Br J Dermatol 2026; **00**:1–12
https://doi.org/10.1093/bjd/ljaf524
Advance access publication date: 16 January 2026

BJD
British Journal of Dermatology
Paediatric Dermatology

Associations between early-life exposures and the infant skin microbiome

Conor Broderick[1], Casper S Poulsen[2], Mathis H Hjelmsø[2], Tom Marrs[3,4], Suzana Radulovic[3,4], Kirsty Logan[3,4], Xuanji Li[5,6], Ziqi Wu[5,7], Søren J Sørensen[5], Bouchra Ezzamouri[1], Helen Alexander[1], Nanna Fyhrquist[8,9], Harri Alenius[8,10], Madhumita Bhattacharyya[11,12], Avidan U Neumann[12,13], Gideon Lack[3], Michael Perkin[14], Klaus Bønnelykke[2], Jakob Stokholm[2,15] and Carsten Flohr[1]

[1]Unit for Paediatric and Population-Based Dermatology Research, St John's Institute of Dermatology, King's College London and Guy's and St Thomas' Hospitals, London, UK
[2]COPSAC, Copenhagen Prospective Studies on Asthma in Childhood, Herlev and Gentofte Hospital, University of Copenhagen, Copenhagen, Denmark
[3]Paediatric Allergy, Department of Women and Children's Health, King's College London, London, UK
[4]Children's Allergies, Evelina London, Guy's and St Thomas' NHS Foundation Trust, St Thomas' Hospital, London, UK
[5]Section of Microbiology, University of Copenhagen, Copenhagen, Denmark
[6]Section of Microbiology, School of Life Sciences, University of Zhejiang, Hangzhou, China
[7]School of Environmental Science and Engineering, Southern University of Science and Technology, Shenzhen, China
[8]Institute of Environmental Medicine, Karolinska Institutet, Stockholm, Sweden
[9]Karlstad University, The Faculty of Health, Science and Technology, Karlstad, Sweden
[10]Human Microbiome Research Program (HUMI), Medical Faculty, University of Helsinki, Helsinki, Finland
[11]Environmental Medicine, Technical University Munich, Augsburg, Germany
[12]Environmental Medicine, Faculty of Medicine, University of Augsburg, Augsburg, Germany
[13]Institute of Environmental Medicine, Helmholtz Munich, Augsburg, Germany
[14]Population Health Research Institute, St George's, University of London, London, UK
[15]Section of Microbiology and Fermentation, Department of Food Science, University of Copenhagen, Frederiksberg, Denmark

Correspondence: Carsten Flohr. Email: carsten.flohr@kcl.ac.uk

C.B. and C.S.P. are co-first authors.

Abstract

Background Factors influencing the early-life skin microbiome and the association with atopic dermatitis (AD) are relatively unexplored.

Objectives To evaluate associations with the infant skin microbiome during the first year of life.

Methods Three-month-old infants from the Enquiring About Tolerance (EAT) birth cohort were examined for AD at enrolment and at 1 and 3 years of age. Parent-completed questionnaires, transepidermal water loss (TEWL) and *FLG* mutation status were evaluated. Bacterial swabs were collected from the elbow crease and volar forearm from 148 infants at 3 months and 1 year of age, and the microbiome composition was characterized using *16S* rRNA gene sequencing (V3–V4 region).

Results Shannon diversity was significantly higher at the forearm compared with the elbow. *Staphylococcus*, *Acinetobacter* and *Streptococcus* were the most abundant genera across time and body site. Microbiome community composition was primarily associated with body site and age ($P \leq 0.001$ for both). Other significant associations were found with ethnicity ($P=0.009$), *FLG* status ($P \leq 0.001$), urban vs. rural residence ($P=0.005$), older siblings ($P=0.04$) and bath product use at 3 months ($P=0.01$) but not with pets ($P=0.16$), systemic antibiotics ($P=0.27$) or bathing frequency ($P=0.11$). The microbiome was associated with elevated TEWL ($P=0.004$ at 3 months and $P \leq 0.001$ at 1 year) and with concurrent AD ($P=0.03$ at 3 months, $P \leq 0.001$ at 1 year). *Streptococcus parasanguinis* was significantly less abundant in the nonlesional skin of infants with AD at 3 months.

Conclusions In addition to age and body site, the infant skin microbiome is associated with heritable factors, the home environment, hygiene practices and with the presence of AD.

An author video to accompany this article is available online.

Accepted: 13 December 2025

Lay summary

At birth, our skin encounters the environment for the first time. We become carriers of 'microbes', including bacteria, viruses and fungi. Eczema affects around 1 in 4 children in the UK. It is caused by various genetic and environmental factors. Previous studies have found a strong link between eczema and the bacteria living on our skin, known as the skin 'microbiome'.

In this study, we wanted to find out whether any of the environmental factors linked with eczema also affected the skin microbiome. We studied 3-month-old babies from a UK clinical trial called 'Enquiring About Tolerance'. The babies' parents answered questions about their home, bathing habits and their baby's health. The babies were examined for eczema until they were 3 years old. We collected skin swabs from their elbows and forearms. We did this again when they were 1 year old. We then processed the swabs to study the skin microbiome. We found that the body part (elbow or forearm) and the baby's age were the key factors shaping the skin microbiome. We found other factors were linked with the skin microbiome. These included the baby's genetics, having a parent or sibling with eczema, living in a city or urban area, and using soaps and/or shampoos regularly. We did not find any links between the skin microbiome and with having pets, using antibiotics or how often the baby had a bath.

Finally, we found a link between the skin microbiome and whether the baby had eczema. But, we did not find the same bacteria that we expect in older children and adults with eczema.

What is already known about this topic?

- The skin microbiome varies by body site, and changes with age, particularly during the first year of life and at puberty.
- Delivery method affects the skin microbiome immediately after birth, but factors shaping the evolving infant skin microbiome are less well defined.
- Atopic dermatitis (AD) is associated with alterations in the skin microbiome, with increased *Staphylococcus aureus* abundance, but the relationship between the infant skin microbiome and the development of AD remains uncertain.

What does this study add?

- The skin microbiome composition was more strongly associated with body site and age than with heritable and hygiene factors, or urban vs. rural living.
- Microbiome composition was associated with skin barrier impairment, and with AD, but specific taxonomic associations were inconsistent, and *S. aureus* was infrequently identified.
- More associations were identified with the microbiome at the elbow crease than with the volar forearm, which will inform future research.

The skin microbiome consists of the communities of bacteria, fungi and viruses residing on our skin. Body site is the primary determinant of the bacterial skin microbiome, because specific bacteria thrive in moist, dry and sebaceous body sites.[1,2] Once established, our individualized bacterial microbiome remains relatively stable over time, despite continuous interactions with environmental factors and external microbes.[2–4] Our understanding of factors that shape the development of the early-life skin microbiome is limited. Delivery method has been shown to influence the skin microbiome immediately after birth.[5–7] Additionally, age, pubertal status, contact with other family members, and with soil and the wider living environment all contribute to the composition of the skin microbiome.[8–11]

People with atopic dermatitis (AD) are frequently colonized with *Staphylococcus aureus* and have a propensity to skin infections.[12–14] Beyond *S. aureus*, culture-independent microbiome studies have provided evidence of wider bacterial dysbiosis in patients with AD, as well as associations between the microbiome and AD severity and flares.[15,16] Perturbations of the early-life skin microbiome may predate the development of AD.[17–19] However, whether the early-life microbiome plays an active or bystander role in AD pathogenesis remains to be elucidated. This study aimed to characterize the infant skin microbiome, and to evaluate for associations with environmental and hygiene-related exposures, as well as associations with AD.

Patients and methods

Enquiring About Tolerance (EAT) was a population-based randomized trial of a dietary intervention aiming to prevent food allergy in 1303 term-born, 3-month-old infants.[20] Questionnaires gathered information on the infants' general health, home environment and family history. A numerical 'hygiene score' was generated based on parent-reported bathing and hygiene factors (Table S1; see Supporting Information).[21,22] Infants' home locations were categorized as urban or rural, using satellite-derived land-use data of the environment surrounding the home postcode (Figure S1; see Supporting Information).[23] Infants were examined for AD at enrolment (3 months), and at 1 and 3 years of age. AD was defined according to the photographic protocol of the International Study of Asthma and Allergies in Childhood (ISAAC) phase II, based upon the UK working-party diagnostic criteria.[24] AD severity was recorded using the SCORing Atopic Dermatitis (SCORAD) index at each clinical assessment.[25] AD severity was dichotomized as mild vs. moderate-to-severe (SCORAD $\geq$ 15), as previously described.[26]

The study was registered with the International Standard Randomized Controlled Trial Number Register (https://doi.org/10.1186/ISRCTN14254740). Further background and study methodology are available in Appendix S1 (see Supporting Information).

Sample processing and *16S* rRNA gene amplicon sequencing

EAT enrolled participants from November 2009. Beginning in December 2011, skin swabs were collected from consecutive infants at enrolment and at the scheduled 1-year visits, according to a standardized procedure. Infants with longitudinal samples at 3 months and 1 year were selected for this analysis (n=148). A comparison of infants who participated in the EAT microbiome study with the overall EAT cohort is provided in Table S2 (see Supporting Information). Microbiome samples were collected from the left volar forearm ('forearm') and antecubital fossa ('elbow') using a sterile, moistened Isohelix buccal DNA collection swab (Cell Projects, Kent, UK). An environmental control swab was collected for each visit. Further details of sample collection and laboratory and bioinformatic workflows are provided in Appendix S1, Table S3 and Figures S2–S6 (see Supporting Information).

Statistical analysis

All analyses were performed using R version 4.3.1 (R Foundation for Statistical Computing, Vienna, Austria), as described in full in Appendix S1. Shannon diversity and $\log_{10}$-transformed relative abundance were evaluated using linear mixed-effects models to account for the longitudinal and paired skin samples. Genus relative abundances were compared using Wilcoxon signed-rank tests, corrected for multiple hypotheses, using 'rabuplot' (https://github.com/jstokholm/rabuplot/). Species differential abundance testing, subset to specific body site and age combinations, was performed with function 'da.ds2' (an implementation of 'DESeq2', package 'DAtest')[27] comparing species abundances by age, urban vs. rural status and AD diagnosis, using a Wald test with sequencing run as a covariate. Bray–Curtis dissimilarity metrics were calculated using Hellinger-transformed and total-sum-scaled counts, and associations with the microbiome community composition were evaluated with permutational multivariate analysis of variance (PERMANOVA) ('adonis2' function, package 'vegan'; https://github.com/vegandevs/vegan/), using a blocked design, to account for body site and age. Ethnicity was parent-reported and dichotomized as White vs. non-White for PERMANOVAs. A more granular or inclusive approach to ethnicity classification was not possible due to the small numbers of infants with Black, Asian, mixed or other heritage. It is important to acknowledge that the EAT study was performed more than 15 years ago, and subsequently there has been substantial progress and increased emphasis on diversity and representation in research.

Results

Description of the cohort

Table 1 presents the demographic details of 148 participants in EAT who contributed longitudinal skin swabs. Most infants were White (n=128/148; 86.5%) and were born by vaginal delivery (n=116/148; 78.4%). Fifty-four infants (36.5%) resided in highly urban environments. While 81.8% of infants (n=121/148) had at least one parent with a history of atopy, only 9.5% (n=12/126) carried a loss of function (LOF) mutation in *FLG*.

Forty-five infants (30.4%) had AD at enrolment at 3 months, and 71 (48.0%) had active AD at least once before 3 years of age. AD was more prevalent in boys than girls at 3 months of age [38.2% (n=29/76) vs. 22.2% (n=16/72); P=0.049]. There was a trend towards a higher rate of AD among Asian, Black mixed and other heritage infants vs. White infants (P=0.07). *FLG* LOF was associated with AD at 3 months [18% (n=7/40) vs. 6% (n=5/86)], but statistical significance was borderline (P=0.05). AD rates at enrolment were balanced with regard to other demographic characteristics.

At enrolment, most infants were bathed 2–4 times weekly (n=79/141; 56.0%). Infants with AD at 3 months were bathed more frequently (more than once weekly) than those without AD, although the statistical significance was borderline [95% (n=41/43) vs. 83% (n=81/98); P=0.06]. Bath product use (soap, shampoo, bubble bath) was equivalent among infants with and without AD at enrolment (P=0.58). There was no association between the 3-month hygiene score quintile and AD at 3 months of age (P=0.35).

Infant skin microbiome varies with body site and age

Six samples with < 2000 reads were removed during quality control, resulting in a total of 586 clinical samples. Following contamination removal, 25 517 amplicon sequence variants (ASVs) were found. Eighty-eight per cent of ASVs were annotated at genus-level and 69.9% at species-level. Body site and age were the main determinants of alpha and beta diversity (Figure 1a, b). Shannon diversity was higher at the forearm than at the elbow, and at 1 year than at 3 months (Figure 1a). The microbiome community composition was determined more by differences between the two body sites, relative to differences by age (Figure 1b). Sequencing run and depth were also strongly associated with the community composition (Table S4; see Supporting Information), but these associations were attenuated following the removal of contaminant ASVs (Figure S4), and were accounted for in subsequent analyses.

Staphylococcus was the most abundant genus (mean relative abundance 25.2%) [Figure 1c; Figure S7, Table S5 (see Supporting Information)], followed by *Acinetobacter*, *Streptococcus*, *Corynebacterium*, *Moraxella*, *Pseudomonas* and *Bifidobacterium*. Linear mixed models demonstrated that body site and age influenced the relative abundance of these seven genera (Figure 1d). *Staphylococcus* was significantly more abundant at the elbow compared with the forearm ($P_{site} < 0.001$). *Staphylococcus* relative abundance decreased between 3 months and 1 year of age ($P_{time} < 0.001$), more notably at the elbow ($P_{interaction} = 0.04$) (Figure 1c, d). *Streptococcus* relative abundance was significantly higher at the forearm ($P_{site} < 0.001$) and increased over time at both sites ($P_{time} < 0.001$; $P_{interaction} > 0.99$). Additional model results are presented in Table S6 (see Supporting Information).

Table 1 Baseline demographics of infants from the Enquiring About Tolerance (EAT) study included in the microbiome analysis, according to atopic dermatitis (AD) status at enrolment (aged 3 months)

	No AD at enrolment ($n=103$; 69.6%)	AD at enrolment ($n=45$; 30.4%)	*P*-value
Demographics and birth history			
Sex			0.049
Female	56 (54.4)	16 (35.5)	
Male	47 (45.6)	29 (64.4)	
Ethnicity (parent-reported)			0.07
White	93 (90.3)	35 (77.8)	
Mixed heritage	8 (7.8)	6 (13.3)	
Asian or Asian British	1 (1.0)	1 (2.2)	
Black or Black British	1 (1.0)	1 (2.2)	
Other heritage	0 (0)	2 (4.4)	
FLG status			0.05
Wildtype	81 (94.2)	33 (82.5)	
Any LOF mutation	5 (5.8)	7 (17.5)	
Missing	17	5	
Caesarean delivery	21 (20.4)	11 (24.4)	0.67
Family history (self-reported)			
Maternal AD	34 (33.0)	17 (37.8)	0.58
Maternal atopy[a]	62 (60.2)	25 (55.6)	0.72
Paternal AD	20 (19.4)	14 (31.1)	0.14
Paternal atopy[a]	57 (55.3)	27 (60.0)	0.72
Older sibling(s)	51 (49.5)	27 (60.0)	0.28
Number of siblings			0.47
0	52 (50.5)	18 (40.0)	
1	38 (36.9)	19 (42.2)	
≥ 2	13 (12.6)	8 (17.8)	
Sibling(s) with AD	24 (23.3)	16 (35.6)	0.16
Home environment			
Urban–rural classification (Urban) (%)			0.58
Rural	67 (65.0)	27 (60.0)	
Urban	36 (35.0)	18 (40.0)	
Pets in the home	56 (54.4)	18 (40.0)	0.15
Cat(s) (% of those with cats)	33 (80.5)	8 (19.5)	
Dog(s) (% of those with dogs)	18 (60.0)	12 (40.0)	
Exposure to cigarette smoke	12 (11.7)	7 (15.6)	0.60
'Hard' domestic water ($CaCO_3$ concentration ≥ 257 mg L^{-1})	52 (50.5)	22 (48.9)	> 0.99
Systemic antibiotic use[b]			
Antibiotics before enrolment	13 (13.3)	5 (11.6)	> 0.99
Missing	5	2	
Antibiotics in the 30 days preceding the 1-year visit	23 (23.2)	4 (9.5)	0.07
Missing	4	3	
Bathing and hygiene variables (3-month questionnaire)			
Bathing frequency			0.10
Once a week or less	17 (17.3)	2 (4.7)	
2–4 times a week	49 (50.0)	30 (69.8)	
5–6 times a week	8 (8.2)	3 (7.0)	
Daily or more than daily	24 (24.5)	8 (18.6)	
Missing	5	2	
Infrequent (weekly or less) bathing	17 (17.3)	2 (4.7)	0.06
Missing	5	2	
Bath product use (soap, shampoo, bubble bath)	60 (61.2)	24 (55.8)	0.58
Missing	5	2	
Frequency of handwashing/hand wiping			0.30
Not at all	11 (11.2)	9 (20.9)	
1–2 times a day	72 (73.5)	27 (62.8)	
3–4 times a day	15 (15.3)	7 (16.3)	
≥ 5 times a day	0	0	
Missing	5	2	
Wet wipes used to clean hands	23 (26.4)	7 (20.6)	0.64
Missing	16	11	

Data are presented as *n* (%). Microbiome samples were collected from infants enrolled sequentially in EAT; however, only infants with swabs taken both at 3 months and 1 year were analysed. *P*-values were calculated using Fisher's exact test. LOF, loss of function. [a]Asthma, AD, hay fever or food allergy. [b]Systemic antibiotic use was based on parental recall and did not specify which antibiotic was used, the indication or the duration of treatment.

We obtained similar findings at species level. *Staphylococcus epidermidis* was most abundant overall and predominated at the elbow (Figure S8; see Supporting Information). Notably, *S. aureus* was infrequently identified at 3 months ($n=27$ infants) and at 1 year ($n=23$ infants), and with a mean relative abundance of 0.07% (range

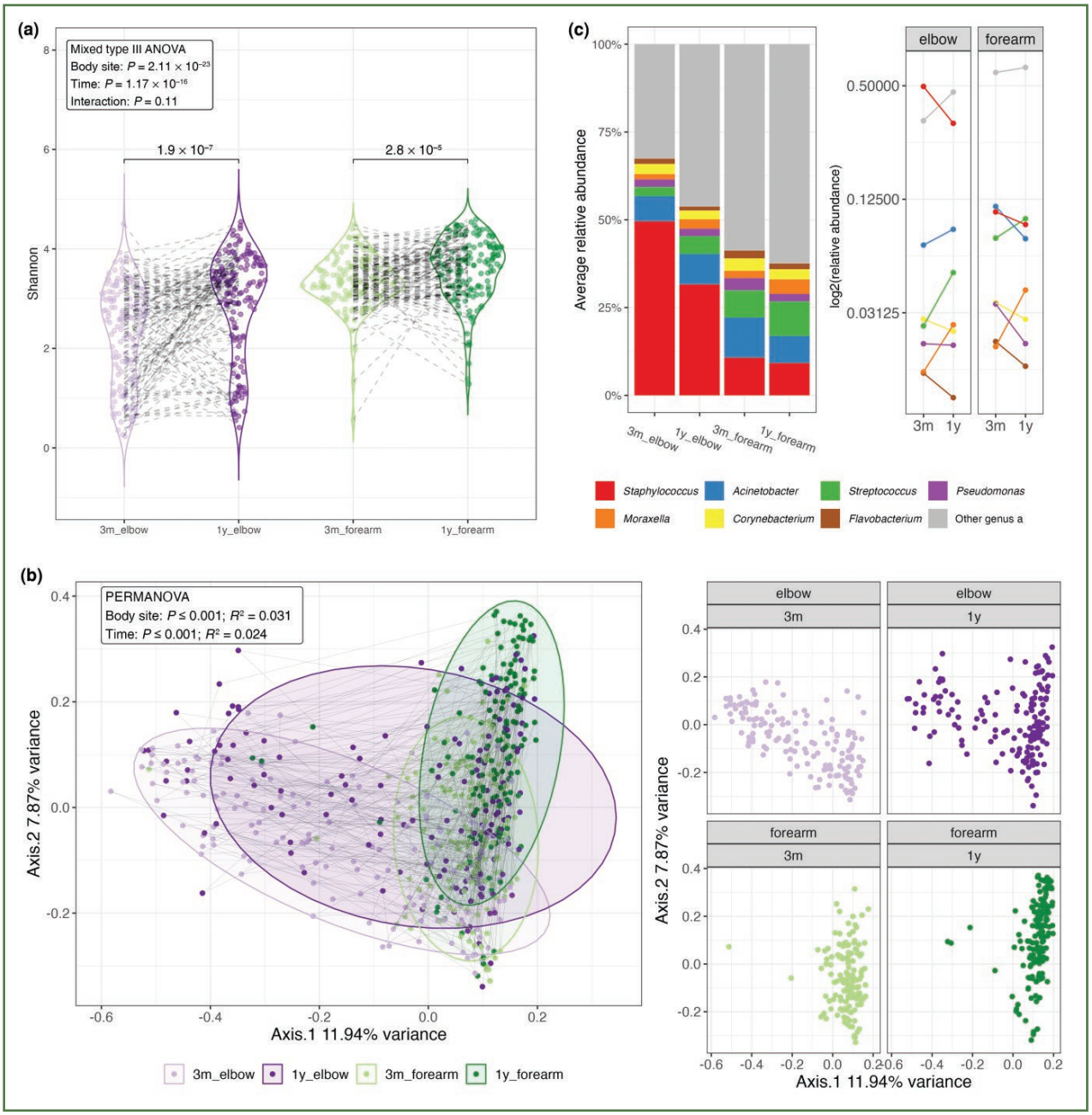

Figure 1 The infant skin microbiome is highly influenced by body site and age. (a) Violin plots of Shannon diversity according to the infants' age ('time') and body site sampled. Paired samples from each timepoint are joined by dashed lines. A linear mixed-effects model (text box) demonstrated the significant effect of body site and infants' age on Shannon diversity, adjusting for sequencing depth, natural logarithm-transformed sequencing depth and sequencing run as fixed effects, and the individual infants as random effects. The interaction (between age and body site) was not statistically significant. Shannon diversity was significantly higher at 1 year compared with at 3 months at the elbow and the forearm. *P*-values in the plot were generated using Wilcoxon signed-rank tests, adjusted for multiple hypothesis testing (Holm method). (b) Principal coordinate analysis (PCoA) ordination demonstrating that genus-level community composition (Bray–Curtis dissimilarity index) was more strongly associated with body-site than with infants' age. R^2 and *P*-values (inset) were generated using permutational multivariate analysis of variance (PERMANOVA) based on 999 permutations constrained by age and body site, and adjusted for sequencing run and sequencing depth. Count data were Hellinger transformed and total sum scaled prior to PERMANOVA. (c) Stacked bar plots and line plots and (d) violin plots depict the relationship between the relative abundance of the seven most abundant genera, the infant's age and body site sampled. Linear mixed models [D] were generated using transformed relative abundances, to facilitate visualization of right-skewed data (log_{10}-transformation following the addition of a pseudocount of 0.01). Linear mixed models were generated with sequencing depth, natural logarithm-transformed sequencing depth and sequencing run as fixed effects, and the individual infants as random effects. *Staphylococcus* was the most abundant genus overall, particularly at the elbow at 3 months. *Staphylococcus* abundance was significantly associated with age and with body site, and abundance decreased between 3 months and 1 year at the elbow, while abundance remained relatively stable at the forearm, as evidenced by the significant interaction between body site and age from the linear mixed model. Conversely, while *Streptococcus* abundance was significantly associated with age and with body site, and was higher at the forearm than at the elbow both in 3-month and 1-year samples, the interaction between age and body site was not statistically significant, which signified a consistent relationship between *Streptococcus* abundance and body site and age. 1y, 1 year; 3m, 3 months. (*Continued*)

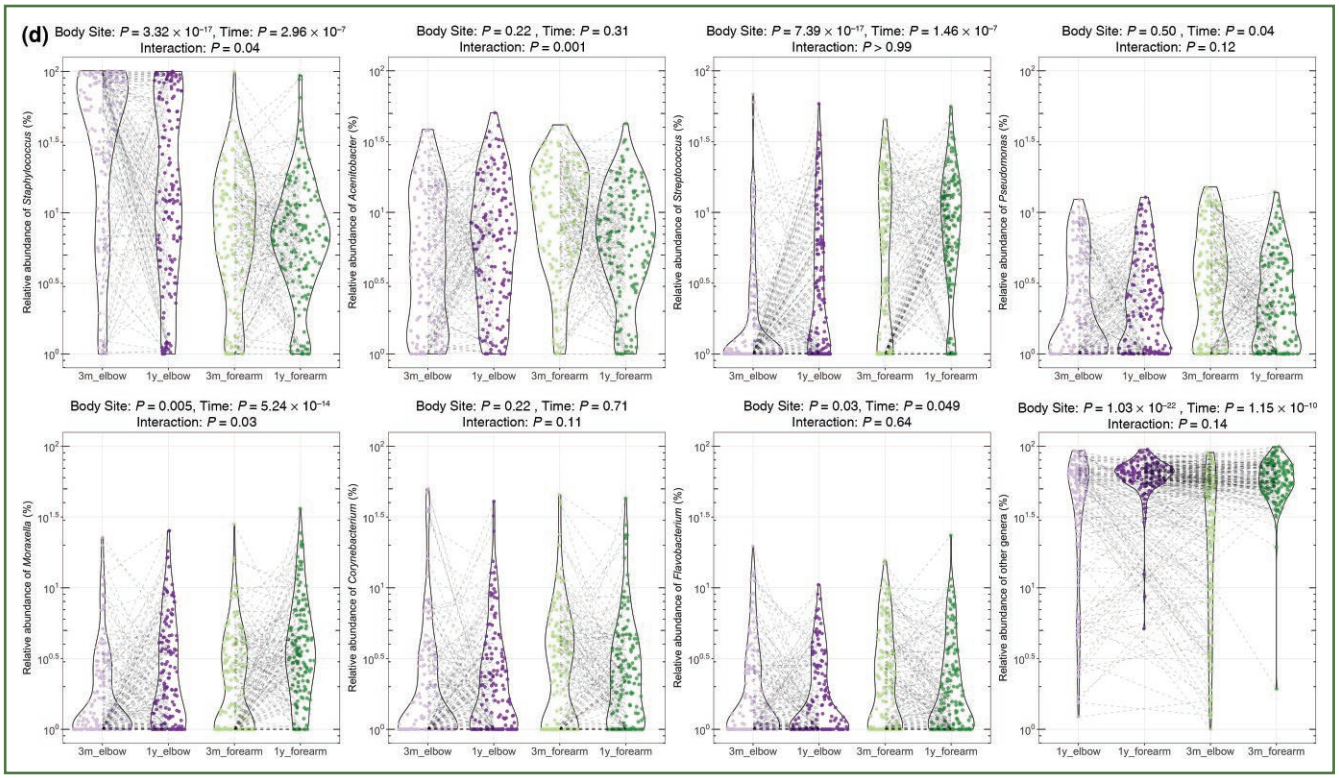

Figure 1 (*Continued*)

0.002–2.27%). Differential abundance testing identified numerous species that varied with time, at each body site, including various *Streptococcus* species (Figures S9, S10 and Table S7; see Supporting Information).

Associations between the early-life skin microbiome and intrinsic and extrinsic factors, including hygiene practices

Figure 2 presents the PERMANOVA results of the evaluation of associations between the species-level community composition and intrinsic and heritable factors, and extrinsic factors encompassing the household and living environment and personal hygiene practices (Tables S8–S10; see Supporting Information). We found significant variations in the infant skin microbiome according to the infant's sex ($P=0.046$), ethnicity ($P=0.009$), *FLG* LOF status ($P\leq0.001$), paternal history of AD ($P=0.03$), paternal atopy ($P=0.04$) and having a sibling with AD ($P=0.004$). Delivery method, maternal history of AD and maternal atopy were not significantly associated with the microbiome composition ($P>0.05$).

The overall skin microbiome composition was associated with an objective urban vs. rural classification of the infant's home environment at 3 months ($P=0.005$), and the association with the elbow microbiome at 1-year was highly significant ($P\leq0.001$). At 3 months, *Corynebacterium* was more abundant at the forearm in infants from rural areas [$P=0.03$; Figure S11 (see Supporting Information)]; however, this finding was not significant following adjustment for multiple hypotheses ($q=0.20$). Rural living was associated with the abundance of four taxa in the 3-month forearm samples (Figure S12b; see Supporting Information). Meanwhile, at the elbow at 1 year, we found a significant association between an urban vs. rural environment and the abundance of three commensal *Staphylococcus* species, among others (Figure S12c). There were no differentially abundant species at other time–site combinations (Figures S12a, d; see Supporting Information).

A significant association was identified with the number of older siblings ($P=0.04$). Cigarette smoke exposure was only associated with the microbiome composition at the forearm in 1-year-old children ($P=0.03$). There was no association with pet ownership at 3 months. There was also no association between the microbiome composition and systemic antibiotic use.

Bathing and personal hygiene factors were associated with significant variations in the infant skin microbiome (Table S10), particularly bath product use ($P=0.01$), frequency of hand wiping and handwashing ($P=0.005$), and using baby wipes to clean infants' hands ($P=0.005$). There was no significant association between community composition and domestic water hardness ($P=0.14$). Bathing frequency at 3 months was not associated with the microbiome ($P=0.11$). However, we identified a borderline significant association when bathing frequency was dichotomized ('once per week or less' vs. more frequent bathing, $P=0.05$), a variable previously associated with AD and barrier dysfunction in this cohort.[28] There was also a significant association with the hygiene score (numerical hygiene score: $P=0.04$; quintile of hygiene score: $P=0.001$). Many of the significant associations with the overall microbiome composition were not identified when PERMANOVAs were performed using only the swabs from each age and body site combination (Tables S8–S10).

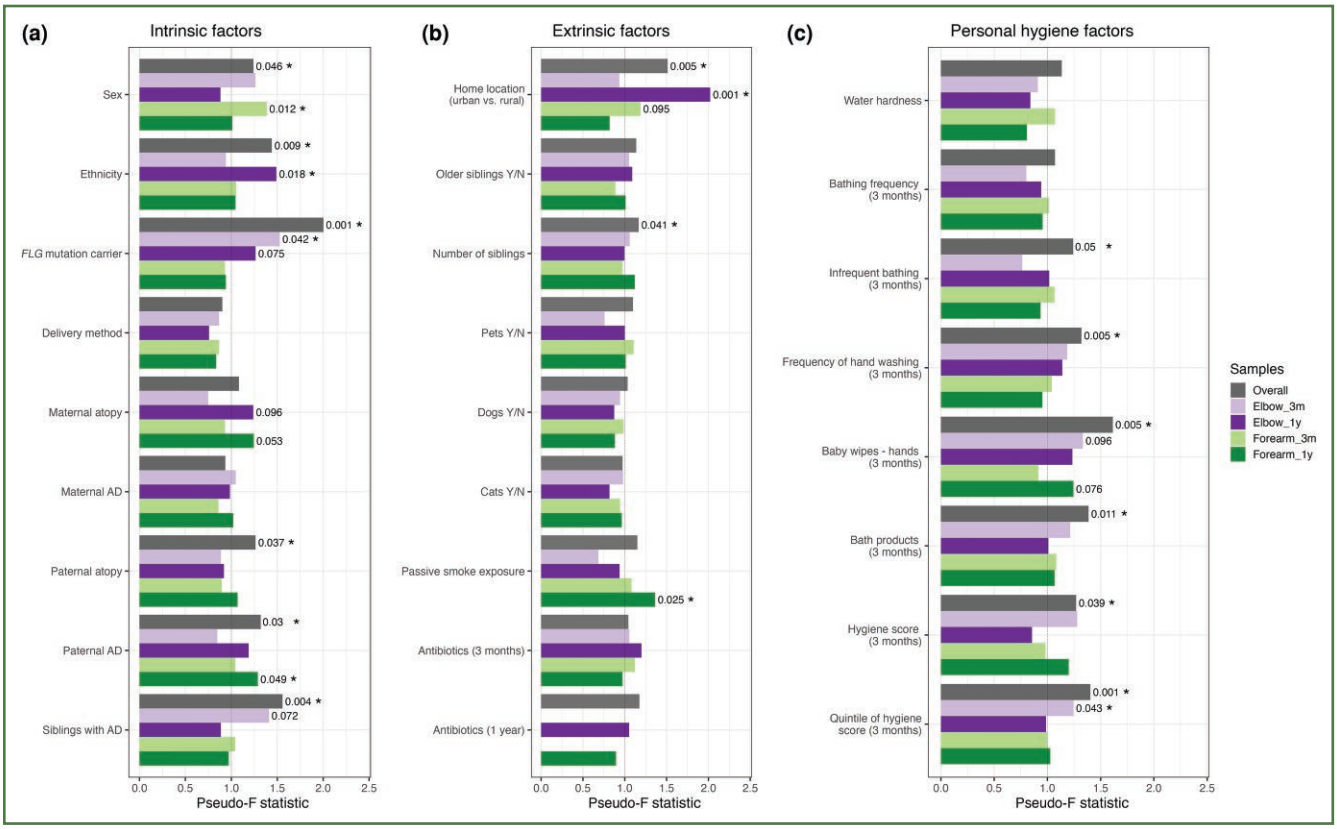

Figure 2 The early-life skin microbiome is associated with a variety of intrinsic and heritable, extrinsic and hygiene-related factors. Bar plots of pseudo-F statistics and *P*-values evaluating the association between the species-level composition of the infant skin microbiome and (a) intrinsic and heritable factors; (b) extrinsic factors, including the home environment and systemic antibiotic use; and (c) personal hygiene factors. Permutational multivariate analysis of variance (PERMANOVA) was performed using the Bray–Curtis dissimilarity index, assessing the marginal effect of each variable, with 999 permutations (constrained by age and body site) and adjusted for sequencing run and sequencing depth. Bar plots were annotated with *P*-values ≤ 0.10, with significant *P*-values (≤ 0.05) highlighted with an asterisk (*). Data were Hellinger transformed and total-sum scaled prior to PERMANOVA. Overall PERMANOVA results (calculated using all samples) are presented as grey bars, while associations with age and body site-specific results are presented with purple and green bars. In (b), overall PERMANOVA regarding antibiotic use in the month preceding the 1-year visit used 1-year samples only. In (c), infrequent bathing refers to bathing once weekly or less. Exposure to bath products involved parent-reported use of soap, shampoo and/or bubble bath. The EAT hygiene score was calculated according to the responses regarding weekly bathing frequency, daily hand and face cleaning frequency, and the use of baby wipes to clean infants' hands, and was evaluated as a linear descriptor of hygiene practice and categorized into quintiles. Full PERMANOVA results are presented in Tables S8–S10. 1y, 1 year; 3m, 3 months; AD, atopic dermatitis; N, no; Y, yes.

Presence of atopic dermatitis in infancy is associated with overall composition of the skin microbiome

Table 2 summarizes the prevalence of AD and associated characteristics, including distribution, age of onset and severity. AD was more frequent in 3-month-old and 1-year-old infants vs. 3-year-old children [30.4% (n=45/148) and 28.4% (n=42/148) vs. 14.7% (n=21/143)]. AD severity remained mostly mild-to-moderate during the study; only nine infants had a SCORAD index ≥ 15. The median SCORAD index was highest at 3 years [median 11.50 (interquartile range 6.30–39.20)].

Shannon diversity was significantly lower in those with AD at 3 months [P=0.03; Figure S13 (see Supporting Information)], and specifically at the elbow (P=0.004) but not the forearm (P=0.59). There was no association between raised transepidermal water loss (TEWL) and Shannon diversity at 3 months (Figure S14; see Supporting Information). At 1 year, there was no association between Shannon diversity and AD (Figure S15; see Supporting Information); however, Shannon diversity was significantly lower in infants with raised TEWL at 1 year [P=0.008; Figure S16 (see Supporting Information)], specifically at the elbow (P=0.02).

Given the fluctuating nature of AD over time, we assessed associations between AD and the skin microbiome composition at corresponding timepoints: AD at 3 months with 3-month samples and AD at 1 year with 1-year samples [Figure 3; Tables S11, S12 (see Supporting Information)]. At 3 months, AD was associated with the overall microbiome composition (P=0.03). AD at the elbow at 3 months was associated with the overall microbiome composition (P=0.008), and specifically with the microbiome at the elbow (P=0.02). Similar patterns emerged at 1 year (Figure 3b). AD was associated with the overall microbiome composition (P≤0.001) and with the microbiome composition at the elbow (P=0.02) but not the forearm (P=0.22). AD at the elbow at 1 year was associated with the overall microbiome composition (P=0.04) but not with the elbow microbiome (P=0.15), as was seen at 3 months. AD on the forearm was only associated with the overall microbiome at 1 year (P=0.006). We found a borderline association between AD

Table 2 Diagnosis and severity of atopic dermatitis (AD) and skin barrier impairment in infants up to 3 years of age ($n=148$)

	Yes (%)	No (%)	Missing (*n*)
3-month variables			
AD at 3 months	45 (30.4)	103 (69.6)	0
Active AD on forearms (% of those with AD)	5 (11.1)	40 (88.9)	0
Active AD at antecubital fossae (% of those with AD)	6 (13.3)	39 (86.7)	0
SCORAD, median (IQR)	3.70 (3.60– 7.10)	NA	
Moderate-to-severe AD (SCORAD ≥ 15)	3 (6.7)	42 (93.3)	103
TEWL, median (IQR)	12.72 (10.77–15.26)		
Raised TEWL (≥ 15.0 g m^{-2} h^{-1})	43 (29.1)	105 (70.9)	
1-year variables			
AD at 1 year	42 (28.4)	106 (71.6)	0
Active AD on forearms (% of those with AD)	8 (19.0)	34 (81.0)	0
Active AD at antecubital fossae (% of those with AD)	10 (23.8)	32 (76.2)	0
SCORAD, median (IQR)	7.70 (3.73–14.37)	NA	
Moderate-to-severe AD (SCORAD ≥ 15)	9 (21.4)	33 (78.6)	106
TEWL, median (IQR)	14.06 (11.81–17.07)		16
Raised TEWL (≥ 16.1 g m^{-2} h^{-1})	44 (33.3)	88 (66.7)	16
3-year and longitudinal variables			
AD at 3 years	21 (14.7)	122 (85.3)	5
Active AD on forearms (% of those with AD)	6 (28.6)	15 (71.4)	5
Active AD at antecubital fossae (% of those with AD)	9 (42.9)	12 (57.1)	5
SCORAD, median (IQR)	11.50 (6.30– 39.20)	NA	0
Moderate-to-severe AD (SCORAD ≥ 15)	9 (47.4)	10 (52.6)	129
AD at least once by 3 years (cumulative AD ever)	71 (48.6)	75 (51.4)	2
Age of onset of AD			2
Early-onset AD (present at 3 months)	45 (30.8)		
Late-onset AD (developed after 3 months)	26 (17.8)		
Unaffected by AD by 3 years of age	75 (51.4)		

Data are presented as *n* (%) unless otherwise stated. The total SCORing of Atopic Dermatitis (SCORAD) index was recorded at each visit in infants with AD (range 0–103). A SCORAD ≥ 15 was used to define moderate to severe AD, as per our previous publication.[26] Transepidermal water loss (TEWL) was measured on clinically unaffected skin on the left volar forearm in triplicate, and the mean value was calculated for each infant, at each visit. Results were dichotomized ('raised TEWL') if the value was above the upper quartile of TEWL measurements in infants without visible AD at that age [median TEWL at 3 months 12.4 g m^{-2} h^{-1} (IQR 10.4–15.0); median TEWL at 1 year 13.7 g m^{-2} h^{-1} (IQR 11.4–16.1).[26,44] Further information is provided in Appendix S1. IQR, interquartile range; NA, not applicable.

severity and the overall microbiome composition at 1 year ($P=0.06$). Despite small numbers, AD severity was associated with the elbow microbiome at 1 year ($P=0.04$). In longitudinal analysis (Figure 3c), the microbiome during the first year of life was not associated with the presence of AD at 3 years of age ($P=0.11$).

Skin barrier impairment at 3 months ($P=0.004$) and 1 year ($P\leq0.001$) was associated with the microbiome composition. Frequency of moisturizer use was not associated with the skin microbiome composition.

Taxonomic differences associated with atopic dermatitis in infancy

Staphylococcus was more abundant at the elbow in infants with AD ($P=0.04$), but this finding lost significance after adjustment for multiple testing ($q=0.21$) (Figure S17; see Supporting Information). One-year-old infants with no AD had significantly higher *Pseudomonas* relative abundance at the elbow, surviving adjustment ($P=0.003$, $q=0.025$). Other associations with AD at 1 year were not statistically significant after adjustment.

At the species level, 3-month-old infants with AD had lower *Streptococcus parasanguinis* abundance, at the elbow and the forearm (Figures S18, S19, Table S13; see Supporting Information). AD was associated with lower *Corynebacterium vitaeruminis* abundance at the elbow at 3 months (Figure S18) and at the forearm at 1 year (Figure S20; see Supporting Information). Other taxa were differentially abundant only at single time–site combinations (Figures S18–S21; see Supporting Information). *Staphylococcus aureus* was not frequently identified and was thus excluded from the differential abundance analysis following species filtering. Other *Staphylococcus* species were not significantly associated with the presence of AD (Table S13).

Discussion

We studied the evolution of the skin microbiome of term-born infants during the first year of life by sequencing longitudinal skin swabs from a moist and a dry ecological niche. Body site and age were the most significant determinants of the skin microbiome in this early-life cohort. Sex, ethnicity, *FLG* LOF status, paternal AD, urban vs. rural living and hygiene practice score demonstrated significant associations in the overall microbiome composition analysis and in certain body site and age-specific analyses. Other associations were only significant with the overall microbiome composition, including the number of older siblings, having a sibling with AD, bathing frequency and using bath products. Delivery method, the presence of pets and hard domestic water were not associated with the microbiome composition. The association between the skin microbiome and AD was stronger at 1 year than at 3 months. There was no association between the skin microbiome during the first year of life and the presence of AD at 3 years.

Our study contributes to the limited body of evidence relating to the early-life skin microbiome. Low biomass sites, such as the skin, require rigorous contamination control

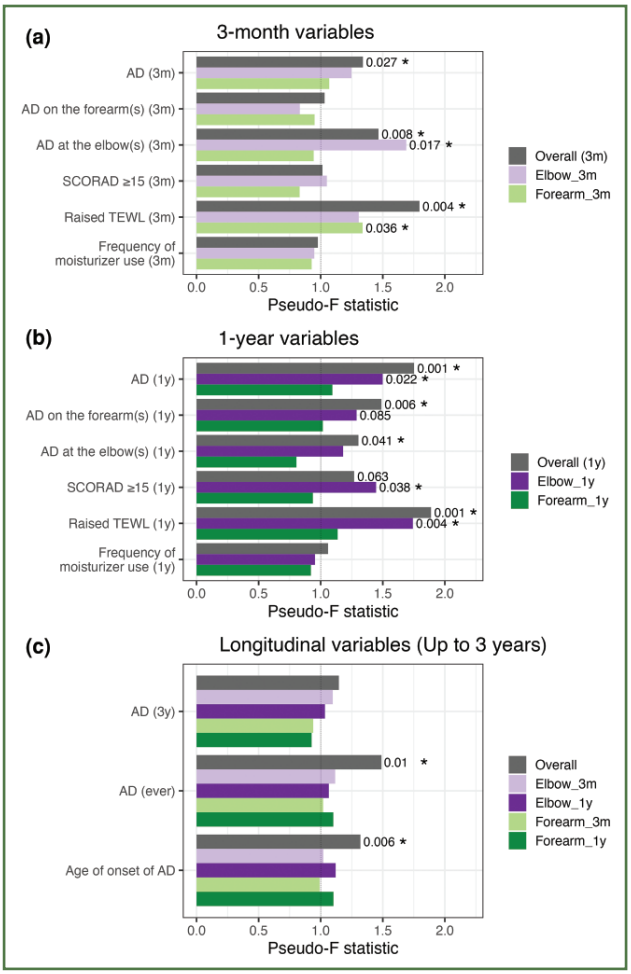

Figure 3 Atopic dermatitis (AD), AD severity and skin barrier impairment are associated with the composition of the infant skin microbiome. Bar plots of pseudo-F statistics and *P*-values evaluating the association between the species-level composition of the infant skin microbiome and AD and related factors at (a) 3 months, (b) at 1 year and (c) longitudinally up to 3 years of age. Permutational multivariate analysis of variance (PERMANOVA) was performed using the Bray–Curtis dissimilarity index, assessing the marginal effect of each variable, with 999 permutations (constrained by age and body site), and adjusted for sequencing run and sequencing depth. Bar plots were annotated with *P*-values ≤ 0.10, with significant *P*-values (≤ 0.05) highlighted with an asterisk (*). Data were Hellinger transformed and total-sum scaled prior to PERMANOVA. Overall PERMANOVA results (calculated using all samples) are presented as grey bars, while associations with specific age and body site-specific results are presented with purple and green bars. (a, b) Time-congruous analyses evaluating associations between clinical outcomes and the microbiome at the same time point (either 3-month or 1-year outcomes and respective 3-month and 1-year samples only). In (c), 'atopic dermatitis (3y)' refers to the presence of AD on examination at 3 years of age, while 'atopic dermatitis (ever)' refers to the diagnosis of AD at any point between enrolment and 3 years of age. Age of onset categorizes infants as early-onset AD (present at 3 months), late-onset (present at 1 year or later) or unaffected (by 3 years of age). The complete PERMANOVA results are presented in Tables S11 and S12. 1y, 1 year; 3m, 3 months; SCORAD, SCORing of Atopic Dermatitis index; TEWL, transepidermal water loss.

measures throughout sampling and sequencing procedures. Environmental control samples collected during skin sampling were particularly important during quality control.

Taxonomic identification beyond genus using *16S* rRNA gene sequencing methods is often restricted by shared sequence homology.[29,30] To improve taxonomic resolution, we used AnnotIEM to leverage multiple annotation databases.[31–33] A similar approach has been used by an independent research group, supporting this methodology.[34] Shotgun metagenomic sequencing may allow more definite species- and strain-level analysis, as well as interrogation of the functional importance of the skin microbiome.

Thirty per cent of infants already had active AD at the first microbiome sampling at 3 months. In order for future studies to answer whether the microbiome plays a direct role in AD development,[35] samples need to be collected prior to disease initiation, as well as longitudinally thereafter to monitor changes throughout the disease trajectory.

Finally, the associations with intrinsic, environmental and hygiene-related factors were systematically evaluated using the marginal effects of each variable in separate permutational models. PERMANOVA results were adjusted for sequencing run and sequencing depth, with permutations constrained by body site and the infants' age, to address the significant impact of these variables on the skin microbiome.

A small number of studies have evaluated for associations between AD and the infant skin microbiome.[7,15,17–19,36–42] Prior to our study, only Rapin *et al.* comprehensively evaluated associations with AD-associated epidemiological factors and skin barrier changes.[7] PreventADALL was an allergy-focused population-based randomized trial with longitudinal skin sampling, allowing direct comparison with our results. However, there were important methodological differences, including sampling body site (outer upper arm in PreventADALL), and technical factors, including *16S* rRNA gene sequencing primer choices (V4 in PreventADALL).

Rapin *et al.* demonstrated an association with delivery method on day 1 of life, which was no longer significant by 3 months of age, in keeping with our results, as well as the existing literature.[5–7,17] While we found no association with pets in the home at 3 months, Rapin *et al.* reported that exposure to dogs during pregnancy was associated with the microbiome composition.[7]

We objectively classified the home environment using satellite data and demonstrated significant associations between urban vs. rural living and the composition of the microbiome, similar to Rapin *et al.*,[7] who found an association with a highly urban birth location. In our study, rural infants were more likely to be White (93.6% vs. 74.1%, $P=0.002$) and to live with pets (56.4% vs. 38.9%; $P=0.06$); otherwise, the groups were similar. Urban vs. rural living was most significantly associated with the 1-year elbow microbiome, which we attribute to infants' developmental milestones, where older infants increasingly interact with their surroundings, and hence more environmental microbes. Previous studies have indicated an association between an objective urban vs. rural classification and the skin microbiome in older children,[11] as well as the infant gut and airway microbiome.[23] Future work should investigate how demographic, socioeconomic and environmental factors (e.g. air pollution) shape microbial differences in urban vs. rural areas, as this may provide insights into higher rates of AD in urban populations.

FLG mutation status and skin barrier dysfunction were significantly associated with the skin microbiome in EAT.

PreventADALL found no significant association with *FLG* mutations,[7] but we characterized *FLG* more comprehensively, and the PreventADALL analysis may have been underpowered.

We found a significant association between the microbiome composition and the presence of concurrent AD both at 3 months and 1 year. However, we did not identify strong signals for any AD-associated genera or species. The low prevalence and abundance of *S. aureus* in this study was noteworthy, despite the high prevalence of AD, as there is a vast literature linking AD and *S. aureus* in older children and adult populations.[12,15,16,43] The scarcity of *S. aureus* in our cohort may reflect the by-and-large mild AD in the EAT cohort, and that we did not purposely swab lesional skin.[43] However, our findings are consistent with a growing body of literature that does not implicate *S. aureus* in infant AD.[7,17–19,40] Sampling from active dermatitis (lesional skin) may be informative regarding any potential link between *S. aureus* and infant AD.

In summary, the infant skin microbiome was primarily influenced by body site and age, and was associated with a variety of epidemiological factors relevant to AD, including urban vs. rural living, siblings, family history of atopy, and infant bathing and hygiene practices. We identified significant associations between the skin microbiome and AD. The differential abundance of bacterial species in infants with AD, and in infants from urban vs. rural environments, warrants further study. This research contributes to our understanding of the 'biodiversity hypothesis', which suggests that the reduction in personal and environmental biodiversity is responsible for the increased prevalence of chronic inflammatory diseases such as AD and allergies.

Author contributions

Conor Broderick (Formal analysis, Visualization, Writing—original draft [lead], Investigation, Methodology [equal], Software [supporting]), Casper Sahl Poulsen (Formal analysis, Software, Visualization [lead], Investigation, Methodology, Writing—original draft [equal]), Mathis H. Hjelmsø (Formal analysis, Investigation, Software [supporting], Writing—review & editing [equal]), Tom Marrs (Investigation, Methodology, Writing—review & editing [equal]), Suzana Radulovic (Investigation, Writing—review & editing [equal]), Kirsty Logan (Data curation, Project administration, Writing—review & editing [equal]), Xuanji Li (Investigation, Writing—review & editing [equal], Software [supporting]), Ziqi Wu (Investigation, Writing—review & editing [equal], Software [supporting]), Søren J. Sørensen (Resources, Writing—review & editing [equal], Supervision [supporting]), Bouchra Ezzamouri (Investigation, Methodology [supporting], Writing—review & editing [equal]), Helen Alexander (Investigation, Methodology [supporting], Writing—review & editing [equal]), Nanna Fyhrquist (Project administration [supporting], Writing—review & editing [equal]), Harri Alenius (Funding acquisition, Writing—review & editing [equal]), Madhumita Bhattacharyya (Investigation, Methodology, Software [supporting], Writing—review & editing [equal]), Avidan U. Neumann (Methodology, Resources [supporting], Writing—review & editing [equal]), Gideon Lack (Conceptualization, Funding acquisition, Methodology, Resources, Writing—review & editing [equal]), Michael Perkin (Conceptualization, Funding acquisition, Methodology, Resources, Writing—review & editing [equal], Supervision [supporting]), Klaus Bønnelykke (Conceptualization, Supervision [supporting], Funding acquisition, Resources, Writing—review & editing [equal]), Jakob Stokholm (Conceptualization, Funding acquisition, Investigation, Methodology, Resources, Software, Writing—original draft, Writing—review & editing [equal], Supervision [lead]), and Carsten Flohr (Conceptualization, Resources, Supervision [lead], Funding acquisition, Investigation, Methodology, Writing—original draft, Writing—review & editing [equal]).

Funding sources

The main components of the Enquiring About Tolerance (EAT) study were jointly funded by the Food Standards Agency (UK) (grant code T07051) and the Medical Research Council (grant MC_G1001205). The skin-related aspects of the study were supported by a Clinician Scientist Award from the UK National Institute for Health and Care Research (NIHR) held by C.F. (NIHRCS/01/2008/009). This project also received funding from the Innovative Medicines Initiative 2 Joint Undertaking (JU) under grant agreement no. 821511 (BIOMAP). The JU receives support from the European Union's Horizon 2020 research and innovation programme and EFPIA (European Federation of Pharmaceutical Industries and Associations). This publication reflects only the authors' views, and the JU is not responsible for any use that may be made of the information it contains. The granting agencies covered costs and played no role in the manuscript preparation.

Conflicts of interest

S.R. receives salary support from grants from the National Institute of Allergy and Infectious Diseases [NIAID, National Institutes of Health (NIH)]. X.L. is supported by BIOCODEX INTERNATIONAL GRANT 2022. G.L. is part of the scientific advisory board for Aimmune, Novartis and DBV Technologies; is Joint Director of the Food Allergy Immunotherapy Centre; a shareholder in DBV Technologies and Mission MightyMe; and has received grants from the NIAID (NIH), the Medical Research Council and the National Peanut Board. C.F. is Chief Investigator of the UK National Institute for Health Research-funded TREAT (ISRCTN15837754) and SOFTER (ClinicalTrials.gov NCT03270566) trials, as well as the UK–Irish Atopic eczema Systemic TherApy Register (A-STAR; ISRCTN11210918) and a Principle Investigator in the European Union (EU) Horizon 2020-funded BIOMAP Consortium (http://www.biomap-imi.eu/). He also leads the EU Trans-Foods consortium. He has also received compensation from the *BJD* (reviewer and Section Editor) and EuroGuiDerm (guidelines lead). In addition, C.F. has received support from Almirall, Amgen, Apogee, Bioderma, Pfizer and Sanofi for travel, lectures and consultancy. The other authors declare no conflicts of interest.

Data availability

De-identified *16S* sequencing data are available at the European Nucleotide Archive (ENA) under accession number ERP188919 (https://www.ebi.ac.uk/ena/

browser/view/ERP188919). R code has been deposited on GitHub (https://github.com/BIOMAP-EATCOPSAC/EAT-Brodericketal2026).

Ethics statement

Ethics approval was granted by the Guy's and St Thomas' Hospital Research Ethics Committee (reference 08/H0802/93).

Patient consent

Written informed consent for participation in the EAT clinical trial, including publication of the study findings, was obtained from each participant's parent(s) or guardian(s).

Supporting Information

Additional Supporting Information may be found in the online version of this article at the publisher's website.

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Co-primary endpoints PASI 90 and IGA 0/1 at Week 16 were met.**Secondary endpoints. †N= mNRI, missing data were imputed with mNRI (patients with missing data following treatment discontinuation due to lack of efficacy or a TRAE were counted as non-responders; multiple imputation methodology was used for other missing data). ‡43.9% (n=189/431), and 43.4% (n=116/267) of biologic-naïve and TNFi-IR PsA patients achieved the primary endpoint of ACR 50 at Week 16 in BE OPTIMAL and BE COMPLETE, respectively (vs 10.0% [n=28/281] and 6.8% [n=9/133] placebo, p<0.0001); 54.5% (n=235/431) and 51.7% (n=138/267) maintained it at Week 52 (NRI).[4–6]

ACR 50, ≥50% response in the American College of Rheumatology criteria; **AS**, ankylosing spondylitis; **CRP**, C-reactive protein; **DMARD**, disease-modifying antirheumatic drug; **HS**, hidradenitis suppurativa; **IGA**, Investigator's Global Assessment; **(m)NRI**, (modified) non-responder imputation; **MRI**, magnetic resonance imaging; **nr-axSpA**, non-radiographic axial spondyloarthritis; **NSAID**, non-steroidal anti-inflammatory drug; **PASI 75/90/100**, ≥75/90/100% improvement from baseline in Psoriasis Area and Severity Index; **PsA**, psoriatic arthritis; **PsD**, psoriatic disease; **PsO**, psoriasis; **TNFi-IR**, tumour necrosis factor-α inhibitor – inadequate responder; **TRAE**, treatment-related adverse event.

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