Early Human Development 210 (2025) 106373

Contents lists available at ScienceDirect

Early Human Development

journal homepage: www.elsevier.com/locate/earlhumdev

Research paper

Associations of birth size with BMI trajectories and fluctuation across adolescence and adulthood: A longitudinal study of two Finnish twin cohorts

Alvaro Obeso [a,b,*], Aline Jelenkovic [b], Gabin Drouard [c], Jaakko Kaprio [c], Karri Silventoinen [a]

[a] Helsinki Institute for Demography and Population Health, University of Helsinki, Helsinki, Finland
[b] Department of Genetics, Physical Anthropology and Animal Physiology, Faculty of Science and Technology, University of the Basque Country (UPV/EHU), Bilbao, Spain
[c] Institute for Molecular Medicine Finland, HiLIFE, University of Helsinki, Helsinki, Finland

A B S T R A C T

Introduction: The influence of intrauterine conditions on later weight gain remains unclear. We examined the associations of birth size characteristics (weight, length, and ponderal index (PI)) with body mass index (BMI) trajectories and fluctuation in adolescence and adulthood using a twin design, which provides insights into the role of genetic and environmental factors.

Data and methods: Data from two Finnish twin cohorts including 9850 twin individuals (48 % males) were used. Weight and length or height were measured at birth and at different ages during adolescence and adulthood (11.5–37 years in FinnTwin12; 16–34 years in FinnTwin16). BMI trajectories across different stages of adolescence and adulthood were calculated as the difference in BMI divided by the time elapsed between measurements. BMI fluctuation was assessed as the variance of BMI trajectories at each stage. Linear regression models were used to examine the associations of birth size characteristics with BMI trajectories and fluctuation in adolescence and adulthood. Interactions between baseline BMI and birth characteristics were assessed. Within-pair analysis was performed to assess whether the identified associations persist while controlling for genetic effects.

Results: BMI trajectories during early adolescence were positively associated with birth PI and negatively with birth weight and length. BMI trajectories during middle adolescence were positively associated with birth length and negatively with birth weight. PI showed a negative association with BMI trajectories in late adolescence. Moreover, BMI fluctuation in adulthood was negatively associated with birth weight and length. No significant interactions were found between birth size characteristics and baseline BMI in explaining BMI trajectories and fluctuation at different stages of adolescence and adulthood. Among the identified associations, none remained significant in within-pair analysis.

Conclusion: Our findings suggest that birth size has a long-term influence on BMI development. However, these associations may not be due to the intrauterine environment but may rather indicate the role of shared genetic factors.

1. Introduction

According to the World Health Organization, the prevalence of obesity has nearly tripled over the past 40 years, with approximately 3.12 billion people (around 40 % of the global population) classified as overweight or obese [1,2]. Body mass index (BMI) is a widely used measure to assess weight status and is strongly associated with various health issues and cause-specific mortality [3]. BMI is influenced by a variety of factors such as biological, developmental, environmental, and behavioral factors, as well as their mutual interactions [4]. The relevance and extent to which these factors contribute may vary during distinct life stages [5,6].

Several studies have suggested the importance of the intrauterine environment for the development of obesity in later life mediated by various mechanisms [7,8]. Factors such as prenatal maternal stress [9,10], maternal BMI [11], and placental characteristics like weight [12,13], volume, and surface area [12] can influence the fetal environment, affecting birth size characteristics and, consequently, BMI later in life [14–17]. Direct measures of fetal size during pregnancy are rarely available, but birth size characteristics can be used as proxy indicators of prenatal conditions. While birth length has been found to be positively associated with BMI during childhood, such association has been reported to be negative during adolescence and adulthood [18]. Studies indicate that birth weight is positively associated with BMI in later years [19,20]. Similarly, ponderal index (PI) at birth has been found to be positively associated with BMI in later stages [19,21]. Together, these

* Corresponding author at: Helsinki Institute for Demography and Population Health, University of Helsinki, Helsinki, Unioninkatu 33, 00170, Finland.
E-mail address: alvaro.obeso@helsinki.fi (A. Obeso).

https://doi.org/10.1016/j.earlhumdev.2025.106373
Received 12 May 2025; Received in revised form 5 August 2025; Accepted 21 August 2025
Available online 23 August 2025

studies indicate that different birth size characteristics may be associated differently with later body weight and that these associations may vary throughout the life course.

However, the challenge in analyzing the effects of intrauterine conditions on later body weight lies in distinguishing these conditions from the effect of the maternal genotype, which is partly inherited by the offspring. Maternal genetic variants explained 22 % of the birth weight variation in women of European descent [13]. Another study showed that placental weight and fetal growth share genetic factors, with genetic correlations ranging from 0.2 to 0.6 [22]. Previous research has reported heritability estimates of 23–32 % for birth length, 17–23 % for birth weight, and 9–17 % for PI [23]. Genetic factors also play a significant role in explaining individual differences in BMI, as shown by twin [5,6] and genome-wide association studies [24–27]. Furthermore, gestational age as a key determinant of birth size characteristics can contribute to these associations. Previous studies have explored the relationship between gestational age and BMI in childhood [28,29], adolescence [30], and adulthood [31,32], reporting a positive association in childhood, no significant relationship in adolescence, and mixed findings in adulthood.

Twin pregnancies offer a unique opportunity to examine how specific intrauterine conditions influence the associations between birth size characteristics and later BMI trajectories and fluctuation. Twins have the same gestational age, largely experience the same postnatal environment, and in the case of monozygotic (MZ) twins, share practically the same DNA sequence. However, each fetus develops under unique fetoplacental conditions, including variations in nutrient and oxygen supply, which can differ significantly between co-twins [33]. These conditions are influenced by the type of placenta (monochorionic or dichorionic), which can further affect intrauterine development. In monochorionic twins, where both fetuses share a single placenta, there is a greater likelihood of unequal nutrient and oxygen distribution between the twins, potentially leading to discordant growth patterns [34]. In contrast, dichorionic twins, with separate placentas, are less likely to experience such disparities [35]. Consequently, comparing within-pair associations in MZ twins, who include both monochorionic and dichorionic pregnancies, and dizygotic (DZ) twins, who are all dichorionic, can provide information on the effects of intrauterine conditions, placental type, and genetic factors on the association between birth size characteristics and later BMI development.

Twin design has been previously used to analyze the associations between birth size characteristics and later BMI [24]. However, less is known about the associations between birth size characteristics and subsequent weight gain, as well as BMI fluctuation across different life stages. Understanding patterns of BMI change over time provides valuable insight into developmental and health-related processes. A large positive BMI trajectory—indicating a rapid increase in BMI across a specific developmental stage—may reflect excessive weight gain, which has been linked to an increased risk of obesity-related metabolic disorders such as insulin resistance, type 2 diabetes, and cardiovascular disease [36,37]. In addition to the overall direction of BMI change, BMI fluctuation—captured through variance or successive differences in BMI over time—can reflect instability in weight regulation. High fluctuation may indicate underlying physiological dysregulation, such as poor metabolic control or hormonal imbalances, and has been associated with inconsistent health behaviors (e.g., irregular eating patterns, physical activity, or psychological stress), as well as increased risk for adverse cardiometabolic outcomes [38–40]. Therefore, analyzing both BMI trajectories and fluctuations allows for a more detailed understanding of how weight patterns across key developmental periods relate to later health outcomes.

We aimed to address these gaps by investigating associations between birth size characteristics and BMI trajectories at various stages of adolescence and adulthood, as well as fluctuation across two broader life stages (adolescence and adulthood) using two Finnish twin cohorts. The twin design allowed us to examine the contribution of intrauterine conditions to these associations while controlling for genetic factors. We also explored interactions between baseline BMI and birth size characteristics to analyze in more detail how these traits act together in shaping BMI trajectories and fluctuation.

2. Data and methods

2.1. Cohorts

The study cohort was derived from two independent Finnish population based, longitudinal twin studies. The FinnTwin12 (FT12) cohort included all twins born in Finland during 1983–1987. The measurements were taken at approximate ages of 11.5, 14, 17.5, 24, and 37 years (response rates: 43 % – 92 %; maximum $N = 5362$) [41–43]. The FinnTwin16 (FT16) cohort included all twins born in Finland during 1974–1979. The measures were taken at approximate ages of 16, 17, 18, 25, and 34 years (response rates: 72 % – 95 %; maximum $N = 5659$) [44,45]. In each wave, the participants self-reported their current weight and height used for the calculation of BMI (kg/m^2). Subsets of twin pairs were enrolled in laboratory protocols for which DNA was obtained and zygosity was confirmed. When DNA was unavailable, zygosity was based on responses about physical similarities from the baseline questionnaire. This method was evaluated for 395 same-sex twin pairs from the FT12 study, and 97 % of the zygosity classifications based on the questionnaire were confirmed by DNA testing, indicating high reliability [45].

Separate questionnaires were sent to the parents of the twins, who provided information on birth length and weight used for the calculation of PI (kg/m^3). The parents also reported the gestational age of the twin pregnancy and whether there were one or two placentas. All DZ pairs are dichorionic, while MZ pairs were classified as mono- and dichorionic based on the parental reports. Among MZ pairs, 80 % were classified as monochorionic, which is probably an overestimate as a single placenta can be hard to distinguish from a conjoined placenta. Nearly 8 % of parents could not recall the gestational age. The mean reported gestational age was 36.9 (SD 2.6) weeks, with a distribution similar to that in the Birth Registry of Finland [46].

Initially, participants with at least two BMI measures in a specific stage of adolescence or adulthood were included, resulting in 9992 twin individuals. Impossible values and outliers for BMI trajectories, fluctuation, and birth size characteristics were checked through visual inspection of scatter plots leading to a final sample of 9850 twin individuals. For association analyses, individuals with their co-twin ($N = 8900$) and without their co-twin (singletons) ($N = 950$) were included. The number of individuals included in the association analyses varied by developmental stage and outcome. Specifically, for the association analyses with BMI trajectories, 4277 individuals were included for early adolescence, 9056 for middle adolescence, 4949 for late adolescence, 6358 for emerging adulthood, and 4542 for early adulthood. For the association analyses of BMI fluctuation, 8663 individuals were included for adolescence and 4344 for adulthood. For within-pair analyses, only complete twin pairs were included ($N = 4450$ pairs).

2.2. BMI trajectories and fluctuation calculation

The BMI trajectories were calculated using a delta method, which subtracts the BMI values at two timepoints and then divides the results by the time elapsed between the two measurements. BMI trajectories were calculated for three stages of adolescence (early adolescence: from 11 to 14 years old; middle adolescence: from 14 to 17 years old; late adolescence: from 17 to 18 years old) and two stages of adulthood (emerging adulthood: from 17.5 or 18 to 24 years old; early adulthood: from 24 to 34/37 years old) based on previous studies [47]. The last measure of each stage was the first measure of the subsequent stages. The selected age ranges correspond to key periods of growth, puberty, and maturation that influence BMI development. During early

adolescence (11–14 years), growth is rapid due to the hormonal changes of puberty [48]. In middle adolescence (14–17 years), most physical growth is complete, though brain development continues [48]. In late adolescence (17–18 years), major life transitions (especially those related to home, school, work, and personal identity) become more prominent while males may continue physical development [48]. Emerging adulthood (18–24 years) represents peak physiological functioning, including strength, sensory abilities, and cardiovascular health [49]. Additionally, this period has been identified as a high-risk period for excess weight gain, with several studies reporting the greatest increase in obesity prevalence occurring during this life stage [50,51]. Finally, early adulthood (24–37 years) is more stable but may involve gradual weight increase related to aging and lifestyle. The stage-specific approach allowed us to examine the rates of BMI change during key developmental transitions, reflecting potentially sensitive periods for growth and weight gain.

To calculate BMI fluctuation, we calculated the variance of BMI trajectories across two different stages. The BMI fluctuation in adolescence were defined based on BMI trajectories in early, middle, and late adolescence. The BMI fluctuation in adulthood was defined based on BMI trajectories of emerging adulthood and early adulthood. To examine the robustness of our BMI fluctuation measure, we additionally calculated BMI fluctuation using the Root Mean Square of Successive Differences (RMSSD), a method that captures the magnitude of successive BMI changes over time and that has been previously used for BMI [53] as well as other body composition measures [54]. We then assessed the correlation between RMSSD and our originally proposed approach. These results are presented in Supplementary Table 1. These analyses were conducted using the R software (version 4.2.3) and packages lme4 (version 1.1–34), lmerTest (version 3.1–3), modelsummary (version 1.4.3), dplyr (version 1.1.4) and optimx (version 2023–10.21).

2.3. Association and interaction analysis

First, the associations between: (i) birth size characteristics (length, weight, and PI) with BMI trajectories at different stages of adolescence and adulthood and (ii) birth size characteristics with BMI fluctuation in adolescence and adulthood were quantified. The analysis focused on specific developmental stages, rather than modeling BMI changes continuously across the entire lifespan. BMI trajectories were treated as distinct outcome variables, since they represent summary measures rather than repeated observations. Second, significant associations were analyzed for interaction analysis to determine if baseline BMI at each specific stage influences the associations between BMI trajectories at the same stage and the birth size characteristics. Linear regression models with individual-specific BMI trajectories and BMI fluctuation as dependent variables, and individual-specific birth size characteristics, sex, zygosity, stage-specific baseline BMI, stage-specific baseline age, and cohort identifier as independent variables were used. Interactions between stage-specific baseline BMI and birth size characteristics were included in the model. To account for the non-independence of observations within twin pairs, a family identifier was used as a clustering variable, and the model was estimated using cluster-robust standard errors to address within-family correlations. The analyses were conducted with and without sex stratification. Interactions between sex and the birth size characteristics were tested to investigate potential differences between males and females.

Sensitivity analysis was conducted to account for the potential influence of gestational age and placenta type on intrauterine conditions, which may confound the associations between birth size characteristics and BMI trajectories and fluctuation in adolescence and adulthood. Gestational age and placenta type were used as independent variables in the linear regression models along with the interaction of birth size characteristics with zygosity and type of placenta. Before carrying out sensitivity analysis, individuals with no data or impossible data for gestational age and placenta type were removed. These analyses were performed using the R software (version 4.2.3) and the R packages dplyr (version 1.1.4), modelsummary (version 1.4.3), sandwich (version 3.0–2) and lmtest (version 0.9–40).

2.4. Within-pair analysis

To control for the potential influence of genetic factors on the identified associations, within-pair analysis was performed using linear regression models [24]. We calculated within-pair differences (Δ) between the BMI trajectories, BMI fluctuation, and the birth size characteristics from previously found significant associations. We also calculated the mean of the baseline BMI values for each pair. Then, we performed linear regression models with within-pair birth size characteristics difference as the independent variable and within-pair BMI trajectories and fluctuation differences as the outcome variable. The specific models are displayed in the caption of the corresponding table. Initially, all complete twin pairs (a total of 4450 pairs) were included, followed by stratification by zygosity (1514 MZ pairs and 2936 DZ pairs). MZ twins share virtually identical DNA sequences, while DZ twins share 50 % of genetic variance. Differences in birthweight and later BMI within MZ pairs can only be influenced by environmental factors that are unique to individuals (i.e. the intrauterine conditions), whereas differences within DZ pairs can also be influenced by genetic factors [52,53]. Therefore, a stronger association in DZ than in MZ twins may indicate that the relationship between birthweight and later BMI development is confounded by genetic factors. To test whether the differences between MZ and DZ twins in effect sizes may be due to sample error, interactions between zygosity and birth size characteristics were calculated. These analyses were performed using the R software (version 4.2.3) and the R packages dplyr (version 1.1.4), modelsummary (version 1.4.3), sandwich (version 3.0–2) and lmtest (version 0.9–40).

Differences between co-twins may be small, especially for traits strongly influenced by genetic factors, potentially leading to low statistical power in within-pair models. To evaluate the range of variability in trait differences, we identified twin pairs whose absolute difference in BMI trajectory exceeded 0.1 units across the previously defined age intervals of 11–14 ($N = 111$ discordant MZ pairs and 252 discordant DZ pairs), 14–17 ($N = 185$ and 447, respectively), 17–18 ($N = 93$ and 216, respectively), 18–24 ($N = 235$ and 609, respectively), and 24–34/37 years ($N = 208$ and 492, respectively). The variances of the trait differences per zygosity groups are reported in Supplementary Table 6. These findings suggest significant variability in trait differences, indicating that within-pair models are likely adequately powered to detect associations, except for birth length, where differences between co-twins were minimal (< 1 cm).

3. Results

Table 1 presents the descriptive statistics of birth size characteristics, BMI trajectories, and BMI fluctuation during adolescence and adulthood by sex and cohort. In both cohorts, birth characteristics were greater in boys than in girls. BMI trajectories were higher in males in all stages of adolescence and adulthood in the FT16 cohort, while in the FT12 cohort, females had higher values in early adolescence (11–14 years of age) and early adulthood (24–37 years of age), and males in the other stages. For BMI fluctuation, in the FT12 cohort, females had higher values in adolescence while males had higher values in adulthood. In the FT16 cohort, the BMI fluctuation values were higher in males in adolescence and adulthood. We observed sex differences in both cohorts for BMI trajectories at all stages and for BMI fluctuation in adulthood.

The distribution of gestational age and chorionicity in the FT12 and FT16 cohorts reveals similar patterns. In both cohorts, the majority of individuals were born at term, with a peak in gestational ages between 36 and 38 weeks. The FT12 cohort showed a higher proportion of twin births at these gestational ages, especially for males and females born at 38 weeks. The FT16 cohort also followed this trend, with a similar

Table 1
Means and standard deviations (SD) of birth characteristics and BMI trajectories and fluctuation across different stages of life in FT12 and FT16 cohorts by sex.

	Males		Females		Sex differences (*p*-value)
	Mean	SD	Mean	SD	
FinnTwin12	*N* = 2579		*N* = 2528		
Birth characteristics					
Length (m)	0.48	0.02	0.47	0.01	0.18
Weight (kg)	2.71	0.53	2.66	0.53	0.25
Ponderal Index (kg/m^3)	25.78	1.80	25.25	1.91	0.98
BMI trajectories (kg/m^2 per year)					
Early adolescence (11–14 years old)	0.64	0.63	0.70	0.73	5.12e-03
Middle adolescence (14–17 years old)	0.67	0.60	0.45	0.61	<2.00e-16
Late adolescence (17–18 years old)	–	–	–	–	–
Emerging adulthood (17/18–24 years old)	0.35	0.37	0.24	0.35	2.36e-13
Early adulthood (24–40 years old)	0.16	0.22	0.22	0.26	5.78e-04
BMI fluctuation (kg/m^2 per year)					
Adolescent (11–18 years old)	0.46	0.97	0.47	0.89	0.76
Adulthood (18–40 years old)	0.10	0.21	0.08	0.17	7.43e-03
FinnTwin16	*N* = 2749		*N* = 2970		
Birth characteristics					
Length (m)	0.49	0.03	0.47	0.03	0.29
Weight (kg)	2.67	0.55	2.65	0.53	0.17
Ponderal Index (kg/m^3)	25.31	2.45	24.26	5.28	0.81
BMI trajectories (kg/m^2 per year)					
Early adolescence (11–14 years old)	–	–	–	–	–
Middle adolescence (14–17 years old)	0.70	1.34	0.27	1.18	<2.00e-16
Late adolescence (17–18 years old)	0.50	0.93	0.22	0.88	<2.00e-16
Emerging adulthood (17/18–24 years old)	0.34	0.37	0.23	0.39	<2.00e-16
Early adulthood (24–40 years old)	0.20	0.25	0.18	0.29	1.04e-05
BMI fluctuation (kg/m^2 per year)					
Adolescent (11–18 years old)	1.63	4.38	1.34	3.98	0.67
Adulthood (18–40 years old)	0.13	0.34	0.12	0.36	4.02e-08

Caption: The BMI trajectory estimates were obtained by Delta slope method based on BMI at two consecutive points, while fluctuation were derived from standard deviation of different BMI trajectories. The sex difference was studied using linear regression models (for ages: Age at n wave ~ Sex + Zygosity. For birth size characteristics: Birth size characteristics ~ Sex + Zygosity + Twin order. For BMI: BMI at n wave ~ Age at n wave + Sex + Zygosity + Birth length + Birth weight. For BMI trajectories during adolescence: BMI trajectories during adolescence ~ BMI intercept for adolescence + Adolescence age baseline + Zygosity + Sex + Birth weight. For BMI trajectories during adulthood: BMI trajectories during adulthood ~ BMI intercept for adulthood + Adulthood age baseline + BMI trajectories during adolescence + BMI intercept for adolescence + Zygosity + Sex + Birth weight. For BMI fluctuation during adolescence: BMI fluctuation during adolescence ~ BMI intercept for adolescence + BMI trajectories during adolescence + Zygosity + Sex. For BMI fluctuation during adulthood: BMI fluctuation during adulthood ~ BMI intercept for adulthood + BMI trajectories during adolescence + Zygosity + Sex. Abbreviations: SD: standard deviation, BMI: body mass index.

distribution but slightly higher counts for twin births at 38 weeks (Supplementary Table 2). Regarding the type of placenta, both cohorts predominantly comprised dichorionic twins (72 % in both cohorts) (Supplementary Table 3).

The associations of birth characteristics with BMI trajectories and fluctuation in the pooled data from the FT12 and FT16 cohorts are shown in Table 2. In the pooled data of males and females, all birth characteristics (length, weight, and PI) were significantly associated with BMI trajectories in early adolescence: greater birth length and weight were associated with lower BMI trajectories, while higher PI was associated with higher BMI trajectories (length: β = −12.93; 95 % CI: −16.50 to −2.10; weight: β = −0.28; 95 % CI: −0.53 – -0.17; PI: β = 0.09; 95 % CI: 0.02–0.19). Birth length was positively associated with BMI trajectories in middle adolescence (β = 9.19; 95 % CI: 0.03–15.40) and negatively associated with BMI fluctuation in adulthood (β = −3.31; 95 % CI: −7.81 to −0.46 for the variance-based method and β = −1.60; 95 % CI: −2.58 to −0.62 for the RMSSD method). Birth weight was also associated with BMI fluctuation in adulthood (β = −0.17; 95 % CI: −0.48 to −0.05 for the variance-based method and β = −27.10; 95 % CI: −45.52 to −8.69 for the RMSSD method) and BMI trajectories in early adulthood (β = −0.13; 95 % CI: −0.37 – -0.02). Higher PI was also associated with lower BMI in late adolescence (β = −0.03; 95 % CI: −0.18 – -0.02). The interactions between sex and birth size characteristics were not statistically significant (*p*-value: 0.09–0.97), indicating no evidence of different associations between males and females.

The sensitivity analysis including gestational age and type of placenta showed that five from the previously found significant associations remained significant after these adjustments. The interactions between (i) zygosity and birth size characteristics and (ii) placenta type and birth size characteristics were not statistically significant (Supplementary Table 4). We then conducted an interaction analysis between birth size characteristics and baseline BMI for BMI trajectories and fluctuation for all significant associations. However, none of these interactions were statistically significant (Table 3).

Finally, we conducted within-pair analysis for the significant associations of BMI trajectories and fluctuation with birth size characteristics (Table 4). The associations were calculated first including all complete twin pairs and then stratified by zygosity. None of the previously found eight significant associations remained significant. Moreover, we observed no statistically significant interaction with zygosity. Alongside the within-pair analysis results, intra-pair mean differences for birth size characteristics are presented in Table 4. For all three birth size characteristics, within-pair differences were smaller in DZ twin pairs compared to MZ twin pairs.

4. Discussion

The present study examined the associations between BMI trajectories at five life stages (three over adolescence and two over adulthood) and fluctuation at two broader life stages (adolescence and adulthood) with birth size characteristics (length, weight, and PI). The results were largely similar in males and females, and no evidence for sex differences in these associations was found. When pooling males and females, eight significant associations were identified between BMI trajectories and fluctuation with birth characteristics (three of them with birth length, three with birth weight, and two with PI). However, no interactions between birth size characteristics and BMI at baseline in explaining variability in BMI trajectories or fluctuation were found. Finally, none of the eight identified associations remained statistically significant in the within-pair analysis. This suggests that the intrauterine environmental factors differing between co-twins, such as nutrient supply through different vascularization, may not explain the associations between birth size and BMI development. Furthermore, no interaction between

Table 2
Associations of birth characteristics with BMI trajectories per 10 years across different stages of the adolescence and adulthood and fluctuation per 10 years across two stages of life by sex.

Variables of study	All ($N = 9850$)			Males ($N = 4723$)			Females ($N = 5127$)			Birth size characteristic-sex interaction
	β	95 % CI		β	95 % CI		β	95 % CI		p-value
		LL	UL		LL	UL		LL	UL	
Birth length										
BMI trajectories in early adolescence	−12.93	−16.50	−2.10	−9.12	−17.30	2.80	−15.44	−21.70	−0.30	0.77
BMI trajectories in middle adolescence	9.19	0.03	15.40	9.45	−3.60	20.10	8.84	−2.58	16.94	0.43
BMI trajectories in late adolescence	−5.01	−11.31	7.38	−5.30	−15.90	12.06	−3.83	−12.24	12.18	0.22
BMI trajectories in emerging adulthood	−4.32	−6.73	0.21	−2.50	−7.43	2.86	−4.43	−8.71	−0.03	0.93
BMI trajectories in early adulthood	−2.07	−4.81	0.83	−2.39	−5.95	1.77	−1.55	−5.73	2.08	0.65
BMI fluctuation$_1$ in adolescence	3.32	−24.46	14.92	7.56	−36.93	18.64	−3.26	−27.64	28.55	0.47
BMI fluctuation$_2$ in adolescence	0.20	−0.10	0.60	0.23	−0.10	0.29	−0.07	−0.11	0.08	0.51
BMI fluctuation$_1$ in adulthood	−3.31	−7.81	−0.46	−2.36	−8.57	2.86	−5.19	−9.92	−0.16	0.90
BMI fluctuation$_2$ in adulthood	−1.60	−2.58	−0.62	−0.89	−3.26	1.30	−3.88	−6.12	−0.07	0.92
Birth weight										
BMI trajectories in early adolescence	−0.28	−0.53	0.17	−0.17	−0.63	0.46	−0.36	−1.63	0.87	0.97
BMI trajectories in middle adolescence	0.67	0.27	1.07	0.69	0.07	1.31	0.63	0.02	1.06	0.32
BMI trajectories in late adolescence	−0.23	−0.72	0.23	−0.61	−1.09	2.37	0.10	−0.91	0.81	0.09
BMI trajectories in emerging adulthood	−0.16	−0.36	0.01	−0.03	−1.66	1.24	−0.29	−0.41	−0.03	0.08
BMI trajectories in early adulthood	−0.13	−0.37	−0.02	−0.10	−1.15	0.68	−0.19	−0.47	−0.06	0.79
BMI fluctuation$_1$ in adolescence	0.27	−1.36	0.73	0.07	−2.54	1.67	0.51	−1.60	2.16	0.66
BMI fluctuation$_2$ in adolescence	−0.70	−8.03	6.50	−0.05	−1.80	0.89	−1.24	−2.24	−0.96	0.70
BMI fluctuation$_1$ in adulthood	−0.17	−0.48	−0.05	−0.01	−1.76	0.96	−0.34	−0.63	−0.16	0.35
BMI fluctuation$_2$ in adulthood	−27.10	−45.52	−8.69	−3.45	−7.80	3.24	−34.80	−48.20	−16.65	0.42
Ponderal Index										
BMI trajectories in early adolescence	0.09	0.02	0.19	0.08	0.07	0.23	0.11	−0.07	0.28	0.58
BMI trajectories in middle adolescence	0.03	−0.06	0.19	0.08	−0.26	0.11	−0.01	−0.17	0.12	0.17
BMI trajectories in late adolescence	−0.03	−0.18	−0.02	−0.05	−2.06	−0.23	−0.01	−0.12	0.10	0.26
BMI trajectories in emerging adulthood	0.01	−0.09	0.07	0.02	−0.05	0.09	−0.01	−0.09	0.04	0.21
BMI trajectories in early adulthood	−0.01	−0.08	0.02	0.01	−0.03	0.08	−0.02	−0.14	−0.02	0.41
BMI fluctuation$_1$ in adolescence	0.02	−0.06	0.05	−0.06	−0.21	0.20	0.11	−0.31	0.20	0.26
BMI fluctuation$_2$ in adolescence	0.06	−0.01	0.10	−0.09	−0.23	0.21	0.08	−0.17	0.19	0.32
BMI fluctuation$_1$ in adulthood	−3.72e-03	−0.08	0.03	0.02	−0.04	0.12	−0.02	−0.10	0.06	0.12
BMI fluctuation$_2$ in adulthood	−0.01	−0.20	0.06	0.04	−0.06	0.14	−0.04	−016	0.10	0.18

Caption: Linear regression model examining the association of BMI trajectories and fluctuation (obtained by variance method [1] and Root Mean Square of Successive Differences [2]) at different stages and birth size characteristics are displayed along with robust standard errors clustered by family ID (using the HC3 estimator) to account for within-family correlations at the twin family level. The model has as an outcome BMI trajectories and fluctuation in different stages and includes covariates for Birth size characteristics, Twin order, Cohort ID, Baseline BMI, Age baseline, Zygosity and Sex when including all the individuals. The interaction between birth size characteristic and sex was included as a covariate when checking for sex interactions. Finaly, sex was dropped as a covariate when analyzing males and females separately. **Abbreviations:** BMI: Body mass index; β: Association estimate; CI: Confidence interval; LL: Lower limit; UL: Upper limit.

birth size characteristics and baseline BMI in explaining BMI trajectories and fluctuation was found. Previous studies have reported positive phenotypic associations between different birth size characteristics and BMI in adolescence and adulthood [19,20,54,55]. However, to the best of our knowledge, no previous studies on the association of birth size characteristics and BMI trajectories and fluctuation have been published.

Our results provide evidence of the heterogeneous nature of associations between birth size characteristics and longitudinal BMI outcomes across different life stages. While larger birth size (in terms of birth weight and length) was positively associated with BMI during adolescence and adulthood [19], the associations with BMI trajectories and fluctuation were negative. This suggests that individuals born larger may experience slower increases in BMI over time, particularly during adolescence, in contrast to those born smaller, who may show faster or more pronounced BMI trajectories. This discrepancy underscores the complexity of the relationship between early life factors and later growth patterns, suggesting that birth size characteristics may have a differential effect on static BMI measures compared to dynamic BMI trajectories and fluctuation.

The diverse and occasionally opposing associations between birth size and BMI trajectories probably stem from intricate growth and developmental processes. Early growth patterns, such as catch-up or catch-down growth, can impact BMI differently depending on the timing during development [56]. Changes in gene expression throughout development may affect how genetic and environmental factors shape growth trajectories [57]. Environmental factors like nutrition and physical activity also have varying impacts across different life stages [58,59]. These biological and environmental mechanisms underscore the dynamic and stage-specific nature of how birth size influences BMI trajectory and fluctuations over time.

In the sensitivity analysis, including gestational age and placenta type as additional covariates along with the interactions of birth size characteristics with zygosity and type of placenta, the associations observed between BMI trajectories and birth size characteristics were largely consistent with the main analysis, with no substantial changes in

Table 3
Linear regression model results to assess interactions between birth variables and BMI baseline affecting the trajectories and fluctuation in BMI per 10 years when pooling males and females data from FT12 and FT16.

	Birth size characteristic			BMI baseline			Birth size characteristic: BMI baseline		
	β	95 % CI		β	95 % CI		β	95 % CI	
		LL	UL		LL	UL		LL	UL
Birth length									
BMI trajectories in early adolescence	41.10	−8.83	91.10	0.63	−0.70	1.96	−2.91	−5.76	0.1
BMI trajectories in middle adolescence	−29.62	−84.35	25.10	−1.66	−2.95	0.37	1.85	−0.87	4.59
BMI fluctuation in adulthood	24.68	−6.87	56.27	0.87	−0.17	1.57	−1.31	−2.79	0.16
Birth weight									
BMI trajectories in middle adolescence	−1.61	−4.44	1.22	−1.09	−1.48	−0.70	0.11	−0.03	0.25
BMI trajectories in early adulthood	−0.95	−1.99	0.07	−0.16	−0.28	−0.03	0.03	−0.01	0.10
BMI fluctuation in adulthood	0.39	−1.22	2.01	0.32	0.01	0.52	−0.02	−0.10	0.05
Birth Ponderal Index									
BMI trajectories in early adolescence	−7.71e-03	−0.46	0.44	−0.08	−1.55	−0.21	4.83e-03	−0.02	0.03
BMI trajectories in late adolescence	−0.24	−0.90	0.41	−0.70	−1.50	0.10	9.70e-03	−0.02	0.04

Caption: Linear regression model examining the association of BMI trajectories and fluctuation at different stages and birth size characteristics are displayed along with robust standard errors clustered by family ID (using the HC3 estimator) to account for within-family correlations at the twin family level. The model has as an outcome BMI trajectories and fluctuation in different stages and includes covariates for Birth size characteristics, Twin order, Cohort ID, Baseline BMI, Age baseline, Zygosity, Sex and the interaction between Birth size characteristics and Baseline BMI. **Abbreviations:** β: Association estimate; LL: Lower limit; UL: Upper limit; CI: Confidence interval; BMI: body mass index.

Table 4
Within pair analysis for previously found significant associations between birth size characteristics and BMI trajectories per 10 years in different stages of adolescence and adulthood and fluctuation in two stages of life.

	All twin pairs (N = 4450 pairs)			Monozygotic twin pairs (*N* = 1514 pairs)			Dizygotic twin pairs (*N* = 2936 pairs)			Birth size characteristic-zygosity interaction
	β	95 % CI		β	95 % CI		β	95 % CI		
		LL	UL		LL	UL		LL	UL	p-value
All participants										
Birth length										
Intrapair differences	−0.01			−0.02			−5.69e-03			–
BMI trajectories in early adolescence	−8.45	−39.37	28.74	−7.41	−27.55	12.73	−5.29	−27.23	19.44	0.84
BMI trajectories in middle adolescence	3.58	−29.19	31.31	6.79	−10.03	24.46	−15.87	−37.42	5.68	0.85
BMI fluctuation in adulthood	−2.50	−15.16	25.02	4.45	−9.68	18.38	−10.93	−24.44	2.58	0.57
Birth weight										
Intrapair differences	−8.4e-04			−1.80e-03			−3.50e-04			–
BMI trajectories in middle adolescence	0.40	−1.69	1.30	0.39	−0.58	1.48	−0.63	−1.74	0.74	0.39
BMI trajectories in early adulthood	−0.07	−1.28	0.14	−0.18	−0.61	0.24	−0.39	−0.99	0.18	0.13
BMI fluctuation in adulthood	−0.03	−0.30	1.05	0.21	−0.57	0.97	−0.41	−0.98	0.15	0.40
Birth Ponderal Index										
Intrapair differences	5.36e-03			5.16e-03			5.46e-03			–
BMI trajectories in early adolescence	−0.02	−0.39	0.20	−0.01	−0.21	0.12	−0.08	−0.27	0.11	0.52
BMI trajectories in late adolescence	−0.01	−0.24	0.10	−0.01	−0.13	0.10	−0.15	−0.36	0.09	0.60

Caption: Within-pair analysis was conducted using linear regression models in which each twin's outcome was regressed on the difference in exposure variables within the twin pair. The models used were: Δ BMI trajectory ~ Sex of the pair + Zygosity + Mean Baseline BMI + Baseline Age + Δ Birth size characteristic + Twin order of the pair + Cohort ID + Family ID when including all twin pairs. The interaction between Δ Birth size characteristic and zygosity was included as a covariate in the previous model when checking for zygosity interactions. Finaly, zygosity was dropped as a covariate from the initial model, when analyzing MZ and DZ separately. **Abbreviations:** β: Association estimate; LL: Lower limit; UL: Upper limit; CI: Confidence interval; BMI: body mass index.

direction or strength. The inclusion of gestational age and placenta type did not significantly alter the relationships, indicating that these intrauterine conditions had minimal impact on the overall findings. Nevertheless, other intrauterine conditions, such as maternal nutrition [60], placental function [12,13,61], and hormonal environment [62], may play a crucial role in shaping both birth size characteristics and subsequent BMI development. These early-life influences could contribute to the observed differences in BMI trajectories, potentially affecting metabolic programming and growth regulation mechanisms that persist in adolescence and adulthood.

Finally, no associations between birth characteristics and BMI trajectories remained significant in within-pair analysis, suggesting that intrauterine environmental factors that differ between twin fetuses do not contribute to this association. While environmental factors underpinning the correlations between birth size characteristics and later BMI have been reported in previous studies [54], to our knowledge, the current study is the first to investigate how environmental effects may underpin associations between birth size characteristics and BMI trajectories and fluctuation. It is possible that genetic factors, rather than environmental ones, primarily influence the relationship between birth

size characteristics and BMI across the lifespan. Alternatively, the environmental factors acting at the individual level may differ from those influencing twins, particularly in a shared environment. In twin pairs, both genetic and environmental factors are more tightly controlled, which could mean that the unique environmental factors (those experiences or exposures that differ between individuals within twin pairs) may play a more subtle role in shaping BMI trajectories. On the other hand, the shared environmental factors, such as maternal health behavior or socioeconomic conditions, may be more important for the associations between birth size characteristics and BMI trajectories and fluctuation later in life.

The current study has several strengths and limitations. We used population-based data collected over two decades, covering a period from puberty to early adulthood and including five measurement time points in two different cohorts. Examination of BMI change rates during distinct developmental stages allowed us to focus on specific periods that may be more sensitive to early-life influences. Calculating BMI trajectories based on changes between two consecutive time points per stage provided localized estimates of growth, yielding valuable insights into developmental BMI dynamics that might be missed by modeling overall trajectories alone. However, two or three measurements may be insufficient to capture accurate BMI trajectories and fluctuation. This limitation could lead to an underestimation of true variations, particularly for individuals who experience rapid weight loss or gain over short periods. To assess the effect of these sparse measurements on the accuracy of fluctuation estimates, we conducted additional sensitivity analyses using alternative methods. This involved calculating the standard deviation of available BMI measures and examining their correlations (Supplementary table 5). These analyses indicated that BMI fluctuation estimates may differ depending on the number of available measurements, especially during adolescence. This underscores the importance of more frequent assessments to accurately capture the dynamics of BMI fluctuation.

It is important to note that the use of a shared measurement point between two stages to obtain BMI trajectories in consecutive stages and BMI fluctuation in consecutive stages may have increased the association estimates and their significance. BMI alone does not provide information about body composition, so including other indicators, such as body fat percentage, would be beneficial. While we controlled for some intrauterine conditions such as gestational age and type of placenta, we did not have information for other potential intrauterine conditions that may influence birth size characteristics and subsequent BMI development. Except for parental education [63], we lack information on childhood socioeconomic status, dietary habits, and physical activity during critical developmental periods. These factors are known to influence long-term BMI trajectories and may partially explain associations attributed to shared environmental influences in our study. Participant attrition over time represents another limitation. In FT12, the participation rate was 87 % in the first wave and declined over subsequent surveys, reaching 43 % in the final survey; in FT16, the participation rate started at 90 % and even though it increased in the two subsequent surveys, it decreased in the final survey, reaching 72 %. This may have affected the generalizability and reliability of the findings. Another limitation is that 80 % of the MZ twins appeared to consist of monochorionic twins, which is likely to be an overestimate. Finally, the relatively small sample size may have reduced the statistical power to detect significant associations and subtle interaction effects. This may be particularly true for within-pair models, where statistical power was likely limited, especially in analyses involving birth length. Thus, it would be valuable to replicate these analyses in larger samples, which could provide a clearer understanding of how different factors interact in shaping BMI trajectories and fluctuation over time.

In conclusion, our findings suggest that birth size characteristics have a long-term influence on BMI trajectories and fluctuation in adolescence and adulthood. However, these associations are driven by genetic factors or environmental factors, both intrauterine and postnatal, that are common for both twins rather than those differing during pregnancy. Since the effect sizes were small, these results should be replicated in other cohorts. The results emphasize the need for further research on early-life factors influencing later weight gain.

CRediT authorship contribution statement

Alvaro Obeso: Writing – original draft, Formal analysis, Data curation, Conceptualization. **Aline Jelenkovic:** Writing – review & editing. **Gabin Drouard:** Writing – review & editing. **Jaakko Kaprio:** Writing – review & editing. **Karri Silventoinen:** Writing – review & editing, Conceptualization.

Author contributions

The study design was developed by AO and discussed with KS. The statistical analyses were performed by AO. JK participated in the data collection. AO wrote the original manuscript. All authors actively participated in the improvement of the manuscript by critically revising it. All the authors read and approved the final version of the manuscript.

Ethics statement

For the FT12 study, the ethics committee of the Helsinki University Central Hospital District (HUS) approved the most recent data collection (wave 5) (HUS/2226/2021, dated September 22, 2021) as well as the previously collected data. For the FT16 study, the ethics committee of the Department of Public Health of the University of Helsinki and the Institutional Review Board (IRB) of Indiana University approved the study. The participants provided informed consent when participating in the surveys.

Funding

AO, KS and JK have been supported by the BETTER4U project, which has received funding from the European Union's Horizon Europe Research and Innovation programme under Grant Agreement n° 101080117, by UK Research and Innovation (UKRI) under the UK government's Horizon Europe funding guarantee (grant number 10093560 for QMUL and 10106435 for BiB) and from the Swiss State Secretariat for Education, Research and Innovation (SERI). Views and opinions expressed are, however, those of the author(s) only and do not necessarily reflect those of the European Union. Phenotype and genotype data collection in the twin cohort has been supported by the Wellcome Trust Sanger Institute, the Broad Institute, ENGAGE – European Network for Genetic and Genomic Epidemiology, FP7-HEALTH-F4–2007, grant agreement number 201413, National Institute of Alcohol Abuse and Alcoholism (grants AA-12502, AA-00145, and AA-09203 to R J Rose; AA15416 and K02AA018755 to D M Dick; and AA015416 to Jessica Salvatore) and the Academy of Finland (grants 100499, 205585, 118555, 141054, 264146 and 308248 to J Kaprio). J Kaprio acknowledges support by the Academy of Finland (grants 265240, 263278) and by the Academy of Finland Centre of Excellence in Complex Disease Genetics (grants 312073, 336823 and 352792).

Declaration of competing interest

The authors declared no potential conflicts of interest with respect to the research, authorship, and/or publication of this article.

Acknowledgements

Not applicable.

Code availability

All R scripts are available from the corresponding author.

Appendix A. Supplementary data

Supplementary data to this article can be found online at https://doi.org/10.1016/j.earlhumdev.2025.106373.

Data availability

The FT12 data is not publicly available due to the restrictions of informed consent. However, the FT12 data is available through the Institute for Molecular Medicine Finland (FIMM) Data Access Committee (DAC) (fimmdac@helsinki.fi) for authorized researchers who have IRB/ethics approval and an institutionally approved study plan. To ensure the protection of privacy and compliance with national data protection legislation, a data use/transfer agreement is needed, the content and specific clauses of which will depend on the nature of the requested data.

References

[1] World Health Organization, Obesity and overweight, June 21, https://www.who.int/news-room/fact-sheets/detail/obesity-and-overweight, 2021.

[2] World Obesity Federation, World obesity atlas. https://data.worldobesity.org/publications/?cat=19, 2023.

[3] World Health Organization, Obesity and overweight, 2020 https://www.who.int/news-room/fact-sheets/detail/obesity-and-overweight.

[4] H.H. Maes, M.C. Neale, L.J. Eaves, Genetic and environmental factors in relative body weight and human adiposity, Behav. Genet. 27 (4) (1997) 325–351.

[5] K. Silventoinen, A. Jelenkovic, R. Sund, Y.M. Hur, Y. Yokoyama, C. Honda, J. Kaprio, Genetic and environmental effects on body mass index from infancy to the onset of adulthood: an individual-based pooled analysis of 45 twin cohorts participating in the COllaborative project of development of anthropometrical measures in twins (CODATwins) study, Am. J. Clin. Nutr. 104 (2) (2016) 371–379.

[6] K. Silventoinen, A. Jelenkovic, R. Sund, Y. Yokoyama, Y.M. Hur, W. Cozen, J. Kaprio, Differences in genetic and environmental variation in adult BMI by sex, age, time period, and region: an individual-based pooled analysis of 40 twin cohorts, Am. J. Clin. Nutr. 106 (2) (2017) 457–466.

[7] I. Rogers, The influence of birthweight and intrauterine environment on adiposity and fat distribution in later life, Int. J. Obes. 27 (7) (2003) 755–777. Lond.

[8] W.H. Dietz, Critical periods in childhood for the development of obesity, Am. J. Clin. Nutr. 59 (5) (1994) 955–959.

[9] C.V. Farewell, J. Puma, Z.M. Thayer, S. Morton, Prenatal stress and early childhood body mass index: a path analysis approach, Matern. Child Health J. 25 (3) (2021) 439–449, https://doi.org/10.1007/s10995-020-03060-9.

[10] A. Matsas, P. Panopoulou, N. Antoniou, A. Bargiota, A. Gryparis, N. Vrachnis, G. Mastorakos, S.N. Kalantaridou, T. Panoskaltsis, N.F. Vlahos, G. Valsamakis, Chronic stress in pregnancy is associated with low birth weight: a Meta-analysis, J. Clin. Med. 12 (24) (2023) 7686, https://doi.org/10.3390/jcm12247686.

[11] J. Smith, K. Cianflone, S. Biron, F.S. Hould, S. Lebel, S. Marceau, O. Lescelleur, L. Biertho, S. Simard, J.G. Kral, P. Marceau, Effects of maternal surgical weight loss in mothers on intergenerational transmission of obesity, J. Clin. Endocrinol. Metab. 94 (11) (2009) 4275–4283, https://doi.org/10.1210/jc.2009-0709.

[12] C.M. Salafia, J. Zhang, A.K. Charles, M. Bresnahan, P. Shrout, W. Sun, E.M. Maas, Placental characteristics and birthweight, Paediatr. Perinat. Epidemiol. 22 (3) (2008) 229–239.

[13] R.N. Beaumont, N.M. Warrington, A. Cavadino, J. Tyrrell, M. Nodzenski, M. Horikoshi, R.M. Freathy, Genome-wide association study of offspring birth weight in 86 577 women identifies five novel loci and highlights maternal genetic effects that are independent of fetal genetics, Hum. Mol. Genet. 27 (4) (2018) 742–756.

[14] C.V. Farewell, J. Puma, Z.M. Thayer, S. Morton, Prenatal stress and early childhood body mass index: a path analysis approach, Matern. Child Health J. 25 (3) (2021) 439–449, https://doi.org/10.1007/s10995-020-03060-9.

[15] B. Leppert, K.M. Junge, S. Röder, et al., Early maternal perceived stress and children's BMI: longitudinal impact and influencing factors, BMC Public Health 18 (2018) 1211, https://doi.org/10.1186/s12889-018-6110-5.

[16] S.M. Teo, R. Segurado, A. Douglass, F.M. McAuliffe, C. Murrin, C.C. Kelleher, C. M. Phillips, Associations between placental parameters and childhood BMI trajectories from birth to nine years of age in the lifeways cross-generation cohort, Am. J. Clin. Nutr. (2025), https://doi.org/10.1016/j.ajcnut.2025.03.016. S0002-9165(25)00144–3. Advance online publication.

[17] K. Baptiste-Roberts, C.M. Salafia, W.K. Nicholson, A. Duggan, N.Y. Wang, F. L. Brancati, Gross placental measures and childhood growth, The journal of maternal-fetal & neonatal medicine: the official journal of the European Association of Perinatal Medicine, the Federation of Asia and Oceania Perinatal Societies, the International Society of Perinatal Obstetricians 22 (1) (2009) 13–23, https://doi.org/10.1080/14767050802415728.

[18] J. Baran, A. Weres, E. Czenczek-Lewandowska, J. Leszczak, K. Kalandyk-Osinko, A. Mazur, Relationship between children's birth weight and birth length and a risk of overweight and obesity in 4-15-year-old children, Medicina (Kaunas) 55 (8) (2019) 487, https://doi.org/10.3390/medicina55080487.

[19] A. Jelenkovic, Y. Yokoyama, R. Sund, K.H. Pietiläinen, Y.M. Hur, G. Willemsen, M. Bartels, T.C.E.M. van Beijsterveldt, S. Ooki, K.J. Saudino, M.A. Stazi, C. Fagnani, C. D'Ippolito, T.L. Nelson, K.E. Whitfield, A. Knafo-Noam, D. Mankuta, L. Abramson, K. Heikkilä, T.L. Cutler, K. Silventoinen, Association between birthweight and later body mass index: an individual-based pooled analysis of 27 twin cohorts participating in the CODATwins project, Int. J. Epidemiol. 46 (5) (2017) 1488–1498, https://doi.org/10.1093/ije/dyx031.

[20] K.H. Pietiläinen, J. Kaprio, M. Räsänen, T. Winter, A. Rissanen, R.J. Rose, Tracking of body size from birth to late adolescence: contributions of birth length, birth weight, duration of gestation, parents' body size, and twinship, Am. J. Epidemiol. 154 (1) (2001) 21–29.

[21] F. Rasmussen, M. Johansson, The relation of weight, length and ponderal index at birth to body mass index and overweight among 18-year-old males in Sweden, Eur. J. Epidemiol. 14 (4) (1998) 373–380, https://doi.org/10.1023/a:1007411613576.

[22] R.N. Beaumont, C. Flatley, M. Vaudel, X. Wu, J. Chen, G.H. Moen, P.R. Njølstad, Genome-wide association study of placental weight identifies distinct and shared genetic influences between placental and fetal growth, Nat. Genet. 55 (11) (2023) 1807–1819.

[23] Y. Yokoyama, A. Jelenkovic, Y.M. Hur, R. Sund, C. Fagnani, M.A. Stazi, K. Silventoinen, Genetic and environmental factors affecting birth size variation: a pooled individual-based analysis of secular trends and global geographical differences using 26 twin cohorts, Int. J. Epidemiol. 47 (4) (2018) 1195–1206.

[24] R. Hardy, et al., Life course variations in the associations between fto and mc4r gene variants and body size, Hum. Mol. Genet. 19 (2010) 545–552. https://www.ncbi.nlm.nih.gov/pubmed/19880856.

[25] J. Min, D.T. Chiu, Y. Wang, Variation in the heritability of body mass index based on diverse twin studies: a systematic review, Obes. Rev. 14 (11) (2013) 871–882.

[26] L. Yengo, J. Sidorenko, K.E. Kemper, Z. Zheng, A.R. Wood, M.N. Weedon, T. M. Frayling, Meta-analysis of genome-wide association studies for height and body mass index in ~700,000 individuals of European ancestry, Hum. Mol. Genet. 27 (20) (2018) 3641–3649.

[27] A.E. Locke, B. Kahali, S.I. Berndt, A.E. Justice, T.H. Pers, F.R. Day, J. Lindström, Genetic studies of body mass index yield new insights for obesity biology, Nature 518 (7538) (2015) 197–206.

[28] G. Roberts, et al., Growth of extremely preterm survivors from birth to 18 years of age compared with term controls, Pediatrics 131 (2) (2013) e439–e445.

[29] E.M. Boyle, et al., Effects of gestational age at birth on health outcomes at 3 and 5 years of age: population based cohort study, BMJ 344 (2012) e896.

[30] J.L. Vinther, C.T. Ekstrøm, T.I.A. Sørensen, et al., Gestational age and trajectories of body mass index and height from birth through adolescence in the Danish National Birth Cohort, Sci. Rep. 13 (2023) 3298.

[31] P. Markopoulou, E. Papanikolaou, A. Analytis, E. Zoumakis, T. Siahanidou, Preterm birth as a risk factor for metabolic syndrome and cardiovascular disease in adult life: a systematic review and meta-analysis, J. Pediatr. 210 (69–80) (2019) e5.

[32] M. Sipola-Leppanen, E. Kajantie, Should we assess cardiovascular risk in young adults born preterm? Curr. Opin. Lipidol. 26 (4) (2015) 282–287.

[33] I. Blickstein, L.G. Keith (Eds.), Multiple Pregnancy: Epidemiology, Gestation, and Perinatal Outcome, CRC Press, 2005.

[34] A.L. Fick, V.A. Feldstein, M.E. Norton, C.W. Fyr, A.B. Caughey, G.A. Machin, Unequal placental sharing and birth weight discordance in monochorionic diamniotic twins, Am. J. Obstet. Gynecol. 195 (1) (2006) 178–183.

[35] A. Victoria, G. Mora, F. Arias, Perinatal outcome, placental pathology, and severity of discordance in monochorionic and dichorionic twins, Obstet. Gynecol. 97 (2) (2001) 310–315.

[36] M. Geserick, M. Vogel, R. Gausche, T. Lipek, U. Spielau, E. Keller, A. Körner, Acceleration of BMI in early childhood and risk of sustained obesity, N. Engl. J. Med. 379 (14) (2018) 1303–1312, https://doi.org/10.1056/NEJMoa1803527.

[37] X. Li, C.D.G. Keown-Stoneman, G. Lebovic, J.A. Omand, K. Adeli, J.K. Hamilton, A. J. Hanley, M. Mamdani, B.W. McCrindle, J.L. Sievenpiper, M.S. Tremblay, J. L. Maguire, P.C. Parkin, C.S. Birken, TARGet Kids! Collaboration, The association between body mass index trajectories and cardiometabolic risk in young children, Pediatr. Obes. 15 (8) (2020) e12633, https://doi.org/10.1111/ijpo.12633.

[38] L. Lissner, P.M. Odell, R.B. D'Agostino, J. Stokes 3rd, B.E. Kreger, A.J. Belanger, K. D. Brownell, Variability of body weight and health outcomes in the Framingham population, N. Engl. J. Med. 324 (26) (1991) 1839–1844, https://doi.org/10.1056/NEJM199106273242602.

[39] S.A. French, A.R. Folsom, R.W. Jeffery, W. Zheng, P.J. Mink, J.E. Baxter, Weight variability and incident disease in older women: the Iowa women's health study, Int. J. Obes. Relat. Metab. Disord. 21 (3) (1997) 217–223, https://doi.org/10.1038/sj.ijo.0800390.

[40] E.J. Rhee, Weight cycling and its Cardiometabolic impact, Journal of obesity & metabolic syndrome 26 (4) (2017) 237–242, https://doi.org/10.7570/jomes.2017.26.4.237.

[41] J. Kaprio, L. Pulkkinen, R.J. Rose, Genetic and environmental factors in health-related behaviors: studies on Finnish twins and twin families, Twin Res. 5 (4) (2002) 366–371.

[42] R.J. Rose, J.E. Salvatore, S. Aaltonen, P.B. Barr, L.H. Bogl, H.A. Byers, J. Kaprio, Finntwin12 cohort: an updated review, Twin Res. Hum. Genet. 22 (5) (2019) 302–311.

[43] M.E. Cooke, E. Lumpe, M. Stephenson, M. Urjansson, F. Aliev, T. Palviainen, S. J. Brislin, M. Piirtola, J. Rabinowitz, A. Latvala, P.B. Barr, E. Vuoksimaa, H.H. M. Maes, R. Viken, R.J. Rose, J. Kaprio, D.M. Dick, S. Aaltonen, J.E. Salvatore, Alcohol use in early midlife: Findings from the age 37 follow-up assessment of the FinnTwin12 cohort, Behav. Genet. 55 (2) (2025) 124–140, https://doi.org/10.1007/s10519-024-10212-y.

[44] M. Kaidesoja, S. Aaltonen, L.H. Bogl, K. Heikkilä, S. Kaartinen, U.M. Kujala, U. Kärkkäinen, G. Masip, L. Mustelin, T. Palviainen, K.H. Pietiläinen, M. Rottensteiner, P.N. Sipilä, R.J. Rose, A. Keski-Rahkonen, J. Kaprio, FinnTwin16: a longitudinal study from age 16 of a population-based Finnish twin cohort, Twin research and human genetics: the official journal of the International Society for Twin Studies 22 (6) (2019) 530–539, https://doi.org/10.1017/thg.2019.106.

[45] A. Jelenkovic, A. Ortega-Alonso, R.J. Rose, J. Kaprio, E. Rebato, K. Silventoinen, Genetic and environmental influences on growth from late childhood to adulthood: a longitudinal study of two Finnish twin cohorts, Am. J. Hum. Biol. 23 (6) (2011) 764–773, https://doi.org/10.1002/ajhb.21208.

[46] U. Sankilampi, M.-L. Hannila, A. Saari, M. Gissler, L. Dunkel, New population-based references for birth weight in singletons by gestational age in Finland, Ann. Med. 45 (5–6) (2013) 446–454.

[47] K. Salmela-Aro, Stages of adolescence, in: Encyclopedia of Adolescence, Academic press, 2011, pp. 360–368.

[48] Theresa Kyle D. N. P., Primary Care Pediatrics for the Nurse Practitioner: A Practical Approach, Springer Publishing Company, 2021.

[49] S. Tyler, Physical development in early adulthood, in: Human Behavior and the Social Environment I, 2020.

[50] A.H. Mokdad, M.K. Serdula, W.H. Dietz, B.A. Bowman, J.S. Marks, J.P. Koplan, The spread of the obesity epidemic in the United States, 1991-1998, Jama 282 (16) (1999) 1519–1522.

[51] C.L. Ogden, M.D. Carroll, L.R. Curtin, M.A. McDowell, C.J. Tabak, K.M. Flegal, Prevalence of overweight and obesity in the United States, 1999-2004, Jama 295 (13) (2006) 1549–1555.

[52] D. Boomsma, et al., Twins and the fetal origins hypothesis: An application to growth data, in: C. Kordon, R.C. Gaillard, Y. Christen (Eds.), Hormones and the Brain. Research and Perspectives in Endocrine Interactions, eds, Springer, Berlin, Heidelberg, 2005, https://doi.org/10.1007/3-540-26940-1_2.

[53] R. Morley, T. Dwyer, Studies of twins: what can they tell us about the fetal origins of adult disease? Paediatr. Perinat. Epidemiol. 19 (2005) 2–7.

[54] K. Silventoinen, K.H. Pietiläinen, P. Tynelius, T.I. Sørensen, J. Kaprio, F. Rasmussen, Genetic and environmental factors in relative weight from birth to age 18: the Swedish young male twins study, International journal of obesity 31 (4) (2007) 615–621, https://doi.org/10.1038/sj.ijo.0803577, 2005.

[55] Z. Yu, S. Han, J. Zhu, X. Sun, C. Ji, X. Guo, The relationship between birth weight and the risk of overweight and obesity in childhood and adolescence: a meta-analysis, BMC Public Health 11 (1) (2011) 542, https://doi.org/10.1186/1471-2458-11-542.

[56] K.K. Ong, R.J. Loos, Rapid infancy weight gain and subsequent obesity: systematic reviews and hopeful suggestions, Acta Paediatr. 95 (8) (2006) 904–908, https://doi.org/10.1080/08035250600719754. Oslo, Norway: 1992.

[57] K. Silventoinen, K.H. Pietiläinen, P. Tynelius, T.I. Sørensen, J. Kaprio, F. Rasmussen, Genetic regulation of growth from birth to 18 years of age: the Swedish young male twins study, Am. J. Hum. Biol. 20 (3) (2008) 292–298, https://doi.org/10.1002/ajhb.20717.

[58] A.K. Dahl, C.A. Reynolds, T. Fall, P.K. Magnusson, N.L. Pedersen, Multifactorial analysis of changes in body mass index across the adult life course: a study with 65 years of follow-up, International journal of obesity (2005) 38 (8) (2014) 1133–1141, https://doi.org/10.1038/ijo.2013.204.

[59] K. Silventoinen, A. Jelenkovic, R. Sund, Y. Yokoyama, Y.M. Hur, W. Cozen, J. Kaprio, Differences in genetic and environmental variation in adult BMI by sex, age, time period, and region: an individual-based pooled analysis of 40 twin cohorts, Am. J. Clin. Nutr. 106 (2) (2017) 457–466.

[60] A.R. Chia, L.W. Chen, J.S. Lai, C.H. Wong, N. Neelakantan, R.M. Van Dam, M.F. F. Chong, Maternal dietary patterns and birth outcomes: a systematic review and meta-analysis, Adv. Nutr. 10 (4) (2019) 685–695.

[61] M.C. Roland, C.M. Friis, N. Voldner, K. Godang, J. Bollerslev, G. Haugen, T. Henriksen, Fetal growth versus birthweight: the role of placenta versus other determinants, PLoS One 7 (6) (2012) e39324, https://doi.org/10.1371/journal.pone.0039324.

[62] L. Page, N. Younge, M. Freemark, Hormonal determinants of growth and weight gain in the human fetus and preterm infant, Nutrients 15 (18) (2023) 4041, https://doi.org/10.3390/nu15184041.

[63] H.R. Lajunen, J. Kaprio, R.J. Rose, L. Pulkkinen, K. Silventoinen, Genetic and environmental influences on BMI from late childhood to adolescence are modified by parental education, Obesity (Silver Spring, Md.) 20 (3) (2012) 583–589, https://doi.org/10.1038/oby.2011.304.