = 2	1.69 (1.43 to 2.00)	1.10 (0.92 to 1.31)	0.63 (0.50 to 0.79)
Parity = 3	3.40 (2.83 to 4.08)	1.99 (1.63 to 2.42)	1.02 (0.81 to 1.30)
Parity = 4	2.80 (2.29 to 3.42)	1.28 (1.03 to 1.60)	0.49 (0.37 to 0.64)
Parity = 5	6.01 (4.64 to 7.78)	2.54 (1.92 to 3.36)	1.20 (0.87 to 1.66)
Parity ≥ 6	6.82 (5.56 to 8.36)	1.81 (1.40 to 2.33)	0.62 (0.46 to 0.84)

BMI, body mass index; WC, waist circumference; WHR, waist-to-hip ratio; WHtR, waist-to-height ratio.

*Adjusted by age, education <8 years, income <\$3000, employed, marital status, daily smoker, smoking cessation, heavy drinker, hypertension, type 2 diabetes, dyslipidaemia, parents obesity, menarche, use of birth control pill, fetal macrosomia, hypertension during pregnancy, gestational diabetes, menopause status, use of postmenopausal hormone replacement therapy.

accumulation.⁷⁵ Furthermore, increased secretions of androgens and decreased secretion of oestrogens in abdominally obese women might be a consequence of HPA hyperactivity as result of socioeconomic and psychosocial stress, unhealthy lifestyles and traits of depression and anxiety.^{19 74–78} Therefore, the increase in abdominal obesity in Chilean women, especially in those with low SES, might have a negative impact on parity through neuroendocrine alterations. If this is the case the reduced population fertility seems to be acting in synergy with family planning strategies. Although this hypothesis, that some have called “the civilisation syndrome”,¹⁹ addresses the null or inverse association between parity and abdominal obesity, it does not suggest per se the existence of any protection against the components of metabolic syndrome. To clarify this issue further investigation is needed.

Although the SFP sample should be considered representative of Chilean-Hispanic women (similarly distributed to the ones described in the National Health Survey, with an average weight and height in women of 65.7 kg and 155.6 cm, respectively³³), this study is limited by its cross-sectional design which does not allow inferences about causal implications of the associations or a clear definition of the possible effects of age and cohort composition. On the other hand, no epidemiological study should be expected to contribute more than what is

contained in its design.⁷⁹ Therefore many of our conjectures are speculative and require further investigation using prospective designs in different populations. Although anthropometric data and metabolic risk conditions such as AHT, T2DM and dyslipidaemia were directly measured, sociodemographic factors and gynaeco-obstetric variables were self-reported and may be subject to information bias. Additionally, variables that can potentially modify these results such as dietary intake and physical activity were not available, although it is known that the prevalence of sedentary lifestyle of Chilean women has reached 90.8%.³³ One of the difficulties of including too many variables in the regression models is the statistical problem of multicollinearity. It is important to consider that multicollinearity could be a problem only when covariates may measure the same aspects or phenomena. In our study, multicollinearity was negligible for parity and the other covariates. Even though age unavoidably correlates with most of the covariates, VIF was consistently less than 10, a general threshold to define statistical significance in multicollinearity diagnostic tests.^{34 35} On the other hand, there was a substantial gain in the determination coefficient (R^2) in each model of additive covariates mutually adjusted, which supports a not redundant impact of age in the full model. For this reason, a specific treatment of age (for example, centred or categorical values) was considered unne-

What is already known on this subject

- ▶ Parity has been associated with the development of overweight and obesity in women. Nevertheless, it would have a modest impact and would be intertwined with numerous factors.
- ▶ Currently, there are several anthropometric measurements of obesity. Abdominal adiposity has been identified as a major marker of obesity-related metabolic risk factors.
- ▶ Previous studies have focused on the association of parity with weight gain and body mass index (BMI). Virtually no data are available today about the relation between parity and anthropometric measures of abdominal obesity in Latin American-Hispanic women.

What this study adds

- ▶ After controlling for age differences and 18 potential confounders, we established a cross-sectional linear association between parity and BMI in Chilean-Hispanic women. Nevertheless, the association with anthropometric measurements of abdominal adiposity was negligible after multivariate adjustments.
- ▶ Using population-specific cut-offs for anthropometric measurements of obesity, parous women showed less probability of abdominal obesity than nulliparous women.
- ▶ A dose-response gradient was observed in odds ratios for BMI with increasing parity, but not for anthropometric measurements of abdominal obesity.
- ▶ Parity would increase adipose tissue in women, but not necessarily following an abdominal pattern. Whether parity exerts any protective role in the distribution of adipose tissue and obesity-related metabolic complications is a conjecture that needs further investigation.

necessary. Finally, we should note that in this cross-sectional design it was not possible to determine if weight gain occurred during pregnancy or at any other period of life. Since the prevalence of obesity in the group studied is high, it is possible that some of the women presented with obesity before their first pregnancy and that parity had a smaller impact on their obesity than other factors. Indeed, the associations of anthropometric measures of adiposity with macrosomia suggest the presence of a previous state of overweight as a powerful confounder for parity-related obesity in cross-sectional studies. A recent prospective study showed that some blood components that may cross the placental barrier and are associated with the metabolic syndrome such as dyslipidaemia and increased insulin blood levels are predictors of macrosomia, independently of maternal BMI.⁶⁰ However, as discussed above, we cannot rule out reverse causality between visceral obesity, metabolic alterations and reproductive problems.

In conclusion, this paper addresses challenging issues concerning the association of parity and different anthropometric measures of obesity. Parity seems to modestly influence BMI, but does not seem to be related to WC, WHR or WHtR in Chilean-Hispanic women after controlling for confounders. Whether parity exerts any protective role in the distribution of adipose tissue and obesity-related metabolic complications is a conjecture that needs further investigation.

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