Dietary Sugar and Atopic Dermatitis in a Longitudinal Birth Cohort

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Importance: The association of diet with atopic dermatitis (AD) in children is understudied and may present an opportunity to optimize AD management in a cost-effective and low-risk manner. **Objective:** The aim of this study was to determine the extent to which dietary sugar is associated with AD period prevalence and severity in a longitudinal pediatric cohort. **Design, setting, and participants:** This was a longitudinal cohort study of children from the Avon Longitudinal Study of Parents and Children with food frequency questionnaire data to estimate dietary carbohydrate and sugar at 1, 3, 5, 7, 10, and 13 years. **Exposure:** The exposure was dietary sugar as a proportion of total caloric intake. **Main outcome and measure:** The primary outcome was AD based on a maternal- or self-reported questionnaire that asked about disease activity and severity over the past 12 months. Logistic regression models adjusted for sex, race, maternal delivery age, highest parental education level, social class assessed through parental occupation, body mass index, total caloric intake, and maternal history of AD. **Results:** The study population included 5372 unique participants, 50% of whom were female, and 20−30% of whom reported AD at any time point. No significant associations were found at ages 1, 3, 5, and 7 years. At age 13 years, logistic regression revealed that a 10% increase in dietary sugar as a proportion of total caloric intake was associated with a 22% (95% confidence interval = 7−40%) increase in odds of AD overall. There was a dose−response relationship with disease severity: there was a 19% (95% confidence interval = 0−42%) increase in the odds of mild AD and 32% (95% confidence interval = 5−86%) increase in the odds of moderate−severe AD. When examining subtypes of dietary sugar, the effect was limited to nonmilk extrinsic sugars. **Conclusions and relevance:** Given the known health benefits, reduction of nonmilk sugars could be studied as a cost-effective and low-risk intervention for AD in late childhood and early adolescence.

Keywords: ALSPAC, Atopic dermatitis, Clinical research, Dietary sugar, Epidemiology

INTRODUCTION

Atopic dermatitis (AD) is one of the most common inflammatory skin conditions worldwide, with an annual period prevalence of up to 20% in children. It is important for clinical practice to understand whether children with AD would benefit from dietary assessment and modification because the episodic nature of the disease is problematic for patients, and current treatments do not address triggers for flares.

Prior studies have shown increased dietary carbohydrates and refined sugars to be associated with AD in adults but have not explored this association in children (Ito et al, 2019; Solvoll et al, 2000). Our study aimed to address this gap in the literature using a series of cross-sectional analyses among the Avon Longitudinal Study of Parents and Children (ALSPAC) birth cohort followed over 13 years to examine the association between dietary sugar and AD period prevalence in a pediatric population. We also examined whether sugar intake was associated with the severity of AD and whether the association changed with age.

RESULTS

Our study included 5372 unique participants in total who were asked dietary questions about sugar and AD over a minimum of one 0−10-month time period. There were 811 participants at age 1 year, 687 at age 3 years, 622 at age 5 years, 4414 at age 7 years, 4544 at age 10 years, and 3833 at age 13 years (Table 1). Participant numbers were smaller at the earlier ages because only 10% of children born in the last 6 months—known as children in focus—of the recruitment phase were selected to take part in a substudy that lasted until age 5 years (Boyd et al, 2013).

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Abbreviations: AD, atopic dermatitis; ALSPAC, Avon Longitudinal Study of Parents and Children; CI, confidence interval; IMS, intrinsic and milk sugar; NMES, nonmilk extrinsic sugar

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Table 1. Patient Characteristics

Characteristic	1 y		3 y		5 y		7 y		10 y		13 y	
	n = 811	% or mean (SD)	n = 687	% or mean (SD)	n = 622	% or mean (SD)	n = 4414	% or mean (SD)	n = 4544	% or mean (SD)	n = 3833	% or mean (SD)
BMI (kg/m^2)	785	17.02 (1.32)	675	16.43 (1.36)	609	16.09 (1.56)	4383	16.22 (2.09)	4497	18.21 (3.14)	3819	20.36 (3.51)
Sex	811		687		622		4414		4544		3833	
Female	362	44.64	299	43.52	281	45.18	2217	50.23	2291	50.42	1958	51.08
Race	787		667		605		4028		4098		3469	
Non-White	19	2.41	12	1.80	14	2.31	142	3.53	150	3.66	129	3.72
Highest education of parent[1]	798		675		612		4098		4166		3526	
CSE	57	7.14	36	5.33	30	4.90	278	6.78	326	7.83	252	7.15
Vocational	41	5.14	32	4.74	30	4.90	221	5.39	226	5.42	186	5.28
O level	211	26.44	179	26.52	158	25.82	1038	25.33	1042	25.01	846	23.99
A level	300	37.59	267	39.56	250	40.85	1473	35.94	1482	35.57	1275	36.16
Degree	189	23.68	161	23.85	144	25.53	1088	26.55	1090	26.16	967	27.42
Mother had AD	811		687		622		4414		4544		3833	
Yes	211	26.02	182	26.49	181	29.10	972	22.02	995	21.90	835	21.78
Social class[2]	777		653		596		4024		4068		3448	
Professional	124	15.96	108	16.54	89	14.93	642	15.95	647	15.90	588	17.05
Managerial and technical	373	48.01	318	48.70	297	49.83	1952	48.51	1942	47.74	1666	48.32
Skilled nonmanual	197	25.35	165	25.27	150	25.178	1005	24.98	1033	25.39	847	24.56
Skilled manual	44	5.66	38	5.82	33	5.54	300	7.46	311	7.65	250	7.25
Partly skilled	30	3.86	19	2.91	22	3.69	115	2.86	122	3.00	89	2.58
Unskilled	9	1.16	5	0.77	5	0.84	10	0.25	13	0.32	8	0.23
Exposures												
Total caloric intake (calories)	811	1098 (227)	687	1350 (260)	622	1516 (293)	4414	1708 (316)	4544	1849 (379)	3833	1947 (519)
Carbohydrate (% kcal/day)	811	0.50 (0.06)	687	0.52 (0.05)	622	0.54 (0.05)	4414	0.54 (0.05)	4544	0.53 (0.05)	3833	0.53 (0.06)
Sugar (% kcal/day)	811	0.28 (0.06)	687	0.27 (0.06)	622	0.27 (0.06)	4414	0.26 (0.06)	4544	0.24 (0.06)	3833	0.23 (0.07)
Intrinsic milk sugar (% kcal/day)	811	0.15 (0.05)	687	0.10 (0.04)	622	0.09 (0.04)	4414	0.07 (0.03)	4544	0.06 (0.03)	3833	0.06 (0.04)
Nonmilk extrinsic sugar (% kcal/day)	811	0.13 (0.06)	687	0.17 (0.06)	622	0.18 (0.06)	4414	0.18 (0.06)	4544	0.18 (0.06)	3833	0.17 (0.07)
Outcomes												
Presence of AD	797		667		589		3923		4111		3459	
Yes	195	24.47	180	26.99	172	29.20	1183	30.16	1052	25.59	714	20.64
AD severity subtypes	739		667		589		3919		4108		3456	
Mild	104	14.07	125	18.74	117	19.86	874	22.30	806	19.62	521	15.08
Moderate/severe	34	4.60	55	8.25	55	9.34	305	7.78	243	5.92	190	5.50

Abbreviations: AD, atopic dermatitis; A level, advanced level; BMI, body mass index; CSE, certificate of secondary education; O level, ordinary level.

[1]Based on British-based education levels. Vocational training is occupation-based training. O level represents 11 years of academic study and marks the end of the secondary education cycle. O level and CSE are equivalent. A level represents an additional 2 years of study (13 in total) after the O level is obtained and is an admission requirement for university to pursue a degree.

[2]Based on highest parental occupation level at time of initial study enrolment.

In primary analyses, there was only an association between dietary sugars and AD at age 13: the OR for AD for each 10% increase in dietary sugars as a proportion of total caloric intake was 1.22 (95% confidence interval [CI] = 1.03−1.44) (Table 2). There was a dose−response relationship with AD severity: the OR for mild AD was 1.19 (95% CI = 1.00−1.42) compared with no AD, whereas the OR for moderate/severe AD was 1.32 (95% CI = 0.99−1.75) (Figure 1).

In secondary analyses exploring subcategories of dietary sugars (intrinsic and milk sugars [IMSs] and nonmilk extrinsic sugars [NMESs]), we did not find associations between dietary IMSs and the odds of AD at any age (Table 2). We found positive associations between increased dietary NMESs and AD odds at ages 10 (OR = 1.15, 95% CI = 1.00−1.32) and 13 (OR = 1.27, 95% CI = 1.07−1.51) years, with a dose−response relationship with disease severity only at age 13 years (Figure 1).

DISCUSSION

In this pediatric cohort study, we did not find associations between carbohydrates and AD but found that increased dietary sugar, specifically NMESs, was associated with increased AD odds at ages 10 and 13 years: for each 10% increase in dietary NMESs, there was a 15−27% increase in the odds of prevalent AD at those ages.

These results are consistent with those of an existing case-control study on dietary sugar and AD in adults, which showed that higher intake of refined sugar in male patients was associated with the odds of moderate/severe AD ($P = .014$) (Solvoll et al, 2000). Another case-control study showed that adult patients with AD had higher intake of carbohydrates than healthy controls (Ito et al, 2019). Although there is less literature on sugar and AD in children, our results are also consistent with findings from the International Study of Asthma and Allergies in Childhood, where fast food consumption was associated with an increase in AD odds in adolescents aged 13−14 years but not in individuals aged 6−7 years (Ellwood et al, 2013). Extrinsic sugars in sweetened/soft drinks (common in fast food) have previously been shown to be the largest contributors to NMES intake in adolescents (Gibson et al, 2016; Lai et al, 2019).

Dietary guidelines generally recommend reducing NMES intake (Great Britain Department of Health, 1989). Although all sugars are of equivalent energy density, IMSs are not targeted for reduction because they exist within a cellular matrix with micronutrients and/or fiber and are less likely to cause tooth decay. In contrast, NMESs are targeted for reduction on the premise that they are cariogenic and can compromise diet quality by displacing micronutrients from the diet (Gibson et al, 2016).

Limitations

Our study is limited by the self-report nature of food frequency questionnaires. We used data from a questionnaire that was previously validated and recommended for nutritional research in the United Kingdom but only had annual assessments and did not include measures of overall diet quality (Northstone and Emmett, 2005). The

Table 2. OR for Atopic Dermatitis

	OR (95% CI) of AD							
	Carbohydrates (per 10% kcal/day)		Total Sugars (per 10% kcal/day)		Intrinsic and Milk Sugars (per 10% kcal/day)		Nonmilk Extrinsic Sugars (per 10% kcal/day)	
Age, y	Unadjusted	Adjusted	Unadjusted	Adjusted	Unadjusted	Adjusted	Unadjusted	Adjusted
1 (n = 811)	1.10 (0.83–1.45)	1.08 (0.79–1.46)	1.05 (0.80–1.39)	0.98 (0.72–1.35)	1.16 (0.83–1.63)	1.07 (0.73–1.57)	0.95 (0.72–1.25)	0.95 (0.70–1.28)
3 (n = 687)	1.12 (0.73–1.55)	1.07 (0.73–1.57)	0.87 (0.63–1.20)	0.78 (0.54–1.10)	1.04 (0.67–1.62)	0.88 (0.55–1.43)	0.86 (0.62–1.18)	0.84 (0.59–1.18)
5 (n = 622)	1.44 (1.00–2.06)	1.36 (0.92–2.01)	1.20 (0.87–1.67)	1.06 (0.74–1.53)	1.11 (0.66–1.82)	1.11 (0.63–1.97)	1.16 (0.83–1.61)	1.02 (0.71–1.46)
7 (n = 4414)	1.08 (0.94–1.25)	1.09 (0.94–1.27)	1.11 (0.98–1.25)	1.13 (0.99–1.29)	1.19 (0.96–1.47)	1.11 (0.88–1.38)	1.05 (0.92–1.19)	1.10 (0.96–1.26)
10 (n = 4544)	1.03 (0.90–1.18)	1.09 (0.94–1.27)	1.10 (0.98–1.24)	1.13 (0.99–1.29)	0.96 (0.77–1.19)	0.98 (0.77–1.25)	1.12 (0.99–1.27)	**1.15 (1.00–1.32)**
13 (n = 3833)	1.14 (1.00–1.31)	1.13 (0.98–1.31)	**1.20 (1.06–1.35)**	**1.22 (1.03–1.44)**	1.00 (0.79–1.27)	0.95 (0.74–1.23)	**1.22 (1.07–1.38)**	**1.27 (1.07–1.51)**

Abbreviations: AD, atopic dermatitis; BMI, body mass index; CI, confidence interval.
The OR for AD versus no AD was determined on the basis of 10% increment increases in carbohydrate/sugar/intrinsic milk sugar/nonmilk extrinsic sugar intake as a percentage of overall caloric intake. Analyses were adjusted for sex, race, maternal delivery age, highest parental education level, social class assessed through parental occupation, BMI, total caloric intake, and parental history of AD. The statistically significant ORs are in boldface.

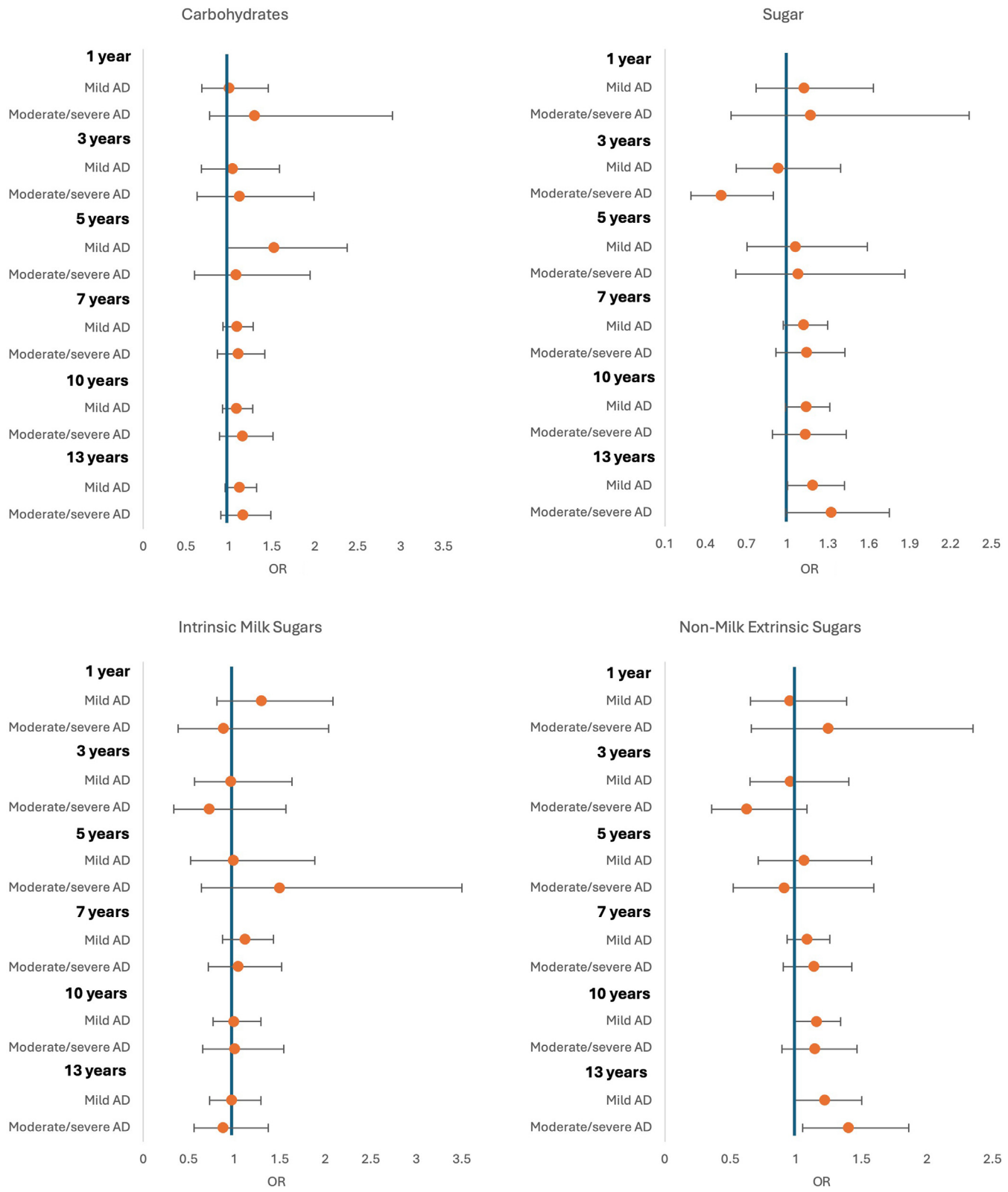

Figure 1. OR for AD by severity across 6 time points. The OR for AD by severity (vs no AD) was determined on the basis of 10% increment increases in carbohydrate/sugar/intrinsic and milk sugar/nonmilk extrinsic sugar intake as a percentage of overall caloric intake. Whiskers illustrate the 95% CI. Analyses were adjusted for sex, race, maternal delivery age, highest parental education level, social class assessed through parental occupation, BMI, total caloric intake, and parental history of AD. The only significant association was found with moderate-to-severe AD at age 13 years (OR = 1.32 [95% CI = 0.99–1.75]). AD, atopic dermatitis; BMI, body mass index; CI, confidence interval.

study is limited in generalizability because the data come from a mostly White population from the United Kingdom. Finally, the study is limited by missing data: only 5% of participants had data for 2 or more time points; therefore, we were not able to reliably examine dietary patterns over time or evaluate the impact of cumulative sugar intake (Table 3).

Our study suggests that dietary sugar is not associated with AD in early childhood, although there may be a positive association between increased nonmilk extrinsic dietary sugars and odds of AD in older childhood. This finding is consistent with nutrition guidelines to limit intake of added sugars. Future studies are needed to confirm this association and evaluate whether overall dietary patterns and diet quality have an impact on this association.

MATERIALS AND METHODS

Our study used data from the ALSPAC cohort, an ongoing longitudinal birth cohort with repeated measures of AD activity and disease severity in addition to social and environmental variables. We included children alive at the age of 1 year who had AD data for at least 1 of the following time points: 1, 3, 5, 7, 10, and 13 years (Table 4).

Eligibility and recruitment

Pregnant women who resided in a defined geographical area in southwest England with an expected delivery date between April 1, 1991 and December 31, 1992 were eligible for inclusion in ALSPAC. Recruitment was performed through advertisements in the media and distribution of information cards in health centers. A total of 14,541 (71.8% of eligible) women were recruited antenatally, and data are available for 13,988 children who were alive at age 1 year (Boyd et al, 2013; Fraser et al, 2013). The study website contains additional data details: http://www.bristol.ac.uk/alspac/researchers/our-data/. The hypothesis and design of this study are not registered prospectively in a publicly accessible source. Our study sample was limited to children who were alive at age 1 year and included assessments through age 13 years. Patterns of attrition in the cohort and intermittent participation have been described elsewhere (Boyd et al, 2013). We assumed that data were missing at random and unlikely to be systematically related to the exposure and outcome and used a complete case analysis.

Exposure

The primary exposures were total carbohydrate and total sugar intake calculated as a proportion of total caloric intake. The secondary exposures were IMS and NMES intake as a percentage of total caloric intake. These variables were collected by food frequency questionnaire completed by the child's main caregiver or the child themself (at ages 10 and 13 years) at each time point. For most foods, the mother was asked to indicate how often her child was currently consuming each, using the following options: (i) never or rarely, (ii) once in 2 weeks, (iii) 1–3 times a week, (iv) 4–7 times a week, and (v) more than once a day (Northstone and Emmett, 2005). Nutrient content was calculated for each food/drink consumed and combined to produce average daily carbohydrate and sugar intakes (Emmett, 2009). The United Kingdom Department of Health defines extrinsic sugars as sugars not located within the cellular structure of food (ie, intrinsic sugars) and are further divided into extrinsic milk sugars (lactose, naturally present in milk and dairy foods) and NMESs. IMS refers to the combination of intrinsic sugars and extrinsic milk sugars. The term NMES (used in the United Kingdom) is synonymous with the term "free sugars" used by the World Health Organization, defined as "all monosaccharides and disaccharides added to foods […] plus sugars naturally present in honey, syrups and fruit juices" (Great Britain Department of Health, 1989).

Table 3. Pattern of Missing Data

Time Points with Missing Data	Number of Participants	%
0	88	1.64
1	97	1.81
2	120	2.23
3	1323	24.63
4	1480	27.55
5	2264	42.14
Total	5372	100

Table 4. Atopic Dermatitis and Diet Data Time Points

Time Point	1	2	3	4	5	6
Atopic dermatitis	18 mo	42 mo	57 mo	81 mo	128 mo	166 mo
Diet	18 mo	43 mo	61 mo	84 mo	120 mo	156 mo

Outcome

The primary outcome was the 12-month period prevalence of AD at each age. Prevalence was chosen as the main outcome because we hypothesized that diet was most likely to play a role in disease activity and severity. We did not additionally examine incidence because ALSPAC does not include precise dates of disease onset.

The secondary outcome was AD severity over the last 12 months. Both measures were based on a maternal-reported questionnaire. Responses to AD severity were categorized as "mild" and "moderate/severe."

Covariates

We adjusted for potential confounders, including sex, race or ethnicity, maternal delivery age, highest parental education level, social class assessed through parental occupation, body mass index, total caloric intake, and maternal history of AD. Race or ethnicity data were collected from the ALSPAC study database and were originally collected to assess the representability of the study population compared with the general population (Boyd et al, 2013). Because the study took place in Avon, United Kingdom, an area with a largely White population, the race or ethnicity categories were designated as "White" and "non-White," without further delineation of specific categories within the "non-White" category. We assessed for multicollinearity by checking the variance inflation factors for all variables included in the analysis, none of which exceeded the accepted threshold of 10.

Statistical analyses

In primary analyses, we performed multinomial logistic regression to assess the association between each 10% increment increase in carbohydrate and sugar intake as a proportion of total caloric intake and the 12-month period prevalence of AD at ages 1, 3, 5, 7, 10, and 13 years. We also performed multinomial logistic regression to assess the association between each 10% increment increase in carbohydrate and sugar intake as a proportion of total caloric intake

and AD disease severity (with disease severity subgroups of "mild" and "moderate/severe") at the same ages.

In secondary analyses exploring dietary sugar subgroups, we repeated the analyses mentioned earlier for each 10% increment increase in dietary IMSs and NMESs as a proportion of total caloric intake.

We corrected for multiple comparisons using an approach derived from the Hochberg step-up method (Efird and Nielsen, 2008). Data were analyzed between January 2024 and June 2024.

ETHICS APPROVAL AND INFORMED CONSENT

Ethical approval for the study was obtained from the Avon Longitudinal Study of Parents and Children Ethics and Law Committee and then Local Research Ethics Committees. Informed consent for the use of data collected through questionnaires and clinics was obtained from participants following the recommendations of the Avon Longitudinal Study of Parents and Children Ethics and Law Committee at the time.

DATA AVAILABILITY STATEMENT

Avon Longitudinal Study of Parents and Children data access is through a system of managed open access. Datasets related to this article can be found at http://www.bristol.ac.uk/alspac/researchers/our-data/.

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CONFLICT OF INTEREST

KA is a consultant to Target RWE and receives research grants to her institution from Pfizer. The remaining authors state no conflict of interest.

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AUTHOR CONTRIBUTIONS

Conceptualization: JS, KA; Data Curation: MY; Formal Analysis: JS, MY, KA; Funding Acquisition: KA; Methodology: JS, MY, KA; Writing - Original Draft Preparation: JS, KA; Writing - Review and Editing: JS, KA, MY, S-PW, HK, AL, SML, ELV

DECLARATION OF GENERATIVE ARTIFICIAL INTELLIGENCE (AI) OR LARGE LANGUAGE MODELS (LLMS)

The author did not use AI/LLM in any part of the research process and/or manuscript preparation.

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