

# Maternal and Early-Life Nutritional Determinants of Early Adiposity Rebound in Moroccan Children

Bouchra Tarchoun, Soad Khal-Layoun, Btihaj Al Ibrahim, Nisrine Yassine, Boutaina Kasdi, Manar Makhoulouk, Zakaria Abidli, Abdellatif Bour.

## ABSTRACT

### Background

Childhood obesity represents a growing global public health challenge, with early-life metabolic programming playing a critical role in shaping long-term cardiometabolic health. Early adiposity rebound (EAR), defined as a premature increase in body mass index (BMI) after its physiological nadir in early childhood, has emerged as a strong predictor of later obesity and cardiometabolic disorders. Increasing evidence suggests that nutritional and metabolic exposures during the first 1000 days of life may influence the timing of adiposity rebound.

### Material and methods

This study aimed to investigate maternal and early-life nutritional determinants associated with EAR among Moroccan children. A cross-sectional analytical study was conducted in 2025 and included 100 mother-child pairs recruited from the Pèpinière Health Center in Salé, Morocco. Anthropometric measurements and early-life exposure data were collected using structured questionnaires and maternal-child health records. Associations between EAR and potential determinants were first evaluated using chi-square tests and subsequently examined using multivariate logistic regression analysis.

### Results

The prevalence of EAR was 38%. Early introduction of complementary feeding before six months (OR = 3.12; 95% CI: 1.35–7.21;  $p = 0.008$ ), early wheat introduction (OR = 2.74; 95% CI: 1.18–6.35;  $p = 0.019$ ), formula feeding (OR = 6.85; 95% CI: 2.52–18.60;  $p < 0.001$ ), and low birth weight (OR = 4.96; 95% CI: 1.41–17.43;  $p = 0.012$ ) were significantly associated with EAR. Maternal and perinatal factors also contributed to EAR risk, including maternal overweight at pregnancy onset (OR = 3.27; 95% CI: 1.36–7.86;  $p = 0.008$ ), cesarean delivery (OR = 5.21; 95% CI: 1.82–14.88;  $p = 0.002$ ), gestational diabetes (OR = 4.48; 95% CI: 1.65–12.16;  $p = 0.003$ ), and limited prenatal care (OR = 3.91; 95% CI: 1.55–9.83;  $p = 0.040$ ).

### Conclusion

These findings highlight the critical role of nutritional and metabolic exposures during the first 1000 days of life in shaping early adiposity trajectories. Strengthening maternal health care and promoting appropriate infant feeding practices may therefore represent key strategies for preventing early adiposity rebound and reducing long-term obesity risk.

### Keywords

Early adiposity rebound; first 1000 days; childhood obesity; cardiometabolic risk; Morocco.

## INTRODUCTION

Early-life growth trajectories play a fundamental role in shaping long-term metabolic health. Among the developmental milestones observed during childhood, adiposity rebound represents a critical transition in body composition, corresponding to the second rise in body mass index (BMI) following its physiological decline during early childhood. When this rebound occurs at an unusually early age, a phenomenon known as early adiposity rebound (EAR), it has been consistently associated with an increased risk of obesity and cardiometabolic disorders later in life<sup>1,2,3</sup>.

Although EAR becomes clinically detectable during early childhood, accumulating evidence suggests that its biological origins may emerge much earlier in development. This perspective aligns with the framework of the Developmental Origins of Health and Disease (DOHaD), which proposes that environmental exposures during sensitive developmental windows can shape long-term physiological and metabolic outcomes<sup>4,5,6</sup>. In particular, the first 1000 days of life; from conception to the end of the second year, represent a critical period of developmental plasticity during which nutritional, metabolic, and environmental influences may permanently affect disease susceptibility<sup>7,8</sup>.

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The importance of this early-life period has been widely recognized by international public health organizations such as the World Health Organization and UNICEF, which identify the first 1000 days as a key opportunity to promote optimal child development and prevent malnutrition, obesity, and long-term non-communicable diseases<sup>9,10</sup>. This perspective was further reinforced in 2015 during the World Congress of the DOHaD Society in Cape Town, which led to the adoption of the Cape Town Manifesto, calling for the integration of early-life programming concepts into biomedical research, clinical practice, and public health policies worldwide<sup>11</sup>.

Within this framework, maternal metabolic conditions and early nutritional exposures are considered important determinants of offspring adiposity trajectories. Maternal overweight, gestational diabetes, and altered intrauterine metabolic environments may influence fetal metabolic programming through mechanisms involving hyperglycemia, fetal hyperinsulinemia, and changes in nutrient transfer<sup>12,13</sup>. After birth, infant feeding practices and early growth patterns may further influence metabolic regulation. Breastfeeding, for example, has generally been associated with more favorable adiposity outcomes than formula feeding, while rapid postnatal catch-up growth following low birth weight has been linked to increased cardiometabolic risk later in life<sup>14,15</sup>.

Despite growing international evidence, studies investigating early-life determinants of EAR remain limited in North African populations. In Morocco, only a few investigations have examined maternal and early nutritional factors associated with EAR within a comprehensive first-1000-days framework. Identifying these determinants is essential for developing preventive strategies adapted to local nutritional and socioeconomic contexts<sup>15</sup>.

The present study therefore aimed to investigate maternal and early-life nutritional determinants associated with early adiposity rebound among children in Salé, Morocco.

## MATERIAL AND METHODS

This cross-sectional analytical study was conducted over a six-month period in 2025 at the Urban Health Center of Tabriquet in Salé, Morocco. The study aimed to investigate maternal and early-life nutritional determinants associated with early adiposity rebound (EAR) during the first 1000 days of life. Mother-child pairs attending pediatric consultations during the study

period were eligible for inclusion. Children aged 1 to 7 years at the time of assessment were included. A total of 100 mother-child pairs were included in the study.

Data were collected using a structured questionnaire administered to mothers and supplemented with information obtained from maternal and child health records.

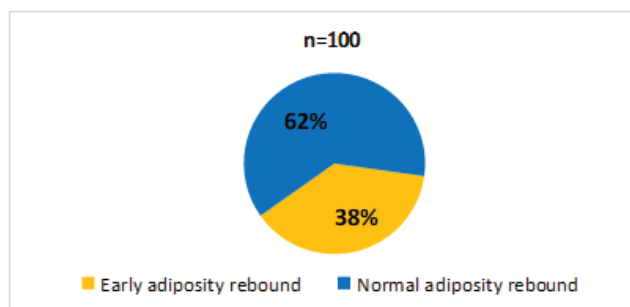
Anthropometric measurements were performed following standardized procedures. Body weight was measured using a calibrated bioelectrical impedance scale, and height was measured with a portable stadiometer. Body mass index (BMI) was calculated as weight in kilograms divided by height in meters squared ( $\text{kg}/\text{m}^2$ ). Body fat percentage was assessed using bioelectrical impedance analysis. Early adiposity rebound (EAR) was identified based on BMI trajectory assessment.

Statistical analyses were performed using SPSS software. Associations between EAR and categorical variables were assessed using the chi-square test. Variables significant in bivariate analysis were included in a multivariate logistic regression model. Results were reported as odds ratios (OR) with 95% confidence intervals (95% CI). A  $p$ -value  $< 0.05$  was considered statistically significant.

## RESULTS

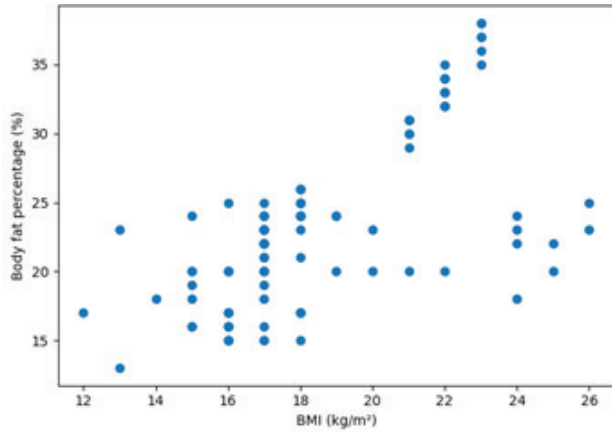
### Characteristics of the study population

A total of 100 children were included in the study. The mean age was  $43.96 \pm 14.65$  months (range: 11–72 months). The mean body weight was  $17.98 \pm 5.21$  kg and the mean height was  $0.94 \pm 0.15$  m. The average Body Mass Index (BMI) was  $18.64 \pm 3.09$   $\text{kg}/\text{m}^2$ , with values ranging from 12 to 26  $\text{kg}/\text{m}^2$  (figure 2). The mean body fat percentage was  $23.40 \pm 6.26\%$ , ranging from



**Figure 1.** Prevalence of Early Adiposity Rebound among the study population.

13% to 38%. Early Adiposity Rebound was observed in 38% of the children (n = 38). Among these children, 27 were girls and 11 were boys, indicating that EAR was significantly more frequent in girls than in boys ( $\chi^2 = 11.93, p = 0.001$ ).



**Figure 2.** Relationship between body mass index (BMI) and body fat percentage among children in the study population.

### Nutritional factors associated with Early Adiposity Rebound

The association between early-life nutritional factors and early adiposity rebound (EAR) was assessed using chi-square tests (Table 1).

Age at complementary feeding introduction was significantly associated with EAR. Early complementary feeding (<6 months) was more frequent among children with EAR compared with those with normal adiposity rebound ( $\chi^2 = 16.27, p < 0.001$ ).

Similarly, age at wheat introduction showed a significant association with EAR. Early introduction before six months was more common among children with EAR than among those with normal adiposity rebound ( $\chi^2 = 12.62, p < 0.001$ ).

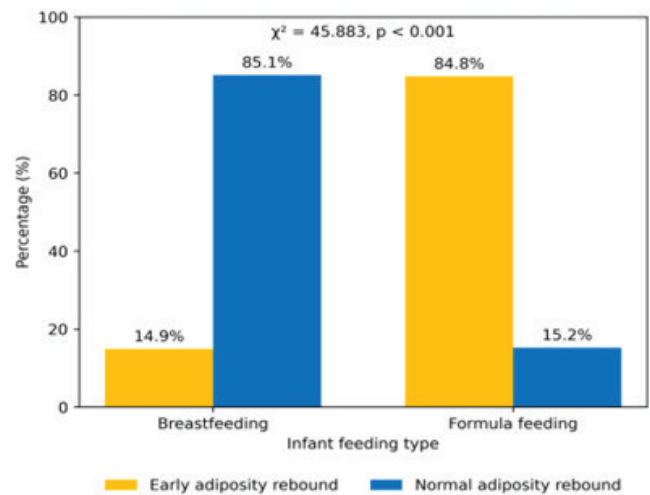
Infant feeding practices were strongly associated with EAR. Formula feeding was markedly more frequent among children with EAR, whereas breastfeeding predominated among children with normal adiposity rebound ( $\chi^2 = 45.88, p < 0.001$ ) (Figure 3).

Birth weight was also significantly associated with EAR. Low birth weight (<2.5 kg) occurred more frequently among children with EAR than among those

with normal adiposity rebound ( $\chi^2 = 27.38, p < 0.001$ ).

**Table 1.** Association between nutritional factors and EAR

Variable	Category	EAR n (%)	Normal n (%)	$\chi^2$	p value
Complementary feeding age	<6 months	31 (55.4)	25 (44.6)	16.275	<0.001
	≥6 months	7 (15.9)	37 (84.1)		
Age at wheat introduction	<6 months	28 (54.9)	23 (45.1)	12.620	<0.001
	≥6 months	10 (20.4)	39 (79.6)		
Infant feeding type	Breastfeeding	10 (14.9)	57 (85.1)	45.883	<0.001
	Formula feeding	28 (84.8)	5 (15.2)		
Birth weight	<2.5 kg	16 (94.1)	1 (5.9)	27.378	<0.001
	2.5–4 kg	22 (26.5)	61 (73.5)		



**Figure 3.** Association between infant feeding type and Early Adiposity Rebound.

### Maternal and perinatal factors associated with early adiposity rebound

Several maternal and perinatal characteristics were significantly associated with early adiposity rebound (EAR) (Table 3).

Maternal contraceptive pill use prior to pregnancy was more common among mothers of children with EAR compared with those with normal adiposity rebound ( $\chi^2$

= 17.26,  $p < 0.001$ ) (figure 4A).

Maternal weight at pregnancy onset was strongly associated with EAR, with a higher proportion of overweight mothers among children with EAR, whereas normal adiposity rebound was predominantly observed among children of mothers with normal weight ( $\chi^2 = 56.67$ ,  $p < 0.001$ ).

Mode of delivery was also significantly related to EAR. Cesarean section was markedly more frequent among children presenting EAR, whereas vaginal delivery predominated among children with normal adiposity rebound ( $\chi^2 = 52.01$ ,  $p < 0.001$ ).

Gestational age at delivery showed a significant association with EAR, with preterm birth occurring more frequently among children with EAR ( $\chi^2 = 27.38$ ,  $p < 0.001$ ).

Maternal supplementation during pregnancy was significantly associated with EAR ( $\chi^2 = 37.73$ ,  $p < 0.001$ ). Different supplementation patterns were observed between the two groups (figure 4B).

Gestational diabetes was strongly associated with EAR ( $\chi^2 = 27.28$ ,  $p < 0.001$ ).

Maternal diet during pregnancy was also related to EAR, with unbalanced dietary patterns being more common among mothers of children with EAR ( $\chi^2 = 5.05$ ,  $p = 0.025$ ).

Finally, prenatal care attendance showed a significant association with EAR, with limited prenatal follow-up being more frequent among children with EAR ( $\chi^2 = 43.33$ ,  $p < 0.001$ ).

**Table 2.** Maternal and perinatal factors associated with EAR

Variable	Category	EAR n (%)	Normal n (%)	$\chi^2$	p value
Maternal contraceptive pill use	Pill use	34 (53.1)	30 (46.9)	17.26	<0.001
	No pill use	4 (11.1)	32 (88.9)		
Maternal weight at pregnancy onset	Underweight	2 (33.3)	4 (66.7)	56.67	<0.001
	Normal weight	5 (8.6)	53 (91.4)		
	Overweight	31 (86.6)	5 (13.3)		
Mode of delivery	Vaginal	9 (13.4)	58 (86.6)	52.01	<0.001
	Cesarean	29 (87.9)	4 (12.1)		

Variable	Category	EAR n (%)	Normal n (%)	$\chi^2$	p value
Gestational age	Preterm	16 (94.1)	1 (5.9)	27.38	<0.001
	Term	22 (26.5)	61 (73.5)		
Maternal supplementation	Iron	19 (90.5)	2 (9.5)	37.73	<0.001
	Folic acid	13 (40.6)	19 (59.4)		
	Vitamin D	0 (0)	5 (100)		
	Iron + folic acid + vitamin D	6 (14.3)	36 (85.7)		
Gestational diabetes	Yes	21 (80.8)	5 (19.2)	27.28	<0.001
	No	17 (23.0)	57 (77.0)		
Maternal diet during pregnancy	Balanced	32 (34.8)	60 (65.2)	5.05	0.025
	Unbalanced	6 (75.0)	2 (25.0)		
Prenatal care visits	< 3 visits	30 (75.0)	10 (25.0)	43.33	<0.001
	>3visits	8 (13.3)	52 (86.7)		

### Multivariate logistic regression analysis

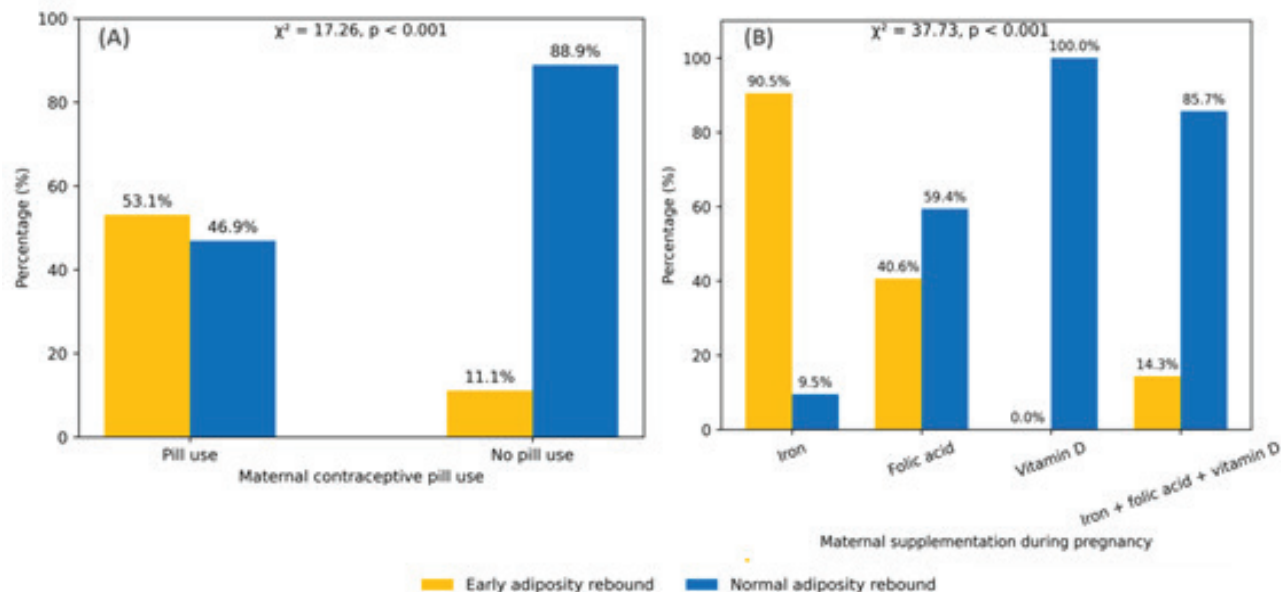
A multivariate logistic regression analysis was conducted to identify independent predictors of early adiposity rebound (Table 3).

Early introduction of complementary feeding before six months was significantly associated with EAR (OR = 3.12, 95% CI: 1.35–7.21,  $p = 0.008$ ). Similarly, early wheat introduction before six months increased the likelihood of EAR (OR = 2.74, 95% CI: 1.18–6.35,  $p = 0.019$ ).

Infant feeding practices were strongly associated with EAR. Children who received formula feeding had a significantly higher risk compared with those who were breastfed (OR = 6.85, 95% CI: 2.52–18.60,  $p < 0.001$ ).

Low birth weight (<2.5 kg) was also independently associated with EAR (OR = 4.96, 95% CI: 1.41–17.43,  $p = 0.012$ ).

Maternal and perinatal factors also played an important role. Maternal overweight at pregnancy onset (OR = 3.27,  $p = 0.008$ ), cesarean delivery (OR = 5.21,  $p = 0.002$ ), gestational diabetes (OR = 4.48,  $p = 0.003$ ), and limited prenatal care (<3 visits) (OR = 3.91,  $p = 0.004$ ) remained significantly associated with EAR after adjustment.

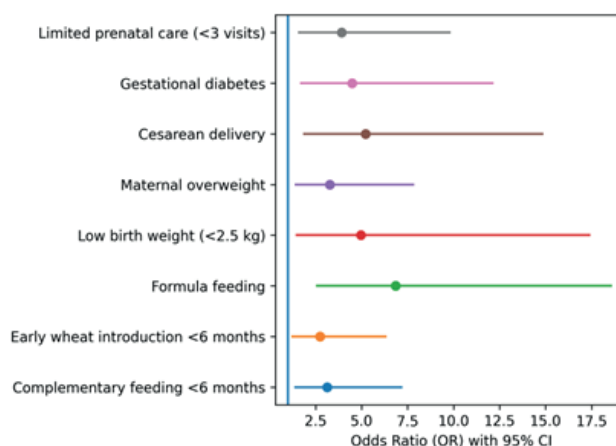


**Figure 4.** Maternal factors associated with Early Adiposity Rebound. (A) Association between maternal contraceptive pill use and EAR. (B) Association between maternal supplementation during pregnancy and EAR.

**Table 3.** Multivariate logistic regression analysis of factors associated with EAR

Variable	OR	95% CI	p value
Complementary feeding <6 months	3.12	[1.35 - 7.21]	0.008 **
Early wheat introduction (<6 months)	2.74	[1.18 - 6.35]	0.019 *
Formula feeding	6.85	[2.52 - 18.60]	<0.001 ***
Low birth weight (<2.5 kg)	4.96	[1.41 - 17.43]	0.012 *
Maternal overweight	3.27	[1.36 - 7.86]	0.008 **
Cesarean delivery	5.21	[1.82 - 14.88]	0.002 **
Gestational diabetes	4.48	[1.65 - 12.16]	0.003 **
Limited prenatal care (<3 visits)	3.91	[1.55 - 9.83]	0,04 *

OR= Odds Ratio ; CI= Confidence Interval ; significance level : (\*)  $p < 0.05$  , (\*\*)  $p < 0.01$  , (\*\*\*)  $p < 0.001$



**Figure 5.** Independent predictors of EAR identified by multivariate logistic regression. Points represent odds ratios (OR) and horizontal lines indicate 95% confidence intervals. The vertical line represents OR= 1 (no association).

## DISCUSSION

The present study investigated maternal, perinatal, and early-life nutritional determinants associated with early adiposity rebound (EAR) among Moroccan children. In our population, EAR was observed in 38% of children, indicating a relatively high prevalence of early adiposity rebound in this setting. EAR has been widely

recognized as an early marker of future obesity and cardiometabolic disorders. The concept refers to the second increase in body mass index (BMI) following its physiological decline during early childhood, and when it occurs prematurely it is strongly associated with an increased risk of later obesity and metabolic diseases<sup>16,17,18</sup>.

In the present study, EAR was more frequent among girls than boys. Similar sex differences in adiposity rebound timing have been reported in several epidemiological studies. Biological differences in fat distribution, hormonal regulation, and growth patterns during early childhood may contribute to earlier adiposity rebound among girls<sup>18,19,20</sup>. Previous cohort studies have also suggested that earlier adiposity rebound among girls may partly explain the higher prevalence of obesity observed among adolescent females in certain populations<sup>19,20</sup>.

Early-life nutritional exposures emerged as major determinants of EAR in our study. In particular, early introduction of complementary feeding before six months was significantly associated with EAR. These findings are consistent with previous research demonstrating that early complementary feeding may increase energy intake during infancy and promote accelerated weight gain during a critical developmental period<sup>21,22,23,24</sup>. Rapid infant weight gain has been consistently identified as one of the strongest predictors of early adiposity rebound and childhood obesity<sup>23,24,25</sup>. Similarly, early introduction of wheat before six months was independently associated with EAR in our population. Although evidence regarding the specific role of wheat introduction remains limited, early exposure to energy-dense complementary foods may increase caloric intake and contribute to rapid infant growth. Early complementary feeding may also influence appetite regulation and metabolic programming during infancy, potentially increasing the risk of later adiposity<sup>21,22,23</sup>.

Infant feeding practices also played a key role in our findings. Children who were formula-fed had a significantly higher risk of EAR compared with those who were breastfed. Numerous studies have reported that breastfeeding is associated with a lower risk of rapid early weight gain and childhood obesity. Breast milk contains bioactive components, including leptin, adiponectin, and growth factors, which may contribute to appetite regulation and metabolic programming

during infancy. In addition, breastfed infants may develop improved self-regulation of energy intake compared with formula-fed infants<sup>26,27,28,29</sup>.

Birth weight also emerged as an important determinant of EAR in our study. Children with low birth weight were significantly more likely to develop EAR. This observation may reflect compensatory catch-up growth frequently observed among infants with intrauterine growth restriction. Rapid postnatal growth following low birth weight has been consistently associated with increased adiposity and cardiometabolic risk later in life<sup>23,30,31,32</sup>. These findings support the Developmental Origins of Health and Disease (DOHaD) hypothesis, which emphasizes the role of early-life exposures in shaping long-term metabolic outcomes<sup>30,31</sup>.

Maternal micronutrient supplementation during pregnancy may also influence early metabolic programming in the offspring. In our study, a significant association was observed between the type of maternal supplementation and the occurrence of early adiposity rebound. Children whose mothers received combined supplementation, including iron, folic acid, and vitamin D, showed a lower prevalence of EAR compared with other supplementation patterns. Micronutrients such as folic acid and vitamin D are known to play key roles in fetal growth, epigenetic regulation, and adipocyte differentiation<sup>33,34,35</sup>. In particular, folate participates in one-carbon metabolism and DNA methylation processes that regulate gene expression related to energy metabolism and adipogenesis<sup>34</sup>. Increasing evidence suggests that epigenetic modifications established during fetal development may influence later BMI trajectories and adiposity risk in childhood<sup>33,34,35</sup>. Likewise, maternal vitamin D status during pregnancy may influence childhood adiposity and metabolic outcomes in offspring<sup>36,37</sup>. Adequate maternal micronutrient status during pregnancy may therefore contribute to healthier metabolic trajectories in early childhood through mechanisms related to developmental metabolic programming<sup>38,39</sup>. However, these findings should be interpreted with caution, as differences in supplementation practices may also reflect variations in maternal nutritional status, prenatal care, or other socioeconomic factors. In addition, Iron supplementation is frequently prescribed in the context of maternal anemia or increased nutritional risk<sup>35,40</sup>. Therefore, the higher prevalence of EAR observed in this group may reflect underlying maternal health

conditions rather than a direct causal effect of iron supplementation itself.

Maternal and perinatal factors were also strongly associated with EAR. Maternal overweight at pregnancy onset significantly increased the likelihood of EAR among offspring. Maternal obesity during pregnancy has been widely recognized as an important contributor to the intergenerational transmission of obesity through genetic, epigenetic, and metabolic mechanisms<sup>15,32,39</sup>. Exposure to an obesogenic intrauterine environment may alter fetal metabolic programming and influence offspring adiposity trajectories<sup>32,39</sup>. Maternal contraceptive pill use was also associated with a higher prevalence of early adiposity rebound in our study. Although the mechanisms underlying this association remain unclear, some studies suggest that hormonal exposure related to contraceptive use may influence maternal metabolic status and hormonal balance prior to pregnancy<sup>41,42</sup>. These factors could potentially affect the intrauterine environment and early metabolic programming of the offspring<sup>11</sup>. In addition, contraceptive use may also reflect broader lifestyle or reproductive health patterns that are indirectly associated with pregnancy outcomes and child growth trajectories.

Similarly, gestational diabetes was independently associated with EAR in our study. Exposure to maternal hyperglycemia during fetal development may influence adipocyte differentiation, insulin sensitivity, and long-term metabolic regulation in offspring. Several studies have reported that children born to mothers with gestational diabetes have an increased risk of obesity and metabolic disorders later in life<sup>13,14,32</sup>.

Mode of delivery also appeared to influence EAR risk. Cesarean delivery was significantly associated with EAR in our population. Previous studies suggest that cesarean section may alter the establishment of the infant gut microbiota, which plays an important role in metabolic regulation and energy homeostasis. Alterations in early microbial colonization have been linked to increased risk of childhood overweight and obesity<sup>12,32,33,43</sup>.

Finally, limited prenatal care was associated with EAR. Inadequate prenatal follow-up may reflect broader socioeconomic disparities affecting maternal nutrition, pregnancy outcomes, and infant feeding practices. Several population-based studies have highlighted the importance of early-life social and environmental

determinants in shaping childhood obesity risk trajectories<sup>15,44,45</sup>.

Despite growing international evidence, research exploring early-life determinants of EAR remains limited in North African populations<sup>15,44,45,46</sup>. In Morocco, only a few studies have investigated the determinants of early adiposity rebound. A study conducted in Salé by Tahiri et al. reported a relatively high prevalence of EAR among Moroccan children and identified maternal obesity, cesarean delivery, and low birth weight as significant associated factors. These findings are broadly consistent with the associations observed in our study and further highlight the importance of maternal metabolic health and early-life conditions in shaping childhood growth trajectories<sup>15</sup>.

Taken together, these findings highlight the critical importance of the first 1000 days of life as a sensitive window for metabolic programming and obesity prevention. Maternal metabolic health, adequate prenatal care, and appropriate infant feeding practices appear to play key roles in determining the timing of adiposity rebound and the subsequent risk of childhood obesity<sup>47,48,49,50</sup>.

Despite these findings, several limitations should be acknowledged. First, the cross-sectional design does not allow causal relationships to be established. Second, the study was conducted in a single health center and included a relatively modest sample size, which may limit the generalizability of the findings. Nevertheless, this study provides valuable insights into early determinants of adiposity rebound within the Moroccan context, where epidemiological evidence remains limited.

Future longitudinal studies are needed to better understand the mechanisms linking early-life exposures to adiposity rebound and to develop targeted interventions aimed at preventing childhood and adult obesity and related cardiometabolic diseases.

## CONCLUSION

This study emphasizes the important role of maternal, perinatal, and early-life nutritional factors in shaping the timing of adiposity rebound among Moroccan children. The relatively high prevalence of early adiposity rebound observed in our population suggests that susceptibility to obesity may begin during the earliest stages of life. Maternal metabolic health, gestational

conditions, and infant feeding practices appear to influence early growth trajectories and metabolic programming. These findings highlight the importance of the first 1000 days as a critical window for obesity prevention. Strengthening maternal health care and promoting optimal infant nutrition may therefore represent key strategies to reduce the long-term risk of childhood obesity.

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