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ORIGINAL ARTICLE

Body mass index trajectories from birth to adolescence and associated factors in the PARIS cohort

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Abstract

Aim: Development of body mass index (BMI) trajectories is essential for understanding childhood overweight, a public health concern. This study aimed to identify BMI trajectories from birth to adolescence and examine associated factors in the Pollution and Asthma Risk: an Infant Study (PARIS) birth cohort.

Methods: Data on height, weight, birth parameters, lifestyle, parental weight status and stress were collected via questionnaires and health check-ups. BMI z-score (BMIz) trajectories were developed using group-based trajectory modelling on anthropometric data collected at least six times. Associated factors were investigated in multivariable multinomial logistic regression models, adjusted for confounders.

Results: Five BMIz trajectories were identified in 540 adolescents. The early high stable BMI trajectory grouped participants who reached overweight status in early childhood. Four trajectories remained within the normal weight status: low stable BMI, continuous decrease BMI, continuous increase BMI, and early increase and slight decrease BMI trajectories. Compared with low stable BMI, high BMI trajectories were associated with higher parental weight status, early rebound age, excessive TV watching, lower food avoidance score, stressful events in early life and parent–child relationship stress.

Conclusion: High BMI trajectories shared several modifiable factors, emphasising the need for multifactorial interventions to tackle the childhood overweight epidemic.

KEYWORDS
adolescent, lifestyle, longitudinal study, overweight, stress

1 | INTRODUCTION

In last decades, childhood obesity has become an important global health concern. From 1990 to 2022, the worldwide prevalence of obesity increased from 1.7% to 6.9% in girls, and from 2.1% to 9.3% in boys.[1] In 2015, approximately 17% of French children aged 6 to 17 years were classified as living with overweight, and 4% as living with obesity.[2] The obesity epidemic puts a substantial burden on individuals, communities, and economies, manifesting in a spectrum of chronic diseases from cardiovascular diseases and hepatic and

Abbreviations: BMI, Body mass index; BMIz, BMI z-score; MVPA, Moderate-to-vigorous physical activity; PARIS, Pollution and Asthma Risk: an Infant Study; SES, Socioeconomic status.

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renal disorders to type 2 diabetes.[3] Multiple long-term studies have consistently indicated that childhood obesity significantly increases the likelihood of developing cardiometabolic diseases in adulthood, especially if there is sustained obesity throughout life.[4,5] Most epidemiological studies on childhood overweight and obesity have considered body mass index (BMI) at a single time point, neglecting any changes as children develop. However, a study published in 2023 indicates that the longitudinal examination of BMI during childhood is more predictive of later overweight or obesity than a single-point measurement.[6]

Childhood obesity results from the intricate interplay of genetic factors and perinatal, environmental, lifestyle and psychosocial elements. Identifying determinants is therefore crucial for understanding the different developmental paths leading to overweight by adolescence. Few studies have prospectively examined childhood BMI trajectories in relation to a broad spectrum of risk factors, albeit they were limited to short study periods.[7,8] In this context, the objective of this study was to construct BMI trajectories from birth to adolescence within the Pollution and Asthma Risk: an Infant Study (PARIS) birth cohort and investigate the associated factors.

Key notes

- Development of body mass index trajectories is essential for a comprehensive understanding of weight status.
- Five body mass index trajectories were identified, two of which exhibited a higher cardiometabolic risk associated with known risk factors and stress in early life and through the life.
- The identification of modifiable associated factors emphasises the need for multifactorial intervention strategies to tackle the childhood overweight epidemic.

2 | METHODS

2.1 | PARIS birth cohort

The PARIS birth cohort comprised 3840 healthy full-term newborn infants recruited between 2003 and 2006 in five Paris maternity hospitals. The follow-up was based on regular questionnaires and health check-ups.[9]

2.2 | Anthropometric measures

Height and weight were extracted from medical records: at birth, 1, 3, 6, 9 and 12 months, at 2, 3, 4, 5, 6, 8–9, 13 and at 15–16 years, and measured in health check-ups at 18 months, 8–9 and 15–16 years. Body mass was measured during check-ups using a standard balance. Height was measured to the nearest 0.1 cm using a mechanical Kern height metric MSF 200 N (Kern & Sohn GmbH). BMI z-scores (BMIz) were estimated according to sex and age with reference to the WHO 2006 (0–5 years) and 2007 (5–19 years) growth standards.[10] Children were classified as living with underweight: < −2 standard deviation (SD), normal weight: ≥ −2SD, <1SD, overweight: ≥ 1SD, <2SD and obesity: ≥ 2SD.

2.3 | Assessment of cardiometabolic parameters

Additionally, other anthropometric parameters, such as body composition, blood pressure, heart rate and blood samples, were collected during the adolescent's check-up.

Body composition was measured by multifrequency bioelectrical impedancemetry using a Tanita MC-780MA segmental analyser (Tanita Corp) and was assessed by the percentage or mass in kilograms (kg) of fat, muscle and lean mass. Waist and hip circumferences were determined to the nearest 0.1 cm. Heart rate, systolic blood pressure and diastolic blood pressure levels were recorded with an Omron HEM-RML31 cuff blood pressure monitor (Omron Healthcare Co. Ltd). The average of three measurements was calculated. Triglycerides (g/L), blood glucose (g/L), total cholesterol (g/L) and High-density lipoprotein (HDL) (g/L) concentrations were determined by an enzymatic method: glycerol-3-phosphate oxidase-phenol-aminophenazone, hexokinase, cholesterol oxidase-phenol-aminophenazone, and a mixture of polyanions and detergents (Roche Cobas 6000 c501, Roche Diagnostics). Low-density lipoprotein (LDL) cholesterol levels (g/L) were calculated using the Friedwald formula.

Cardiometabolic health was assessed throughout a continuous metabolic syndrome risk score.[11] Based on these anthropometric and biological data, cardiometabolic profiles were identified using an unsupervised cluster analysis, revealing 356 adolescents in the healthy profile (67.2%) and 174 in the at cardiometabolic risk profile (32.8%).[12]

2.4 | Associated factors

2.4.1 | Birth parameters

At the maternity hospital, data on the baby's sex, weight and height, parents' socioeconomic status (SES), number of siblings, delivery mode and mother's age were collected. Parents' SES was categorised based on the lowest occupation of both parents. Occupations were divided into two categories being high (high-level white-collar workers) and medium (craftsmen, shopkeepers, intermediate-level white-collar workers) or low (unemployed, students, blue-collar workers, low-level white-collar workers). The mother's age was dichotomised according to the median age of the study population. Active smoking by mothers during pregnancy (no, yes) was documented.

2.4.2 | Parental history

Parental height and weight were collected when participants were 15–16 years, and parental BMI was calculated. Parents' weight status was categorised as: at least one living with overweight/obesity (>25 kg/m^2) and living with underweight/normal weight (BMI ≤25 kg/m^2).

2.4.3 | Physical activity, sedentary behaviour and sleep

The types of extracurricular sport activities that participants practiced at 8–9 years and 15–16 years were documented. The frequency and number of hours per week were determined at 8–9 years and 15–16 years, respectively. Using the Metabolic Equivalent of Task (MET) values for each activity, the intensity of each physical activity was then determined. Moderate-to-vigorous physical activity (MVPA) was defined as ≥3 METs. The practice of MVPA was categorised into three classes based on the median at each age: at 8–9 years, 'no', 'yes, once a week', and 'yes, twice a week'; and at 15–16 years, 'no', 'yes, less than four hours per week', and 'yes, more than four hours per week'. Sedentary lifestyle was analysed by daily screen time, and adolescents were grouped into those who spent 'less than three hours a day' and those who spent 'at least three hours a day' in front of screens. Information on bedtime and wake-up time, on weekdays and on weekends, allowed to determine whether they met the minimum recommendation of 8 h of sleep.

2.4.4 | Diet

The exclusive breastfeeding of children aged between 0 and 3 months was also documented. Adherence to the Mediterranean diet at 8–9 years of age was assessed based on the adapted Mediterranean Diet Quality Index[13]; using the following thresholds: <8 (low/moderate) and ≥8 (high). The food avoidance and food approach scales (from one for low to five for high) were calculated using the Adult Eating Behaviour Questionnaire administered at 15–16 years old. Adolescents also indicated whether they had breakfast every morning, how often they snacked, and how frequently they consumed soda: less than once a week, and at least once a week.

2.4.5 | Body development

Each child's adiposity rebound age was determined as the age at which their BMI began to rise again after reaching its lowest point. Early adiposity rebound was defined as ≤4.5 years.[14] At 15–16 years, adolescents also reported their perceived body development relative to peers of the same age. Their answers were classified as: early, similar to others and late.

2.4.6 | Stress

The occurrence of a stressful event within the family, such as death, job loss, divorce, illness, between 0 and 2 years of age was documented. At 15–16 years, adolescents provided information about any stress or anxiety they had experienced in their relationship with their parents.

2.5 | Statistical analyses

Statistical analyses were performed using Stata, version SE 17 software (Stata Corporation).

BMIz longitudinal trajectories were identified for adolescents with available data for z-score calculation (height, weight, age at measurement) at specific time periods: birth, 18 months, 3 or 4 years, 5 or 6 years, 8–9 or 13 years and 15–16 years. A group-based trajectory modelling using Stata plug-in *Traj* identified distinct trajectory groups with similar body-shape development patterns. Individual participants were assigned to one of the trajectory groups based on their highest estimated probability of belonging. Multiple models were estimated from two to six trajectory groups, initially assuming a quintic relationship, then quartic, cubic or quadratic relationship for any significant polynomial terms.

The selection of the most appropriate model was based on the interpretability and clinical plausibility of the identified trajectory groups and several goodness-of-fit and discrimination indices. These indices included the Bayesian information criteria, log-likelihood, proportion of subjects classified in each class with a posterior probability >0.7, proportion of participants assigned to each trajectory, a priori defined to contain at least 10% of the sample, and odds of correct classification >5.

The baseline characteristics of participating and non-participating adolescents were compared using chi-square, Fisher's exact test or Student's *t*-test. One-way analysis of variance, chi-square, and Bonferroni multiple-comparison tests were used to examine the difference of cardiometabolic parameters between BMIz trajectories.

Factors associated with BMIz trajectories were investigated in multivariable multinomial logistic regression models, adjusted for potential confounders. First, variables with *p* values under 0.2 in univariate analysis were selected (Table S2). Next, a step-by-step approach was used, from an initial model including sex and SES and gradually incorporating the other variables one by one, starting with the one with the lowest *p* value. To study the determinants of two specific trajectories, a multivariable logistic regression model was built in the same manner (Table S3).

Results were expressed as adjusted odds ratios (aOR) with 95% confidence intervals (95% CI). Associations were considered significant when the *p* value was less than 0.05.

3 | RESULTS

This study is based on 540 adolescents with available data to construct BMIz trajectories (Table S1). Parents of participating adolescents had higher SES and post-secondary education rates compared with non-participants, with a higher tendency to be born in France, but there was no difference in their place of residence. Participating adolescents had older mothers and were more likely to have been breastfed at birth, but no differences were found regarding their sex, weight and height at birth or exposure to tobacco smoke (Table 1).

Distribution of BMI at each time point is presented in Table S1. The best-fitting trajectory modelling was obtained for five BMIz trajectories (Figure 1). The trajectory called early high stable BMI grouped 10.0% of adolescents who reached overweight status at a very young age. The other four trajectories remained within the range of normal weight: low stable BMI (10.0%), continuous decrease BMI (27.2%), continuous increase BMI (23.3%) and early increase and slight decrease BMI (29.5%). Children in the low stable BMI trajectory were on average 1.01 SD below the average value of BMIz at birth.

The comparison of cardiometabolic parameters across BMIz trajectories is presented in Table 2. No differences were observed among the five trajectories for height, diastolic blood pressure, heart rate, LDL, HDL and triglycerides. The early high stable BMI trajectory exhibited significantly higher values for weight, BMI, waist and hip circumferences, waist-to-height ratio and body fat, while demonstrating lower values for lean body mass and muscle mass compared with the other trajectories. The early high stable BMI trajectory also exhibited higher values of continuous metabolic syndrome risk score and a higher proportion of adolescents in the at cardiometabolic risk profile compared with the low stable BMI and continuous decrease BMI trajectories. The continuous increase BMI trajectory demonstrated higher weight, BMI, waist and hip circumferences, waist-to-height ratio and continuous metabolic syndrome risk score compared with the low stable BMI and continuous decrease BMI trajectories.

TABLE 1 Baseline characteristics of adolescents from the PARIS birth cohort still being followed up at 15–16 years of age who participated, or did not participate in the BMI trajectory study ($n = 2117$).

Baseline characteristics	Participants ($n = 540$)	Non-participants ($n = 1577$)	*p* value
Male sex, *n* (%)	273 (50.6)	810 (51.4)	0.7
Weight at birth, kg (mean ± SD)	3.4 ± 0.4	3.4 ± 0.4	0.8
Height at birth, cm (mean ± SD)	50.3 ± 1.9	50.2 ± 1.9	0.2
Breastfed between 0 and 3 months, *n* (%)	183 (34.1)	409 (26.5)	0.001
Place of residence at birth			0.9
Paris city, *n* (%)	336 (62.2)	984 (62.4)	
Paris suburbs, *n* (%)	204 (37.8)	593 (37.6)	
Family socioeconomic status[a]			0.002
Low, *n* (%)	98 (18.1)	393 (25.0)	
Medium, *n* (%)	210 (38.9)	609 (38.6)	
High, *n* (%)	232 (43.0)	575 (36.4)	
Parents' level of education[b]			<0.001
Primary, *n* (%)	1 (0.2)	11 (0.7)	
Secondary, *n* (%)	25 (4.6)	157 (10.0)	
Post-secondary, *n* (%)	514 (95.2)	1409 (89.3)	
Geographical origin of parents			0.055
Two parents born in France, *n* (%)	407 (75.4)	1121 (71.1)	
At least one parent born outside France, *n* (%)	133 (24.6)	456 (28.9)	
Mother's age at birth, years (mean ± SD)	33.2 ± 3.9	32.8 ± 4.0	0.01
Mother actively smoked during pregnancy, *n* (%)	42 (7.8)	158 (10.0)	0.1
Smokers at home at birth, *n* (%)	95 (17.8)	316 (20.3)	0.2

Note: *p* value from Chi-square/Fisher's exact test or Student's *t*-test.

Abbreviation: SD, standard deviation.

[a]Lowest among parents.

[b]Highest among parents.

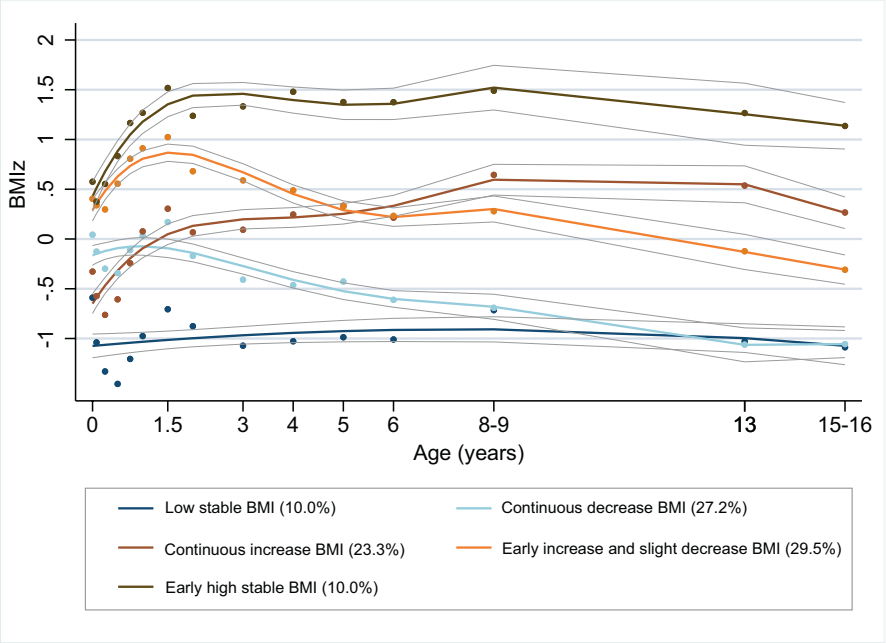

FIGURE 1 BMIz trajectories from birth to adolescence in the PARIS birth cohort.

The factors associated with BMIz trajectories are presented in Table 3, with the low stable BMI trajectory used for reference. Parental history of overweight or obesity and stress were positively associated, while the food avoidance score was negatively associated with continuous increase BMI and early high stable BMI trajectories. These trajectories were (tended to be) associated with watching television (≥3 h/day) and an early rebound age. The early high stable BMI trajectory tended to be linked to maternal active smoking during pregnancy and early body development. Delayed body development tended to be associated with the continuous decrease BMI trajectory. There was no association between any trajectory and sex, SES at birth or MVPA.

The study of factors associated with the early increase and slight decrease BMI trajectory compared with the continuous increase BMI trajectory is illustrated in Table 4. An early age of adiposity rebound was negatively associated with the early increase and slight decrease BMI trajectory, while high adherence to a Mediterranean diet was positively associated with this trajectory. Sex, SES at birth, weight status of the parents and MVPA were not associated with this trajectory.

4 | DISCUSSION

This study is one of only a few that describe body mass trajectories over an extended period, from birth to adolescence. Five trajectories were identified. One trajectory exhibits a rapid increase in BMI from early life, stabilising beyond the threshold for overweight. The other four trajectories evolve differently within the normal BMI range. We have identified several factors mainly associated with the two ascending trajectories, including parental history of overweight or obesity, adiposity rebound, behaviours (eating habits and sedentary lifestyle) and stress experienced at different life stages.

To date, much of the research examining BMIz trajectories has dealt mainly with childhood or adolescence. Few studies have tracked development from birth and early infancy, despite early childhood being a critical period in BMI development. For example, a literature review[15] of 65 papers using group-based trajectory modelling identified only 14 that had investigated BMIz trajectories from birth, the median number of assessment points being eight. Furthermore, most identified trajectories covered school-aged children and rarely included adolescents. Most studies reported three or four trajectories, with some reporting up to seven. Our study identified five BMIz trajectories with at least six regular measurements over an extended period from birth to 15–16 years, providing a good characterisation of BMI dynamics during childhood and adolescence.

The comparison of cardiometabolic parameters among the five trajectories indicates that the two ascending trajectories had anthropometry close to overweight. However, there was no difference in biological parameters, showing relatively favourable metabolic health, in contrast to findings reported in an Australian study describing higher total cholesterol and lower HDL cholesterol at adolescence in an obesity trajectory,[16] which was unobserved in our study. Nevertheless, high childhood BMIz trajectories predict adult obesity and poor metabolic health, emphasising the need to monitor BMIz trajectories in childhood.[17]

The analyses in this study have identified factors over pregnancy, (early) childhood, adolescence, associated with elevated BMIz trajectories. These associated factors include a history of overweight, the period of body development, behaviours and perceived stress.

Parental overweight was associated with the continuous increase BMI and early high stable BMI trajectories. The influence of

TABLE 2 Comparison of cardiometabolic parameters assessed at 15–16 years across BMIz trajectories from birth to adolescence in the PARIS birth cohort.

	Low stable BMI Mean (SD) or *n* (%)	Continuous increase BMI Mean (SD) or *n* (%)	Continuous decrease BMI Mean (SD) or *n* (%)	Early increase and slight decrease BMI Mean (SD) or *n* (%)	Early high stable BMI Mean (SD) or *n* (%)	*p* value*
Height (cm)	171.2 (7.0)	171.1 (8.7)	171.0 (7.8)	172.0 (8.5)	171.7 (9.7)	0.88
Weight (kg)	53.3 (5.6)[a,b,c]	63.1 (10.0)[a,d,e,f]	53.2 (6.4)[d,g,h]	59.1 (7.0)[b,e,g,i]	73.2 (13.2)[c,f,h,i]	<0.001
BMI (kg/m^2)	18.2 (1.7)[a,b,c]	21.5 (3.0)[a,d,e,f]	18.2 (1.6)[d,g,h]	20.0 (1.8)[b,e,g,i]	24.9 (4.2)[c,f,h,i]	<0.001
Weight status						<0.001
Underweight	6 (11.1)	1 (0.8)	18 (12.2)	3 (1.9)	–	
Normal weight	47 (87.0)	101 (80.2)	129 (87.8)	154 (96.9)	27 (50.0)	
Overweight	1 (1.9)	19 (15.1)	–	2 (1.2)	19 (35.2)	
Obesity	–	5 (3.9)	–	–	8 (14.8)	
Waist circumference (cm)	68.4 (5.9)[a,c]	75.1 (8.5)[a,d,f]	68.8 (6.2)[d,g,h]	72.3 (6.0)[g,i]	82.0 (10.0)[c,f,h,i]	<0.001
Hip circumference (cm)	88.5 (5.4)[a,b,c]	94.2 (7.2)[a,d,f]	88.1 (4.9)[d,g,h]	92.3 (5.5)[b,g,i]	102.3 (9.5)[c,f,h,i]	<0.001
Waist-to-height ratio	0.4 (0.0)[a,c]	0.4 (0.0)[a,d,f]	0.4 (0.0)[d,g,h]	0.4 (0.0)[g,i]	0.5 (0.1)[c,f,h,i]	<0.001
Body fat (%)	18.7 (5.8)[c]	22.2 (7.7)[d,f]	18.3 (6.1)[d,h]	20.6 (6.2)[i]	27.1 (8.5)[c,f,h,i]	<0.001
Lean body mass (%)	81.0 (5.9)[c]	77.8 (7.7)[d,f]	81.7 (6.2)[d,h]	79.4 (6.2)[i]	72.9 (8.5)[c,f,h,i]	<0.001
Muscle mass (%)	76.9 (5.6)[c]	73.8 (7.3)[d,f]	77.6 (5.9)[d,h]	75.4 (5.9)[i]	69.2 (8.1)[c,f,h,i]	<0.001
SBP (mmHg)	107.6 (9.6)	113.1 (9.7)[d]	109.3 (9.0)[d]	109.9 (8.6)	111.4 (10.8)	<0.001
DBP (mmHg)	66.2 (6.6)	66.1 (5.5)	66.0 (5.7)	65.6 (6.5)	65.6 (6.7)	0.96
Heart rate (pulse/minute)	68.1 (9.1)	66.2 (8.8)	69.8 (10.8)	67.4 (11.7)	65.4 (14.0)	0.05
LDL (g/L)	0.8 (0.2)	0.8 (0.2)	0.8 (0.2)	0.9 (0.3)	0.9 (0.3)	0.18
HDL (g/L)	0.6 (0.1)	0.6 (0.1)	0.6 (0.1)	0.6 (0.1)	0.6 (0.1)	0.63
Triglycerides (g/L)	0.7 (0.3)	0.8 (0.3)	0.8 (0.4)	0.8 (0.4)	0.8 (0.6)	0.65
Blood glucose (g/L)	0.9 (0.1)	0.9 (0.1)	0.9 (0.1)	0.9 (0.1)	0.9 (0.1)	0.04
cMetS risk score	−0.3 (0.4)[a,c]	0.0 (0.5)[a,d]	−0.3 (0.4)[d,g,h]	−0.1 (0.5)[g]	0.1 (0.5)[c,h]	<0.001
Cardiometabolic profiles						<0.001
'healthy'	45 (93.2)	51 (50.5)	100 (87.7)	82 (66.7)	10 (21.7)	
'at cardiometabolic risk'	3 (6.3)	50 (49.5)	14 (12.3)	41 (33.3)	36 (78.3)	

Abbreviations: BMI, body mass index (weight (kg)/height (m)^2; BMIz, body mass index z-score; DBP, diastolic blood pressure; HDL, high-density lipoprotein; LDL, low-density lipoprotein; SBP, systolic blood pressure; SD, standard deviation.

*One-way analysis of variance, Chi-square or Fisher's exact tests were used to compare trajectories. Bonferroni multiple-comparison tests are shown for an adjusted $p < 0.005$ between:

[a]'Continuous increase BMI' and 'Low stable BMI'.

[b]'Early increase and slight decrease BMI' and 'Low stable BMI'.

[c]'Early high stable BMI' and 'Low stable BMI'.

[d]'Continuous decrease BMI' and 'Continuous increase BMI'.

[e]'Early increase and slight decrease BMI' and 'Continuous increase BMI'.

[f]'Early high stable BMI' and 'Continuous increase BMI'.

[g]'Early increase and slight decrease BMI' and 'Continuous decrease BMI'.

[h]'Early high stable BMI' and 'Continuous decrease BMI'.

[i]'Early high stable BMI' and 'Early increase and slight decrease BMI'.

TABLE 3 Associated factors with BMIz trajectories in adolescents from the PARIS birth cohort: Results from the multivariable multinomial regression model (reference: 'Low stable BMI' trajectory).

	Continuous increase BMI			Continuous decrease BMI			Early increase and slight decrease BMI			Early high stable BMI		
	n (%)	aOR	95% CI	*n* (%)	aOR	95% CI	*n* (%)	aOR	95% CI	*n* (%)	aOR	95% CI
Sex												
Male	61 (54.5)	1.00		64 (54.7)	1.00		58 (46.0)	1.00		18 (48.6)	1.00	
Female	51 (45.5)	0.70	0.30–1.64	53 (45.3)	0.53	0.23–1.21	68 (54.0)	1.07	0.47–2.41	19 (51.4)	0.92	0.31–2.69
SES at birth												
High	44 (39.3)	1.00		56 (47.9)	1.00		53 (42.1)	1.00		17 (45.9)	1.00	
Medium/Low	68 (60.7)	0.84	0.39–1.82	61 (52.1)	0.72	0.34–1.53	73 (57.9)	0.88	0.42–1.85	20 (54.1)	0.53	0.20–1.40
Weight status of the parents												
Underweight/normal	39 (34.8)	1.00		56 (47.9)	1.00		57 (45.2)	1.00		12 (32.4)	1.00	
Overweight/obesity	73 (65.2)	**2.55**	**1.19–5.44**	61 (52.1)	1.42	0.68–2.94	69 (54.8)	1.71	0.83–3.54	25 (67.6)	**2.66**	**1.00–7.08**
Mother actively smoked during pregnancy												
No	105 (93.7)	1.00		110 (94.0)	1.00		115 (91.3)	1.00		33 (89.2)	1.00	
Yes	7 (6.3)	3.22	0.35–29.10	7 (6.0)	2.53	0.29–22.13	11 (8.7)	4.17	0.50–34.78	4 (10.8)	8.05	0.77–83.92
Stressful events during ages 0–2												
No	59 (52.7)	1.00		56 (47.9)	1.00		66 (52.4)	1.00		13 (35.2)	1.00	
Yes	53 (47.3)	1.43	0.66–3.10	61 (52.1)	1.52	0.72–3.21	60 (47.6)	1.38	0.66–2.89	24 (64.8)	**3.26**	**1.22–8.68**
Stress related to the parent–child relationship												
No	50 (44.6)	1.00		61 (52.1)	1.00		69 (54.8)	1.00		15 (40.5)	1.00	
Yes	62 (55.4)	**2.40**	**1.09–5.27**	56 (47.9)	1.53	0.71–3.29	57 (45.2)	1.51	0.71–3.22	22 (59.5)	2.44	0.91–6.55
Early rebound age												
No	47 (42.0)	1.00		84 (71.8)	1.00		88 (69.8)	1.00		15 (40.5)	1.00	
Yes	65 (58.0)	**2.21**	**1.02–4.80**	33 (28.2)	0.64	0.30–1.38	38 (30.2)	0.65	0.31–1.39	22 (59.5)	2.46	0.92–6.62
Pubertal development												
Same as the others	80 (71.4)	1.00		73 (62.4)	1.00		93 (73.8)	1.00		24 (64.9)	1.00	
Early	18 (16.1)	1.65	0.50–5.58	16 (13.7)	1.70	0.51–5.64	15 (11.9)	1.11	0.33–3.70	12 (32.4)	3.59	0.96–13.42
Late	14 (12.5)	1.48	0.43–5.13	28 (23.9)	2.76	0.86–8.81	18 (14.3)	1.42	0.43–4.70	1 (2.7)	0.30	0.03–3.08
Time spent watching TV												
<3 h/day	77 (68.7)	1.00		88 (75.2)	1.00		99 (78.6)	1.00		23 (62.2)	1.00	
≥3 h/day	35 (31.3)	2.60	0.98–6.81	29 (24.8)	1.89	0.72–4.94	27 (21.4)	1.49	0.57–3.90	14 (37.8)	**3.71**	**1.20–11.52**
FAV (+1 unit)		**0.27**	**0.12–0.60**		0.85	0.40–1.83		**0.37**	**0.17–0.80**		**0.26**	**0.10–0.71**
Moderate-to-vigorous physical activity												
No	10 (8.9)	1.00		21 (18.0)	1.00		13 (10.3)	1.00		5 (13.5)	1.00	
Yes, <4 h/week	53 (47.3)	2.12	0.66–6.71	50 (42.7)	1.09	0.39–3.05	55 (43.7)	1.98	0.67–5.89	17 (45.9)	1.62	0.39–6.68
Yes, ≥4 h/week	49 (43.8)	2.42	0.75–7.77	46 (39.3)	1.02	0.36–2.91	58 (46.0)	2.42	0.81–7.26	15 (40.6)	1.77	0.42–7.44

Note: Bold values highlight associations with $p < 0.05$.

Abbreviations: aOR, adjusted odds ratio; BMI, body mass index (kg/m^2); BMIz, body mass index z-score; CI, confidence interval; FAV, food avoidance; SES, socioeconomic status.

TABLE 4 Associated factors with 'Early increase and slight decrease BMI' trajectory in adolescents from the PARIS birth cohort: Results from the multivariable logistic regression model (reference: 'Continuous increase BMI' trajectory).

	Early increase and slight decrease BMI		
	n (%)	aOR	95% CI
Sex			
Male	59 (47.6)	1.00	
Female	65 (52.4)	1.69	0.95–3.05
Early rebound age			
No	85 (68.5)	1.00	
Yes	39 (31.5)	**0.32**	**0.18–0.58**
SES at birth			
High	52 (41.9)	1.00	
Medium/Low	72 (58.1)	0.83	0.46–1.48
Weight status of the parents			
Underweight/normal	57 (46.0)	1.00	
Overweight/obesity	67 (54.0)	0.61	0.34–1.08
Mediterranean diet adherence at 8 years			
Low/Moderate	107 (86.3)	1.00	
High	17 (13.7)	**5.67**	**1.54–20.82**
Moderate-to-vigorous physical activity at 8 years			
No	10 (8.1)	1.00	
Yes, <1/week	62 (50.0)	0.87	0.29–2.62
Yes, ≥1/week	52 (41.9)	0.59	0.19–1.76

Note: Bold values highlight associations with $p < 0.05$.
Abbreviations: aOR, adjusted odds ratio; BMI, body mass index (kg/m^2); CI, confidence interval; SES, socioeconomic status.

parent overweight on offspring weight status has been corroborated widely in the literature,[6–8,18–21] and may be caused by genetic, epigenetic and family environment factors.[22]

The link between maternal smoking during pregnancy and both childhood overweight and BMI developmental trajectories has also been well documented in the literature.[7,20,23–26] The suggested prenatal mechanisms of smoking involve developmental hypoxia, reduced uteroplacental blood flow, placental toxicity and effects on growth induced by the various chemicals in cigarette smoke, as well as potential epigenetic factors.[23] Although our results did not indicate a clear association, probably due to the low percentage of maternal smoking during pregnancy (<10%), the early high stable BMI trajectory tended to be associated with maternal smoking during pregnancy.

Concerning the period of body development, the early age of adiposity rebound predicts obesity in later life.[27] In our study, early adiposity rebound was associated, or tended to be associated, with the two ascending continuous increase BMI and early high stable BMI trajectories; these findings are in line with previous studies.[18,28] Fan et al. showed that children with a chronic overweight/obesity trajectory exhibited the highest risk of early pubertal maturation.[29] In our study, since the majority of adolescents had already reached puberty, we focused on individuals' perception of body development compared with others. Adolescents from the early high stable BMI trajectory tended to consider their body development more advanced compared with others, and adolescents from the continuous decrease BMI trajectory tended to consider their body development more delayed compared with others.

Behaviours, such as physical activity, sedentary and eating habits, are well-known risk factors for paediatric obesity,[30] but very few studies have investigated their association with BMIz trajectories.[7,8,18,21,31] While, to date, most research has not found any association,[7,18,21] two recent studies indicated that the overweight trajectory was associated with high sedentary[8] or low physical activity,[31] and the trajectory with decreasing BMI was associated with high physical activity.[8] In our study, a high amount of time spent in front of the television (≥3 h/day) was linked to the high BMIz trajectory, as found in the study by Fan et al.[8] Conversely, no association was observed with physical activity, which can be explained by a very physically active population (about 85% of participants performed MVPA). Concerning eating behaviour, food avoidance score was significantly less common in the continuous increase BMI, early increase and slight decrease BMI and early high stable BMI trajectories than in the low stable BMI trajectory, which is consistent with a longitudinal study reporting that high levels of undereating and picky eating (two behaviours assessed by the food avoidance score) were associated with a lower BMIz.[32]

This study documents an association of early and lifetime stress with BMIz trajectories. The occurrence of stressful events between 0 and 2 years was associated with the early high stable BMI trajectory. Adolescents who reported experiencing stress or anxiety in their relationship with their parents were more at risk of being part of the continuous increase BMI trajectory. Literature reviews have shown a positive association between parental reports of stress and the risk of paediatric obesity.[33,34] The association with early stress may be explained by parental stress, leaving less energy or fewer resources available to actively engage with their children, who thus develop obesity-risk behaviours. The parent–child relationship also seems to play a role; one study showed that child–parent conflict was associated with the obesity trajectory, and that children who had a close relationship with their mothers were less likely to follow the increased BMIz trajectory.[19]

We observed that two trajectories, continuous increase BMI and continuous decrease BMI, cross around the age of 5, as previously described.[6,7,19] To the best of our knowledge, our study shows for the first time factors associated with these trajectories. Indeed, the trajectory with decreasing BMI over time had a lower risk of experiencing an early rebound and adheres more to a Mediterranean diet at the age of 8 compared with the trajectory with increasing BMI over time. This study is consistent with the findings of Fernández-Barrés et al.,[35] who reported that high adherence to a Mediterranean diet during pregnancy is associated with a lower risk of having offspring with an accelerated growth

pattern; the mother's eating behaviour in particular appears to be transmitted to the offspring during childhood.

The follow-up from birth to adolescence allowed the collection of weight and height data for each child at up to 15 data points. Measurements were taken by a healthcare professional during the three health check-ups or reported by parents according to the child's health booklets. BMIz trajectories were established for children who had available data at six key developmental phases: at birth, at 18 months, before the adiposity rebound, after the adiposity rebound, at the onset of adolescence, and during adolescence. According to various statistical quality indices, the trajectories are robust. The covariates were issued from a comprehensive prospectively collected database. At 15–16 years, adolescents completed a questionnaire describing their feelings. Despite the prospective follow-up, behaviours were self-reported leading to a potential overestimation or underestimation. Data on some factors, such as screen time, were collected at 15–16 years which do not allow for causal relationship. Another limitation was the small sample size due to the attrition rate and the data available in adolescence, which may result in a lack of power in the study. As frequently observed in cohort follow-up, participants had a higher SES than non-participants leading to potential selection bias.

5 | CONCLUSION

This study has described five BMIz trajectories from birth to adolescence. One trajectory indicated rapid overweight after birth, while the other four remained within the normal BMI range. Various potentially modifiable determinants, such as maternal smoking, stress, sedentary behaviour and eating habits, seem to be risk associated factors for the two ascending trajectories. These results emphasise the need for multifactorial intervention strategies to reduce the overweight epidemic among children and adolescents.

AUTHOR CONTRIBUTIONS

Léa Lefebvre: Methodology; visualization; writing – review and editing; writing – original draft; validation; formal analysis. **Riem Nur:** Formal analysis. **Thomas Grunemwald:** Investigation. **Karima Hamrene:** Investigation. **Céline Roda:** Methodology; validation; writing – review and editing; supervision; conceptualization. **Isabelle Momas:** Conceptualization; funding acquisition; methodology; validation; writing – review and editing; supervision.

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CONFLICT OF INTEREST STATEMENT

The authors have no conflicts of interest to declare.

DATA AVAILABILITY STATEMENT

Data available on reasonable request.

ETHICS STATEMENT

The French Ethics Committees approved the PARIS study (permission nos. 031153, 051289, ID-RCB, 2009-A00824-53 and 2009-12-04 MS2). Parents and adolescents gave written informed consent.

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