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Association between parental and offspring BMI: results from EPACI Portugal 2012

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Abstract

Objective: To assess the longitudinal association between parental BMI and offspring's BMI, in EPACI Portugal 2012.

Design: Longitudinal study with retrospective collection of children's anthropometry data since birth. Children's anthropometric data were gathered from individual child health bulletins, and parents' anthropometrics were self-reported. Children's and parents' BMI were classified according to WHO cut-offs. Linear mixed models with random intercept and slope for age were applied to quantify the association between parental BMI and children BMI *Z*-score (zBMI).

Setting: EPACI Portugal 2012.

Participants: Representative sample from the Portuguese population (*n* 2230) aged from 12 to 36 months.

Results: 58·9 % of the fathers and 35·6 % of the mothers were overweight (OW) or obese. Prevalence of infants who were, at least, at risk of OW increased from 17·0 % to 30·3 % since birth to 12 months. About half of the mothers with pre-pregnancy OW and obesity (OB) gained gestational weight above the recommendations. The children from mothers with gestational weight gain (GWG) below the recommendations showed a −0·15 SD lower zBMI (95 % CI −0·23, −0·06) in early life, comparing with mothers within GWG recommendations. Children of obese mothers were more likely to present a higher zBMI (0·24 SD, 95 % CI 0·13, 0·35) throughout the first months of life.

Conclusions: A high prevalence of OW and OB was observed in Portuguese young adults and toddlers. Mothers' pre-pregnancy BMI and insufficient GWG had a direct effect on offspring BMI. Early effective interventions are needed in order to prevent the transgenerational transmission of OB.

Keywords
Parents
Offspring
Body mass index
Obesity

Obesity (OB), especially childhood OB, has been growing in recent decades all over the world[1–3]. According to the WHO, 1·9 billion of adults and 41 million of children under the age of 5 years are obese or overweight (OW)[4]. Portugal is not an exception, as the prevalence of OW and OB in adults is 37·5 % and 24·9 %, respectively[5]; moreover, 25 % of the children (<10 years old) and 32·3 % of the adolescents (10–17 years) are OW or obese[5]. Other nationally representative study of 6- to 8-year-old children showed an OW/OB prevalence of 30·7 %[6]. However, toddlerhood is much less studied in Portugal, and until now, no study about OW status in these ages has been conducted.

It is well known that nutrition before conception, as well as during pregnancy and in the first 2 years of life, can influence the long-term nutritional status of the children. Lucas defined this concept as 'programming' in 1998[7]. In fact, different studies show that prenatal and postnatal factors play an important role in the programming of some diseases[8], including future OB[2]. To this regard, the Early Nutrition Project (a multicentre

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and multidisciplinary collaborative research) brought new insights about the impact of early nutritional programming on later life, providing knowledge to invest in practical strategies for OB prevention[9–11].

Regarding prenatal factors, the high pre-pregnancy BMI of parents[12,13], particularly of the mother[14–16], the excessive gestational weight gain (GWG)[16–19] as well as caesarean delivery[20,21] have been described as significant childhood OB risk factors. The association between parental pre-pregnancy and offspring BMI at birth is stronger for mother's BMI than for father's BMI[22]. The most plausible explanation for this difference is fetal overnutrition, since, on the one hand, other influences (such as the major role of mothers in feeding and taking care of the children), which can also impact offspring BMI, are discarded[23] in this period and, on the other hand, father's role has its impact later on in life.

Concerning postnatal factors, high infant birth weight and high infant weight gain during the first years of life are strongly associated with OB risk[14,24]. Children's diet and physical activity[15] and the current parental BMI[25,26] are also associated with OB.

It is also clearly demonstrated that there is an association between parents' and children's OB prevalence, supported by the share of the genetic and of the obesogenic environment[25,27,28].

Currently, it is known that OB shows a tracking from infancy to adulthood[2,9]. Some authors suggest that being OW or obese at the age of 5 years increases the risk of being obese in the future[2]. However, there is a significant lack of knowledge regarding the onset age in which the OB's clinical expression occurs.

Much of the existing evidence was gathered by cross-sectional analysis, based in different studies and in different populations, and there is a lack of studies that explore the influence of parental weight status throughout the first years of life, particularly in European countries. In the particular cases of Southern European countries that present high prevalence of OW[5,6,29,30], it is especially relevant to study the determinants and tracking of OB since earlier ages[31]. Although studies have been conducted in other non-European countries[32,33], the OB prevalence found in adults in such studies was lower than that found in Portugal and therefore the parental influence can be different.

The purpose of this study is to assess the association between parental BMI and offspring's BMI at 0–24 months, in the EPACI Portugal 2012 (Study of the Childhood Feeding Patterns and Growth).

Methodology

Study design and sampling

EPACI Portugal 2012 was a cross-sectional study which purpose was to characterise dietary intake and nutritional status of Portuguese infants and toddlers. Children aged between 12 and 36 months were recruited, and the extraction of anthropometrics from the individual health bulletins, measured since birth, allowed a longitudinal approach of children's growth.

A representative sample of about 1 % of Portuguese toddlers aged 12–36 months (Portugal mainland only) was recruited. Since about 100·000 births per year occurred in Portugal, a sample size of 2000 plus 25 % for refusals was defined. Based on the National Institute of Statistics data, the sample was stratified by Portuguese geographical regions (NUTS II, a territorial classification that divides the country in five areas), in the same proportions of the births.

Considering that the national vaccination plan in Portugal is applied only in the primary health care, the sampling was based on primary health care registries. First, a list with all Portuguese primary health care units was obtained, and then the number of units in each region was randomly selected. This allowed to reach the target number of children for each region, considering that in each unit, twenty children would be assessed. However, some of the health units were very small and had less than twenty eligible children registered. In total, 128 health units were included.

After identifying the health units, respective directors were contacted, invited to participate and asked for the lists of children aged 12–36 months of age registered in their units. In each health unit, twenty-five children were randomly selected and their tutors contacted. If the appointments could not be successfully scheduled in this first selection (e.g. due to incorrect phone numbers, impossibility to participate on the scheduled day, children with chronic disease or severe malformations), more children were randomly selected until twenty-five children were scheduled. Sixty-four percentage of the tutors consented to the scheduling of the assessment and, from these, 72 % were evaluated.

Children's assessment by the research staff occurred on the same day in each health unit.

Data collection

Data on sociodemographic characteristics (including parental age and education level), health history (including diseases, emergencies and hospital admissions) and dietary history (including breast-feeding, artificial formulas, complementary feeding and a FFQ in respect of current dietary intake) were collected by trained interviewers, using a structured protocol in face-to-face, computer-assisted interviews. In Portugal, all children have an individual health bulletin where anthropometrics and health vigilance are registered. Anthropometric information (weight, length/height and head circumference) at birth, 2, 4, 6, 9, 12, 18 and 24 months (depending on each child's age and according to the available measures) was extracted, allowing a longitudinal analysis. These time points were considered since they correspond to those preconised by the National Health System regarding children

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health survey. However, the registries do not always overlap exactly with these ages. As such, for the nutritional status characterisation (zBMI) during the first 2 years of life, we considered an interval of 15 d for the measurement at 2 months, of 30 d for the 4 and 6 months, 45 d for the 9 and 12 months and 60 d for the 18 and 24 months measurement.

Low birth weight was defined as a birth weight lower than 2500 g[34] and macrosomia as a birth weight higher than 3999 g[35,36]. Nutritional status (BMI) of each child, at each age, was characterised and expressed as *Z*-score, using the WHO standard[37], and divided into five categories: underweight (UW) (*Z*-score < -2); normal weight (*Z*-score ≥ -2 and ≤ 1); OW risk (*Z*-score > 1 and ≤ 2); OW (*Z*-score > 2 and ≤ 3) and OB (*Z*-score > 3)[37]. The category designated 'OW + OB' combined the OW and OB categories, which correspond to *Z*-score above 2. The expression 'OW risk onwards' combined the three categories corresponding to a BMI *Z*-score (zBMI) above 1: OW risk, OW and OB.

For the present analysis, we excluded: (a) children with any disease which affects the children's development and growth, such as malformation syndrome, severe cardiac, osteoarticular and chronic disease; and (b) premature (gestational age <37 weeks) or postmature (≥42 weeks) children.

Information on parents' anthropometrics was also collected. Regarding mothers, the pre-pregnancy weight, the gestational age, the GWG and the current weight and height were obtained by self-report. From fathers, the self-reported current weight and height were collected. Posterior telephone contacts were scheduled when the respondents did not know or did not remember some of the information or when they did not have the pregnant bulletin or individual health bulletin at the appointment.

Based on parents' weight and height, BMI was calculated as the ratio between weight (in kg) and the squared height (in metres). BMI was classified according to WHO classification into four categories: UW (BMI $< 18{\cdot}5$ kg/m^2); normal weight (BMI $\geq 18{\cdot}5 \leq 24{\cdot}9$ kg/m^2); OW (BMI ≥ 25 and $\leq 29{\cdot}9$ kg/m^2) and OB (BMI ≥ 30 kg/m^2)[38]. The recommended intervals of GWG were defined for each BMI class according to the Institute of Medicine guidelines[39]. Adequate weight gain range (kg) was 12·5–18 kg in mothers with pre-pregnancy UW; 11·5–16 kg among those who were normal weight; 7–11·5 kg for OW category and 5–9 kg for OB[39]. Weight gain below recommendations was defined as weight gain lower than the minimum value of each interval, and weight gain above recommendations was defined as the weight gain higher than the maximum value of each of the intervals. The variable 'obese mother and father' comprises all children who have obese mother and father, simultaneously.

Only children with complete data (n 1671) concerning child gender, mother age, pre-pregnancy BMI, parental education, parental BMI, weight gain adequacy were considered and included in the linear mixed models. The mean number of measurements was four for each child, and only 6 % of the children had less than three measurements.

Statistical analysis

Prevalence estimates were weighted according to the complex sampling design, considering stratification by Portuguese geographical regions (NUTS III, a territorial classification that divides the country in twenty-eight areas) and cluster effect for the selected Primary Health Care Unit. A level of significance of 5 % was considered. For this analysis, *Statistical Package for the Social Sciences* SPSS® version 24 for Windows® was used.

Linear mixed models with random intercept and slope for age were applied, and the linear regression fixed coefficients were used to quantify the longitudinal association between GWG and children's zBMI and between parental BMI and children's zBMI. All the mixed models were adjusted to mother's age and mother's pre-pregnancy BMI. The mother and father education were also tested but were not included in the final models because they did not present any significant association. Although other potential confounders are referred in the literature, EPACI Portugal 2012 study did not gather these; therefore, this adjustment was not possible. The linear mixed effect models assume missing at random. In this specific analysis, data were not weighted. This analysis was performed using the R package nlme.

Results

A total of 2230 children aged between 12 and 36 months were evaluated in EPACI Portugal 2012. For this analysis, 221 children were excluded: twenty by chronic disease, 178 by prematurity (two had simultaneously a chronic disease and prematurity) and twenty-five because the respective gestational age was not possible to ascertain. A total of 2009 children (n 953; 47·4 % female) were included in this study. From these 2009, 1007 (50·1 %) were aged between 12 and 24 months and 1002 (49·9 %) were older than 24 months. However, the determinants of the children's BMI *Z*-score were analysed only in the 1671 children with complete data for the variables that were included in the analysis.

We compared children included in the analysis (n 1671) with children who did not participate (n 338) regarding gender, mother's age, mother's pre-pregnancy BMI, GWG adequacy, mother's OB, father's OB, and mother's and father's BMI. We only found significant differences, concerning GWG adequacy ($P = 0{\cdot}02$), mother and father BMI ($P < 0{\cdot}01$) and mother age ($P < 0{\cdot}01$).

A very high prevalence of OW/OB in Portuguese young adults ($\overline{X} = 33$ years old for mothers and $\overline{X} = 35$ years old for fathers) was found. At the recruitment, 23·9 % of the mothers and 45·1 % of the fathers were OW; 11·7 % of

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Table 1 Pre-pregnancy nutritional status (BMI), weight gain during pregnancy and weight gain adequacy*

							Weight gain adequacy* (% weighted)			
							<	=	>	
BMI classes	*n*	Pre-pregnancy BMI *n*	% weighted	Weight gain mean (kg)	95 % CI	*P* value	29·4†	37·4†	33·2†	*P* value
Underweight	97	105	5·5	14·9	13·4, 16·3		48·4	26·8	24·8	
Normal weight	1216	1218	62·9	13·6	13·3, 14·0	0·270	34·9	40·7	24·4	<0·001
Overweight	438	431	22·3	12·7	12·0, 13·3		12·0	34·6	53·5	
Obesity	181	179	9·3	8·0	6·8, 9·3		23·1	28·0	49·0	

<, weight gain lower than recommended; =, weight gain as recommended; >, weight gain higher than recommended.
*According Institute of Medicine Recommendations, 2009[38].
†Prevalence in the total sample.
Weight gain range (kg): underweight (12.5–18); normal weight (11.5–16); overweight (7–11.5); obesity (5–9).
The sample was % weighted.

mothers and 13·8 % of fathers were obese. Prevalence of OW plus OB in mothers increased from 31·6 % to 35·6 % between pre-pregnancy and the recruitment for the study (12–36 months after birth). Considering the whole sample, mean BMI before pregnancy was 23·9 kg/m^2 and mean weight gain was 13 kg. Pre-pregnancy nutritional status, weight gain during pregnancy and weight gain adequacy are described in Table 1. In this sample, weight gain during pregnancy was similar among mothers who were UW and those with a normal pre-pregnancy BMI.

GWG means were within recommended intervals[39], except for mothers who were OW before pregnancy, for whom mean GWG was above the interval. The mean of GWG was closer to the inferior limit of the recommended interval in women who were UW or normal weight before pregnancy and was just slightly superior and closer to the superior limit for OW and obese women before pregnancy, respectively (Table 1).

A third (37·4 %) of women had adequate weight gain during pregnancy, while 29·4 % increased weight below and 33·2 % increased weight above the recommendations. When stratified by BMI class, almost 75 % of the mothers who were UW/obese before pregnancy had an inadequate weight gain during pregnancy, whereas in the other BMI classes, the prevalence of inadequate weight gain was lower. About half (53·5 % and 49·0 % of mothers with pre-pregnancy OW and OB, respectively) increased weight above the recommendations during pregnancy, while 48·4 % of mothers who were UW before pregnancy increased weight below the recommendations during pregnancy (Table 1).

Anthropometric characterisation at birth is described in Table 2. The prevalence of infant with low birth weight (*n* 73) and macrosomia (*n* 74) was the same (3·7 %).

Nutritional status from birth until 24 months can be observed in Table 3. The prevalence of infants who are at OW risk onwards almost doubled from birth to 12 months (17·0 % to 30·3 %), and the sum of OW and OB almost tripled in the same interval (2·2 % *v.* 5·8 %). From 12 to 24 months, the increase in the prevalence of the sum of OW and OB was 1·7 %.

The longitudinal association between GWG during pregnancy and zBMI of the children in the first 24 months of life can be observed in Table 4. No interaction between the growth trajectory and sex was observed. Mothers with GWG below the recommendations had a lower progression of the children's zBMI (−0·15 SD, 95 % CI −0·23, −0·06), comparing with mothers with GWG within recommendations. Higher mothers' BMI before pregnancy was associated with a higher progression of children's zBMI (0·03 SD, 95 % CI 0·02, 0·03). There was also an inverse association between maternal age and the progression of children's zBMI (−0·01 SD, 95 % CI −0·01, 0·00). All these associations were independent over time (0·03 SD, 95 % CI 0·03, 0·03). When considering length for age as outcome, a positive association was found regarding weight gain above recommendations, but not with weight gain below the recommendations.

The association between the parents' BMI at the recruitment and the zBMI of children since birth can be observed in Table 5. Children of obese mothers were more likely to have a higher increase in zBMI (0·24 SD, 95 % CI 0·13, 0·35), but fathers' OB had no effect on children's zBMI trajectory (0·08 SD, 95 % CI −0·02, 0·18). When both mother and father were obese, an interaction with time was verified and the children's zBMI grew faster (0·02 SD, 95 % CI 0·00, 0·03). When considering length for age as outcome, the association with mother's OB is not observed.

Discussion

The prevalence of OW/OB among Portuguese adult population is very high (62·4 %)[5], and EPACI shows an alarming increase of OW prevalence in the first 2 years of life. Comparing nutritional status of parents and children, we observed a positive association between mothers' OB and children's zBMI, whereas no association between fathers' OB and children's zBMI was found.

EPACI shows a high prevalence of OW/OB in Portuguese young adults (45·1 % of the fathers and 23·9 % of the mothers are OW and 13·8 % and 11·7 % are

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Table 2 Anthropometry at birth (mean (95 % CI))

	Total	Mean	95 % CI	Female gender	Mean	95 % CI	Male gender	Mean	95 % CI
Weight (g)	*n* 2003*	3247	3227, 3268	*n* 948	3197	3173, 3221	*n* 1055	3292	3255, 3330
Height (cm)	*n* 1996*	48·8	48·7, 49·0	*n* 943	48·4	48·3, 48·5	*n* 1053	49·2	49·0, 49·4
Head circumference (cm)	*n* 1977*	34·4	34·3, 34·5	*n* 937	34·1	34·0, 34·2	*n* 1040	34·7	34·6, 34·8
zBMI (SD)	*n* 1993*	0·08	0·04, 0·13	*n* 941	0·15	0·09, 0·21	*n* 1052	0·02	−0·05, 0·10

*Total does not sum up 2009 due to missing data.The sample was % weighted.

obese, respectively). Nonetheless, these results are slightly lower than those found in the IAN-AF[5]. When comparing the results of both of these studies by gender, similar prevalence was found in men (OW: 41·8 % in IAN-AF *v.* 45·1 % in EPACI; OB: 19·7 % *v.* 13·8 %, respectively), but there were greater differences in women (OW: 31 % in IAN-AF *v.* 23·9 % in EPACI, OB: 23·7 % *v.* 11·7 %, respectively). Notwithstanding, in IAN-AF, anthropometrics were measured, whereas in the present study, these were self-reported. As observed in other studies, when nutritional data are self-reported, it is common to observe an underestimation of the weight[2,9] and this could be the reason for the differences found in women's OW/OB prevalence.

As described in previous studies[40,41], the prevalence of OW in men is always significantly higher than that of women (at least more than 12 %)[40,41], which is in accordance with what we found in our study. This may perhaps be justified with a higher social pressure of body image in women[39].

Mothers' BMI is a strong predictor of the newborn's nutritional status[18,42,43]. In the present study, 31·6 % of women were OW/OB before conception, which is consistent with another Portuguese study (30·1 %)[44]. Furthermore, in women who were UW or with a normal pre-pregnancy BMI, mean weight gain during pregnancy was closer to the inferior limit of Institute of Medicine-recommended guidelines[39], whereas in women who were OW and obese before pregnancy, this was just slightly superior/closer to the superior limit. These results are according to the existing research, which shows that pre-pregnancy nutritional status influences the mother's weight gain during pregnancy[45] and has an impact on future fetal growth and health[46,47]. For instance, if a mother is obese before pregnancy, this will increase the risk of some health complications in the mother/newborn dyad, including fetal macrosomia and later OB[48].

Weight gain during pregnancy also has an impact on the nutritional status of the fetus and on the future health of the child. In the present study, 37·4 % of women showed an adequate weight gain during pregnancy[39], a result similar to another Portuguese study (35·8 %)[49]. However, comparing with the same study, in EPACI, a higher proportion of women gained weight during pregnancy below the recommendations (29·4 % *v.* 22·6 %) and a lower proportion of women had a GWG above the recommendations (33·2 % *v.* 41·7 %).

When stratified by BMI class, EPACI data showed that three quarters of the mothers who were UW and obese before pregnancy had an inadequate weight gain during pregnancy (above or below), which is consistent with the results found by Henriques *et al.*[49]. In the EPACI study, almost half of the mothers who had pre-pregnancy OW and OB increased weight above the recommendations during pregnancy, while very few increased weight below the recommendations. In addition, almost half of UW mothers before pregnancy increased weight below the

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Table 3 Nutritional status characterisation (zBMI)* during the first 2 years of life (*n* (%); CI 95 %)

	Birth *n* 1993	2 months *n* 1593	4 months *n* 1775	6 months *n* 1694	9 months *n* 1747	12 months *n* 1665	18 months *n* 1204	24 months *n* 679
UW								
n	47	51	46	36	15	8	5	3
weighted	2·4	3·2	2·6	2·1	0·8	0·5	0·4	0·4
95 % CI	1·7, 3·3	2·3, 4·4	1·8, 3·6	1·4, 3·2	0·5, 1·5	0·2, 1·0	0·2, 1·0	0·2, 0·9
NW								
n	1607	1356	1480	1398	1299	1152	754	446
weighted	80·6	85·1	83·4	82·5	74·4	69·2	62·6	65·7
95 % CI	78·4, 82·7	83·2, 86·8	81·4, 85·3	80·5, 84·4	71·8, 76·7	66·2, 72·1	59·6, 65·6	61·6, 69·5
OW risk								
n	295	167	217	220	357	408	334	179
weighted	14·8	10·5	12·2	13·0	20·5	24·5	27·7	26·5
95 % CI	13·2, 16·5	8·9, 12·4	10·5, 14·1	11·3, 14·9	18·4, 22·7	22·1, 27·0	25·0, 30·6	23·0, 30·2
OW + OB								
n	44	19	32	40	76	97	111	517
weighted	2·2	1·2	1·8	2·3	4·3	5·8	9·2	7·5
95 % CI	1·6, 3·1	0·7, 1·9	1·2, 2·6	1·7, 3·2	3·4, 5·6	4·5, 7·6	7·6, 11·2	5·1, 10·7

zBMI, BMI *Z*-score; UW, underweight; NW, normal weight; OW risk, overweight risk; OW, overweight; OB, obesity.
*According WHO, 2006[36].
The sample was % weighted.
UW: zBMI < –2; NW: zBMI ≥ –2 e ≤ 1 OW risk: zBMI > 1 e ≤ 2; – OW + OB: zBMI > 2 (WHO Classification).

Table 4 Linear mixed model on children zBMI (SD)* (*n* 1671)

	β	95 % CI	
	Est.	Lower	Upper
Intercept	–0·38	–0·65	–0·12
Weight gain according to the recommendations†	Ref		
Weight gain below recommendations†	–0·15	–0·23	–0·06
Weight gain above recommendations†	0·02	–0·06	0·10
Age (months)	0·03	0·03	0·03
Mother Pre-pregnancy BMI (kg/m^2)	0·03	0·02	0·03
Mother age (years)	–0·01	–0·01	0·00
	σ‡	Corr§	
Intercept	0·57		
Age slope	0·03	–0·02	
Residual	0·69		

zBMI, BMI *Z*-score.
*According to WHO, 2006[36].
†According to Institute of Medicine Recommendations, 2009:[38] Weight Gain Range (kg): underweight (12·5–18); normal weight (11·5–16); overweight (7–11·5); obesity (5–9).
‡Standard deviation of the random effects.
§Correlation between the random effects; with random intercept and slope for age; the model was adjusted to mother age and mother pre-pregnancy BMI.

Table 5 Linear mixed model on children zBMI (SD)* (*n* 1671)

		95 % CI	
	β	Lower	Upper
Mother			
Non obese†	Reference		
Obese†	0·24	0·13	0·35
Age	0·03	0·03	0·03
Father			
Non obese†	Reference		
Obese†	0·08	–0·02	0·18
Age	0·03	0·03	0·03
Mother and father			
Non obese†	Reference		
Obese†	0·12	–0·11	0·34
Age	0·03	0·03	0·03
Non obese† × age	Reference		
Obese† × age	0·02	0·00	0·03

zBMI, BMI *Z*-score.
*According to WHO, 2006[36].
†According to WHO, 2000[37].
With random intercept and slope for age; the model was adjusted to mother age and mother pre-pregnancy BMI.

recommendations during pregnancy. These results are consistent with the study of Deputy *et al.*, in which the UW women had also the highest prevalence of weight gain below the recommendations (39·3 %) and the OW/OB women had the highest prevalence of weight gain above the recommendations (64·1 % and 63·5 %, respectively)[50].

Previous studies showed that excessive weight gain during pregnancy is associated with several adverse pregnancy outcomes, such as large-for-gestational-age births, childhood OW and OB and maternal postpartum weight retention[17,51,52]. In the EPACI study, there was not a significant different trajectory in the zBMI of children whose mothers gained excessive weight during pregnancy, when comparing with offspring of women with adequate GWG. However, it should be noted that, in this analysis, the included sample had nearly 10 % more mothers with adequate GWG, compared with the sample that was excluded. If a higher proportion of mothers with an excessive GWG during pregnancy had been included, it is possible that the association between mothers who gained gestational weight above the recommendations and children's BMI would be significant.

On the other hand, women who gain weight below the recommendations are more likely to have small-for-gestational age children[50,52,53], and EPACI results were in line with

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this. In fact, we found that mothers with GWG below recommendations showed a lower children's zBMI trajectory, comparing with mothers with GWG within recommendations, which can protect from later childhood OB. EPACI children had a mean birth weight very similar to the Generation XXI study[54], but an inferior prevalence of low birth weight when compared with another Portuguese study (3·7 % *v.* 6·8 %)[55]. Regarding macrosomia, EPACI found precisely the same prevalence observed by Moreira *et al.*[55]. Anthropometry at birth, in particular birth weight, reflects the intrauterine environment[56], more specifically the genetic background and nutritional and environmental experiences and is also a predictor of future health.

A high prevalence and an early expression of OW and OB in Portuguese children were found in EPACI. In fact, the OW risk onwards prevalence almost doubled from birth to 12 months (17 % to 30·3 %), the greatest increase occurring at 9 months, which corresponds to the first follow-up after the recommended beginning of complementary feeding (at 5–6 months). Similar data were observed in a Chinese cohort[33], where this increase was even more evident, almost tripling from birth to 12 months (10 % – 30 %). This very significant increase of the OW risk prevalence in a short period of time suggests that environment plays a more important role than genetic[57,58]. The developmental origins of health and diseases, also known as Barker Hypothesis, explain the association between environmental events in early life and the risks in future health through epigenetic modifications[59,60]. According to the current knowledge, the first years of life are a period of vulnerability for the OB persistency and future disease programming[61]. Literature shows an intergenerational transmission of OB[62], justified by the sharing of the genetic and obesogenic environment[25,27,28] and also by behavioural factors and intrauterine environment[19].

A significant association between parental weight status and offspring's BMI seems to exist. In fact, according to the EPACI data, an association between mothers' BMI and children's zBMI was visible and mothers' OB showed a significant effect on a higher zBMI in children. In line with Portuguese results, the referred study from a Chinese cohort[33] showed that pre-pregnancy maternal BMI had a significant effect on offspring's BMI. Similarly to EPACI results, a birth cohort study in Malaysia, following mothers throughout pregnancy, demonstrated that a high pre-pregnancy BMI may increase offspring's BMI in the first year of life[32].

Fathers' OB had no effect on offspring's BMI. In fact, according to the results presented above, there was an association between maternal BMI and offspring's zBMI trajectory, but the same does not happen when paternal BMI is analysed. These results can be justified by the influence of intrauterine mechanisms and experiences that occur during pregnancy, such as the overnutrition hypothesis, which predict OB in the future[63]. Curiously, when the presence of OB in both parents was considered, there was no effect on children's zBMI trajectory, but an interaction effect with time was observed.

Contrary to our results, a study in an Australian cohort observed an association between paternal and offspring's BMI. This study, conducted at the age of 14 years, also demonstrated a higher association between maternal and offspring's BMI than between paternal and offspring's BMI[64]. Despite we are comparing nutritional status in older ages of the children, it is known that parental OB shows a tracking until adult age. In addition, and relating to early ages, the Chinese cohort[33] also showed that maternal BMI had more impact in children's growth in the first 24 months and that father's BMI had less impact, even though it had influence in children's growth. A study from Malaysia[32], which focus was the first 12 months of life, corroborates EPACI data and also found an association between mothers' pre-pregnancy BMI and children's BMI.

Notwithstanding, our findings are not in consonance with the results found by Smith[57], based in the Avon Longitudinal Study of Parents and Children and by Fleten *et al.*[65], based in a Norwegian Mother and Child Cohort Study, which showed a similar association between BMI of both parents and offspring childhood BMI. These authors argue that these results probably indicate that the association between maternal and offspring's BMI is more related to the share of the genetic and environment factors rather than with the influence of intrauterine programming[57,65], since if the intrauterine environment had a relevant influence, this association between mothers and offspring would be superior and stronger than the paternal association[57].

In a pandemic OB context, where the influence of the first months of life seems unequivocal, it is crucial to revisit the cut-off points and to consider as OW zBMI above 1 in children from birth to the age of 5 years, since current recommendations only consider OW above *Z*-score 2 at this age range[37]. However, the OW cut-off point of zBMI above 1 for children under 5 years of age is already considered by some authors, such as Mei *et al.*[33], and is already used in clinical practice. Furthermore, in older children, above 5 years of age[66], this same cut-off point of zBMI above 1 classifies children as OW. Thus, for all reasons mentioned above, EPACI results considered as OW risk onwards children with zBMI above 1.

A direct comparison of our results with those of other studies is more difficult because of differences in the birth cohorts, the ages at which examinations were performed, the methods used for the measurement of adiposity, the definitions of OB and the reference populations used to adjust measurements for age and sex. Nevertheless, the influence of maternal BMI on offspring OW and OB since early ages seems unquestionable.

Moreover, we tested the association of parental BMI in the two first years of life, whereas in some of the referred studies, older ages were used. Nonetheless, it should be noted that in the above referred studies, the maternal pre-pregnancy BMI was used, while in our study, the current mother BMI was used.

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Concerning the strengths of this study, one is the national representative sample ($n > 2000$) and the fact that this was the first longitudinal assessment in a national representative sample. Another strength of our study relates to the protocol for data collection, which was prepared by specialists (paediatricians and nutritionists) and the data being gathered in face-to-face interviews by trained interviewers following standardised procedures. In addition, our results brought more knowledge about OB prevention and the influence of pre-pregnancy BMI and GWG on offspring's OW and consequently in long-term health, which reinforced the need for early prevention (since preconception).

In contrast, some weaknesses must also be considered. The parental BMI data were self-reported and, as pointed out, when weight is self-reported, it is usual to have underestimation[2,9]. It is possible that stronger effects could appear if measured anthropometrics were considered. Furthermore, children's data were taken from individual health bulletins, and some inconsistencies were found in data. In order to minimise inconsistent data, an exhaustive and logical verification of the database was made, before the data treatment was performed.

Conclusions

A high prevalence of OW and OB was observed in Portuguese young adults. An early onset and a high prevalence of OW and OB were observed in Portuguese toddlers.

Older mothers and mothers with low GWG showed a lower offspring's zBMI trajectory throughout the first 2 years of life. High pre-pregnancy BMI and obese mothers showed a higher offspring's zBMI trajectory. Almost half of OW and obese women increased their gestational weight above the recommendations.

Urgent and early, ideally before conception, effective interventions are needed in order to prevent the transgenerational transmission of OB. It is imperative to implement surveillance plans and changes of attitude, especially in primary health care and particularly concerning women in fertile age and pregnant.

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