


Do socioeconomic factors impact atopic dermatitis outcome? A single-center study

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Abstract

Background: Race and socioeconomic status are thought to influence the severity of atopic dermatitis (AD), but findings differ between countries and measures used. The role of social determinants of health versus biologic factors in causing these differences is poorly understood.

Objective: We hypothesized that spatially-derived factors correlate with AD severity and patient-reported outcome (PRO) in a pediatric cohort from Chicago, USA.

Methods: Children with AD and caregivers were enrolled from February 2018 to April 2019 in this single-site cross-sectional study. Severity was self- and physician-assessed using validated measures. Patient addresses were geocoded and linked to census tract IDs. Deprivation index (DI) was calculated using variables of the 2018 American Community Survey.

Results: Among 216 children aged 5–17 years old, 111 (51.4%) lived in urban, 104 (48.1%) suburban, and one (0.5%) in rural areas. Race was self-classified as White in 31.0%, Black 24.5%, other or mixed 25.0%, and Asian 19.4%; 24.5% were Hispanic. Median DI was 0.32 (range 0.03–0.72), with higher scores indicating more deprivation. DI correlated with insurance type, family income, ethnicity, race, and parental education, and weakly with selected PRO T-scores. However, no correlations between any AD severity score and DI, race, ethnicity, income, education, or insurance type were found.

Conclusion: The impact of socioeconomic factors on AD severity in our study population was less pronounced than expected. This could be because of regional differences, including access to high-quality care. The role of access as a deciding factor in the impact of socioeconomic status on AD outcome deserves further investigation.

KEYWORDS

access, atopic dermatitis, deprivation index, health disparities, healthcare, Medicaid, patient-reported outcomes, public insurance, race/ethnicity, socioeconomic factors

1 | INTRODUCTION

Atopic dermatitis (AD) is one of the most common dermatoses in children worldwide, affecting all races and socioeconomic groups. The influence of socioeconomic status (SES) on AD has been subject of research, with findings varying between countries, age groups, and the SES measures used.¹ While some studies found that children from higher socioeconomic classes were more frequently affected,^{2,3} other studies suggested that family income greater than \$100,000 and higher parental education were associated with less severe AD, and yearly income <\$30,000 and lower parental education with more severe AD.⁴ Children in urban areas appear more frequently affected by AD than those in rural areas, although definitions of “urban” may differ between studies and countries. Regarding race, Black and Hispanic children may be more severely affected, with higher rates of poor disease control.^{4–7} Potential non-biological explanations for these racial differences are poor access to healthcare and resources,⁸ structural racism manifesting as bias in the healthcare setting,^{4,9} and masking of erythema in skin of color, leading to inaccurate assessment of severity by healthcare providers and hence inadequate therapy.^{10,11} Higher frequencies of asthma, another common atopic disease, have been reported in Black children,¹² and have been shown to correlate with exposure to smoking.¹³ Smoking, apart from being a direct disease modifier, can also be considered a SES factor, as it is more common in lower socioeconomic classes. The role of social determinants of health versus biologic factors in these described protective and risk factors of AD is poorly understood, but large-scale studies indicate that genetic ancestry is an insufficient explanation.¹⁴

Our aim was to analyze associations between AD severity and patient-reported outcomes (PRO) with patient location and its corresponding “deprivation”¹⁵ in a well-characterized pediatric AD cohort from a highly diverse US metropolitan area. We hypothesized that spatially derived factors, in particular the material community deprivation index (DI),¹⁵ correlate with AD severity and PRO. As secondary outcomes, the potential influence of additional population characteristics, such as smoke exposure and atopic comorbidities, and access to specialized care on AD severity will be discussed.

2 | MATERIALS AND METHODS

In this cross-sectional, single-center study, children with AD and a parent were enrolled in pediatric dermatology and allergy clinics at the Ann and Robert H. Lurie Children's Hospital of Chicago from February 2018 to April 2019 (baseline visit of the prospective AAD-PEPR cohort study, IRB #2016–201; #2022–5670).¹⁶ The parent and children ≥12 years old signed IRB-approved consents and assents respectively; a REDCap database was used for data collection. Inclusion criteria were presence of AD and age 5–17 years; patients could be in a clinical trial at the time; and they could have other forms of atopy and other clinical conditions (this was recorded). AD severity was self- and physician-assessed using the validated AD scores validated Investigator Global Assessment (vIGA^{17,18}), scoring atopic

dermatitis (SCORAD), eczema area and severity index (EASI), affected body surface area (BSA), and patient oriented eczema measure (POEM). Children ≥8 years old and caregivers of all children (proxy response) independently completed validated PRO questionnaires (PRO Measurement Instrumentation System/PROMIS, www.healthmeasures.net). Collected SES information included geographic location, health insurance type, family income, parent education level, and household size. To further explore population characteristics, information on atopic comorbidities, smoke exposure, duration of breastfeeding, and pets was retrieved. Patient home addresses were matched with census tract IDs and DI was calculated using variables of the 2018 American Community Survey. The DI is scored as 0–1, with higher scores indicating greater deprivation.¹⁵ Analyses were performed in GraphPad Prism version 9.5.0 for MacOS (GraphPad Software, San Diego, California USA, www.graphpad.com) and R¹⁹ using descriptive statistics, ANOVA, *t*-tests, Mann-Whitney *U* tests, Spearman correlations for continuous variables, and Point-Biserial Correlation for continuous and categorical variables. Odds ratios were computed through multiple logistic regression. Significance level was set at 0.05. QGIS was used to map geocoded data (www.qgis.org).

3 | RESULTS

Among 216 children (126 female) with a median age of 10.5 years (range 5–17), 31% self-classified as White and 24.5% were Hispanic (Table 1). Approximately equal numbers of children had private versus Medicaid health insurance; Medicaid insurance was more common in Black or mixed race versus Asian or White children (Table 1; $p < .0035$). The majority had moderate or severe AD at time of enrollment based on vIGA (Table 2, Figure 2), which correlated significantly with all other AD scores (Figure S1). There were no significant differences in AD severity across racial and ethnic groups (Figure 1A; Table 2). Disease onset was reported within the first year of life for 56.5%. Enrollment took place at their first visit at the Children's clinic for 56 patients (25.9%) and at a follow-up visit for 129 (59.7%; no information for $n = 31$). Among children with low, medium, and high DI according to interquartile ranges (low ≤ 0.22 , 24.1%; medium 0.23–0.47, 50.5%; high ≥ 0.48 , 25.5%), patients with low and medium DI were included more often during their first visit (30.4% and 55.5% first visit vs. 21.7% and 47.3% follow-up for those with information available). Patients with the highest DI were more often enrolled at a follow-up visit (14.3% vs. 31.0%).

The median DI was 0.32 (range 0.03–0.72). The distribution of patient homes on the map highlights areas of lower socioeconomic status (SES)/higher DI and higher SES/lower DI (Figure 2). There were moderate correlations of DI with insurance type ($\rho = 0.43$) and family income ($\rho = -0.42$) and weak correlations with ethnicity ($\rho = -0.37$), parental education ($\rho = 0.27$), and race ($\rho = 0.16$) (Figure 1B,F). However, DI was not correlated with any AD severity score (as an example, see SCORAD in Figure 1E). DI correlated, but weakly to very weakly, with PROMIS scores for psychological stress ($\rho = 0.20$), sleep disturbance ($\rho = 0.16$), sleep-related impairment ($\rho = 0.24$), depression

TABLE 1 Demographic characteristics of the 216 patients in the cohort.

Variable	Total (n [%])	Asian (n [%])	Black (n [%])	Other/Mixed (n [%])	White (n [%])	Hispanic (n [%])
n (% of total)	216	42 (19.4)	53 (24.5)	54 (25.0)	67 (31.0)	55 (25.5)
Age/years						
5–7 years	57 (26.1)	10 (23.8)	18 (34.0)	10 (18.5)	19 (28.4)	10 (18.2)
8–11 years	73 (33.5)	14 (33.3)	16 (30.2)	24 (44.4)	19 (28.4)	21 (38.2)
12–17 years	86 (39.4)	18 (42.9)	19 (35.8)	20 (37.0)	29 (43.3)	24 (43.6)
Sex						
Male	90 (41.7)	19 (45.2)	35 (66.0)	24 (44.4)	29 (43.3)	23 (41.8)
Female	126 (58.3)	23 (54.8)	18 (34.0)	30 (55.6)	38 (56.7)	32 (58.2)
Disease onset (years, mean ± SD)	2.5 ± 3.2	1.7 ± 2.5	2.4 ± 3.1	2.7 ± 3.4	3.0 ± 3.6	3.0 ± 3.3
Household income						
<\$10,000	9 (4.2)	0 (0.0)	6 (11.3)	1 (1.9)	2 (3.0)	1 (1.8)
\$10,000 to < \$25,000	19 (8.8)	4 (9.5)	8 (15.1)	2 (3.7)	5 (7.5)	6 (10.9)
\$25,000 to <\$50,000	36 (16.7)	2 (4.8)	11 (20.8)	18 (33.3)	5 (7.5)	17 (30.9)
\$50,000 to <\$100,000	72 (33.3)	18 (42.9)	21 (39.6)	18 (33.3)	15 (22.4)	20 (36.4)
>\$100,000	52 (24.1)	10 (23.8)	4 (7.5)	9 (16.7)	29 (43.3)	6 (10.9)
No response	28 (13.0)	8 (19.0)	3 (5.7)	6 (11.1)	11 (16.4)	5 (9.1)
Parental education						
Some high school	15 (7.1)	1 (2.4)	5 (9.8)	5 (9.3)	4 (6.3)	6 (10.9)
High school	29 (13.8)	1 (2.4)	9 (17.6)	8 (14.8)	11 (17.2)	10 (18.2)
Some college	32 (15.2)	2 (4.9)	11 (21.6)	10 (18.5)	9 (14.1)	14 (25.5)
College	80 (38.1)	20 (48.8)	18 (35.3)	23 (42.6)	19 (29.7)	20 (36.4)
Graduate school	54 (25.7)	17 (41.5)	8 (15.7)	8 (14.8)	21 (32.8)	5 (9.1)
Residential setting						
Urban	111 (51.4)	16 (38.1)	36 (67.9)	34 (63.0)	25 (37.3)	38 (69.1)
Suburban	104 (48.1)	26 (61.9)	17 (32.1)	20 (37.0)	41 (61.2)	16 (29.1)
Rural	1 (0.5)	0 (0.0)	0 (0.0)	0 (0.0)	1 (1.5)	1 (1.8)
Asthma	81 (37.5)	12 (28.6)	25 (47.2)	22 (40.7)	22 (32.8)	21 (38.2)
One or more atopic comorbidity	156 (72.2)	36 (85.7)	42 (79.2)	33 (61.1)	45 (67.2)	35 (63.6)
Exposure to smoke	17 (8.0)	2 (4.8)	11 (20.8)	0 (0.0)	4 (6.0)	3 (5.5)
Pet with fur or feathers at home	66 (30.6)	12 (28.6)	14 (26.4)	16 (29.6)	24 (35.8)	19 (34.5)
Insurance type						
Private	97 (44.9)	22 (52.4)	18 (34.0)	15 (27.8)	42 (62.7)	12 (21.8)
Medicaid	96 (44.4)	15 (35.7)	32 (60.4)	33 (61.1)	16 (23.9)	36 (65.5)
Combined	3 (1.4)	0 (0.0)	1 (1.9)	0 (0.0)	2 (3.0)	1 (1.8)
No insurance	4 (1.9)	1 (2.4)	1 (2.4)	1 (1.9)	1 (1.5)	1 (1.8)
NA	16 (7.4)	4 (9.5)	1 (1.9)	5 (9.3)	6 (9.0)	5 (9.1)

($p = 0.18$), and peer relationships ($p = -0.19$; all $p < .05$; Figure S1). These scores also correlated significantly with SCORAD. Race, ethnicity, income, and parental education did not significantly influence the child's odds of having moderate-to-severe versus mild AD (Figure 3).

Black children experienced significantly more exposure to smoking (20.8%) than White (6.0%), Asian (4.8%), or mixed-race (0.0%) children ($p < .001$). The median DI was higher for those with smoking exposure (0.42, range 0.15–0.72) than without (0.31, range 0.03–0.69) ($p = 0.12$), but smoking did not correlate with IGA or SCORAD

within our cohort. Black children also more frequently had asthma (47.2%) than children of other races ($p = .22$, Table 1), but the median DI of Black children with and without asthma did not differ ($p = .88$). Having at least one atopic comorbidity, experienced by 76.9% of the patients, varied significantly by racial group (Table 1, $p < .05$) and correlated very weakly with SCORAD ($p = 0.15$, $p < .05$), but not with DI ($p = -0.03$). Neither exposure to pets at home (30.5% of subjects) nor having been breastfed (70.8%; average duration 0.6 years) correlated with AD severity or DI.

TABLE 2 Deprivation index, atopic dermatitis severity measures, and patient-reported outcome measures of the cohort separated for races.

Variable	Total	Asian	Black	Other/mixed	White	Hispanic
<i>n</i> (% of total)	216	42 (19.4)	53 (24.5)	54 (25.0)	67 (31.0)	55 (25.5)
Deprivation index	Mean \pm SD (range)	0.34 \pm 0.16 (0.03–0.72)	0.27 \pm 0.12 (0.09–0.56)	0.41 \pm 0.14 (0.20–0.72)	0.25 \pm 0.13 (0.03–0.64)	0.44 \pm 0.13 (0.17–0.65)
<i>Atopic dermatitis severity measures</i>						
vIGA <i>n</i> (%)	6 (2.8)	1 (2.4)	3 (5.7)	1 (1.9)	1 (1.5)	1 (1.8)
1—Almost clear						
2—Mild	42 (19.4)	5 (11.9)	8 (15.1)	13 (24.1)	16 (23.9)	14 (25.5)
3—Moderate	124 (57.4)	26 (61.9)	29 (54.7)	28 (51.9)	41 (61.2)	32 (58.2)
4—Severe	44 (20.4)	10 (23.8)	13 (24.5)	12 (22.2)	9 (13.4)	8 (14.5)
SCORAD	Mean \pm SD (range)	50.5 \pm 16.2 (15.1–86.6)	52.1 \pm 14.4 (15.0–77.5)	48.1 \pm 14.3 (12.1–90.5)	46.9 \pm 14.3 (9.2–78.8)	47.0 \pm 18.4 (12.1–90.5)
EASI	Mean \pm SD (range)	18.9 \pm 12.7 (0.8–66.0)	16.7 \pm 10.5 (1.4–43.0)	17.3 \pm 15.7 (0.2–60.0)	13.6 \pm 8.1 (0.7–43.2)	15.7 \pm 15.0 (0.2–60.0)
% BSA	Mean \pm SD (range)	30.3 \pm 19.9 (3.5–90.5)	26.9 \pm 14.6 (2.5–59.7)	26.8 \pm 23.4 (0.5–82.5)	22.7 \pm 13.9 (0.5–76.5)	24.4 \pm 22.3 (0.5–90.5)
<i>Patient-reported outcome measures (only proxy responses were used for statistics)</i>						
Itch NRS	Mean \pm SD (range)	5.6 \pm 2.6 (0–10)	6.2 \pm 2.6 (0–10)	5.8 \pm 2.6 (0–10)	5.5 \pm 2.5 (0–10)	6.0 \pm 2.4 (1–10)
POEM	Mean \pm SD (range)	15.7 \pm 7.0 (0–28)	14.6 \pm 6.7 (0–27)	15.5 \pm 7.4 (2–28)	14.6 \pm 7.2 (1–27)	14.4 \pm 7.9 (2–27)
CDLQI	Mean \pm SD (range)	9.7 \pm 6.8 (0–30)	10.2 \pm 6.6 (0–25)	10.0 \pm 7.0 (0–30)	10.5 \pm 7.5 (0–28)	10.1 \pm 7.1 (0–28)
PROMIS itch	T-Score \pm SD (range)	44.0 \pm 9.6 (22.6–65.2)	44.8 \pm 8.7 (22.6–62.2)	41.9 \pm 10.4 (22.6–65.2)	42.8 \pm 8.9 (25.5–62.0)	44.4 \pm 10.1 (33.6–65.2)
PROMIS stigma	T-Score \pm SD (range)	42.6 \pm 9.3 (26.3–70.3)	42.9 \pm 9.4 (26.3–64.5)	43.6 \pm 10.0 (26.3–70.3)	41.4 \pm 9.4 (26.3–66.7)	43.1 \pm 9.1 (26.3–70.3)
PROMIS Psych. Stress	T-Score \pm SD (range)	53.1 \pm 9.5 (39.6–77.0)	52.4 \pm 9.8 (39.6–71.9)	53.3 \pm 9.1 (39.6–69.6)	72.3 \pm 10.0 (39.6–72.3)	52.5 \pm 9.4 (39.6–69.6)
PROMIS sleep disturbance	T-Score \pm SD (range)	61.3 \pm 10.2 (38.7–85.8)	61.7 \pm 9.3 (44.0–79.1)	61.0 \pm 11.1 (38.7–83.8)	60.5 \pm 9.1 (44.0–83.8)	60.9 \pm 11.3 (38.7–83.8)
PROMIS sleep-related impairment	T-Score \pm SD (range)	55.0 \pm 11.2 (37.9–78.4)	56.0 \pm 11.5 (37.9–78.4)	54.5 \pm 11.1 (37.9–75.5)	52.8 \pm 11.67 (37.9–77.1)	54.0 \pm 10.9 (37.9–74.4)
PROMIS global health	T-Score \pm SD (range)	45.3 \pm 10.1 (24.1–66.1)	45.0 \pm 10.2 (26.8–66.1)	43.8 \pm 9.5 (26.1–66.1)	47.1 \pm 10.3 (24.1–66.1)	43.9 \pm 9.6 (26.1–64.0)
<i>PROMIS parent proxy-25 profile v2.0</i>						
Anxiety	T-Score \pm SD (range)	47.9 \pm 9.9 (36.3–83.7)	45.8 \pm 8.6 (36.3–63.0)	46.8 \pm 10.3 (36.3–83.7)	48.7 \pm 8.3 (36.3–68.8)	49.1 \pm 10.3 (36.3–83.7)
Depressive symptoms	T-Score \pm SD (range)	46.8 \pm 8.6 (37.2–76.8)	45.5 \pm 7.5 (37.2–61.1)	46.6 \pm 9.1 (37.2–76.8)	47.7 \pm 8.8 (37.2–65.3)	47.3 \pm 8.5 (37.2–65.3)
Fatigue	T-Score \pm SD (range)	47.7 \pm 9.7 (37.0–80.6)	47.7 \pm 9.5 (37.0–80.6)	48.2 \pm 9.8 (37.0–70.9)	46.6 \pm 9.7 (37.0–66.3)	48.4 \pm 10.0 (37.0–72.1)
Mobility	T-Score \pm SD (range)	50.8 \pm 6.7 (19.6–55.9)	50.6 \pm 6.3 (37.9–55.9)	51.7 \pm 6.7 (35.9–55.9)	48.6 \pm 7.5 (19.6–55.9) ^a	49.1 \pm 7.6 (19.6–55.9) ^a
Pain interference	T-Score \pm SD (range)	52.1 \pm 8.8 (39.2–74.7)	52.9 \pm 8.8 (39.2–74.7)	51.8 \pm 8.9 (39.2–74.7)	51.0 \pm 8.3 (39.2–67.6)	51.5 \pm 9.1 (39.2–70.4)
Peer relationships	T-Score \pm SD (range)	49.1 \pm 9.5 (19.1–60.8)	50.4 \pm 7.9 (33.7–60.8)	47.5 \pm 9.3 (25.9–60.8)	51.1 \pm 9.3 (33.5–60.8)	47.9 \pm 10.0 (19.1–60.8)

Note: Versions of PROMIS tools used (if not stated above): PROMIS Itch—PIQ-C-Proxy; PROMIS Stigma—PROMIS Stigma Scale for Skin Diseases—Proxy; PROMIS Psychological Stress Experiences—PROMIS SF v1.0—Parent Proxy Psych Stress Experience 4a; PROMIS Sleep Disturbance—PROMIS SF v1.0 Sleep Disturbance 8b Parent Proxy Reported; PROMIS Sleep-related Impairment—PROMIS SF v1.0 Sleep-Related Impairment 8a Parent Proxy Reported; PROMIS Global Health—PROMIS Parent Proxy Scale v1.0—Global Health 7.

Abbreviations: BSA, body surface area; CDLQI, children's dermatology life quality index; EASI, eczema area and severity index; vIGA, validated Investigator Global Assessment; POEM, patient oriented eczema measure; SCORAD, scoring atopic dermatitis.

^aSignificant difference between other/mixed and White, and between Hispanic and non-Hispanic.

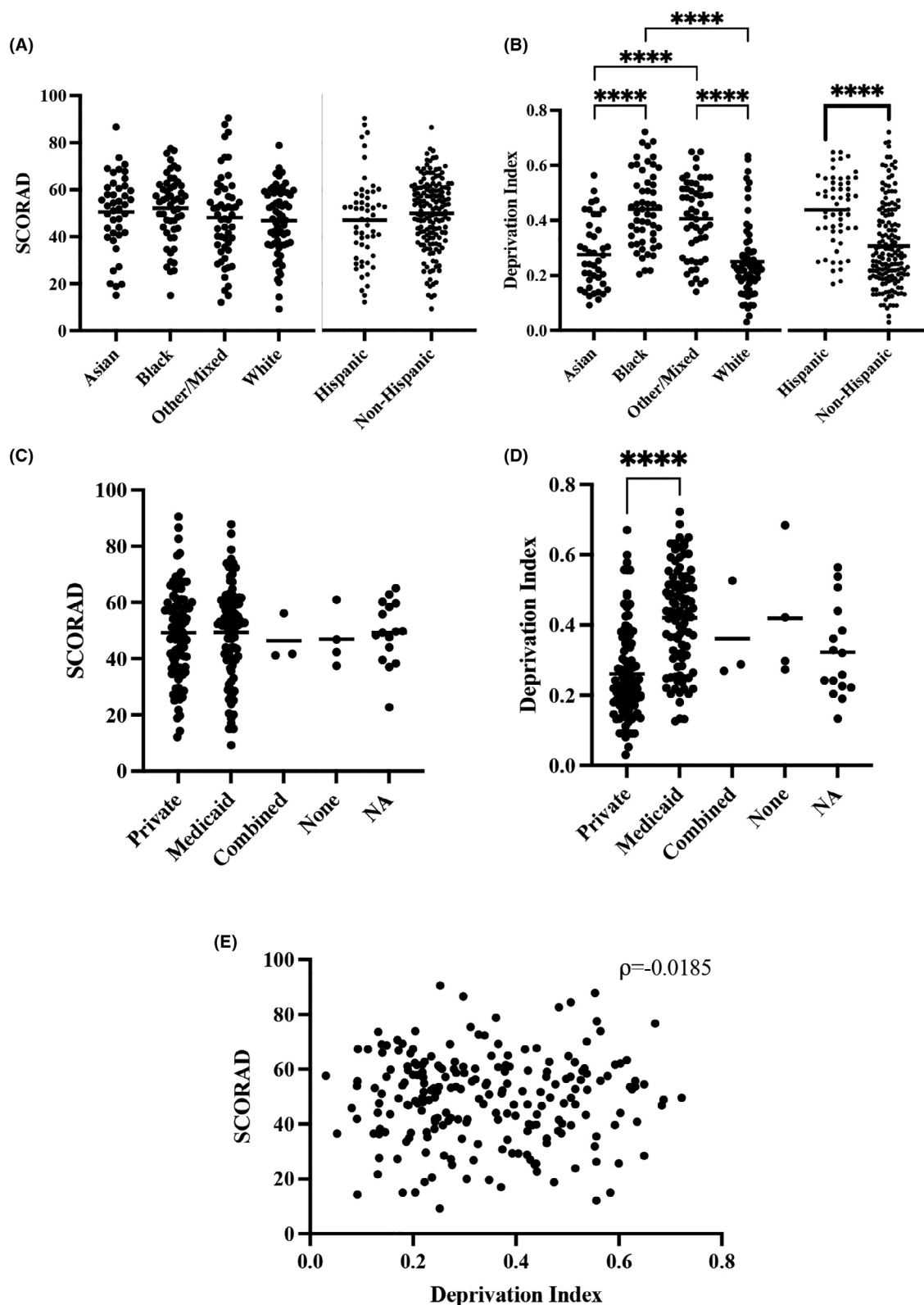


FIGURE 1 Race and ethnicity did not influence atopic dermatitis (AD) severity, as measured by SCORAD (A), but significantly impacted deprivation index (DI) (B). Health insurance type did not influence AD severity (C), but Medicaid-enrolled patients had significantly higher DI than those in private insurance (D). SCORAD was not correlated with DI (E). **** $p \leq .0001$.

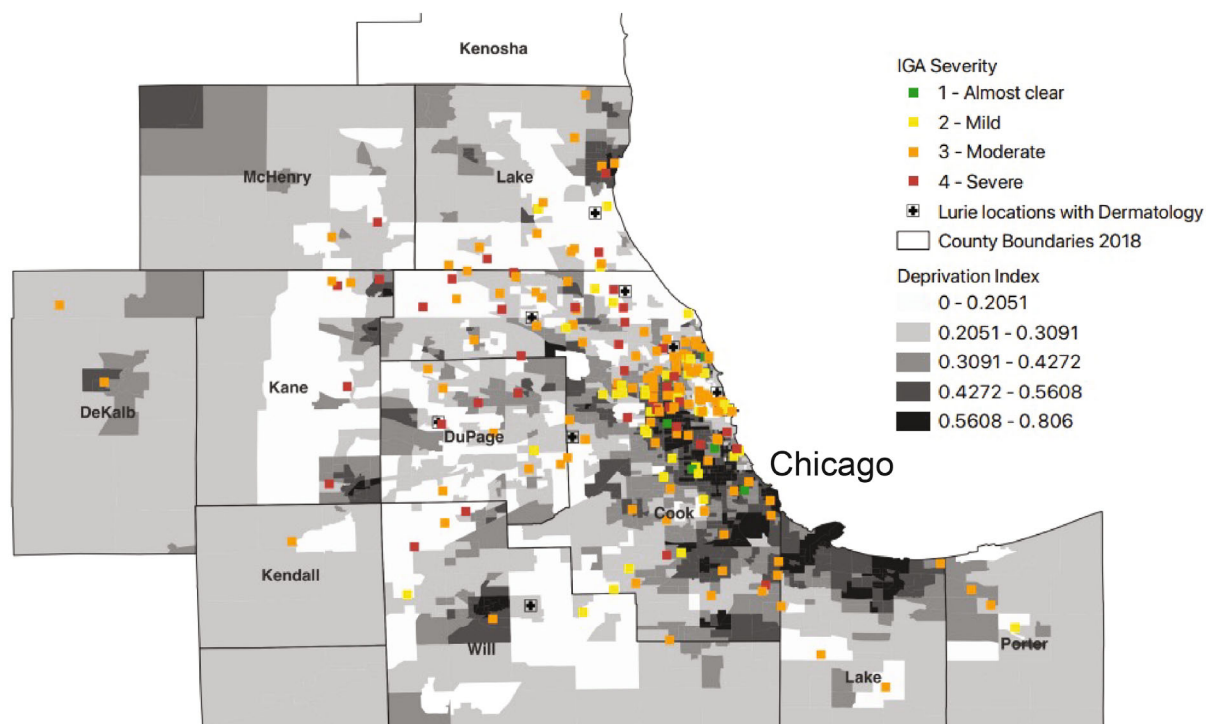


FIGURE 2 Map of the deprivation index (DI) for Chicago and its surrounding counties; Patients are represented according to their disease severity per validated Investigator Global Assessment (vIGA) at their home addresses. DI is depicted in gray shades, and darker gray means more deprived. There is no relationship between DI and vIGA.

4 | DISCUSSION

Established in 2015, the DI is a SES measure derived from data publicly available for every US region. It is a useful tool, as it considers the median household income, educational attainment, proportions of households with income below the poverty level, without health insurance, receiving public assistance, and percentage of vacant houses in an area, and combines those factors into one composite score.¹⁵

In our cohort, DI correlated best with insurance type, family income, and ethnicity, highlighting that it captures SES well. We hypothesized that DI would influence the severity of AD, but were unable to show this relationship. The lack of correlation between DI and AD severity, including patient-reported severity, contradicts previous findings of SES associations in pediatric and adult AD cohorts in other geographical regions, although these studies used other SES measures. In a cross-sectional study of 201 children from North Carolina, Black and Hispanic children had higher odds of having severe AD.⁴ These findings were considered SES-related, specifically attributed to structural racism as measured by a residential segregation (Black/White) index per county, derived from ACS data. We did not use this index, as 96.8% of our study population lived within one county. As a limitation, the North Carolina study used a convenience sample which overrepresented the Black and Hispanic children compared to the local population, which may have biased the results.⁴ While in our cohort, Black and Hispanic children had the highest DI, they did not have higher AD scores than children of other races/

ethnicities. A prospective longitudinal study of 1437 children in Massachusetts found that Black race and non-Hispanic ethnicity were associated with higher AD incidence and persistence, with increased odds ratios after adjusting for maternal education and neighborhood income.⁷ Another birth cohort study from Michigan found an association of Black race and AD and type I-sensitizations after adjusting for maternal educational status and household income.²⁰ Both of these studies found race and ethnicity to be the main factor predisposing for AD, rather than SES, while a broader-scale national study identified older age, lower household income, and fair or poor maternal health as strongly associated with moderate-to-severe AD, in addition to ethnicity and race in multivariate logistic regression models.⁵ Interestingly, while we also saw higher frequencies of asthma in Black children as shown in other studies,¹² we did not find SES, measured by DI, to make a difference in asthma. A possible explanation for the differences between the cohorts, including ours, is that the impact of SES may vary by region, as does AD prevalence overall,²¹ highlighting the importance of studying further populations in different settings. Of note, our cohort was located in a geographically relatively confined metropolitan area with a broad mix of races, ethnicities, and SES, as indicated by DI. Still, there is a possibility that despite this variability in SES, not all patients with high DI/low SES requiring care actually reached the participating site.

In the United States, private and public health insurance (Medicaid) are further indicators of SES, and insurance type may limit access to care, especially to specialty care.²² In Chicago, 59% of children have private insurance, 37.9% public insurance, and 3.0% are

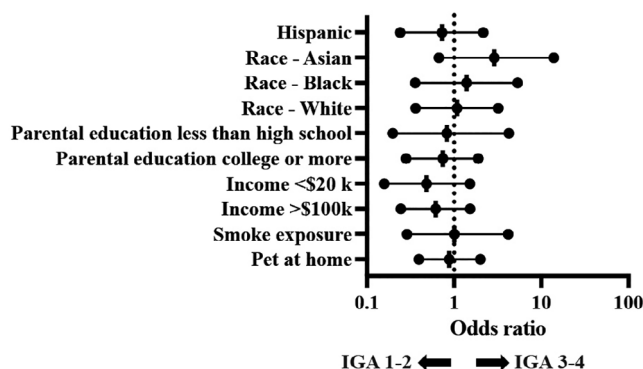


FIGURE 3 Odds ratio (OR) plot for impact of demographic and home factors on patients' odds of having moderate-to-severe (vIGA 3-4) compared with mild atopic dermatitis (vIGA 1-2). Plotted are ORs with upper and lower 95%-confidence intervals. There were no significant associations at the 95% confidence level for either a positive or negative relationship with the depicted factors.

uninsured.^{23,24} Although US children with AD who have Medicaid insurance are known to be less likely to see a dermatologist,²² the children in our cohort who had Medicaid (44.4%) made up a higher proportion than to be expected from mere demographics.^{23,24} This suggests that access to our Chicago-based institution's Pediatric Dermatology clinics is equivalent or better for children with public insurance than with private insurance, as often occurs for academic programs at hospitals dedicated to care of children. This approach, in the long run, is likely to improve AD severity on follow-up visits. In fact, children with higher DI were more proportionally more often recruited at a follow-up visit compared to low DI, so our results may have been biased by already initiated treatments prior to inclusion.

Another limitation of this study was its performance at a single site with experts in AD management, which could have led to preselection bias; we cannot know whether children with higher DI presented to other specialized providers and were thus not captured in this study. However, our cohort had a wide range of DI with large socioeconomic variation, mixture of racial groups, and balance of urban versus suburban residences. Indeed, the cohort mirrored the racial structure of Chicago (45.3% White, 29.2% Black, 7.9% Other/Mixed, 6.8% Asian, and 28.7% Hispanic²⁵), although with overrepresentation of Asians and those of mixed race. Other potential limitations were predominance of subjects with moderate-to-severe AD and the lack of a comparator of children with AD seen regionally at a center without AD expertise, for example, a free community clinic or emergency department.

In conclusion, our data do not support our hypothesis that spatially-derived factors correlate with AD severity and PROs. Nevertheless, it should be recognized that the children who were included had sufficient access to specialists in management of pediatric AD, and that this access may potentially mitigate the impact of SES. Multi-center studies that include various healthcare settings are needed to further test our hypothesis that healthcare access is an important factor, along with socioeconomic factors affecting both severity and outcome of pediatric AD.

AUTHOR CONTRIBUTIONS

Antonia Reimer-Taschenbrecker, Moriel Daniel, and Stephanie M. Rangel had full access to all of the data in the study and take responsibility for the integrity of the data and the accuracy of the data analysis. Concept and design: Antonia Reimer-Taschenbrecker and Moriel Daniel. Acquisition, analysis, or interpretation of data: Antonia Reimer-Taschenbrecker, Moriel Daniel, Stephanie M. Rangel, and Amy S. Paller. Drafting of the manuscript: Antonia Reimer-Taschenbrecker and Moriel Daniel. Critical revision of the manuscript for important intellectual content: Antonia Reimer-Taschenbrecker, Moriel Daniel, Stephanie M. Rangel, and Amy S. Paller. Statistical analysis: Antonia Reimer-Taschenbrecker, and Moriel Daniel. Supervision: Amy S. Paller.

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CONFLICT OF INTEREST STATEMENT

Amy S. Paller: Investigator: AbbVie, Applied Pharma Research, Dermavant, Eli Lilly, Incyte, Janssen, Krystal, Regeneron, UCB. Consultant: Aegerion Pharma, Azitra, BioCryst, Boehringer-Ingelheim, Bristol Myers Squibb, Castle Creek, Eli Lilly, Janssen, Krystal, LEO Pharma, Novartis, Regeneron, Sanofi/Genzyme, Seanergy, TWI Biotechnology, UCB. Data Safety Monitoring Board: AbbVie, Abeona, Catawba, Galderma, InMed. Antonia Reimer-Taschenbrecker, Moriel Daniel, and Stephanie M. Rangel have no conflicts of interest to declare.

DATA AVAILABILITY STATEMENT

The data that support the findings of this study are available from the corresponding author upon reasonable request.

PATIENT CONSENT STATEMENT

All parents and children ≥ 12 years old signed IRB-approved consents and assents respectively before study inclusion.

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SUPPORTING INFORMATION

Additional supporting information can be found online in the Supporting Information section at the end of this article.

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