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Early-pregnancy BMI, maternal gestational weight gain, and asthma and allergic diseases in children

Vincent Ojwang'[1,2,3] | Bright I. Nwaru[1,4,5] | Takkinen Hanna-Mari[1,6,7] | Tapanainen Heli[6] | Minna Kaila[8,9,10] | Suvi Ahonen[1,6] | Onni Niemelä[11] | Anna-Maija Haapala[12] | Jorma Ilonen[13,14] | Jorma Toppari[15,16] | Heikki Hyöty[11,17] | Riitta Veijola[18] | Mikael Knip[8,19] | Suvi M. Virtanen[1,5,7]

Correspondence
Vincent Ojwang', Department of Community Health and Behavioral Sciences, School of Medicine, Masinde Muliro University of Science and Technology, 190-50100 Kakamega, Kenya.
Email: vincent.ojwang@tuni.fi; vincent.ojwang@yahoo.com

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Abstract

Background: Association of early pregnancy body mass index (BMI) and maternal gestational weight gain (GWG), and asthma and allergic disease in children is unclear.

Methods: We analyzed data from 3176 mother–child pairs in a prospective birth cohort study. Maternal anthropometric measurements in the first and last antenatal clinic visits were obtained through post-delivery questionnaires to calculate early pregnancy BMI and maternal GWG. Asthma and allergic diseases in children by the age of 5 years was assessed using a validated questionnaire. Furthermore, serum samples were analyzed for IgE antibodies to eight allergens. We applied Cox proportional hazards and logistic regression analyses to estimate the association of early pregnancy BMI and maternal GWG (as continuous variables and categorized into quarters), and asthma, atopic eczema, atopic sensitization, and allergic rhinitis in children.

Results: Neither early pregnancy BMI nor maternal GWG was associated with asthma and allergic disease in children when analyzed as continuous variables. However, compared to the first quarter of GWG (a rate <0.32 kg/week), mothers in the third quarter (rate 0.42–0.52 kg/week) had children with significantly higher odds of developing atopic eczema (adjusted OR 1.49, 95% CI [1.13–1.96]) by 5 years of age.

Conclusion: Association of early pregnancy BMI and maternal GWG, and asthma and allergic disease in children, is inconsistent. High maternal GWG may be associated with increased odds of atopic eczema.

KEYWORDS
allergic rhinitis, asthma, atopic eczema, atopic sensitization, BMI, children, early pregnancy, gestational weight gain

For affiliations refer to page 8.

1 | BACKGROUND

Excessive early pregnancy weight and maternal gestational weight gain (GWG) are the most common morbidities in pregnancy, with consequent adverse health outcomes. [1] Simultaneously, asthma and allergic diseases are the most common early-onset morbidities in children.[2,3]

Diseases arise from complex interactions of genetic with environmental factors, often beginning in utero and during the perinatal and infant periods.[4,5] However, the role of obesity and/or excessive GWG in the development of asthma and allergic diseases in the offspring has remained unclear.[3,6,7]

Though high early pregnancy BMI has been linked to asthma in the offspring,[8] the evidence regarding maternal GWG is limited[9]; with some studies reporting no association of maternal GWG and asthma in children.[4,10] Furthermore, there is heterogeneity in the definitions of both GWG, and asthma and allergic diseases.[11,12]

High early pregnancy BMI has been directly associated with excessive maternal GWG.[13] However, some studies show that underweight women tend to experience higher weekly weight gain during pregnancy than those with higher early pregnancy BMI.[14] This suggests that early pregnancy BMI may modify maternal GWG.

Maternal nutritional status both before and during pregnancy on infant development and possible disease outcomes is of great interest due to the significant impact of early development (especially during the critical phase of organ development) on long-term disease risk in both mother and child.[1] To effectively prevent and alleviate the impact of these disease conditions, it is imperative to comprehensively elucidate the factors involved in their causation.[15,16]

The present work aims to study the association of early pregnancy BMI and maternal GWG; and asthma, allergic rhinitis, atopic eczema, and atopic sensitization in children in a large population-based birth cohort followed up to the age of 5 years.

Key message

While maternal GWG may be related with atopic eczema in children in our study; the association of early pregnancy BMI and maternal GWG, and asthma and allergic diseases in children remains inconsistent. Further research is needed to establish plausible causal associations and the underlying mechanisms. This is essential to the development of effective preventive strategies and interventions during pregnancy to improve health outcomes of both mother and child.

2 | METHODS

2.1 | Study population

The population under study consist of participants in the Finnish Type 1 Diabetes (T1D) Prediction and Prevention Project (DIPP) Nutrition Study.[17] The DIPP Study is an ongoing prospective population-based birth cohort started in 1994 to investigate the prevention of T1D.[18] The Nutrition Study investigates the role of maternal nutrition during pregnancy and lactation and the child's diet, in the development of childhood T1D, asthma and allergy, and obesity.

The current study includes families with newborn infants with HLA-DQBI-conferred susceptibility to T1D from the University Hospital areas of Oulu and Tampere born between September 1996 and September 2004. In the dietary follow-up at the age 5 of years, 4075 children were invited to participate in the allergy component of the DIPP Nutrition Study. Parents completed a questionnaire modified from the International Study of Asthma and Allergies in Children (ISAAC) questionnaire and consented to drawing of blood samples from their children for analysis of serum immunoglobulin E (IgE) levels to eight specific allergens. Our final study population comprised of 3176 mother–child pairs. The study procedures were approved by the local ethics committees and parents provided written informed consent.

2.2 | Anthropometric measurements

We collected data on maternal height, weight, and weeks of gestation at the first and last antenatal clinic visits (on average at the 10th and 39th weeks of gestation, respectively) using a maternal questionnaire sent to the families following delivery. In cases where the first weight was taken after 10 weeks of gestation, we extrapolated the weight at week 10 from the weight difference during their first and last antenatal clinic visit. We assumed weight gain in the 2nd and 3rd trimesters of pregnancy to be linear in fashion.[19]

We calculated early pregnancy BMI by dividing the mother's weight (kg) during the first antenatal clinic visit by the square of her height (m^2). We then categorized this into quarters (<21.5, 21.5–23.4, 23.5–26.2, and >26.2 kg/m^2). The rates of maternal GWG were calculated by dividing the difference in mother's weight (kg) during the first and last antenatal clinic visit by the number of weeks between the first and last antenatal clinic visits. We further categorized this into quarters (<0.32, 0.32–0.41, 0.24–0.52, and >0.52 kg/week).

2.3 | Socio-demographic measurements

We included parity (0, 1, 2, ≥ 3 previous deliveries), gestational age (<37 vs. ≥ 37 weeks), mode of delivery (vaginal vs. Caesarean section), child's birth weight (<2500 vs. ≥2500 g), and maternal smoking during pregnancy (no vs. yes) as potential confounding factors a priori. We obtained this information from the birth registries of Oulu and Tampere University Hospitals. Data on maternal age (<25,

25–29, 30–34, and ≥35 years) was obtained from the structured questionnaire administered after delivery.

2.4 | Definition of outcomes

We defined *asthma* as physician-diagnosis, in combination with persistent wheezing symptoms and/or the use of asthma medication during the preceding 12 months. This definition of persistent asthma has previously been validated against the Social Insurance Institute reimbursement database on anti-asthmatic medication.[20] Age at diagnosis of asthma was assessed using the question "At what age was asthma diagnosed?" *Allergic rhinitis* was defined as sneezing, nasal congestion, or rhinitis not caused by respiratory tract infections, accompanied with itching of the eye, and tearing in the preceding 12 months. *Atopic eczema* was defined as atopic eczema ever diagnosed by a physician.

2.5 | Immunologic measurements

Atopic sensitization was defined as positive serum IgE concentration of >=0.35 kU/L to any of the following food or inhalant allergens: egg, cow's milk, fish, wheat, house dust mite, cat, timothy grass, and birch. Serum IgE analyses were carried out using the ImmunoCAP fluoroenzyme immunoassay (Phadia Diagnostics, Uppsala, Sweden).

2.6 | Statistical methods

We summarized the study population background characteristics using counts and proportions. Differences in the background characteristics across early pregnancy BMI and maternal GWG were compared using Student's *t*- test or one-way analysis of variance (ANOVA).

To assess the association of early pregnancy BMI and maternal GWG and asthma, we used Cox proportional hazards regression analyses. We applied logistic regression analyses to study the association of early pregnancy BMI and maternal GWG, and allergic rhinitis, atopic eczema, and atopic sensitization.

To account for sibling dependence, we used (a) marginal analysis with a working independence assumption and a robust sandwich estimator (SE) of variance in the Cox regression analysis; and (b) generalized estimating equations (GEE) with the SE of variance in logistic regression analysis. We mutually adjusted for early pregnancy BMI and maternal GWG; while also adjusting for maternal age, maternal history of asthma/rhinitis, number of previous deliveries, maternal smoking during pregnancy, gestational age, birth weight, sex, and mode of delivery.

All the background characteristics were analyzed as categorical variables in the statistical models. Early pregnancy BMI and maternal GWG were treated both as continuous and categorical variables. A two-sided $p < .05$ indicated statistical significance. The analyses were carried out using SAS Enterprise Guide 8.3 (SAS Institute Inc., Cary, North Carolina, USA).

3 | RESULTS

For this work, complete outcome data on asthma, atopic eczema, and allergic rhinitis by the age of 5 years were available for 3176 mother–child pairs. Data on serum IgE to eight food and inhalant allergens were available for 3085 children. Altogether, 162 (5%) children had developed asthma, 689 (22%) had atopic eczema, 390 (12%) had allergic rhinitis, and 1143 (37%) had atopic sensitization. Table 1 shows the prevalence of asthma and allergic diseases by early pregnancy BMI, maternal GWG and population distribution of the background variables.

The mean early pregnancy BMI was 24.4 kg/m^2 (SD 4.3) while the mean maternal GWG was 0.4 kg/week (SD 0.2). Younger mothers exhibited lower early pregnancy BMI and higher GWG than older mothers (Table 1). Women who smoked during pregnancy were more likely to have higher early pregnancy BMI than women who did not smoke during pregnancy. Mothers who had previously delivered had higher early pregnancy BMI and lower GWG than those who had not previously delivered. Delivery before 37 completed weeks of gestation was associated with higher GWG.

Mothers to male babies typically had higher GWG than mothers to female babies. Additionally, Caesarian section delivery was common among mothers with higher, rather than in those with lower early pregnancy BMI. Table 2 shows the differences in mean early pregnancy BMI and maternal GWG by population distribution of the background variables.

3.1 | Early pregnancy BMI, GWG, and asthma and allergy outcomes

Early pregnancy BMI and maternal GWG, analyzed both as continuous and categorical variables, were not associated with the risk of persistent asthma, allergic rhinitis, and atopic sensitization in children. However, mothers in the third quarter of GWG (a rate of 0.42–0.52 kg/week), when compared with those in the first quarter (a rate of <0.32 kg/week), had children with 49% higher odds of developing atopic eczema by 5 years of age (adjusted OR 1.49, 95% CI 1.13–1.96). (Table 3).

4 | DISCUSSION

We report an inconsistent association of early pregnancy BMI and maternal GWG and the development of asthma and allergic diseases in our study of children in a large population-based cohort. While we noted that a high rate of GWG was associated with higher odds of developing atopic eczema, there was no consistent association of

TABLE 1 Population distribution of maternal and child characteristics across asthma and allergic diseases at 5 years of age.

Population Characteristic	Total ($N = 3176$) *n* (%)	Asthma ($n = 162$) *n* (%)	Atopic Eczema ($n = 689$) *n* (%)	Atopic Sensitization[a] ($n = 1143$) *n* (%)	Allergic Rhinitis ($n = 390$) *n* (%)
Early pregnancy BMI (kg/m^2)					
1st Quarter: <21.5	731 (23)	38 (23)	150 (22)	269 (24)	84 (22)
2nd Quarter: 21.5–23.4	756 (24)	39 (24)	166 (24)	265 (23)	104 (27)
3rd Quarter: 23.5–26.2	742 (23)	42 (26)	167 (24)	273 (24)	96 (25)
4th Quarter: >26.2	752 (24)	35 (22)	170 (25)	267 (23)	86 (22)
Missing information	195 (6)	8 (5)	36 (5)	69 (6)	20 (5)
Gestational Weight Gain (kg/week)					
1st Quarter: <0.32	698 (22)	36 (22)	136 (20)	257 (22)	78 (20)
2nd Quarter: 0.32–0.41	755 (24)	31 (19)	161 (23)	256 (22)	89 (23)
3rd Quarter: 0.42–0.52	747 (24)	36 (22)	197 (29)	269 (24)	99 (25)
4th Quarter: >0.52	760 (24)	51 (31)	152 (22)	284 (25)	101 (26)
Missing information	216 (7)	8 (5)	43 (6)	77 (7)	23 (6)
Maternal Age (years)					
<=24	505 (16)	31 (19)	89 (13)	179 (16)	60 (15)
25–29	1124 (35)	53 (33)	270 (39)	425 (37)	141 (36)
30–34	957 (30)	50 (31)	215 (31)	327 (29)	121 (31)
>=35	590 (19)	28 (17)	115 (17)	212 (19)	68 (17)
Maternal History of Asthma/ Rhinitis					
No	1426 (45)	51 (31)	301 (44)	456 (40)	138 (35)
Yes	1201 (38)	106 (65)	366 (53)	503 (44)	241 (62)
Missing information	549 (17)	5 (3)	22 (3)	184 (16)	11 (3)
Number of Previous Deliveries					
0	1435 (45)	69 (43)	333 (48)	531 (46)	197 (51)
1	994 (31)	59 (36)	217 (31)	351 (31)	109 (28)
2	442 (14)	24 (15)	94 (14)	161 (14)	58 (15)
>=3	276 (9)	7 (4)	40 (6)	93 (8)	24 (6)
Missing information	29 (1)	3 (2)	5 (1)	7 (1)	2 (1)
Maternal smoking during pregnancy					
No	2810 (88)	145 (90)	619 (90)	997 (87)	352 (90)
Yes	260 (8)	13 (8)	51 (7)	107 (9)	30 (8)
Missing information	106 (3)	4 (2)	19 (3)	39 (3)	8 (2)
Gestational Age					
<37	198 (6)	19 (12)	37 (5)	66 (6)	19 (5)
>=37	2948 (93)	142 (88)	647 (94)	1069 (94)	370 (95)
Missing information	30 (1)	1 (1)	5 (1)	8 (1)	1 (0)
Birth weight					
<2500	144 (5)	13 (8)	24 (3)	43 (4)	12 (3)
>=2500	3011 (95)	148 (91)	661 (96)	1095 (96)	378 (97)
Missing information	21 (1)	1 (1)	4 (1)	5 (0)	0 (0)
Sex					
Male	1657 (52)	98 (60)	376 (55)	626 (55)	227 (58)
Female	1519 (48)	64 (40)	313 (45)	517 (45)	163 (42)
Mode of delivery					
Vaginal	2732 (86)	136 (84)	600 (87)	991 (87)	338 (87)

TABLE 1 (Continued)

Population Characteristic	Total (N = 3176) n (%)	Asthma (n = 162) n (%)	Atopic Eczema (n = 689) n (%)	Atopic Sensitization[a] (n = 1143) n (%)	Allergic Rhinitis (n = 390) n (%)
Caesarean section	423 (13)	25 (15)	85 (12)	147 (13)	52 (13)
Missing information	21 (1)	1 (1)	4 (1)	5 (0)	0 (0)

[a]Total N = 3085 for atopic sensitization.

early pregnancy BMI and maternal GWG; and asthma and allergic diseases in the children by the age of 5 years.

Our results are consistent with previous research on the potential link between early pregnancy BMI and maternal GWG, and childhood asthma and allergic disease that have yielded inconsistent, contradictory findings.[4,9,10] Researchers have demonstrated a lack of association of early pregnancy BMI and maternal GWG and asthma and allergic disease outcomes in children[4,6,10] Other studies have, however, shown that both early pregnancy obesity and very high or very low maternal GWG render children susceptible to significant risk of asthma and allergic diseases.[7,10,21]

While the traditional "atopic march" posits a sequential progression from eczema to food allergies, allergic rhinitis, and asthma[22] recent research has challenged this progression, suggesting a more complex interplay of factors.[23] We observed an association between GWG and atopic eczema but regarding asthma and allergic rhinitis, although there was a tendency towards putative associations, these were not statistically significant. This inconsistency with the findings for asthma and rhinitis in contrast to the more robust finding for atopic eczema might be due to several factors. Firstly, the complex and heterogeneous nature of atopic diseases, with varying clinical presentations and underlying mechanisms,[24] may influence the strength of associations with GWG. Secondly, the development of allergic rhinitis and asthma often involves a combination of atopic and non-atopic factors,[25] potentially obscuring the impact of maternal GWG.

A large population-based cohort study found that compared to normal early pregnancy BMI, maternal obesity was associated with increased risk of asthma in children but decreased hazards of anaphylaxis and dermatitis. Maternal underweight was however associated with increased hazards of dermatitis in children in that study.[6] Rosenquist et al[8] conclude that high early pregnancy BMI is associated with increased risk of asthma in children. In their cohort study, they categorized early pregnancy BMI into (i) underweight [<18.5], (ii) normal [18.5–24.9], (iii) overweight [25–29.9], (iv) obese class 1 [30–34.9], and (v) obese class 2/3 [≥35]. When they modeled continuous pre-pregnancy BMI using cubic splines with knots at BMI category boundaries, the risk of asthma in the child generally increased linearly with increasing early pregnancy BMI. Using cubic splines allows the flexibility of capturing a wider range of complex nonlinear relationships compared to linear models while maintaining the smoothness of the fitted curve. In our study, we did not model continuous early-pregnancy BMI using splines. We categorized BMI into quarters rather than established criteria to better capture the nuanced effects of maternal weight across the spectrum and to allow for a more granular analysis of maternal GWG. This approach aligns with recent literature, suggesting the need to revisit and potentially adjust GWG recommendations based on the severity of obesity.[26,27]

While early pregnancy BMI was associated with an increased risk of persistent wheeze in children in their study, Polinski et al found that excessive maternal GWG was not associated with asthma and atopic diseases in children.[9] In contrast, our findings suggest that a high rate of maternal GWG may play a role in the development of atopic eczema in children. This could be a spurious finding due to the increased risk of a type I error when making multiple statistical tests. We did not apply the Bonferroni correction to adjust the probability (p) values due to our relatively large sample size and a modest number of outcomes since it would make little difference in the magnitude of the effect sizes we observed.[28] Furthermore, several studies have similarly indicated that a high maternal GWG is associated with atopic eczema in children.[21,29,30]

Obesity in pregnancy has been postulated to program the risk of chronic non-communicable disorders later in life due to remodeling of organ structures during early development.[1,2,5,31] The inflammation induced by excessive GWG may affect the development of pediatric allergic diseases in the developing fetus through pro-inflammatory pathways and increased cytokine levels. [2,4,11] Recent research suggests that early pregnancy BMI has a stronger impact than GWG on inflammatory markers thought to influence asthma.[10] Child overweight/obesity has also been shown to have a mediating effect on the association between early pregnancy BMI and childhood asthma.[32,33]

Limitations in our study should be taken into consideration while interpreting the findings. Though based on maternal healthcare visits, the mothers reported their own weight measurements after delivery. This could introduce social desirability bias and recall bias. However, any errors due to recall appear to be non-differential across the asthma and allergy outcomes. As maternal weight status is not a known risk-factor for atopic disease, the impact of social desirability bias and recall bias is most likely minimal.[34]

Secondly, since our study is observational, we cannot ascertain causality. Additionally, we cannot rule out the possibility of residual confounding. To account for variations in the timing of antenatal clinic visits and final weight measurements among mothers in our cohort study, we used the rate of maternal GWG instead of total weight gain which is expected to enhance the accuracy of our findings.

Thirdly, since our study population consisted of families with HLA-DQBI-conferred susceptibility to T1D, our findings may not

TABLE 2 Study population differences by mean early pregnancy BMI and maternal GWG.[a]

Characteristic	Total, *n* (%)	Early pregnancy BMI (kg/m^2)		Gestational Weight Gain (kg/wk)	
		Mean (*SD*)	*p*-value	Mean (*SD*)	*p*- value
Early pregnancy BMI (kg/m^2)					<.001
1st Quarter: <21.5	731 (23)			0.45 (0.13)	
2nd Quarter: 21.5–23.4	756 (24)			0.45 (0.14)	
3rd Quarter: 23.5–26.2	742 (23)			0.44 (0.15)	
4th Quarter: >26.2	752 (24)			0.37 (0.19)	
Gestational Weight Gain (kg/wk)			<.001		
1st Quarter: <0.32	698 (22)	26.5 (5.5)			
2nd Quarter: 0.32–0.41	755 (24)	23.7 (3.7)			
3rd Quarter: 0.42–0.52	747 (24)	23.5 (3.6)			
4th Quarter: >0.52	760 (24)	24.0 (3.5)			
Maternal Age (years)			<.001		<.001
<=25	505 (16)	23.9 (4.6)		0.46 (0.16)	
25–29	1124 (35)	24.1 (4.0)		0.44 (0.15)	
30–34	957 (30)	24.5 (4.2)		0.42 (0.16)	
>=35	590 (19)	25.4 (4.6)		0.38 (0.16)	
Maternal History of Asthma/ Rhinitis			.315		.154
No	1426 (45)	24.4 (4.1)		0.43 (0.16)	
Yes	1201 (38)	24.5 (4.4)		0.42 (0.16)	
Number of Previous Deliveries			<.001		<.001
0	1435 (45)	24.1 (4.2)		0.46 (0.16)	
1	994 (31)	24.4 (4.1)		0.42 (0.15)	
2	442 (14)	24.9 (4.7)		0.39 (0.16)	
>=3	276 (9)	25.5 (4.2)		0.38 (0.16)	
Maternal smoking during pregnancy			.016		.639
No	2810 (88)	24.3 (4.2)		0.43 (0.16)	
Yes	260 (8)	25.2 (5.2)		0.43 (0.18)	
Gestational Age (weeks)			.88		.005
<37	198 (6)	24.4 (3.8)		0.47 (0.20)	
>=37	2948 (93)	24.4 (4.3)		0.43 (0.16)	
Birth weight (g)			.894		.143
<2500	144 (5)	24.5 (3.9)		0.45 (0.21)	
>=2500	3011 (95)	24.4 (4.3)		0.43 (0.16)	
Sex			.466		.037
Male	1657 (52)	24.5 (4.3)		0.43 (0.16)	
Female	1519 (48)	24.3 (4.2)		0.42 (0.16)	
Mode of delivery			<.001		.475
Vaginal	2732 (86)	24.3 (4.2)		0.43 (0.16)	
Caesarean section	423 (13)	25.2 (4.7)		0.43 (0.17)	

[a]Using Student's *t*-test for variables with two classes, and one-way ANOVA for variables with 3 or more classes.

TABLE 3 Association of early pregnancy body mass index (BMI) and maternal gestational weight gain (GWG) and asthma and allergic diseases in children by age 5 years.[a]

	Asthma ($n=142$)		Atopic Eczema ($n=606$)		Atopic Sensitization ($n=859$)		Allergic Rhinitis ($n=348$)	
Characteristic	**AHR (95% CI)**	***p*-value**	**AOR (95% CI)**	***p*-value**	**AOR (95% CI)**	***p*-value**	**AOR (95% CI)**	***p*-value**
Early pregnancy BMI (kg/m^2) (Continuous)	0.99 (0.96–1.03)	0.754	1.00 (0.98–1.03)	0.791	1.00 (0.98–1.02)	0.739	1.01 (0.98–1.04)	.393
Gestational Weight Gain (kg/wk) (Continuous)	1.72 (0.59–5.04)	0.322	1.16 (0.63–2.11)	0.634	0.91 (0.52–1.59)	0.739	1.33 (0.63–2.83)	.457
Early pregnancy BMI (kg/m^2) (Categorical)		0.772		0.504		0.913		.640
1st Quarter: <21.5	Ref		Ref		Ref		Ref	
2nd Quarter: 21.5–23.4	0.99 (0.62–1.60)		1.01 (0.77–1.32)		1.01 (0.79–1.29)		1.19 (0.85–1.65)	
3rd Quarter: 23.5–26.2	1.12 (0.69–1.82)		1.10 (0.84–1.45)		1.06 (0.83–1.37)		1.17 (0.83–1.65)	
4th Quarter: >26.2	0.87 (0.52–1.45)		1.21 (0.92–1.59)		0.98 (0.76–1.26)		1.02 (0.72–1.46)	
Gestational Weight Gain (kg/wk) (Categorical)		0.113		0.009		0.822		.895
1st Quarter: <0.32	Ref		Ref		Ref		Ref	
2nd Quarter: 0.32–0.41	0.76 (0.45–1.26)		1.11 (0.84–1.46)		0.92 (0.72–1.17)		1.05 (0.74–1.48)	
3rd Quarter: 0.42–0.52	0.89 (0.53–1.47)		1.49 (1.13–1.96)		1.02 (0.79–1.31)		1.13 (0.79–1.62)	
4th Quarter: >0.52	1.30 (0.82–2.05)		0.98 (0.74–1.30)		1.00 (0.78–1.29)		1.11 (0.79–1.57)	

[a]Adjusted for maternal age, maternal history of asthma/rhinitis, number of previous deliveries, maternal smoking during pregnancy, gestational age, birth weight, sex, mode of delivery and place of birth. Early pregnancy BMI and the rate of GWG rate were mutually adjusted for each other. We used Cox proportional hazards model to assess asthma, and Logistic regression for the allergy end-points.

apply to the general population. The complex interplay between T1D and atopic diseases has been a subject of considerable interest. Traditionally, the T-cell helper type 1 (Th1)/ Th2 paradigm suggested a reciprocal relationship, with T1D associated with a Th1-dominant immune response and atopic diseases with a Th2-dominant profile.[35] Recent studies have however challenged this view, revealing a more intricate association. [36] Though some studies support a protective effect of atopy against T1D, some indicate a positive association,[37] yet other researchers found no associations between T1D and atopic dermatitis, allergic rhinitis, or asthma.[38,39] Additionally, the coexistence of both Th1- and Th2-mediated responses in certain conditions further complicates the picture.[35,36] Maternal obesity, excessive GWG, and gestational diabetes mellitus (GDM) correlate with low-grade chronic inflammatory response.[40] However, it has been suggested that the effect of GDM on fetal and birth outcomes decreases after controlling for early pregnancy BMI and maternal GWG.[41,42]

Fourthly, our study cohort was recruited over 20 years ago. Though some recent studies report no significant change in early pregnancy BMI and maternal GWG, especially in women with pregestational diabetes,[43] there has been a rise in maternal obesity globally over the past two decades.[44,45] This potentially influences the association of GWG and asthma and atopic diseases in children. While our findings provide valuable insights, replication in a more recent population is warranted to further elucidate these associations in the context of contemporary maternal BMI trends.

Lastly, we had few numbers of subjects for the analyses using categorical variables. Though we did not perform power calculations a priori, that does not limit our ability to detect even smaller effect sizes.[46,47]

5 | CONCLUSION

The association between early pregnancy BMI and maternal GWG and asthma and allergic diseases in children, is not consistent. It is imperative to consider high maternal GWG as a potential risk factor for atopic eczema in children. Healthcare providers should counsel pregnant women on healthy weight gain and support them to achieve optimal weight goals in pregnancy.

AUTHOR CONTRIBUTIONS

Vincent Ojwang': Conceptualization; methodology; validation; visualization; writing – original draft; writing – review and editing. **Bright I. Nwaru:** Conceptualization; methodology; validation; visualization; writing – review and editing. **Takkinen Hanna-Mari:** Visualization; formal analysis; data curation; validation; writing – review and editing. **Tapanainen Heli:** Formal analysis; validation; visualization; writing – review and editing. **Minna Kaila:** Funding acquisition; supervision; writing – review and editing; project administration. **Suvi Ahonen:** Data curation; writing – review and editing; validation. **Onni Niemelä:** Investigation; writing – review and editing; resources. **Anna-Maija Haapala:** Investigation; writing – review and editing; resources. **Jorma Ilonen:** Investigation; writing – review and editing. **Jorma Toppari:** Investigation; writing – review and editing. **Heikki Hyöty:** Investigation; writing – review and editing. **Riitta Veijola:** Writing – review and editing; investigation; validation. **Mikael Knip:** Writing – review and editing; validation; investigation. **Suvi M. Virtanen:** Conceptualization; methodology; validation; supervision; visualization; project administration; writing – review and editing; funding acquisition.

AFFILIATIONS

[1]Faculty of Social Sciences, Health Sciences Unit, Tampere University, Tampere, Finland
[2]SEVIN's Institute for Health Research, Kisumu, Kenya
[3]Department of Community Health and Behavioral Sciences, School of Medicine, Masinde Muliro University of Science and Technology, Kakamega, Kenya
[4]Krefting Research Centre, Institute of Medicine, University of Gothenburg, Gothenburg, Sweden
[5]Wallenberg Centre for Molecular and Translational Medicine, University of Gothenburg, Gothenburg, Sweden
[6]Department of Public Health and Welfare, Finnish Institute for Health and Welfare, Helsinki, Finland
[7]Center for Child Health Research, Tampere University and Tampere University Hospital, Tampere, Finland
[8]Tampere University Hospital, Wellbeing Services County of Pirkanmaa, Tampere, Finland
[9]Department of Pediatrics, Tampere University Hospital, Tampere, Finland
[10]Public Health Medicine, University of Helsinki and Helsinki University Hospital, Helsinki, Finland
[11]Department of Laboratory Medicine and Medical Research Unit, Seinajoki Central Hospital and University of Tampere, Tampere, Finland
[12]Fimlab laboratories, Pirkanmaa Hospital District, Tampere, Finland
[13]Immunogenetics Laboratory, Institute of Biomedicine, University of Turku, Turku, Finland
[14]Department of Clinical Microbiology, Turku University Hospital, Turku, Finland
[15]Institute of Biomedicine, Research Centre of Integrative Physiology and Pharmacology, University of Turku, Turku, Finland
[16]Department of Pediatrics, Turku University Hospital, Turku, Finland
[17]Department of Virology, Faculty of Medicine and Life Sciences, University of Tampere, Tampere, Finland
[18]Department of Pediatrics, PEDEGO Research Unit, Medical Research Centre, Oulu University, Hospital and University of Oulu, Oulu, Finland
[19]Research Program for Clinical and Molecular Metabolism, Faculty of Medicine, University of Helsinki, Helsinki, Finland

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CONFLICT OF INTEREST STATEMENT

No conflicts of interest to report.

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ORCID

Vincent Ojwang' https://orcid.org/0000-0002-7797-030X
Bright I. Nwaru https://orcid.org/0000-0002-2876-6089
Minna Kaila https://orcid.org/0000-0002-9645-4925
Mikael Knip https://orcid.org/0000-0003-0474-0033

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