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Comparison between type A and type B early adiposity rebound in predicting overweight and obesity in children: a longitudinal study

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Abstract

Early adiposity rebound (EAR) predicts paediatric overweight/obesity, but current approaches do not consider both the starting point of EAR and the BMI trajectory. We compared the clinical characteristics at birth, age 3–5 and 6–8 years of children, according to the EAR and to its type (type A/type B-EAR). We assessed the children's odds of being classified as overweight/obese at age 6–8 years, according to the type of EAR as defined at age 3–5 years. As part of this two-wave observational study, 1055 children were recruited and examined at age 3–5 years. Antenatal and postnatal information was collected through interviews with parents, and weight and height from the health records. Type A and type B-EAR were defined in wave 1 according to the BMI nadir and the variation of BMI *z*-score between the starting point of the adiposity rebound and the last point on the curve. At 6–8 years (wave 2), 867 children were followed up; 426 (40·4 %) children demonstrated EAR. Among them, 172 had type A-EAR, higher rates of parental obesity ($P < 0{\cdot}05$) and greater birth weight compared with other children ($P < 0{\cdot}001$). Odds for overweight/obesity at 6–8 years, when adjusting for antenatal and postnatal factors, was 21·35 (95 % CI 10·94, 41·66) in type A-EAR children and not significant in type B-EAR children (OR 1·76; 95 % CI 0·84, 3·68) compared with children without EAR. Classification of EAR into two subtypes provides physicians with a reliable approach to identify children at risk for overweight/obesity before the age of 5 years.

Key words: **Early adiposity rebound: Childhood: Obesity: BMI trajectory: Perinatal risk factors**

Due to the high prevalence of paediatric obesity worldwide, despite a trend towards a stabilisation of this epidemic[1], early clinical screening must be improved in a preventive perspective. Amongst other factors, the detection of an adiposity rebound (AR), defined as 'the point at which the BMI increases after its nadir'[2,3], before the age of 5·5 years, named 'early AR'[2,4] (EAR), has been defined as a reliable indicator for future obesity[2,4–6].

Adipose tissue undergoes physiological changes during the early years of life, with a rapid increase during the first year due to the growing size of the adipocytes and then a decrease during the following years, to remain stable until the age of 6 years. Thereafter, body fat shows a second phase of rapid growth, named 'AR', which was first reported and documented by Rolland-Cachera *et al.*[4]. According to these and other authors, there is an association

Abbreviations: AR, adiposity rebound; CUG, catch-up growth; EAR, early adiposity rebound; IOTF, International Obesity Task Force.

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between the age at which AR occurs and adiposity at age 16 years[4,7]. Later studies also documented the importance of EAR, highlighting its association not only with cardiometabolic health development[8] but also with maturation disorders, among others[9,10]. Several studies have investigated the potential risk factors associated with the occurrence of this EAR, particularly focusing on antenatal and postnatal factors[11,12]. For instance, maternal and paternal obesity are now recognised as strong risk factors for accelerated weight growth trajectory[13,14].

Undeniably, the description of EAR and its associated antenatal and postnatal risk factors constituted a major step for the understanding and management of childhood overweight, whose prevalence is still dramatically increasing worldwide[15]. In France, 17 % of children and adolescents aged 6–17 years are overweight, including 4 % who are obese[16]. However, the current definition of the EAR does not seem to be sufficiently discriminating. Accordingly, although Hughes *et al.* reported that 58 % of children identified as having an EAR before the age of 3·6 years were actually overweight at 15 years old, only 22 % of those identified with an EAR by the age of 5 years actually became overweight at 15 years old[17].

In 2006, Rolland-Cachera *et al.*[2] described two profiles of early rebounders, namely 'fat children', who start their EAR at the top of their corpulence chart, and 'lean children', who start their EAR at the bottom of their corpulence chart (using French curves[18]). On the other hand, Thibault *et al.*, in 2010, identified in a French cohort of 1424 children aged 8–9 years that the combination of the criterion 'overweight at 3–4 or 5–6 years' and 'increase in BMI > 1 kg/m^2 between 3–4 and 5–6 years' was a good predictor of the risk of overweight at 8–9 years[19]. Based on these studies, and given that only a modest proportion of children identified with EAR actually become overweight/obese[17], we aimed to refine the definition of EAR using French curves related to the International Obesity Task Force (IOTF) references[18]. Thus, we considered both the BMI *z*-score at the starting point of the EAR (BMI nadir) and the BMI trajectory. Indeed, it remains unclear whether the velocity of the BMI curve during EAR systematically predicts future overweight/obesity or not. Therefore, in order to properly identify children most at risk for future overweight/obesity, we proposed a classification of EAR into two classes, based on both initial BMI and BMI trajectory, namely: type A-EAR and type B-EAR.

The objectives of the present study were: 1) to compare the clinical characteristics at birth, at age 3–5 years and at age 6–8 years of children, according to the presence or absence of EAR and, according to the type of EAR (type A and type B-EAR) and 2) to assess the children's odds of being classified as overweight/obese at age 6–8 years, according to the type of EAR as defined at age 3–5 years. We further hypothesised that children exhibiting a type A or type B-EAR would exhibit similar clinical characteristics at birth, and that exhibiting a type A-EAR at age 3–5 years would be associated with a greater risk of overweight/obesity at age 6–8 years when adjusting for antenatal and postnatal factors, compared with children from the non-EAR group.

Materials and methods

Design and population

This longitudinal, multicentre, observational study was conducted in the Department of Haute Saone, France, among children born between 1 January 2003 and 1 May 2005. As described below, two waves of evaluations were performed: the first at 3–5 years of age and the second at 6–8 years of age. Fig. 1 presents the flow chart of the study.

Wave 1: a total of 1159 French children were enrolled in public and private pre-schools, as part of the annual medical

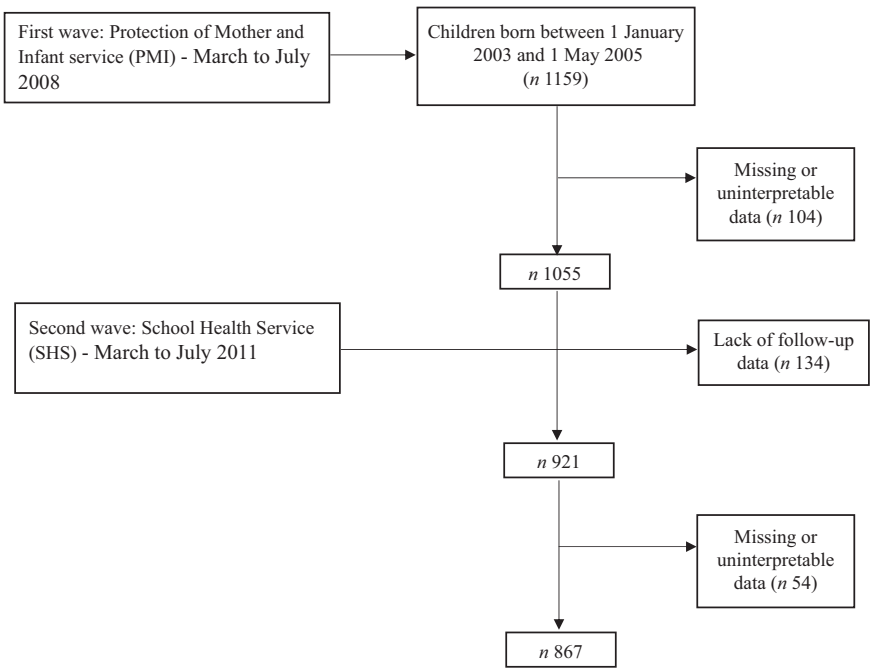

Fig. 1. Flow chart of the study population.

examination at school. Between March and July 2008, data collection including antenatal and postnatal information was collected from the children's health record and from interview with the parents. Anthropometric measurements were performed by health professionals (nurses from the Protection of Mother and Infant service).

Wave 2: 3 years later, between March and July 2011, we performed a second set of measurements similar to those of wave 1, as part of the annual medical examination. In total, 921 children from the initial overall sample completed the second wave of measurements.

During each wave, anthropometric measurements of the children were performed and antenatal and postnatal information were collected during an interview with at least one parent.

We excluded children with secondary obesity such as syndromic, iatrogenic or endocrine obesity and children whose parents failed to attend the initial medical exam.

This study was approved by the Ethics Committee of the University Hospital of Besançon, France (under the number 13/22), by the French national advisory committee for the processing of data in health research (number 14.705) and by the national commission for data protection (number 914632). Legal representatives of participating children were informed of the procedures and provided written informed consent before enrolment of their child in the study.

Antenatal and postnatal information

Data related to birth and the first years of life were retrospectively collected from the children's health record and during the interviews with parents in the first wave:

Gestational age (extremely preterm (<28 weeks), very preterm (28–31 weeks), moderate preterm (32–33 weeks), late preterm (34–36 weeks) and term babies (≥37 weeks)) and body weight status at birth (hypotrophy, eutrophy or macrosomia, respectively, defined as birth weight <10th percentile, 10th–90th percentile or >90th percentile of the reference values) were collected and classified according to French references[20,21]. The mother's smoking status during pregnancy was collected during the interview. Body weight and height of the father and the mother (apart from pregnancy) were self-reported by the parents and used to calculate their BMI. The presence or absence of gestational diabetes and pregnancy-induced hypertension was self-reported by the parents.

Postnatal data concerned how the infant was fed (infant formula feeding or maternal breast-feeding and, if yes, for how long). Body weight and height of the child, taken from the child's health record, at birth and at 3, 9, 12, 24, 36 and 48 months, were collected to establish a graph of BMI.

Anthropometric measurements and body weight determination

Anthropometric measurements were conducted by health professionals during wave 1 and wave 2, as part of the annual medical examination. Body weight was measured to the nearest 0·1 kg using a calibrated scale, and height was determined to the nearest 0·01 m using a standing stadiometer for each child. BMI was calculated as body weight divided by the square of the height in m (kg/m^2). BMI z-score, specific for age and sex, was calculated by the LMS method[22]. Gestational age at birth for birth weight was calculated according to French reference data[20]. Body weight status was classified into one of the three categories according to the IOTF references[22]: thinness (<IOTF-17), overweight ($25 < IOTF \leq 30$) and obesity (>IOTF-30). A classification according to the WHO references[23] was also performed, and the corresponding data are presented in online Supplementary Tables S1–S3.

Determination of the early adiposity rebound. BMI generally peaks during the first year of life and subsequently declines, reaching a minimum around the age of 6 years[2]. The point of minimal BMI value is the start of the AR. Each AR starting before the age of 5·5 years is usually considered as EAR[4]. EAR was evaluated by two investigators: one trained and one untrained, in a blinded fashion, using BMI z-scores and French curves which refer to the IOTF references[18].

Determination of early adiposity rebound type A and type B. EAR was sub-classified into two types, namely: type A-EAR and type B-EAR.

Type A-EAR was defined according to two situations relative to the position of the BMI nadir in relation to the median (corresponding to a BMI z-score = 0 or to the 50th percentile):

1) If the BMI nadir is equal to or above the median (BMI z-score ≥ 0), a type A-EAR is defined when there is an increase of at least 0·5 units in BMI z-score between the starting point of the AR (BMI nadir) and the last point.
2) If the BMI nadir is below the median (BMI z-score < 0), a type A-EAR is defined when there is an increase equal to or greater than 1 unit in BMI z-score between the starting point of the AR (BMI nadir) and the last point.

Type B-EAR was defined according to one situation: If the BMI nadir is below the median (BMI z-score < 0), a type B-EAR is defined when there is an increase greater than 0·5 but lower than 1 unit in BMI z-score between the starting point of the AR (BMI nadir) and the last point (see Fig. 2).

Subjects were thus classified as non-EAR group or EAR group. Within the EAR group, two subgroups were distinguished, namely type A-EAR group and type B-EAR group.

Determination of the catch-up growth. Catch-up growth (CUG) was defined at 2 years when a difference of >0·67 standard deviations was observed between the 24-month BMI z-score and the birth-BMI z-score [11].

Statistical analysis

All statistical analyses were performed with SAS software, version 9.4 (SAS Institute, Inc.). Discrete variables are expressed as numbers and percentages, and continuous variables as mean values and standard deviations unless otherwise stated. Comparisons between non-EAR and EAR groups and between non-EAR, type A-EAR and type B-EAR groups were performed using the χ^2 or Fisher's exact test, Student's t test or ANOVA according to the type of variable. *Post hoc* tests were performed as appropriate (Scheffe's test).

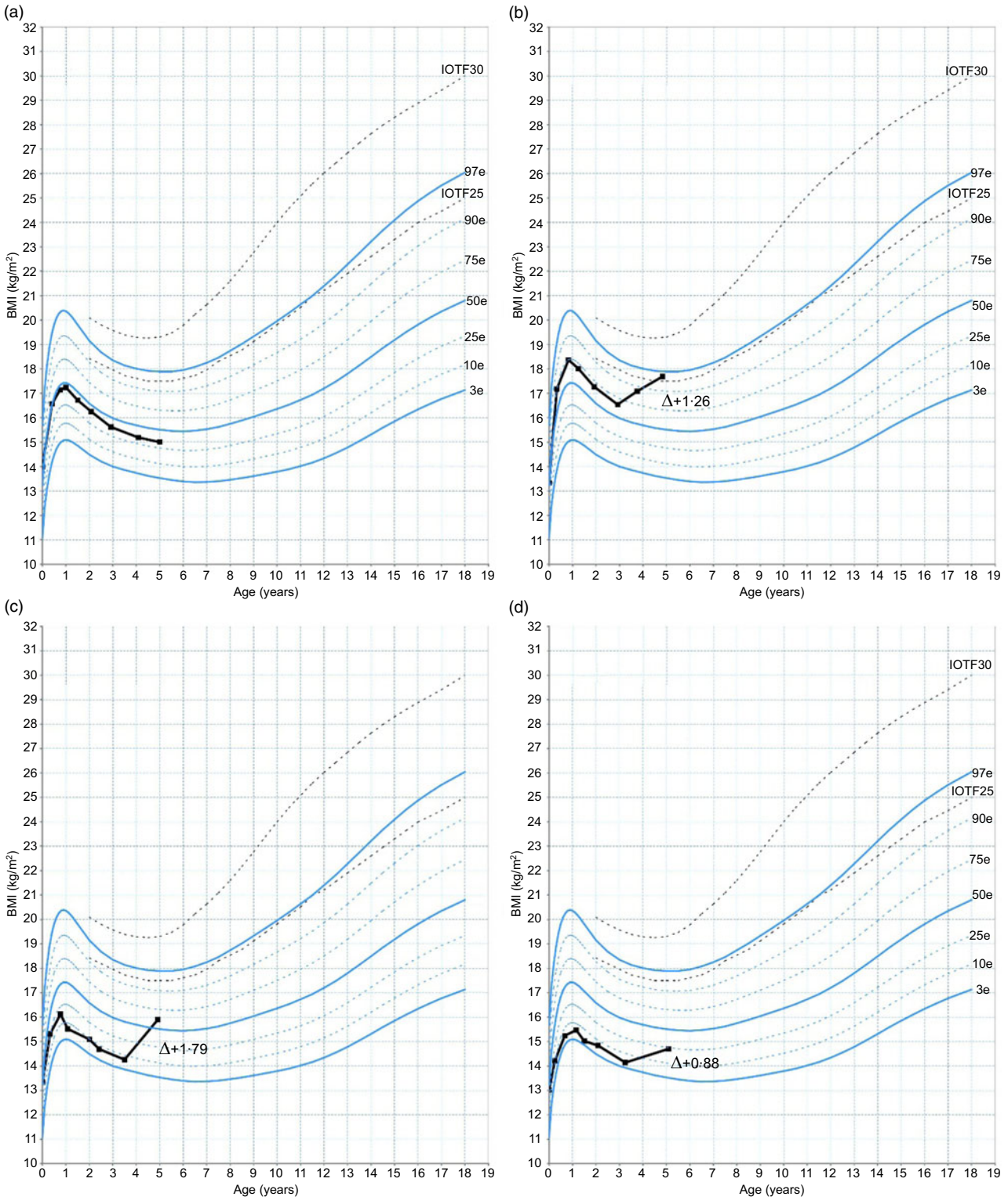

Fig. 2. Examples of non-early adiposity rebound (EAR) and EAR types presented on a French curve, according to the starting point of the EAR and the BMI trajectory. (a) No EAR; (b and c) type A-EAR; (d) type B-EAR. Δ, Difference in BMI *z*-score between last measure and starting point of the EAR; IOTF, International Obesity Task Force.

To determine the predictive factors of overweight and obesity in children, we first performed bivariate logistic analysis. Variables with a level of significance of ≤0·20 in the bivariate analysis were included in the multivariate model, which was analysed with stepwise logistic regression. Interaction effects were sought for all variables included in the model. Model discrimination was assessed by the C-index, which is equal to the AUC. Calibration was assessed by the Hosmer–Lemeshow

goodness-of-fit statistic. All reported P values are two-sided. A P value < 0·05 was considered statistically significant.

Results

Characteristics of the population

In total, 1159 children were enrolled in the study; of these, 104 subjects were excluded from further analysis because of missing data or uninterpretable BMI trajectories. A total of 1055 children (509 girls and 546 boys) with a mean age of 4·4 (SD 0·4) years and a mean BMI of 15·7 (SD 1·5) kg/m^2 were thus retained for analysis. At wave 2, a total of 921 subjects were seen. Fifty-four subjects were excluded, and 867 subjects (422 girls and 445 boys) were retained for final analysis, at a mean age of 7·5 (SD 0·4) years and a mean BMI of 16·5 (SD 2·3) kg/m^2.

Among the 1055 interpreted BMI trajectories, agreement between the two investigators for EAR determination was found for 960 (91 %) children. A third observer was called on to resolve discrepancies for ninety-five subjects.

EAR, determined during the first wave, was observed in 426 children (40·4 % of the population; 225 girls, 201 boys) who constitute the EAR group, while 629 subjects (284 girls and 345 boys) did not exhibit an EAR (non-EAR group, Table 1).

Among the EAR group, 172 children (40·4 %; 93 girls, 79 boys) were identified as presenting type A-EAR and 254 subjects (59·6 %; 132 girls, 122 boys) exhibited type B-EAR (Table 2).

Non-early adiposity rebound and early adiposity rebound groups

Antenatal and postnatal information. Gestational age, size at birth including height, body weight and body weight status at birth were not different between the two groups. Mother's smoking status during pregnancy, BMI of the parents, prevalence of pregnancy-induced hypertension, breast-feeding and gestational diabetes were also similar between groups (Table 1).

Characteristics at age 3–5 years. Children from the EAR group presented higher BMI (P < 0·0001) than children from the non-EAR group. A similar prevalence of CUG was observed in the two groups. In the EAR group, only two children were thin (0·5 %), *v.* 35 (5·6 %) in non-EAR children. Among those with EAR, 57 (13·4 %) children were already overweight and 22 (5·2 %) were obese. Regarding the non-EAR group, 27 (4·3 %) children were overweight with a BMI trajectory at the top of the chart since early infancy, but none was obese (Table 1). Data according to the WHO references are shown in online Supplementary Table S1.

Characteristics at age 6–8 years. Children from the EAR group presented higher BMI (P < 0·0001) than those from the non-EAR group. In the EAR group, no child was thin, while 5 (1·0 %) children from the non-EAR group were thin. In the EAR group, 77 (21·7 %) children were overweight and 35 (9·9 %) were obese. Among the non-EAR group, only 41 (8·0 %) children were overweight and 4 (0·8 %) were obese (Table 1). Data according to the WHO references are shown in online Supplementary Table S1.

Non-, type A- and type B-early adiposity rebound groups

Antenatal and postnatal information. Subjects with type A-EAR had a greater gestational age compared with children from the non-EAR group (P < 0·001) and compared with those from the type B-EAR group (P < 0·001).

At birth, height was greater in the type A-EAR group compared with the type B-EAR group (P < 0·001) and to the non-EAR group (P < 0·001). Birth weight was significantly higher in the type A-EAR group compared with the type B-EAR and non-EAR groups (P < 0·001, Table 2).

Characteristics at age 3–5 years. Age was similar between the three groups. BMI was significantly greater in the type A-EAR group compared with the type B-EAR and the non-EAR groups (P < 0·0001, Table 2).

Rates of CUG were 49 and 40·9 % in the type A-EAR and non-EAR groups, respectively, *v.* 28·5 % in the type B-EAR group (P < 0·0001).

Concerning body weight status, in the type A-EAR group, 57 (33·1 %) children presented overweight and 22 (12·8 %) presented obesity and no child presented thinness. In the type B-EAR group, 2 (0·8 %) children presented thinness and no child presented overweight or obesity (Table 2). Data according to the WHO references are shown in online Supplementary Table S2.

Characteristics at age 6–8 years. Age was similar between the three groups. BMI was higher in the type A-EAR group compared with the type B-EAR and non-EAR groups (P < 0·0001, Table 2).

Concerning body weight status, in the type A-EAR group, 56 (40·0 %) children were overweight and 33 (23·6 %) children presented obesity. In the type B-EAR group, 21 (9·8 %) children were overweight and 2 (0·9 %) children presented obesity. In both groups, no child presented thinness (Table 2). Data according to the WHO references are shown in online Supplementary Table S2.

When combining the type A and type B-EAR groups, 112 children were overweight/obese. 79·5 % of them (n 89) were from the type A-EAR group and 20·5 % (n 23) from the type B-EAR group.

Risk factors for overweight and obesity at age 6–8 years

Bivariate analysis. Gestational age, body weight status at birth (eutrophy, macrosomia and hypotrophy), gestational diabetes and pregnancy-induced hypertension were not associated with overweight/obesity at age 6–8 years.

Risk factors for overweight/obesity at age 6–8 years by bivariate analysis were exposure to prenatal smoking (OR 1·98; 95 % CI 1·22, 3·21), maternal obesity (OR 3·00; 95 % CI 1·82, 4·95), paternal overweight (OR 1·80; 95 % CI 1·20, 2·71), paternal obesity (OR 3·51; 95 % CI 1·99, 6·19) and CUG (OR 2·40; 95 % CI 1·65, 3·50). Risk of overweight/obesity was decreased by breast-

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Table 1. Characteristics of the non-early adiposity rebound (EAR) and EAR groups according to the International Obesity Task Force (IOTF) definition (Mean values and standard deviations; numbers and percentages)

	Non-EAR group		EAR group		
	n	%	*n*	%	*P*
Antenatal and postnatal information					
n	629		426		
Sex (% boys)		54·9		47·2	0·015
Gestational age (weeks)					0·235*
Mean		39·2		39·3	
SD		2·1		2·1	
Gestational age					0·439‡
<28 weeks	2	0·4	3	0·8	
28–32 weeks	7	1·3	4	1·1	
32–36 weeks	34	6·3	16	4·2	
>37 weeks	497	92·0	358	93·9	
Size at birth					
Height (cm)					0·261*
Mean		49·3		49·5	
SD		2·9		2·8	
Body weight (kg)					0·952*
Mean		3·2		3·2	
SD		0·6		0·5	
Body weight status at birth					0·577†
Eutrophy	498	79·7	341	80·4	
Macrosomia	36	5·8	29	6·8	
Hypotrophy	91	14·5	54	12·8	
Mother's smoking status during pregnancy					0·317†
Not smoker	359	80·3	248	83·2	
Smoker	88	19·7	50	16·8	
BMI of the mother					0·476†
<25 kg/m^2	420	72·4	271	69·0	
25–30 kg/m^2	99	17·1	78	19·8	
≥30 kg/m^2	61	10·5	44	11·2	
BMI of the father					0·180†
<25 kg/m^2	313	56·4	192	50·3	
25–30 kg/m^2	192	34·6	151	39·5	
≥30 kg/m^2	50	9·0	39	10·2	
Gestational diabetes					0·515†
No	382	91·8	259	93·2	
Yes	34	8·2	19	6·8	
Pregnancy-induced hypertension					0·620†
No	390	94·4	263	95·3	
Yes	23	5·6	13	4·7	
Breast-feeding					0·443†
No	265	44·0	168	40·7	
<6 months	256	42·4	192	46·5	
≥6 months	82	13·6	53	12·8	
Clinical parameters at age 3–5 years					
Age (years)					0·513*
Mean		4·4		4·4	
SD		0·4		0·5	
BMI (kg/m^2)					<0·0001*
Mean		15·3		16·3	
SD		1·2		1·7	
Catch-up growth					0·197†
No	302	59·1	247	63·3	
Yes	209	40·9	143	36·7	
Body weight status					<0·0001‡
Thinness (<IOTF-17)	35	5·6	2	0·5	
Normal weight (17 < IOTF ≤ 25)	567	90·1	345	81·0	
Overweight (25 < IOTF ≤ 30)	27	4·3	57	13·4	
Obese (>IOTF-30)	0	0	22	5·2	
Clinical parameters at age 6–8 years					
n	513		354		
Sex (% boys)		54·6		46·6	0·021
Age (years)					0·641*
Mean		7·5		7·5	
SD		0·4		0·4	

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Table 1. (*Continued*)

	Non-EAR group		EAR group		
	n	%	*n*	%	*P*
BMI (kg/m^2)					<0·0001*
Mean	15·9		17·4		
SD	1·6		2·9		
Body weight status					<0·0001‡
Thinness (<IOTF-17)	5	1·0	0	0	
Normal weight (17 < IOTF ≤ 25)	463	90·2	242	68·4	
Overweight (25 < IOTF ≤ 30)	41	8·0	77	21·7	
Obese (>IOTF-30)	4	0·8	35	9·9	

* Student's *t* test for quantitative variables for comparison between non-EAR and EAR groups.
† χ^2 Test for qualitative data analysis, for comparison between non-EAR and EAR groups.
‡ Fisher's exact test for qualitative data analysis, for comparison between non-EAR and EAR groups.

Table 2. Characteristics of the non-early adiposity rebound (EAR), type A and type B-EAR groups according to the International Obesity Task Force (IOTF) definition
(Mean values and standard deviations; numbers and percentages)

	Non-EAR group (0)		Type A-EAR group (1)		Type B-EAR group (2)			Dunn's test (*P*)		
	n	%	*n*	%	*n*	%	*P*	1 *v.* 0	1 *v.* 2	2 *v.* 0
Antenatal and postnatal information										
n	629		172		254					
Sex (% boys)		54·9		45·9		48·0	0·046			
Gestational age (weeks)							0·003†	***	***	NS
Mean		39·2		39·8		39·1				
SD		2·0		1·3		2·4				
Gestational age (weeks)							0·088§			
<28	2	0·4	0	0	3	1·3				
28–32	7	1·3	0	0	4	1·8				
32–36	34	6·3	3	2·0	13	5·7				
>37	497	92·0	149	98·0	209	91·3				
Size at birth										
Height (cm)							0·015†	***	***	NS
Mean		49·3		50·0		49·1				
SD		2·9		2·0		3·2				
Body weight (kg)							<0·0001†	***	***	NS
Mean		3·2		3·4		3·1				
SD		0·6		0·4		0·6				
Body weight status at birth							0·070‡			
Eutrophy	498	79·7	143	83·6	198	78·3				
Macrosomia	36	5·8	15	8·8	14	5·5				
Hypotrophy	91	14·5	13	7·6	41	16·2				
Mother's smoking status during pregnancy							0·484‡			
Not smoker	359	80·3	152	84·4	96	81·4				
Smoker	88	19·7	28	15·6	22	18·6				
BMI of the mother							0·026‡			
<25 kg/m^2	420	72·4	92	60·1	179	74·6				
25–30 kg/m^2	99	17·1	38	24·8	40	16·7				
≥30 kg/m^2	61	10·5	23	15·0	21	8·7				
BMI of the father							0·027‡			
<25 kg/m^2	313	56·4	69	45·4	123	53·5				
25–30 kg/m^2	192	34·6	60	39·5	91	39·5				
≥30 kg/m^2	50	9·0	23	15·1	16	7·0				
Gestational diabetes										
No	382	91·8	100	92·6	159	93·5	0·777‡			
Yes	34	8·2	8	7·4	11	6·5				
Pregnancy-induced hypertension							0·737‡			
No	390	94·4	104	96·3	159	94·6				
Yes	23	5·6	4	3·7	9	5·4				
Breast-feeding							0·713‡			
No	265	44·0	63	38·6	105	42·0				
<6 months	256	42·4	79	48·5	113	45·2				
≥6 months	82	13·6	21	12·9	32	12·8				

Table 2. (*Continued*)

	Non-EAR group (0)		Type A-EAR group (1)		Type B-EAR group (2)			Dunn's test (*P*)		
	n	%	*n*	%	*n*	%	*P*	1 *v.* 0	1 *v.* 2	2 *v.* 0
Clinical parameters at age 3–5 years										
Age (years)							0·302†			
Mean		4·4		4·4		4·5				
SD		0·4		0·5		0·4				
BMI (kg/m^2)							<0·0001†	***	***	NS
Mean		15·3		17·8		15·3				
SD		1·2		1·7		0·7				
Catch-up growth							0·0001‡			
No	302	59·1	79	51·0	168	71·5				
Yes	209	40·9	76	49·0	67	28·5				
Body weight status							<0·0001§			
Thinness (<IOTF-17)	35	5·6	0	0	2	0·8				
Normal weight (17 < IOTF ≤ 25)	567	90·1	93	54·1	252	99·2				
Overweight (25 < IOTF ≤ 30)	27	4·3	57	33·1	0	0				
Obese (>IOTF-30)	0	0	22	12·8	0	0				
Clinical parameters at age 6–8 years										
n	513		140		214					
Sex (% boys)		54·6		46·7		46·4	0·070			
Age (years)							0·476†			
Mean		7·5		7·5		7·5				
SD		0·4		0·4		0·4				
BMI (kg/m^2)							<0·0001†	***	***	NS
Mean		15·9		19·5		16·0				
SD		1·6		3·1		1·7				
Body weight status[b]							<0·0001§			
Thinness (<IOTF-17)	5	1·0	0	0	0	0				
Normal weight (17 < IOTF ≤ 25)	463	90·2	51	36·4	191	89·3				
Overweight (25 < IOTF ≤ 30)	41	8·0	56	40·0	21	9·8				
Obese (>IOTF-30)	4	0·8	33	23·6	2	0·9				

* $P < 0·05$, ** $P < 0·01$, *** $P < 0·001$.
† ANOVA for quantitative data analysis and ‡ χ^2 test or § Fisher's exact test for qualitative data analysis for comparison between non-EAR, type A-EAR and type B-EAR groups. *Post hoc* (Scheffe's test) for comparison between groups.

feeding (duration of at least 6 months), compared with infants receiving infant formula (OR 0·42; 95 % CI 0·21, 0·84).

Concerning EAR, children in the EAR group had an increased risk of overweight/obesity at age 6–8 years (OR 4·81; 95 % CI 3·29, 7·03) compared with those in the non-EAR group. In subgroup analyses, compared with those in the non-EAR group, children in the type A-EAR group had an increased risk of overweight/obesity at age 6–8 years (OR 18·15; 95 % CI 11·45, 28·76), whereas those in the type B-EAR group did not have a significantly increased risk (OR 1·25; 95 % CI 0·74, 2·13) of overweight/obesity at age 6–8 years (Table 3).

Results of the bivariate analyses according to the WHO definitions are presented in online Supplementary Table S3.

Stepwise multivariate analysis. Risk factors for overweight/obesity at age 6–8 years by stepwise multivariate logistic regression analysis were mother's smoking status during pregnancy (OR 2·31; 95 % CI 1·15, 4·65), maternal obesity (OR 2·84; 95 % CI 1·33, 6·06), paternal overweight (OR 2·29; 95 % CI 1·12, 4·28), paternal obesity (OR 2·81; 95 % CI 1·12, 7·06) and CUG (OR 2·18; 95 % CI 1·23, 3·88) and type A-EAR (OR 21·35; 95 % CI 10·94, 41·66). The C-index for the model was 0·846 (Table 3). According to the WHO definitions, the risk factors for overweight/obesity at age 6–8 years by stepwise multivariate logistic regression analysis were macrosomia (OR 5·68; 95 % CI 2·33, 13·86), mother's smoking status during pregnancy (OR 2·73; 95 % CI 1·46, 5·12), maternal obesity (OR 2·59; 95 % CI 1·28, 5·24), paternal overweight (OR 2·14; 95 % CI 1·26, 3·63), type A-EAR (OR 13·96; 95 % CI 7·45, 26·18) and type B-EAR (OR 3·39; 95 % CI 1·94, 5·92). The C-index for the model was 0·831 (online Supplementary Table S3).

Discussion

The prevalence of paediatric overweight and obesity remains alarming worldwide[15], accompanied by early metabolic[24], sleep[25,26] and functional[27] impairments, amongst others, calling for effective preventive and treatment strategies.

In the present study, 40·4 % of the children were classified as having an EAR. This prevalence is in line with other studies, such as Hughes *et al.*[17], who reported that 27·2 % of British children experienced EAR, and Ip *et al.*[12], who found a prevalence of about 58·3 % in Latino children.

Although the occurrence of EAR is recognised as a predictive marker for future paediatric overweight and obesity, there is a need to reinforce our screening methods and to identify accurate and reliable predictors. In the present study, only 26·3 % of children identified as having an EAR actually became overweight/obese at age 6–8 years. This low prevalence highlights the need

Table 3. Association between risk factors and overweight and obesity at age 6–8 years*†
(Odds ratios and 95 % confidence intervals)

	Unadjusted OR	95 % CI	*P*	Final model adjusted OR	95 % CI	*P*
Gestational age (weeks)			0·746	Did not enter		–
<32	0·72	0·16, 3·27				
32–36	0·75	0·31, 1·81				
>37	1					
Body weight status at birth			0·075	Did not enter		–
Eutrophy	1					
Macrosomia	1·53	0·81, 2·90				
Hypotrophy	0·59	0·33, 1·07				
Mother's smoking status during pregnancy			0·006			0·003
Not smoker	1			1		
Smoker	1·98	1·22, 3·21		2·31	1·15, 4·65	
BMI of the mother (kg/m^2)			<0·0001			0·037
<25	1			1		
25–30	1·36	0·85, 2·17		0·81	0·37, 1·78	
≥30	3·00	1·82, 4·95		2·84	1·33, 6·06	
BMI of the father (kg/m^2)			<0·0001			0·012
<25	1			1		
25–30	1·80	1·20, 2·71		2·29	1·12, 4·28	
≥30	3·51	1·99, 6·19		2·81	1·12, 7·06	
Gestational diabetes			0·699	Did not enter		–
No	1					
Yes	1·16	0·55, 2·48				
Pregnancy-induced hypertension			0·823	Did not enter		–
No	1					
Yes	0·90	0·34, 2·37				
Breast-feeding			0·048			0·580
No	1					
<6 months	0·87	0·60, 1·27				
≥6 months	0·42	0·21, 0·84				
Catch-up growth			<0·0001			0·008
No	1			1		
Yes	2·40	1·65, 3·50		2·18	1·23, 3·88	
EAR			<0·0001	Did not enter		–
No	1					
Yes	4·81	3·29, 7·03				
Type of rebound			<0·0001			<0·0001
No EAR	1			1		
Type A-EAR	18·15	11·45, 28·76		21·35	10·94, 41·66	
Type B-EAR	1·25	0·74, 2·13		1·76	0·84, 3·68	

*C-index = 0·846, Hosmer–Lemeshow = 0·7109.
† The candidate variables for the multivariate model were those with a level of significance of ≤0·20 in the bivariate analysis.

to refine the definition of EAR, in order to detect only children at high risk of later overweight/obesity.

Based on our clinical expertise, the present work proposes a sub-classification of EAR, considering not only early occurrence of EAR but mainly its velocity and the shape of its trajectory. Indeed, it seems important to differentiate between children whose BMI *z*-score is equal to or below 0 at the occurrence of the EAR and who show an alarming increase in their growth curve (type A); and those who have a lower BMI *z*-score with a low-to-moderate increase in their curve (type B). In this context, we aimed to compare the clinical characteristics at birth, age 3–5 and 6–8 years of children, according to the presence or absence of EAR, and according to the type of EAR (type A and type B-EAR), and to assess the children's odds of being classified as overweight/obesity at age 6–8 years, according to the type of EAR defined at age 3–5 years.

Regarding the clinical characteristics at birth, children with type B-EAR exhibited slightly lower height and body weight compared with children from the type A-EAR group and children from the non-EAR group, despite similar gestational age and maternal smoking status during pregnancy. Although these children could not be considered as small for gestational age, we could nonetheless expect to observe a substantial prevalence of CUG in this group, since 70–90 % of small for gestational age children usually experience this catch-up[28–30]. Surprisingly, only 28·5 % of children with type B-EAR experienced CUG, compared with almost half of the type A-EAR group. This is of particular importance since CUG is recognised as a risk factor for overweight/obesity in childhood, as previously reported[31,32]. Regarding the antenatal and postnatal factors, we found that the prevalence of both maternal and paternal obesity was greater in children with type A-EAR compared with the other children, which confirms the strong relation between parental obesity, EAR and risk of later overweight/obesity of the offspring.

When assessing the children's risk factors of being classified with overweight/obesity at age 6–8 years, some of the previously reported early-life risk factors for obesity during childhood, such as gestational age, body weight status at birth, gestational

diabetes and pregnancy-induced hypertension, were not found to be associated with weight gain in later childhood. Moreover, multivariate logistic regression analysis did not allow us to observe a protective effect of breast-feeding. However, we did not assess whether breast-feeding was exclusive or not, which can explain why we did not observe a protective effect of breast-feeding against overweight/obesity. While previous studies questioned the potential relationships between these factors and the development of paediatric obesity, results remain conflicting and further evidence is needed[33–36].

As expected[11,37], parental body weight status represents a strong risk factor for paediatric overweight/obesity and children born to obese mothers or fathers, respectively, have a 2·84- or 2·81-fold increase in the risk of exhibiting overweight/obesity at the age of 6–8 years. Parental obesity may enhance the risk of obesity not only through genes involved in homoeostatic regulation, appetite suppression and the control of energy balance[38] but also through shared familial characteristics such as food preferences[39] or physical inactivity[40].

We found that exposure to prenatal smoking was associated with an increased risk of overweight/obesity in children (adjusted OR 2·31; 95 % CI 1·15, 4·65). Other authors have reported that infants whose mothers smoked during pregnancy had not only a preserved ponderal index[41] but also more fat mass and less fat-free mass than infants of non-smokers[42,43]. Maternal smoking may also result in lower fetal growth which, in turn, is counterbalanced by faster postnatal weight gain[44]. This quicker postnatal weight gain is associated with the development of overweight[45] and, in accordance with the literature[11,37], we found that CUG represents a risk factor for overweight/obesity at age 6–8 years.

Finally, we observed that children in the EAR group had a 4·8-fold increase in the risk of overweight/obesity at age 6–8 years compared with those in the non-EAR group. AR, which is the point at which the BMI increases after its nadir[2,3], is considered 'early' in the French paediatric population when it occurs before the age of 5·5 years[2,4]. Since its first definition in 1984 by Rolland-Cachera *et al.*[4], numerous studies have confirmed its strong relationship with later overweight/obesity[2,4–6], as well as with cardiometabolic health[46]. Over the last decade, some authors[47,48] have suggested that EAR was a risk factor for later fatness only because it identifies children whose BMI was already high at the occurrence of the rebound, without considering the shape and trajectories of the curve. However, as emphasised by Rolland-Cachera & Péneau[49] and by Rolland-Cachera & Cole[50], BMI at the age of the AR is usually close to the 50th centile of the reference population (corresponding to a BMI *z*-score close to 0), showing that an EAR is not necessarily associated with high BMI values at rebound. Although its reliability to precisely determine later overweight and obesity remains to be confirmed, EAR is a major tool for physicians to target children at risk.

As such, by differentiating type A from type B-EAR according to the nadir of BMI and the trajectory of the children's BMI curves, we found that children found to have type A-EAR show a 21-fold increase in the risk of overweight and obesity at age 6–8 years compared with children from the non-EAR group, after adjusting for mother's smoking status during pregnancy, for CUG and for the body weight of the mother and father. In children defined with type B-EAR, the risk for overweight and obesity was not increased, compared with children from the non-EAR group. Additionally, overweight and obesity were diagnosed at age 6–8 years among 51·7 % children with type A-EAR, while only 9·1 % were found to be overweight/obese in the type B-EAR group.

In this way, among the 426 children initially identified with EAR, only 40·4 % of them (i.e. those who were classified as type A-EAR) were considered as being at high risk for future overweight/obesity. This supports the utility of this classification, which is more sensitive for the identification of children at risk of overweight and obesity, and provides physicians with an easier way to efficiently screen for and manage paediatric obesity. As a reminder, we observed 91 % agreement between the two investigators for EAR determination.

Importantly, it must be emphasised that while children presenting a type B-EAR did not exhibit a risk for overweight/obesity compared with children from the non-EAR group, we cannot ensure that they are metabolically healthier than children with type A-EAR. Rolland-Cachera & Péneau[49] reported that the two main BMI trajectories, respectively, 'lean' and 'fat' children experiencing EAR may be associated with different health risks. Indeed, overweight children with high birth weight followed by high lean body mass may have lower health risks[49,51], while rapid weight gain after AR in thin infants is associated with insulin resistance and CHD[49,52].

This study has some limitations that deserve to be considered when interpreting our results. First, antenatal and postnatal factors were collected following interviews with at least one parent, which might limit the accuracy of the information collected (e.g. gestational diabetes, pregnancy-induced hypertension, BMI of the parents, etc.). Additionally, breast-feeding and smoking status during the pregnancy were also self-reported by the parents. Regarding breast-feeding, we cannot ensure that it was exclusive and this lack of information may explain why breast-feeding did not appear to be protective against overweight and obesity in multivariate analysis.

Second, assessment of fat mass and fat-free mass would have been relevant in order to refine the AR assessment[53], which was not possible due to the design of the study.

Third, we evaluated EAR at the age 3–5 years instead of at the exact age of 5·5 years. This study was run in the context of preventive medicine, in early childhood, at the time of pre-school, and data were collected as part of the annual medical examination performed in pre-school children. Consequently, we were unable to perform data collection at the exact age of 5·5 years. However, the 3–5 year age group is of particular interest, since evidence shows that the earlier the EAR, the more severe the obesity [12,54].

Finally, it would be relevant, in further studies, to transpose the present method to BMI curves by using percentiles to define type A and type B-EAR, in order to make it accessible to a wider public.

In the current context of high prevalence of paediatric obesity worldwide, effective strategies for early diagnosis of overweight/obesity are needed. Based on our clinical expertise, we propose a classification of EAR, using IOTF charts, including both the starting point of the EAR and the BMI trajectory, differentiating

between children whose BMI *z*-score is ≥ 0 at the occurrence of the EAR and/or who show an alarming increase in their growth curve (type A-EAR), and children who have a lower initial BMI and who exhibit a low-to-moderate increase in their curve (type B-EAR). Following this approach, we were able to target almost 80 % of the children initially identified with EAR at 3–5 years, who became overweight/obese at 6–8 years. Differentiating type A from type B-EAR might represent a universal, easy and sensitive way for physicians to identify and improve the early screening of children at high risk of obesity.

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Supplementary material

For supplementary material referred to in this article, please visit https://doi.org/10.1017/S0007114520000987

References

1. Rokholm B, Baker JL & Sørensen TIA (2010) The levelling off of the obesity epidemic since the year 1999 – a review of evidence and perspectives. *Obes Rev* **11**, 835–846.
2. Rolland-Cachera MF, Deheeger M, Maillot M, *et al.* (2006) Early adiposity rebound: causes and consequences for obesity in children and adults. *Int J Obes (Lond)* **30**, Suppl. 4, S11–S17.
3. Dietz WH (1997) Periods of risk in childhood for the development of adult obesity – what do we need to learn? *J Nutr* **127**, 1884S–1886S.
4. Rolland-Cachera MF, Deheeger M, Bellisle F, *et al.* (1984) Adiposity rebound in children: a simple indicator for predicting obesity. *Am J Clin Nutr* **39**, 129–135.
5. Kato N, Isojima T, Yokoya S, *et al.* (2018) Earlier BMI rebound and lower pre-rebound BMI as risk of obesity among Japanese preschool children. *Int J Obes* **42**, 52–58.
6. Whitaker RC, Pepe MS, Wright JA, *et al.* (1998) Early adiposity rebound and the risk of adult obesity. *Pediatrics* **101**, E5.
7. Williams SM & Goulding A (2009) Patterns of growth associated with the timing of adiposity rebound. *Obesity (Silver Spring)* **17**, 335–341.
8. Di Gravio C, Krishnaveni GV, Somashekara R, *et al.* (2019) Comparing BMI with skinfolds to estimate age at adiposity rebound and its associations with cardio-metabolic risk markers in adolescence. *Int J Obes (Lond)* **43**, 683–690.
9. Sun Y, Fang J, Yang R, *et al.* (2017) Prospective association between early adiposity rebound and adolescent development in girls. *Zhonghua Yu Fang Yi Xue Za Zhi* **51**, 796–800.
10. Marakaki C, Karapanou O, Gryparis A, *et al.* (2017) Early adiposity rebound and premature adrenarche. *J Pediatr* **186**, 72–77.
11. Reilly JJ, Armstrong J, Dorosty AR, *et al.* (2005) Early life risk factors for obesity in childhood: cohort study. *BMJ* **330**, 1357.
12. Ip EH, Marshall SA, Saldana S, *et al.* (2017) Determinants of adiposity rebound timing in children. *J Pediatr* **184**, 151.e2–156.e2.
13. Toschke AM, von Kries R, Beyerlein A, *et al.* (2008) Risk factors for childhood obesity: shift of the entire BMI distribution vs. shift of the upper tail only in a cross sectional study. *BMC Public Health* **8**, 115.
14. Dorosty AR, Emmett PM, Cowin IS, *et al.* (2000) Factors associated with early adiposity rebound. *Pediatrics* **105**, 1115–1118.
15. Han JC, Lawlor DA & Kimm SY (2010) Childhood obesity. *Lancet* **375**, 1737–1748.
16. Verdot C, Torres M, Salavane B, *et al.* (2017) Children and adults body mass index in France in 2015. Results of the Esteban Study and trends since 2006. *BEH* **8**, 234–241.
17. Hughes AR, Sherriff A, Ness AR, *et al.* (2014) Timing of adiposity rebound and adiposity in adolescence. *Pediatrics* **134**, e1354–e1361.
18. Programme National Nutrition Santé (2014) *CALIMCO*.
19. Thibault H, Meless D, Carriere C, *et al.* (2010) Early screening criteria for children at risk of overweight. *Arch Pediatr* **17**, 466–473.
20. Salomon L-J, Bernard J-P, de Stavola B, *et al.* (2007) Birth weight and size: charts and equations. *J Gynecol Obstet Biol Reprod (Paris)* **36**, 50–56.
21. Torchin H, Ancel P-Y, Jarreau P-H, *et al.* (2015) Epidemiology of preterm birth: prevalence, recent trends, short- and long-term outcomes (article in French). *J Gynecol Obstet Biol Reprod (Paris)* **44**, 723–731.
22. Cole TJ & Lobstein T (2012) Extended international (IOTF) body mass index cut-offs for thinness, overweight and obesity. *Pediatr Obes* **7**, 284–294.
23. World Health Organization (2007) BMI-for-age (5–19 years). http://www.who.int/growthref/who2007_bmi_for_age/en/ (accessed February 2019).
24. Gueugnon C, Mougin F, Simon-Rigaud M-L, *et al.* (2012) Effects of an in-patient treatment program based on regular exercise and a balanced diet on high molecular weight adiponectin, resistin levels, and insulin resistance in adolescents with severe obesity. *Appl Physiol Nutr Metab Physiol Appl Nutr Metab* **37**, 672–679.
25. Roche J, Gillet V, Perret F, *et al.* (2018) Obstructive sleep apnea and sleep architecture in adolescents with severe obesity: effects of a 9-month lifestyle modification program based on regular exercise and a balanced diet. *J Clin Sleep Med* **14**, 967–976.
26. Roche J, Isacco L, Perret F, *et al.* (2019) Beneficial effects of a lifestyle intervention program on C-reactive protein: impact of cardiorespiratory fitness in obese adolescents with sleep disturbances. *Am J Physiol Regul Integr Comp Physiol* **316**, R376–R386.
27. Chaplais E, Thivel D, Greene D, *et al.* (2015) Bone-adiposity cross-talk: implications for pediatric obesity. A narrative review of literature. *J Bone Miner Metab* **33**, 592–602.

28. Karlberg J & Albertsson-Wikland K (1995) Growth in full-term small-for-gestational-age infants: from birth to final height. *Pediatr Res* **38**, 733–739.
29. Itabashi K, Mishina J, Tada H, *et al.* (2007) Longitudinal follow-up of height up to five years of age in infants born preterm small for gestational age; comparison to full-term small for gestational age infants. *Early Hum Dev* **83**, 327–333.
30. Argente J, Mehls O & Barrios V (2010) Growth and body composition in very young SGA children. *Pediatr Nephrol* **25**, 679–685.
31. Toschke AM, Grote V, Koletzko B, *et al.* (2004) Identifying children at high risk for overweight at school entry by weight gain during the first 2 years. *Arch Pediatr Adolesc Med* **158**, 449–452.
32. Eriksson JG (2007) Epidemiology, genes and the environment: lessons learned from the Helsinki Birth Cohort Study. *J Intern Med* **261**, 418–425.
33. Giles LC, Whitrow MJ, Davies MJ, *et al.* (2015) Growth trajectories in early childhood, their relationship with antenatal and postnatal factors, and development of obesity by age 9 years: results from an Australian birth cohort study. *Int J Obes (Lond)* **39**, 1049–1056.
34. Hakanen T, Saha MT, Salo MK, *et al.* (2016) Mothers with gestational diabetes are more likely to give birth to children who experience early weight problems. *Acta Paediatr* **105**, 1166–1172.
35. Zheng J-S, Liu H, Ong KK, *et al.* (2017) Maternal blood pressure rise during pregnancy and offspring obesity risk at 4–7 years old: the Jiaxing Birth Cohort. *J Clin Endocrinol Metab* **102**, 4315–4322.
36. Wang Y, Gao E, Wu J, *et al.* (2009) Fetal macrosomia and adolescence obesity: results from a longitudinal cohort study. *Int J Obes* **33**, 923–928.
37. Weng SF, Redsell SA, Swift JA, *et al.* (2012) Systematic review and meta-analyses of risk factors for childhood overweight identifiable during infancy. *Arch Dis Child* **97**, 1019–1026.
38. Lenard NR & Berthoud H-R (2008) Central and peripheral regulation of food intake and physical activity: pathways and genes. *Obesity (Silver Spring)* **16**, Suppl. 3, S11–S22.
39. Francis LA, Lee Y & Birch LL (2003) Parental weight status and girls' television viewing, snacking, and body mass indexes. *Obes Res* **11**, 143–151.
40. Parsons TJ, Power C, Logan S, *et al.* (1999) Childhood predictors of adult obesity: a systematic review. *Int J Obes Relat Metab Disord* **23**, Suppl. 8, S1–S107.
41. Miller HC, Hassanein K & Hensleigh PA (1976) Fetal growth retardation in relation to maternal smoking and weight gain in pregnancy. *Am J Obstet Gynecol* **125**, 55–60.
42. Cliver SP, Goldenberg RL, Cutter GR, *et al.* (1995) The effect of cigarette smoking on neonatal anthropometric measurements. *Obstet Gynecol* **85**, 625–630.
43. Harvey NC, Poole JR, Javaid MK, *et al.* (2007) Parental determinants of neonatal body composition. *J Clin Endocrinol Metab* **92**, 523–526.
44. Oken E, Levitan EB & Gillman MW (2008) Maternal smoking during pregnancy and child overweight: systematic review and meta-analysis. *Int J Obes (Lond)* **32**, 201–210.
45. Oken E & Gillman MW (2003) Fetal origins of obesity. *Obes Res* **11**, 496–506.
46. González L, Corvalán C, Pereira A, *et al.* (2014) Early adiposity rebound is associated with metabolic risk in 7-year-old children. *Int J Obes* **38**, 1299–1304.
47. Dietz WH (2000) 'Adiposity rebound': reality or epiphenomenon? *Lancet* **356**, 2027–2028.
48. Cole T (2004) Children grow and horses race: is the adiposity rebound a critical period for later obesity? *BMC Pediatr* **4**, 6.
49. Rolland-Cachera MF & Péneau S (2013) Growth trajectories associated with adult obesity. *World Rev Nutr Diet* **106**, 127–134.
50. Rolland-Cachera MF & Cole TJ (2019) Does the age at adiposity rebound reflect a critical period? *Pediatr Obes* **14**, e12467.
51. Singhal A, Wells J, Cole TJ, *et al.* (2003) Programming of lean body mass: a link between birth weight, obesity, and cardiovascular disease? *Am J Clin Nutr* **77**, 726–730.
52. Eriksson JG, Forsén T, Tuomilehto J, *et al.* (2003) Early adiposity rebound in childhood and risk of type 2 diabetes in adult life. *Diabetologia* **46**, 190–194.
53. Plachta-Danielzik S, Bosy-Westphal A, Kehden B, *et al.* (2013) Adiposity rebound is misclassified by BMI rebound. *Eur J Clin Nutr* **67**, 984–989.
54. Rolland-Cachera MF, Deheeger M, Guilloud-Bataille M, *et al.* (1987) Tracking the development of adiposity from one month of age to adulthood. *Ann Hum Biol* **14**, 219–229.

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