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Growth Patterns in Young Children with Food Allergy: A Matched Cross-Sectional and Longitudinal Study

Yu-Jin Kwon [1,2,†], Sujin Kim [3,†], Eun Gyeong Seol [4], Hye Sun Lee [5], Eunju Lee [5], Yong Ju Lee [4], Junghwan Suh [3] and Min Jung Kim [4,*]

[1] Department of Family Medicine, Severance Hospital, Yonsei University College of Medicine, Seoul 03722, Republic of Korea; digda3@yuhs.ac
[2] Medical NutriCare Innovation Research Institute, Yonsei University, Seoul 03722, Republic of Korea
[3] Department of Pediatrics, Severance Children's Hospital, Yonsei University College of Medicine, Seoul 03722, Republic of Korea
[4] Department of Pediatrics, Yongin Severance Hospital, Yonsei University College of Medicine, Yongin 16995, Republic of Korea
[5] Biostatistics Collaboration Unit, Yonsei University College of Medicine, Seoul 03722, Republic of Korea
* Correspondence: mj1217@yuhs.ac; Tel.: +82-31-5189-8776
† These authors contributed equally to this work.

Abstract

Background/Objectives: Growth in young children with IgE-mediated food allergy (FA) remains unclear, particularly whether deficits are present at diagnosis or emerge during follow-up. We compared growth at diagnosis and subsequent growth trajectories between children with FA and healthy controls. Methods: We retrospectively analyzed children younger than 6 years with IgE-mediated FA (n = 176) and healthy controls (n = 340), each with at least two anthropometric records, at a single center in Korea. Children with FA were matched 1:1 to controls by age and sex using propensity scores (149 pairs). Height, weight, and body mass index (BMI) standard deviation scores (SDS) were calculated using the 2017 Korean National Growth Charts and compared using linear mixed models stratified by age at the first measurement (<1 vs. ≥1 year). Results: Baseline SDS did not differ between groups. Height SDS declined faster in children with FA than in controls (−0.233 vs. −0.102 SDS/year; difference −0.131, 95% confidence interval −0.222 to −0.040; $p = 0.005$), with a significant between-group difference among children aged ≥1 year but not among those aged <1 year. Weight and BMI SDS trajectories did not differ between groups. In exploratory within-FA analyses, infants with an allergy to ≥2 foods showed steeper declines in height (difference −0.164 SDS/year; $p = 0.044$) and weight SDS (difference −0.263 SDS/year; $p < 0.001$) than infants allergic to egg or milk only. Conclusions: Despite comparable growth at diagnosis, children with IgE-mediated FA showed a steeper subsequent decline in height SDS. The clinical significance of this difference and the value of serial growth assessment or targeted nutritional management require prospective evaluation.

Keywords: food allergy; growth; children; propensity score matching; linear mixed model

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1. Introduction

IgE-mediated food allergy (FA) is common in early childhood. Current guidelines recommend avoiding confirmed culprit foods while maintaining tolerated foods and providing age-appropriate, individualized dietary advice [1,2]. However, eliminating nutrient-dense staple foods such as cow's milk, egg, wheat, and soy during infancy and the preschool years, when growth is rapid and nutritional requirements are high, may compromise the intake of

energy, high-quality protein, calcium, vitamin D, and other nutrients [3,4]. Recent studies continue to report lower height or weight indices in children with FA, particularly in those with cow's milk allergy or multiple food allergies [4–7].

Recent clinical reviews continue to identify growth faltering and nutrient inadequacy as important concerns in children with FA and emphasize regular nutritional and growth assessment [8]. In a population-based birth cohort followed from infancy to age 4 years, FA coexisting with eczema and persistent FA, but not resolved FA, were associated with reduced growth [9].

However, the evidence is not uniform. In a cross-sectional study from Singapore, wasting (1.4%) and stunting (5.4%) were uncommon, although stunting was associated with eczema and with IgE-mediated or mixed-type FA [10]. A nonrandomized intervention study also suggested that dietary counseling by a multidisciplinary team can improve anthropometric measures and energy intake in young children with FA [11].

Longitudinal evidence is limited, however. A recent systematic review of children with multiple FAs included 11 studies, of which only two reported longitudinal growth observations [7]. The most relevant prospective birth-cohort study analyzed weight-for-length up to 2 years but did not examine height or length and compared children with single or multiple IgE-mediated FA with unaffected children rather than with each other [12]. It therefore remains unclear whether linear growth diverges from that of matched healthy children after the diagnosis of IgE-mediated FA, how this differs by age, and whether growth trajectories differ within IgE-mediated FA according to the number of causative foods. The roles of atopic comorbidities and nutritional counseling also remain unclear.

Answering these questions requires age- and sex-standardized measures, appropriate controls, and serial measurements because growth and dietary patterns change rapidly with age in early childhood. In Korea, the 2017 National Growth Charts, which incorporate the World Health Organization (WHO) Child Growth Standards for children younger than 3 years and national reference data thereafter, provide a suitable framework for standardized assessment [13,14].

We therefore compared growth at diagnosis between children younger than 6 years with IgE-mediated FA and age- and sex-matched healthy controls, compared height, weight, and BMI SDS trajectories after diagnosis using linear mixed models stratified by age at first measurement, and examined, within the FA cohort, whether trajectories differed by the number of causative foods and other clinical factors, including atopic comorbidities and nutritional counseling.

2. Materials and Methods

2.1. Study Design and Participants

This retrospective matched cross-sectional and longitudinal study included children younger than 6 years (<72 months) diagnosed with IgE-mediated FA at Yongin Severance Hospital between March 2020 and June 2026. Electronic medical records were reviewed for demographic characteristics, allergic diseases, implicated food allergens, anthropometric measurements, nutritional counseling, and relevant perinatal and clinical factors.

Children with FA were eligible if IgE-mediated FA was diagnosed before 6 years of age and at least two anthropometric records were available. IgE-mediated FA was defined as either (1) a positive oral food challenge or (2) a convincing history of an immediate allergic reaction after ingestion of a specific food, together with sensitization to that food (serum food-specific IgE $\geq$ 0.35 kU/L). Sensitization alone, without a compatible clinical reaction, was not considered sufficient for diagnosis [1].

Among children with IgE-mediated FA, causative foods were categorized as egg only, milk only, two foods, or three or more foods and were also dichotomized as egg or milk

only versus two or more foods (≥2 foods). Clinical variables included sex, age, gestational age, birth weight, mode of delivery (normal spontaneous vaginal delivery [NSVD] vs. cesarean section [C/S]), past history (PHx) and family history (FHx) of atopic diseases, pet ownership (dog or cat), allergic comorbidities, and dietary (nutritional) counseling. Allergic comorbidities comprised atopic dermatitis (AD), allergic rhinitis (AR), asthma, and allergic conjuctivitis. The other conditions included urticaria, and gastrointestinal symptoms or disorders not classified elsewhere. When provided, nutritional counseling was delivered by a dietitian from the hospital nutrition team at the time of FA diagnosis, using standardized educational materials and counseling forms for major food allergens (cow's milk, egg, wheat, walnut, peanut, and soy) that had been jointly reviewed by the treating physicians and the nutrition team.

Healthy controls were selected from children who visited the same institution for routine vaccination, well-child health examinations, or general health counseling. Eligible controls were younger than 6 years, had at least two visits with height/length and weight measurements before age 6 years, and had no documented history of FA or major chronic diseases that could affect growth. Laboratory data were not routinely available for healthy controls and were therefore not included in the comparisons between the IgE-mediated FA and control groups.

For both groups, exclusion criteria were (1) ongoing growth surveillance specifically related to prematurity or low birth weight; (2) chronic medical conditions, congenital disorders, or long-term medication use that could substantially affect growth; (3) anthropometric measurements obtained during or immediately before or after a severe acute illness or hospitalization; and (4) unavailable or unreliable height/length or weight measurements (Figure 1). Prematurity or low birth weight was not an exclusion criterion unless the child was undergoing ongoing growth surveillance for these conditions.

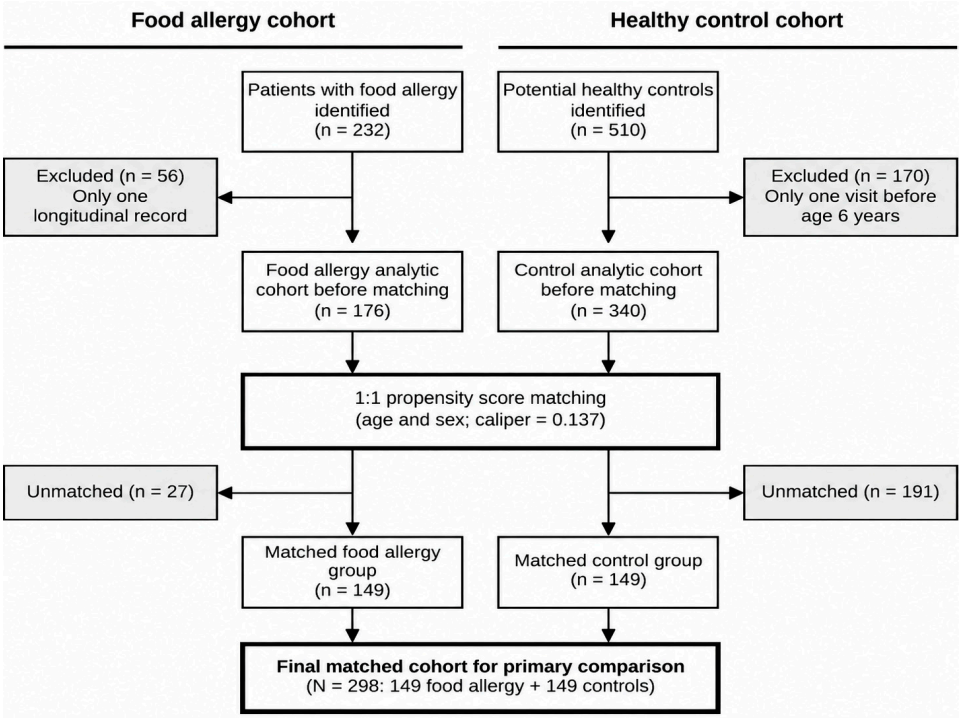


Figure 1. Flow diagram of participant selection and propensity score matching.

2.2. Propensity Score Matching and Index Date

For children with FA, the index date was defined as the date of initial FA diagnosis. For controls, the date of the visit used for matching was designated as the index date. To reduce confounding by age and sex, each child with FA was matched 1:1 to a healthy control by propensity score matching (PSM) based on age and sex, as described in the Statistical Analysis section.

Analyses were additionally stratified according to age at the first measurement into <1 year and ≥1 year. Matching was repeated within each stratum, with calipers of 0.178 for children aged <1 year and 0.065 for those aged ≥1 year.

2.3. Anthropometric Assessment

We extracted all available height, recumbent length, and weight measurements, along with their measurement dates. For children with FA, baseline was defined as the date of diagnosis, and baseline anthropometry was defined as the measurement obtained closest to, and on or before, that date. For controls, baseline was defined as the vaccination or well-child visit used for matching. All measurements from baseline through the end of follow-up were included in the longitudinal analysis. Age- and sex-specific standard deviation scores (SDS; z-scores) were calculated using the 2017 Korean National Growth Charts [13], which incorporate the WHO Child Growth Standards for children aged 0–35 months. Because SDS are continuous measures that retain more information than percentiles, they were used as the outcomes in all longitudinal analyses. The following standardized growth indices were evaluated: height SDS (length SDS for children measured in the recumbent position), weight SDS, and body mass index (BMI) SDS, the last of which could be calculated only from 24 months of age onward.

2.4. Statistical Analysis

Continuous variables are presented as mean ± standard deviation, and categorical variables as number (percentage). Among participants with food allergy, we compared groups by food allergy type using one-way analysis of variance (ANOVA) for continuous variables and the chi-square test for categorical variables, with Bonferroni correction for post hoc pairwise comparisons.

To compare growth trajectories between participants with food allergy and controls, propensity score matching (PSM) was performed based on age and sex using 1:1 nearest-neighbor matching. A caliper width of 0.2 standard deviations of the logit of the propensity score was used (0.137 in the total cohort; 0.178 and 0.065 in participants aged <1 year and ≥1 year, respectively) [15]. Covariate balance after matching was assessed using standardized differences (SDs), with an absolute SD of <0.2 indicating adequate balance between groups [16].

Longitudinal growth trajectories were compared between the food allergy and control groups and, within the food allergy cohort, between participants with a single food allergy (egg or milk only) and those with two or more food allergies using linear mixed models (LMMs). Random intercepts and slopes for time were included to account for within-individual correlation, with an unstructured covariance matrix specified for the random effects. Estimated growth slopes with 95% confidence intervals (CIs) were calculated for each group, and differences in slopes between groups were assessed. Analyses were performed in the total matched cohort and in age strata (<1 year and ≥1 year).

Longitudinal mixed models incorporated all available anthropometric measurements without imputation, under the missing-at-random assumption. Clinical covariates used in the within-FA analyses were analyzed using available cases. Age-stratified and within-FA

subgroup analyses were considered exploratory, and no formal adjustment for multiple comparisons was applied.

In addition, factors associated with growth trajectories among children with FA were evaluated using linear mixed models, with the factor of interest, time, and the factor × time interaction as fixed effects with estimates (β) and 95% CIs reported, and a random intercept and a random slope for time with an unstructured covariance matrix. All analyses were conducted using SAS (version 9.4; SAS Institute Inc., Cary, NC, USA) and R (version 4.6.0; R Foundation for Statistical Computing, Vienna, Austria; http://www.R-project.org), with a p-value < 0.05 considered statistically significant.

3. Results

3.1. Study Population and Propensity Score Matching

After applying the eligibility and data-quality criteria, 176 children with IgE-mediated FA and 340 healthy controls were included before matching (Figure 1). Of the 176 children with IgE-mediated FA, 24 (13.6%) had FA confirmed by a positive oral food challenge, and the remaining 152 (86.4%) were diagnosed based on a convincing history of an immediate reaction to the implicated food together with sensitization to the same food, defined as food-specific serum IgE ≥0.35 kU/L.

Before matching, children with FA were older than controls (1.51 ± 1.40 vs. 0.71 ± 1.14 years; absolute standardized difference [|SD|] 0.629) and more frequently male (68.2% vs. 52.4%; |SD| 0.314; both $p < 0.001$; Table 1). After 1:1 PSM on age and sex, 149 children with FA and 149 controls were matched. The groups were well balanced for age (1.29 ± 1.31 vs. 1.26 ± 1.25 years, $p = 0.852$, |SD| 0.022) and sex (male 66.4% vs. 64.4%, $p = 0.715$, |SD| 0.045). Baseline height SDS (0.46 ± 1.10 vs. 0.42 ± 1.21, $p = 0.790$) and weight SDS (0.66 ± 0.92 vs. 0.66 ± 1.00, $p = 0.958$) were similar between the FA and control groups (Table 1).

Table 1. Baseline demographic and clinical characteristics of the total cohort, before and after propensity score matching.

Variable	Before PSM						After PSM					
	N	Total (N = 516)	Control (N = 340)	Case (N = 176)	*p*-Value	|SD|	N	Total (N = 298)	Control (N = 149)	Case (N = 149)	*p*-Value	|SD|
Sex	516				<0.001	0.314	298				0.715	0.045
Male		298 (57.8)	178 (52.4)	120 (68.2)				195 (65.4)	96 (64.4)	99 (66.4)		
Female		218 (42.2)	162 (47.6)	56 (31.8)				103 (34.6)	53 (35.6)	50 (33.6)		
Baseline age, yr	516	0.98 ± 1.29	0.71 ± 1.14	1.51 ± 1.40	<0.001	0.629	298	1.28 ± 1.28	1.26 ± 1.25	1.29 ± 1.31	0.852	0.022
Height SDS	502	0.42 ± 1.30	0.46 ± 1.36	0.33 ± 1.18	0.292	0.098	286	0.44 ± 1.16	0.42 ± 1.21	0.46 ± 1.10	0.79	0.032
Weight SDS	513	0.61 ± 1.05	0.60 ± 1.11	0.62 ± 0.93	0.82	0.021	296	0.66 ± 0.96	0.66 ± 1.00	0.66 ± 0.92	0.958	0.006
BMI SDS †	76	0.29 ± 1.39	0.23 ± 1.33	0.34 ± 1.44	0.735	0.079	54	0.16 ± 1.11	0.25 ± 1.20	0.06 ± 1.02	0.545	0.166
Gestational age, wk	159			38.18 ± 1.98			134			38.29 ± 1.56		
Birth weight, kg	162			3.19 ± 0.44			136			3.21 ± 0.39		
Food allergy type	176						149					
Egg only				54 (30.7)						51 (34.2)		
Milk only				10 (5.7)						7 (4.7)		
2 foods				48 (27.3)						41 (27.5)		
≥3 foods				64 (36.4)						50 (33.6)		
Food allergy, grouped	176						149					
Egg or milk only				64 (36.4)						58 (38.9)		
≥2 foods				112 (63.6)						91 (61.1)		
Delivery mode	161						135					
NSVD				78 (48.4)						69 (51.1)		
Cesarean section				83 (51.6)						66 (48.9)		
Atopic past history	163						137					
No				46 (28.2)						42 (30.7)		
Yes				117 (71.8)						95 (69.3)		
Atopic family history	161						135					
No				50 (31.1)						40 (29.6)		
Yes				111 (68.9)						95 (70.4)		
Pet ownership	159						133					
None				140 (88.1)						116 (87.2)		
Dog or cat				19 (11.9)						17 (12.8)		

Table 1. *Cont.*

Variable	Before PSM						After PSM				
	N	Total (N = 516)	Control (N = 340)	Case (N = 176)	*p*-Value	|SD|	N	Total (N = 298)	Control (N = 149)	Case (N = 149)	*p*-Value	|SD|
Comorbidities												
Atopic dermatitis	145			61 (42.1)			121			50 (41.3)		
Allergic rhinitis	145			44 (30.3)			121			34 (28.1)		
Asthma	145			12 (8.3)			121			7 (5.8)		
Other	145			103 (71.0)			121			91 (75.2)		
Dietary counseling	165						141					
No				132 (80.0)						110 (78.0)		
Yes				33 (20.0)						31 (22.0)		

Continuous variables are presented as mean ± standard deviation, and categorical variables are presented as number (%). Case: participants with food allergies. Control: participants without food allergies. Propensity score matching (PSM) was performed based on age and sex using 1:1 nearest-neighbor matching with a caliper width of 0.2 standard deviations of the logit of the propensity score (0.137). † Baseline BMI SDS was calculated only for children aged ≥24 months with available height and weight measurements. Data were available for 76 children before matching (34 controls and 42 children with food allergy) and 54 children after matching (28 controls and 26 children with food allergy). Abbreviations: PSM, propensity score matching; |SD|, absolute standardized difference; SDS, standard deviation score; BMI, body mass index; NSVD, normal spontaneous vaginal delivery; C/S, cesarean section. yr: years. wk: weeks.

In the matched cohort, mean follow-up duration was 1.78 ± 1.46 years in the FA group and 1.69 ± 1.72 years in controls. Among children aged <1 year, the corresponding values were 2.02 ± 1.47 and 1.29 ± 1.50 years, respectively; among those aged ≥1 year, they were 1.72 ± 1.53 and 2.32 ± 1.94 years. The mean number of measurements per child was 5.59 ± 4.29 in the FA group and 5.09 ± 3.52 in controls (Supplementary Table S1).

In the matched FA group, 51 children (34.2%) were allergic to egg only, 7 (4.7%) to milk only, 41 (27.5%) to two foods, and 50 (33.6%) to three or more foods; 61.1% were allergic to ≥2 foods. Among children with available data, atopic dermatitis, allergic rhinitis, and asthma were present in 41.3% (50/121), 28.1% (34/121), and 5.8% (7/121), respectively; 22.0% (31/141) had received dietary counseling. After matching, age ($p = 0.035$) and atopic PHx ($p = 0.020$) differed across the four causative-food groups; the other baseline characteristics did not show statistically significant differences (Table 2). Before matching, age and atopic PHx differed across the food groups (Supplementary Table S2). In the age-stratified analyses, in which matching was repeated within each age stratum, 63 pairs were matched in both groups, with |SD| < 0.2 for age (0.001 and 0.019, respectively) and sex (0.112 and <0.001, respectively; Supplementary Table S3A,B).

Table 2. Baseline characteristics of children with food allergies according to the number of causative foods after propensity score matching.

	N	Total (n = 149)	Egg Only (n = 51)	Milk Only (n = 7)	2 Foods (n = 41)	≥3 Foods (n = 50)	*p*-Value
Sex	149						0.164
Male		99 (66.44)	30 (58.82)	5 (71.43)	25 (60.98)	39 (78.00)	
Female		50 (33.56)	21 (41.18)	2 (28.57)	16 (39.02)	11 (22.00)	
Baseline age (years)	149	1.29 ± 1.31	0.95 ± 1.17	0.84 ± 0.98	1.34 ± 1.39	1.67 ± 1.34	**0.035**
Baseline height SDS	141	0.46 ± 1.10	0.53 ± 1.07	0.33 ± 1.42	0.45 ± 1.06	0.41 ± 1.14	0.938
Baseline weight SDS	149	0.66 ± 0.92	0.69 ± 0.89	0.49 ± 0.79	0.60 ± 0.95	0.71 ± 0.96	0.9
Baseline BMI SDS	26	0.06 ± 1.02	−0.48 ± 0.32	0.93	−0.34 ± 0.80	0.41 ± 1.17	0.21
Gestational age (weeks)	134	38.29 ± 1.56	38.14 ± 1.48	38.50 ± 1.38	38.15 ± 1.17	38.53 ± 1.90	0.585
Birth weight (kg)	136	3.21 ± 0.39	3.17 ± 0.38	3.54 ± 0.29	3.22 ± 0.39	3.21 ± 0.41	0.188
Delivery mode	135						0.805
NSVD		69 (51.11)	26 (55.32)	3 (50.00)	16 (44.44)	24 (52.17)	
C/S		66 (48.89)	21 (44.68)	3 (50.00)	20 (55.56)	22 (47.83)	

Table 2. *Cont.*

	N	Total (n = 149)	Egg Only (n = 51)	Milk Only (n = 7)	2 Foods (n = 41)	≥3 Foods (n = 50)	*p*-Value
Atopic past history	137						**0.020**
No		42 (30.66)	18 (37.50)	3 (50.00)	14 (38.89)	7 (14.89)	
Yes		95 (69.34)	30 (62.50)	3 (50.00)	22 (61.11)	40 (85.11)	
Atopic family history	135						0.714
No		40 (29.63)	17 (35.42)	2 (33.33)	9 (25.00)	12 (26.67)	
Yes		95 (70.37)	31 (64.58)	4 (66.67)	27 (75.00)	33 (73.33)	
Pet ownership	133						0.73
None		116 (87.22)	40 (85.11)	5 (83.33)	31 (86.11)	40 (90.91)	
Dog or cat		17 (12.78)	7 (14.89)	1 (16.67)	5 (13.89)	4 (9.09)	
Comorbidities							
Atopic dermatitis	121	50 (41.32)	21 (52.50)	0 (0.00)	12 (36.36)	17 (39.53)	0.115
Allergic rhinitis	121	34 (28.10)	7 (17.50)	2 (40.00)	12 (36.36)	13 (30.23)	0.229
Asthma	121	7 (5.79)	2 (5.00)	0 (0.00)	1 (3.03)	4 (9.30)	0.72
Others	121	91 (75.21)	29 (72.50)	4 (80.00)	24 (72.73)	34 (79.07)	0.906
Dietary counseling	141						0.915
No		110 (78.01)	38 (77.55)	5 (71.43)	31 (81.58)	36 (76.60)	
Yes		31 (21.99)	11 (22.45)	2 (28.57)	7 (18.42)	11 (23.40)	

Continuous variables are presented as mean ± standard deviation (SD), and categorical variables are presented as numbers (%). Participants with food allergies were compared according to the type of food allergy (egg only, milk only, 2 foods, and ≥3 foods) using one-way analysis of variance (ANOVA) for continuous variables and the chi-square test for categorical variables. Propensity score matching was performed based on age and sex using 1:1 nearest-neighbor matching with a caliper width of 0.2 SD of the logit of the propensity score. Bold *p*-values indicate $p < 0.05$. For baseline BMI SDS, the milk-only subgroup included one child; therefore, an SD could not be calculated for that subgroup. Abbreviations: SDS, standard deviation score; BMI, body mass index; NSVD, normal spontaneous vaginal delivery; C/S, cesarean section.

3.2. Growth Trajectories of Children with Food Allergy and Controls

Figure 2A shows that height and weight SDS declined over time in both groups. Height SDS declined more steeply in children with FA than in controls (estimated slope −0.233 [95% CI −0.296 to −0.169] vs. −0.102 [−0.168 to −0.036]; difference −0.131 [−0.222 to −0.040], $p = 0.005$). In contrast, weight SDS (−0.240 vs. −0.225; difference −0.015 [−0.106 to 0.076], $p = 0.744$) and BMI SDS (−0.125 vs. −0.138; difference 0.013 [−0.090 to 0.115], $p = 0.806$) trajectories did not differ between groups (Supplementary Table S4).

In age-stratified analyses, height SDS declined more steeply in the FA group than in controls among children aged ≥1 year (−0.136 [−0.202 to −0.071] vs. −0.011 [−0.069 to 0.047]; difference −0.125 [−0.213 to −0.038], $p = 0.005$). No between-group differences were found for height SDS ($p = 0.419$) or weight SDS ($p = 0.471$) in children aged <1 year, or for weight SDS ($p = 0.912$) in children aged ≥1 year (Supplementary Table S4).

3.3. Growth Trajectories in Children with Food Allergy According to the Number of Causative Foods

Among matched children with FA, growth trajectories of children with FA to ≥2 foods were compared with those allergic to egg or milk only (Figure 2B). In the total cohort, height SDS ($p = 0.569$), weight SDS ($p = 0.102$), and BMI SDS ($p = 0.930$) trajectories did not differ. In exploratory analyses of children aged <1 year, children with FA to ≥2 foods showed steeper declines in height SDS (−0.353 vs. −0.189; difference −0.164 [−0.325 to −0.004], $p = 0.044$) and weight SDS (−0.394 vs. −0.131; difference −0.263 [−0.410 to −0.115], $p < 0.001$). No differences were observed in children aged ≥1 year (height $p = 0.187$; weight $p = 0.357$) (Supplementary Table S5).

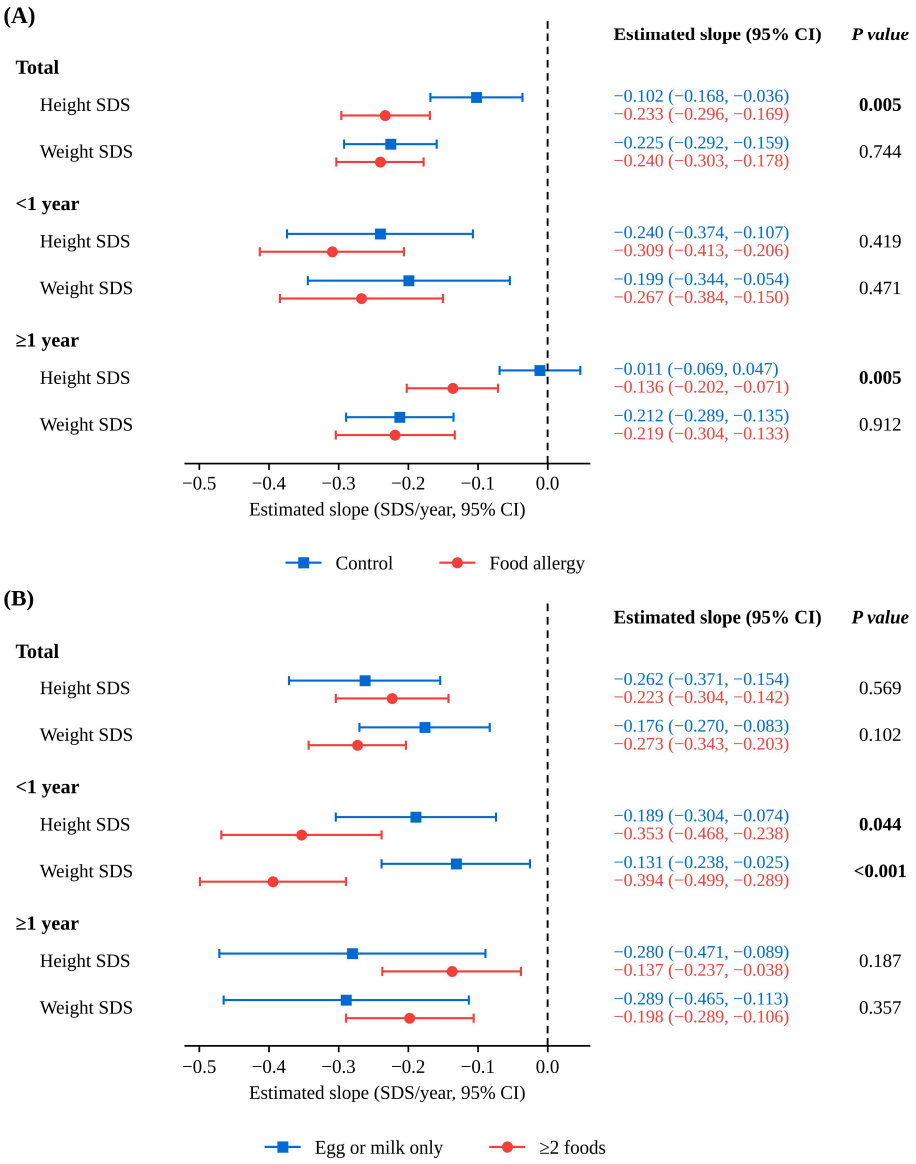


Figure 2. Estimated slopes of height and weight SDS (linear mixed models) in the propensity score-matched cohort: (**A**) Control versus children with food allergies. (**B**) Children with food allergies to egg or milk only versus ≥ 2 foods. Squares indicate estimated slopes, and bars indicate 95% confidence intervals; *p*-values are for the Group × Time interaction. Results are shown for the total cohort and for children aged <1 year and ≥1 year at the first measurement. Results for BMI SDS are provided in Supplementary Tables S4 and S5. CI, confidence interval; SDS, standard deviation score.

3.4. Factors Associated with Growth Trajectories in Children with Food Allergy

Univariable time × variable interaction estimates for height and weight SDS are shown in Table 3, and estimates for BMI SDS are shown in Supplementary Table S6. Compared with egg only, allergy to three or more foods was associated with a steeper decline in weight SDS in the total cohort (β −0.138 [−0.275 to −0.001], $p = 0.049$). Among children aged <1 year, allergy to two foods (β −0.241 [−0.426 to −0.057], $p = 0.010$) and allergy to three or more foods (β −0.345 [−0.542 to −0.149], $p = 0.001$) were associated with steeper weight declines. The corresponding estimates for height SDS did not reach statistical significance (two foods, $p = 0.058$; three or more foods, $p = 0.117$). In the two-category comparison (egg or milk only vs. ≥2 foods), allergy to ≥2 foods was associated with a steeper decline in height SDS among children aged <1 year (β −0.164 [95% CI −0.325 to −0.004], $p = 0.044$).

Table 3. Interaction between time and clinical characteristics on height and weight trajectories in propensity score-matched children with food allergy (linear mixed models).

Variable	Total β (95% CI)	*p*	Age < 1 Year β (95% CI)	*p*	Age ≥ 1 Year β (95% CI)	*p*
Height SDS						
Food allergy (4 categories)						
Egg only	Ref		Ref		Ref	
Milk only	−0.090 (−0.446, 0.266)	0.621	−0.336 (−0.898, 0.226)	0.239	0.079 (−0.617, 0.775)	0.823
2 foods	0.030 (−0.140, 0.200)	0.729	−0.192 (−0.390, 0.007)	0.058	0.083 (−0.235, 0.401)	0.607
≥3 foods	0.031 (−0.129, 0.191)	0.705	−0.179 (−0.403, 0.045)	0.117	0.189 (−0.064, 0.442)	0.142
Food allergy (2 categories)						
Egg or milk only	Ref		Ref		Ref	
≥2 foods	0.039 (−0.096, 0.173)	0.569	**−0.164 (−0.325, −0.004)**	**0.044**	0.143 (−0.070, 0.355)	0.187
Delivery mode: C/S vs. NSVD	0.079 (−0.053, 0.212)	0.239	0.006 (−0.193, 0.205)	0.952	0.034 (−0.132, 0.200)	0.686
Atopic PHx: yes vs. no	−0.191 (−0.337, −0.045)	0.011	−0.174 (−0.397, 0.049)	0.126	−0.047 (−0.235, 0.141)	0.620
Atopic FHx: yes vs. no	−0.012 (−0.161, 0.137)	0.871	0.063 (−0.170, 0.296)	0.595	−0.068 (−0.243, 0.107)	0.444
Pet: dog or cat vs. none	−0.178 (−0.372, 0.016)	0.073	−0.072 (−0.312, 0.169)	0.559	−0.165 (−0.459, 0.129)	0.269
Comorbidities (yes vs. no)						
AD	−0.093 (−0.258, 0.072)	0.271	−0.121 (−0.316, 0.074)	0.221	−0.115 (−0.372, 0.143)	0.381
AR	−0.122 (−0.291, 0.047)	0.158	−0.282 (−0.571, 0.008)	0.056	0.007 (−0.232, 0.246)	0.954
Asthma	−0.082 (−0.372, 0.208)	0.580	−0.326 (−0.928, 0.276)	0.287	−0.088 (−0.449, 0.272)	0.630
Others	0.107 (−0.072, 0.286)	0.241	0.264 (−0.007, 0.536)	0.056	0.063 (−0.175, 0.300)	0.604
Dietary counseling: yes vs. no	−0.062 (−0.229, 0.104)	0.463	−0.084 (−0.289, 0.122)	0.424	−0.121 (−0.416, 0.174)	0.420
Weight SDS						
Food allergy (4 categories)						
Egg only	Ref		Ref		Ref	
Milk only	−0.109 (−0.410, 0.191)	0.476	−0.364 (−0.814, 0.087)	0.113	0.229 (−0.362, 0.821)	0.445
2 foods	−0.072 (−0.218, 0.074)	0.332	−0.241 (−0.426, −0.057)	0.010	0.092 (−0.178, 0.362)	0.502
≥3 foods	−0.138 (−0.275, −0.001)	0.049	−0.345 (−0.542, −0.149)	0.001	0.119 (−0.095, 0.333)	0.275
Food allergy (2 categories)						
Egg or milk only	Ref		Ref		Ref	
≥2 foods	−0.097 (−0.213, 0.019)	0.102	−0.263 (−0.410, −0.115)	<0.001	0.092 (−0.104, 0.287)	0.357
Delivery mode: C/S vs. NSVD	0.032 (−0.090, 0.153)	0.611	0.029 (−0.157, 0.214)	0.763	0.006 (−0.165, 0.178)	0.942
Atopic PHx: yes vs. no	−0.054 (−0.180, 0.071)	0.397	−0.150 (−0.342, 0.042)	0.126	0.140 (−0.041, 0.321)	0.130
Atopic FHx: yes vs. no	0.059 (−0.068, 0.187)	0.360	0.110 (−0.094, 0.314)	0.288	0.000 (−0.172, 0.172)	0.998
Pet: dog or cat vs. none	0.056 (−0.122, 0.234)	0.537	0.123 (−0.107, 0.353)	0.294	−0.015 (−0.322, 0.293)	0.925
Comorbidities (yes vs. no)						
AD	0.089 (−0.048, 0.226)	0.202	0.130 (−0.064, 0.324)	0.187	0.053 (−0.153, 0.259)	0.615
AR	0.004 (−0.142, 0.150)	0.957	−0.159 (−0.441, 0.122)	0.266	0.149 (−0.042, 0.340)	0.126
Asthma	−0.127 (−0.377, 0.123)	0.317	0.047 (−0.489, 0.583)	0.864	−0.231 (−0.529, 0.068)	0.129
Others	0.056 (−0.094, 0.207)	0.463	0.072 (−0.194, 0.337)	0.595	0.062 (−0.141, 0.266)	0.548
Dietary counseling: yes vs. no	0.046 (−0.099, 0.191)	0.536	0.005 (−0.199, 0.209)	0.962	0.129 (−0.090, 0.349)	0.247

Values are β (95% CI) for the Time × variable interaction term from linear mixed models within the propensity score-matched food allergy group, representing the difference in the rate of change in each SDS relative to the reference category. Bold indicates $p < 0.05$. Abbreviations: SDS, standard deviation score; CI, confidence interval; NSVD, normal spontaneous vaginal delivery; C/S, cesarean section; PHx, past history; FHx, family history; AD, atopic dermatitis; AR, allergic rhinitis.

In the total cohort, atopic PHx was associated with a steeper decline in height SDS in the univariable analysis (β −0.191 [−0.337 to −0.045], $p = 0.011$). This association was not statistically significant in either age stratum. C/S delivery, atopic family history, pet ownership, and dietary counseling were not associated with height or weight trajectories (all $p > 0.05$). For BMI SDS, the heterogeneous "other" comorbidity category was associated with a steeper decline in the exploratory analysis (β −0.164 [−0.294 to −0.034], $p = 0.013$; Supplementary Table S6).

4. Discussion

The main finding of this study is that growth differences associated with IgE-mediated FA were not evident at diagnosis but emerged during follow-up. After matching for age and sex, children with FA had height, weight, and BMI SDS that were similar to those of healthy controls at baseline, but their height SDS subsequently declined more steeply, while weight and BMI trajectories did not differ. Within the FA group, infants allergic to two or more foods showed less favorable trajectories in both height and weight. Recent studies have reported

lower height or weight in children with FA, particularly among those with cow's milk allergy or multiple food allergies [4–7], and contemporary reviews continue to highlight poor growth and nutrient deficiency as clinically important risks [8]. In contrast, we found no evidence of a baseline growth deficit after matching for age and sex. This is consistent with a Singapore study in which wasting and stunting were uncommon [10]. Unlike previous reports based largely on cross-sectional comparisons [7], our serial measurements allowed us to distinguish the absence of a baseline deficit from a subsequent change in trajectory. In young children with FA, growth vulnerability may therefore be better captured by a downward shift in trajectory than by anthropometric status at a single visit.

The difference in height trajectory was more apparent in older children. Compared with matched controls, the steeper decline in height SDS was significant in children aged ≥1 year but not in those aged <1 year. This should not be interpreted as evidence that infancy is a low-risk period. Healthy infants also showed a relatively rapid decline in height SDS (−0.240 vs. −0.011 SDS/year in older controls), which may have narrowed the contrast between groups, and our subgroup analyses were not designed to formally test for effect modification by age. These age-specific findings should therefore be considered exploratory. Changes in feeding may also play a role: breast milk or formula may partly buffer the nutritional effects of food avoidance in early infancy, whereas increasing reliance on complementary and family foods may make restriction more consequential later. Rosow et al. similarly observed a less favorable weight-for-length trajectory in children with IgE-mediated FA, particularly after the first year of life [12]. They reported a significant Group × Time interaction for 12–24 months (β −0.033 [95% CI −0.054 to −0.013], $p = 0.001$) but did not analyze height or length [12]. We observed a steeper decline in height SDS (−0.131 SDS/year) without differences in weight or BMI SDS. However, the clinical significance of this difference remains uncertain because age-standardized SDS trajectories cannot be directly translated into absolute height deficits in a cohort with varying ages and follow-up durations. Differences between studies may reflect the outcome measure (weight-for-length vs. height SDS), age window (≤2 vs. <6 years), ethnicity and growth references, and stratification approach (observation age vs. age at first measurement). Our findings are therefore complementary rather than directly interchangeable.

The absence of a clear FA–control difference during infancy did not mean that all infants with FA had similar growth trajectories. Among infants aged <1 year, those with allergies to two or more foods showed steeper declines in both height and weight SDS than those allergic to egg or milk only. The association was particularly evident for weight SDS; the estimated decline was greater for three or more foods than for two foods. This is consistent with recent evidence showing greater nutritional gaps and growth vulnerability in children with multiple food allergies [7,8]. However, the studies synthesized in the systematic review were largely cross-sectional [7]; within a single cohort, we observed that infants with multiple food allergies had steeper height and weight declines than those allergic to egg or milk only. Infancy may be particularly vulnerable to a high food-allergen burden because growth and nutrient requirements are high, complementary feeding is being established, and dietary variety may still be limited [17,18]. Infants with multiple food allergies may therefore represent a nutritionally vulnerable subgroup, even though FA could not be distinguished from controls at this age. However, the number of causative foods is not a direct measure of dietary restriction because we did not record the extent and duration of avoidance.

Among the other clinical factors, a personal history of atopic disease was associated with a steeper decline in height SDS in the univariable analysis of the overall FA cohort. Because atopic PHx was broadly defined and more common in children allergic to three or more foods, it may reflect overall allergic disease burden rather than act as an independent

determinant of growth. Family history of atopy and individual comorbidities, including atopic dermatitis, allergic rhinitis, and asthma, showed no consistent association with height or weight trajectories. Nutritional counseling documented at diagnosis was also not associated with later growth. This does not imply that counseling is ineffective: it was not randomly assigned, was documented in only about one-fifth of the children, and information on repeated counseling, adherence, and subsequent dietary intake was not systematically available. Previous intervention and follow-up studies have reported benefits of structured dietitian-supported care [11,19].

Several mechanisms may contribute to the altered growth trajectories observed in children with FA. Nutritional restriction is likely to be relevant. Foods commonly implicated in early childhood, such as milk and egg, are important sources of high-quality protein, energy, calcium, vitamin D, and other nutrients required for growth [3,4,7,8]. Although total energy intake may be partly compensated for by substitute foods, replacement of protein quality and specific micronutrients may be less complete. Animal-source protein and milk intake have been linked to circulating IGF-I and linear growth in young children [20], and a recent systematic review found predominantly favorable associations between cow's milk consumption and linear growth [21]. Egg supplementation and adequate essential amino acid availability have also been linked to improved linear growth in young children [22,23]. These findings may be relevant to our observation that height trajectories differed between groups despite relatively preserved weight and BMI; however, because we did not assess dietary intake or nutritional status, this explanation remains speculative [24].

The potential nutritional impact may be greater when several foods are involved, particularly during infancy. Restriction of multiple foods can reduce dietary diversity and make adequate substitution more difficult, especially when nutritionally important staple foods are excluded [7,25]. This may help explain why infants with multiple FA in our cohort showed less favorable height and weight trajectories even though FA was not distinguishable from healthy controls during infancy. The number of causative foods, however, should be regarded as a marker of potential dietary burden rather than a direct measure of nutritional restriction because we did not quantify the specific foods avoided, the degree of avoidance, or nutrient intake.

Other pathways have also been proposed. Previous studies have linked chronic inflammatory cytokine signaling to altered activity of the growth hormone–IGF-I axis and the growth plate [26], severe eczema with sleep disturbance to shorter stature [27], and long-term inhaled corticosteroid therapy to a modest reduction in growth velocity, although pooled evidence is inconsistent [28]. In our cohort, individual allergic comorbidities were not consistently associated with height or weight trajectories; thus, our data do not provide direct evidence that inflammation was the primary explanation, although statistical power for individual comorbidities was limited. Feeding difficulties and restrictive eating behaviors are common in children with FA, particularly those with multiple food allergies [29], and the psychosocial burden of FA may narrow dietary variety beyond clinically necessary avoidance [30]. All of these mechanisms, including the nutritional pathways discussed above, remain hypothetical and could not be distinguished in this study.

In this cohort, height and weight SDS within the normal range at diagnosis did not preclude a subsequent decline in height SDS, suggesting that longitudinal growth assessment may provide information not captured by a single measurement. Infants with multiple food allergies or children with a progressive decline in height SDS may warrant closer nutritional assessment and periodic reassessment of continued food avoidance [2,8,31], although the present study did not evaluate the effectiveness of these interventions. The observed between-group difference in height SDS slope was −0.131 SDS/year (95% CI −0.222 to −0.040). This estimate describes a difference in standardized growth trajectories

and does not, by itself, establish a clinically meaningful change in absolute height or an individual child's growth outcome. The observational design also precludes causal conclusions regarding FA or dietary restriction.

This study has several strengths. Its longitudinal design allowed us to assess growth trajectories rather than status at a single time point. Matching on age and sex, standardized SDS based on the Korean National Growth Charts, and linear mixed models that incorporated repeated measurements made full use of the longitudinal data. Examining both the comparison with healthy controls and heterogeneity within the FA group also helped identify children who may be particularly vulnerable.

This study has several limitations. First, its retrospective, single-center design and the requirement for repeated measurements may have introduced selection bias; although measurement frequency was similar after matching, follow-up was longer in children with FA aged <1 year. Second, matching was limited to age and sex; some retrospective clinical covariates were incomplete and analyzed using available cases; and the within-FA comparisons were unadjusted despite baseline differences in age and atopic history, leaving potential residual confounding. Third, detailed dietary intake, the extent and duration of food avoidance, dietary substitutions, tolerance acquisition, and nutritional or biochemical markers were unavailable; therefore, the number of causative foods should be regarded as a marker of food-allergen burden rather than a direct measure of nutritional restriction. Because SDS declined in both groups, regression toward the mean and the transition in growth references at 36 months may have influenced the absolute slopes, while BMI SDS was available only from 24 months of age. Finally, the age- and food-number subgroup analyses involved small samples and multiple comparisons and should be considered exploratory, particularly given the small milk-only subgroup.

5. Conclusions

Children with IgE-mediated FA had growth status comparable to that of matched healthy controls at diagnosis but showed a greater subsequent decline in height SDS. In exploratory age-stratified analyses, this difference was evident among children aged ≥1 year. Exploratory within-FA analyses suggested less favorable height and weight trajectories among infants with multiple food allergies, with a more pronounced association for weight. These findings complement earlier cross-sectional and weight-for-length-based evidence by describing linear growth trajectories against matched controls. The clinical significance of these differences and the value of serial growth assessment or targeted nutritional management require prospective evaluation.

Supplementary Materials: The following supporting information can be downloaded at https://www.mdpi.com/article/10.3390/nu18223831/s1, Table S1: Follow-up characteristics of controls and children with food allergy in the total cohort and by age at the first measurement, before and after propensity score matching; Table S2: Baseline characteristics of children with food allergy according to the number of causative foods before propensity score matching; Table S3A and S3B: Baseline demographic and clinical characteristics of participants aged <1 year and ≥1 year before and after propensity score matching; Table S4: Linear mixed model estimates of growth trajectories by food allergy status (control vs. food allergy) in the propensity score-matched cohort; Table S5: Linear mixed model estimates of growth trajectories according to the number of food allergies (single vs. ≥2 foods) in the propensity score-matched cohort; Table S6: Interaction between time and clinical characteristics on BMI trajectories in propensity score-matched children with food allergy (linear mixed models).

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Data Availability Statement: The data presented in this study are available from the corresponding author upon reasonable request, subject to approval from the Institutional Review Board.

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Abbreviations

The following abbreviations are used in this manuscript:

AD	Atopic dermatitis
AR	Allergic rhinitis
BMI	Body mass index
C/S	Cesarean section
CI	Confidence interval
FA	Food allergy
FHx	Family history
IGF-I	Insulin-like growth factor I
IgE	Immunoglobulin E
LMM	Linear mixed model
NSVD	Normal spontaneous vaginal delivery
PHx	Past history
PSM	Propensity score matching
SDS	Standard deviation score
|SD|	Absolute standardized difference
WHO	World Health Organization

References

1. Santos, A.F.; Riggioni, C.; Agache, I.; Akdis, C.A.; Akdis, M.; Alvarez-Perea, A.; Alvaro-Lozano, M.; Ballmer-Weber, B.; Barni, S.; Beyer, K.; et al. EAACI guidelines on the diagnosis of IgE-mediated food allergy. *Allergy* **2023**, *78*, 3057–3076. [CrossRef] [PubMed]
2. Santos, A.F.; Riggioni, C.; Agache, I.; Akdis, C.A.; Akdis, M.; Alvarez-Perea, A.; Alvaro-Lozano, M.; Ballmer-Weber, B.; Barni, S.; Beyer, K.; et al. EAACI guidelines on the management of IgE-mediated food allergy. *Allergy* **2025**, *80*, 14–36. [CrossRef] [PubMed]
3. D'Auria, E.; Pendezza, E.; Leone, A.; Riccaboni, F.; Bosetti, A.; Borsani, B.; Zuccotti, G.V.; Bertoli, S. Nutrient intake in school-aged children with food allergies: A case-control study. *Int. J. Food Sci. Nutr.* **2022**, *73*, 349–356. [CrossRef] [PubMed]
4. Sackesen, C.; Buyuktiryaki, B.; Gokce, T.; Gogebakan, E.; Gundogdu, B.S.; Bilgic Eltan, S.; Karakoc-Aydiner, E.; Arik Yilmaz, E.; Can, C.; Cengiz, H.; et al. The association of milk and multiple food avoidance with growth parameters in infants and children. *Ann. Allergy Asthma Immunol.* **2024**, *132*, 745–751.e2. [CrossRef] [PubMed]
5. Baek, J.Y.; Jin, J.O.; Kim, J.H.; Jee, H.M.; Shin, Y.H.; Baek, H.S.; Han, M.Y.; Ha, E.K. Growth and micronutrient levels of 6–12 years-old children with food allergies, food sensitisation, and food restrictions. *Acta Paediatr.* **2022**, *111*, 2210–2215. [CrossRef] [PubMed]
6. Mooij-Nieberg, A.M.; ten Hoope, N.A.; van Doorn, O.; Schilperoort, J.; Meyer, R.; de Kroon, M.L.A.; Vlieg-Boerstra, B.J. Poor growth in multi-ethnic children with food allergy in Amsterdam. *Pediatr. Allergy Immunol.* **2025**, *36*, e70262. [CrossRef] [PubMed]

7. Di Cesare, G.; Carciofi, A.; Borgiani, F.; Cappelletti, D.; Correani, A.; Monachesi, C.; Gatti, S.; Lionetti, M.E. Are we meeting the needs? A systematic review of nutritional gaps and growth outcomes in children with multiple food allergies. *Nutrients* **2025**, *17*, 1590. [CrossRef] [PubMed]
8. Venter, C.; Meyer, R.; Bauer, M.; Bird, J.A.; Fleischer, D.M.; Nowak-Wegrzyn, A.; Anagnostou, A.; Vickery, B.P.; Wang, J.; Groetch, M. Identifying children at risk of growth and nutrient deficiencies in the food allergy clinic. *J. Allergy Clin. Immunol. Pract.* **2024**, *12*, 579–589. [CrossRef] [PubMed]
9. Beck, C.; Koplin, J.; Dharmage, S.; Wake, M.; Gurrin, L.; McWilliam, V.; Tang, M.; Sun, C.; Foskey, R.; Allen, K.J. Persistent food allergy and food allergy coexistent with eczema is associated with reduced growth in the first 4 years of life. *J. Allergy Clin. Immunol. Pract.* **2016**, *4*, 248–256.e3. [CrossRef] [PubMed]
10. Chong, K.W.; Wright, K.; Goh, A.; Meyer, R.; Rao, R. Growth of children with food allergies in Singapore. *Asia Pac. Allergy* **2018**, *8*, e34. [CrossRef] [PubMed]
11. Berni Canani, R.; Leone, L.; D'Auria, E.; Riva, E.; Nocerino, R.; Ruotolo, S.; Terrin, G.; Cosenza, L.; Di Costanzo, M.; Passariello, A.; et al. The effects of dietary counseling on children with food allergy: A prospective, multicenter intervention study. *J. Acad. Nutr. Diet.* **2014**, *114*, 1432–1439. [CrossRef] [PubMed]
12. Rosow, R.; Virkud, Y.V.; Martin, V.M.; Young, M.; Su, K.W.; Phadke, N.; Shreffler, W.G.; Yuan, Q. Longitudinal assessment of early growth in children with IgE- and non-IgE-mediated food allergy in a healthy infant cohort. *Ann. Allergy Asthma Immunol.* **2023**, *131*, 362–368.e1. [CrossRef] [PubMed]
13. Kim, J.H.; Yun, S.; Hwang, S.S.; Shim, J.O.; Chae, H.W.; Lee, Y.J.; Lee, J.H.; Kim, S.C.; Lim, D.; Yang, S.W.; et al. The 2017 Korean National Growth Charts for children and adolescents: Development, improvement, and prospects. *Korean J. Pediatr.* **2018**, *61*, 135–149. [CrossRef] [PubMed]
14. WHO Multicentre Growth Reference Study Group. WHO Child Growth Standards based on length/height, weight and age. *Acta Paediatr. Suppl.* **2006**, *450*, 76–85. [CrossRef] [PubMed]
15. Austin, P.C. Optimal caliper widths for propensity-score matching when estimating differences in means and differences in proportions in observational studies. *Pharm. Stat.* **2011**, *10*, 150–161. [CrossRef] [PubMed]
16. Austin, P.C. Balance diagnostics for comparing the distribution of baseline covariates between treatment groups in propensity-score matched samples. *Stat. Med.* **2009**, *28*, 3083–3107. [CrossRef] [PubMed]
17. World Health Organization. *WHO Guideline for Complementary Feeding of Infants and Young Children 6–23 Months of Age*; World Health Organization: Geneva, Switzerland, 2023.
18. Fewtrell, M.; Bronsky, J.; Campoy, C.; Domellöf, M.; Embleton, N.; Fidler Mis, N.; Hojsak, I.; Hulst, J.M.; Indrio, F.; Lapillonne, A.; et al. Complementary feeding: A position paper by the European Society for Paediatric Gastroenterology, Hepatology, and Nutrition (ESPGHAN) Committee on Nutrition. *J. Pediatr. Gastroenterol. Nutr.* **2017**, *64*, 119–132. [PubMed]
19. Poredoš, T.; Vesel Tajnšek, T.; Koren Jeverica, A.; Zajc Avramovič, M.; Markelj, G.; Emeršič, N.; Avčin, T. Comprehensive care and education can improve nutritional and growth outcomes in children with persistent cow's milk, egg, or peanut allergies: A five-year follow-up study of nutritional status. *Nutrients* **2024**, *16*, 48. [CrossRef] [PubMed]
20. Hoppe, C.; Udam, T.R.; Lauritzen, L.; Mølgaard, C.; Juul, A.; Michaelsen, K.F. Animal protein intake, serum insulin-like growth factor I, and growth in healthy 2.5-y-old Danish children. *Am. J. Clin. Nutr.* **2004**, *80*, 447–452. [CrossRef] [PubMed]
21. Saintila, J.; Paz-Olivas, Y. Liquid cow's milk consumption and linear growth outcomes in infancy and childhood: A systematic review. *Nutrients* **2026**, *18*, 2083. [CrossRef] [PubMed]
22. Iannotti, L.L.; Lutter, C.K.; Stewart, C.P.; Gallegos Riofrío, C.A.; Malo, C.; Reinhart, G.; Palacios, A.; Karp, C.; Chapnick, M.; Cox, K.; et al. Eggs in early complementary feeding and child growth: A randomized controlled trial. *Pediatrics* **2017**, *140*, e20163459. [CrossRef] [PubMed]
23. Semba, R.D.; Shardell, M.; Sakr Ashour, F.A.; Moaddel, R.; Trehan, I.; Maleta, K.M.; Ordiz, M.I.; Kraemer, K.; Khadeer, M.A.; Ferrucci, L.; et al. Child stunting is associated with low circulating essential amino acids. *eBioMedicine* **2016**, *6*, 246–252. [CrossRef] [PubMed]
24. Escobedo-Monge, M.F.; Parodi-Román, J.; Escobedo-Monge, M.A.; Marugán-Miguelsanz, J.M. The biological value of proteins for pediatric growth and development: A narrative review. *Nutrients* **2025**, *17*, 2221. [CrossRef] [PubMed]
25. Gratacós Gómez, A.R.; Iniesta Tejera, E.; Bas Bellver, L.; Serrano de Pablo, S.; Clar Castelló, M.; Gomez Torrijos, E. Dietary management of IgE-mediated and non-IgE-mediated food allergies: A systematic review of current evidence and clinical implications. *Nutrients* **2026**, *18*, 3180. [CrossRef] [PubMed]
26. Cammisa, I.; Rigante, D.; Cipolla, C. A theoretical link between the GH/IGF-1 axis and cytokine family in children: Current knowledge and future perspectives. *Children* **2025**, *12*, 495. [CrossRef] [PubMed]
27. Silverberg, J.I.; Paller, A.S. Association between eczema and stature in 9 US population-based studies. *JAMA Dermatol.* **2015**, *151*, 401–409. [CrossRef] [PubMed]

28. Zhao, J.; Dong, A.; Kong, Y.; Xie, T.; Yu, X.; Li, B.; Tan, T.; Mi, J.; Guo, Y. Effects of long-term inhaled corticosteroids on growth velocity and adult height in children with asthma: A systematic review and meta-analysis. *Front. Med.* **2026**, *13*, 1937498. [CrossRef] [PubMed]
29. Hill, S.A.; Nurmatov, U.; DunnGalvin, A.; Reese, I.; Vieira, M.C.; Rommel, N.; Dupont, C.; Venter, C.; Cianferoni, A.; Walsh, J.; et al. Feeding difficulties in children with food allergies: An EAACI Task Force Report. *Pediatr. Allergy Immunol.* **2024**, *35*, e14119. [CrossRef] [PubMed]
30. Bingemann, T.A.; LeBovidge, J.; Bartnikas, L.; Protudjer, J.L.P.; Herbert, L.J. Psychosocial impact of food allergy on children and adults and practical interventions. *Curr. Allergy Asthma Rep.* **2024**, *24*, 107–119. [CrossRef] [PubMed]
31. Meyer, R.; Groetch, M.; Santos, A.; Venter, C. The evolution of nutritional care in children with food allergies—With a focus on cow's milk allergy. *J. Hum. Nutr. Diet.* **2025**, *38*, e13391. [CrossRef] [PubMed]

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