

Socioeconomic status associates with worse asthma morbidity among Black and Latinx adults



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Background: Asthma disproportionately affects African American/Black (AA/B) and Hispanic/Latinx (H/L) patients and individuals with low socioeconomic status (SES), but the relationship between SES and asthma morbidity within these racial/ethnic groups is inadequately understood.

Objective: To determine the relationship between SES and asthma morbidity among AA/B and H/L adults with moderate to severe asthma using multidomain SES frameworks and mediation analyses.

Methods: We analyzed enrollment data from the PeRson EmPowered Asthma RELief randomized trial, evaluating inhaled corticosteroid supplementation to rescue therapy. We tested for direct and indirect relationships between SES and asthma morbidity using structural equation models. For SES, we used a latent variable defined by poverty, education, and unemployment. For asthma morbidity, we used self-reported asthma exacerbations in the year before enrollment

(corticosteroid bursts, emergency room/urgent care visits, or hospitalizations), and Asthma Control Test scores. We tested for mediation via health literacy, perceived stress, and self-reported discrimination. All models adjusted for age, sex, body mass index, ethnicity, and comorbidities.

Results: Among 990 AA/B and H/L adults, low SES (latent variable) was directly associated with hospitalizations ($\beta = 0.24$) and worse Asthma Control Test scores ($\beta = 0.20$). Stress partially mediated the relationship between SES and increased emergency room/urgent care visits and worse asthma control ($\beta = 0.03$ and $= 0.05$, respectively). Individual SES domains were directly associated with asthma morbidity. Stress mediated indirect associations between low educational attainment and unemployment with worse asthma control ($\beta = 0.05$ and $= 0.06$, respectively).

Conclusions: Lower SES is directly, and indirectly through stress, associated with asthma morbidity among AA/B and H/L

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adults. Identification of stressors and relevant management strategies may lessen asthma-related morbidity among these populations. (J Allergy Clin Immunol 2022;150:841-9.)

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Asthma affects more than 25 million individuals in the United States¹ and is associated with substantial morbidity¹ and health care costs.² African American/Black (AA/B) and Hispanic/Latinx (H/L) patients disproportionately bear this burden, and experience higher rates of asthma morbidity compared with Whites.³ These higher asthma morbidity rates among AA/B and H/L are likely due to multifactorial causes related to inequitable distribution of social determinants of health.⁴⁻¹⁴ For example, a recent pediatric study found that associations between hospital readmissions for asthma exacerbations and racial genetic ancestry are mostly mediated by socioeconomic status (SES).¹⁵ SES is a construct that is variably defined by multiple indicators, including income, education, and employment, among others.^{16,17} Several of these SES-related indicators are associated with worse asthma outcomes,¹⁸⁻²¹ including low income,^{22,23} unemployment,²⁴ and low educational attainment.²⁵ Relationships between SES and asthma outcomes may be mediated by other factors in the causal pathway.²⁶ Perceived stress, inadequate health literacy, and perceived discrimination are associated with both low SES²⁷⁻²⁹ and worse asthma outcomes.³⁰⁻³³ However, empirical data about potential mediators of the association between low SES and asthma morbidity are lacking, particularly among AA/B and H/L adults. SES is correlated with race and ethnicity in the United States,³⁴ where AA/B and H/L individuals tend to have lower incomes and attain lower levels of education.³⁵ Thus, most research on the role of SES in asthma morbidity has been unable to disentangle the independent contributions from SES and race/ethnicity in asthma morbidity.³⁶ Moreover, the studies that have accounted for both race/ethnicity and SES have included few AA/B and H/L participants, have been conducted in children, have recruited participants from a single center, have assessed a single SES domain (eg, only assessed income), or have evaluated a limited number of asthma morbidity measures.³⁷⁻⁴⁰ Therefore, an in-depth analysis of SES among AA/B and H/L adults with asthma is warranted. For that reason, we analyzed poverty, unemployment, and education; and the mediatory effects of health literacy, perceived stress, and perceived discrimination on asthma morbidity measures among AA/B and H/L adults with moderate to severe persistent asthma.

METHODS

We used enrollment data from the Patient-Centered Outcomes Research Institute–funded PeRson EmPowered Asthma RELief (PREPARE) trial; a description of the study population, trial aims and methods, and data collected at enrollment has been published.⁴¹ One thousand two hundred one participants were recruited from November 2017 through March 2020 from 19 study sites located across the United States, including Puerto Rico, encompassing a broad range of practice sizes, health care systems, and specialties (eg, allergy/immunology, pulmonology, and primary care clinics), with most recruited from primary care. Recruitment methods varied by site, but participants were commonly directly enrolled from affiliated clinicians' practices. Participants had a physician's diagnosis of asthma for 1 or more years before

Abbreviations used

AA/B:	African American/Black
APGAR:	Activities, Persistent triGgers, Asthma medications and Response to therapy
ASUI:	Asthma Symptom Utility Index
ER:	Emergency room
H/L:	Hispanic/Latinx
PREPARE:	PeRson EmPowered Asthma RELief
SEM:	Structural equation model
SES:	Socioeconomic status

enrollment, and either uncontrolled disease (Asthma Control Test [ACT] score ≤ 19) or 1 or more asthma exacerbations requiring systemic corticosteroids in the year before enrollment. To summarize, PREPARE is a multi-institutional, pragmatic, open-label, randomized trial investigating whether a "Patient-Activated Reliever-Triggered Inhaled CorticoSteroid" (PARTICS) intervention can reduce asthma exacerbations relative to usual care among AA/B and H/L adults with moderate to severe persistent asthma.

For this cross-sectional ancillary study, we used a multidomain SES framework that included a latent variable with 3 measures: poverty, unemployment, and low educational attainment. Poverty status was calculated using income and household size compared against the federal poverty income guidelines.⁴² Poverty guidelines are established for the 48 continental states and typically extended to US territories such as Puerto Rico.^{43,44} Participant incomes were captured in \$10,000/y increments, starting at "less than \$10,000/y" through "more than \$75,000/y." Educational attainment was divided into 3 levels: no high school diploma, high school diploma, or greater than a high school diploma. Employment status was classified as either employed or unemployed.

We tested for effect mediation by perceived stress, health literacy, and perceived discrimination on the effect of SES on asthma morbidity. Self-perceived stress was measured using the 4-item short form of the Perceived Stress Scale⁴⁵; a participant was classified as having high perceived stress if his or her score was in the cohort's top quintile. Health literacy adequacy was determined using the 3-question version of the Brief Health Literacy Scale.⁴⁶ Self-perceived discrimination was measured using the Everyday Discrimination Scale⁴⁷; a participant was classified as having high perceived discrimination if his or her score was in the cohort's top quintile.

We measured our study outcome, asthma morbidity, in several ways. First, we used measures of self-reported asthma exacerbations (outpatient corticosteroid bursts for asthma, emergency room [ER]/urgent care [UC] visits and hospitalizations) in the year before enrollment. We defined steroid bursts and ER/UC visits for asthma as ordinal variables (0, 1, 2, ≥ 3), whereas asthma hospitalizations were dichotomized as 0 versus 1 or more. Second, we assessed scores from 3 participant-completed surveys: the ACT,⁴⁸ a validated 5-item questionnaire that assesses level of asthma control; the Asthma Symptom Utility Index (ASUI),⁴⁹ a validated 10-item questionnaire that assesses preference-based quality of life; and the asthma Activities, Persistent triGgers, Asthma medications and Response to therapy (APGAR),⁵⁰ a validated 5-domain questionnaire that assesses asthma control and is designed for use in primary care.

Statistical analysis

Demographic and clinical characteristics were categorized by poverty status and presented using descriptive statistics, with chi-square tests for categorical variables, and student *t* tests for continuous variables, where appropriate. We fitted structural equation models (SEMs) to determine the relationship of SES (as a 3-domain latent variable, and individually with each measure) and mediators with asthma morbidity measures. The SES latent variable was estimated by analyzing the variance and covariance of its 3 domains—poverty, unemployment, and low educational attainment. We used mediation analyses to assess for indirect (ie, effect of the SES latent variable

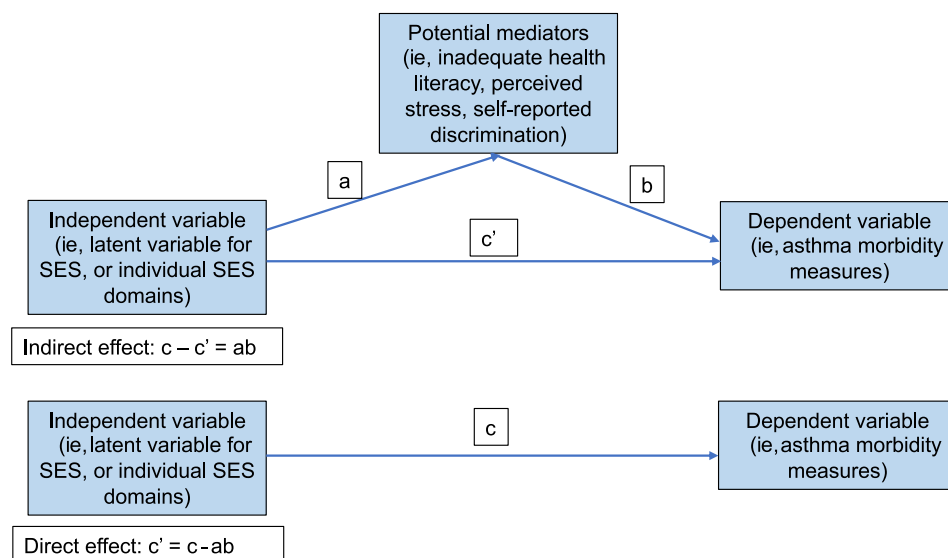


FIG 1. Conceptual diagram of the SEM depicting direct and indirect associations between independent (SES latent variable or its individual domains) and dependent variables (asthma morbidity measures) and potential mediators (inadequate health literacy, perceived stress, and self-reported discrimination). The variable “C” refers to the total effect of the independent variable on the dependent variable, including direct and indirect effects.

on the asthma morbidity outcome via perceived stress, health literacy, or perceived discrimination) and direct pathways (ie, the effect of the SES latent variable directly on the asthma morbidity outcome). Unmeasured mediators may be reflected in the error terms for pathway effect estimates but are otherwise unaccounted for in these models. The strength of association of a pathway was determined using model-based beta coefficients. Beta coefficients for direct pathway estimates equal the SD change in asthma morbidity outcome for 1 SD change in the SES latent variable (or individual SES domain variables) score. Beta coefficients for indirect pathway estimates equal the beta for the pathway between the SES latent variable to the mediator multiplied by the beta for the pathway from the mediator to the asthma morbidity outcome (Fig 1). The contribution of indirect effects through mediation was determined from the product of path coefficients and SE estimates using the delta method proposed by Sobel.⁵¹ Model adequacy was tested through chi-square and adjusted goodness-of-fit measures.⁵² ACT score, ASUI, and APGAR were analyzed as continuous outcomes. Steroid bursts, ER/UC visits, and hospitalizations were grouped according to the categories described earlier and analyzed as continuous, ordinal variables within their respective models. All models were adjusted for age, sex, H/L ethnicity, body mass index, and medical comorbidity counts (heart disease, cancer [excluding nonmelanoma skin], stroke, diabetes, chronic kidney disease, chronic obstructive pulmonary disease, HIV/AIDS, depression, and/or sleep disorders). Additional models were adjusted by smoking status, smoke exposure, and depression (assessed by the Patient Health Questionnaire-2 depression screen).⁵³ To account for federal poverty guidelines being established for the 48 continental states⁴² even if typically extended to Puerto Rico,^{43,44} we conducted sensitivity analyses excluding Puerto Rican residents ($n = 101$).

RESULTS

Data on SES domains, mediators, and asthma morbidity measures were available for 990 PREPARE trial participants; 211 participants preferred not to disclose their income and were thus excluded from this analysis. Table I presents the demographic and clinical characteristics of study participants included in the current analysis, categorized by poverty status (ie, 1 of the 3 SES latent variable domains). Most (83.9%) participants were

female, obese (69.6%), and had 1 or more medical comorbidity (71.0%). Most participants also lived in poverty ($N = 517$ [52.2%]), were unemployed (58.0%), had adequate health literacy (83.8%), experienced 1 or more asthma exacerbation(s) and 1 asthma-related ER visit(s) in the previous year (60.2%), and had uncontrolled asthma (ie, ACT score <20 ; 86.2%). Those living in poverty, relative to those who were not, were more likely to speak Spanish (26.9% vs 14.0%; $P < .001$), be unemployed (73.7% vs 40.8%; $P < .001$) and not a high school graduate (22.2% vs 5.3%; $P < .001$), and to have multiple medical comorbidities (ie, 2 or more: 54.7% vs 42.1%; $P < .001$), inadequate health literacy (23.8% vs 7.8%; $P < .001$), to be depressed (32.7% vs 19.0%; $P < .001$), and to have high self-perceived stress levels (36.9% vs 23.7%; $P < .001$). In addition, those living in poverty, relative to those who were not, were more likely to have been hospitalized for asthma (23.6% vs 14.0%; $P < .001$) and to have worse asthma control (mean ACT score, 14.0 vs 15.3; $P < .001$) but similar rates of ER/UC visits (39.3% vs 35.7% with ≥ 2 ER/UC visits in the year before enrollment; $P = .405$) and corticosteroid bursts for asthma (36.6% vs 37.0% with 2+ corticosteroid bursts for asthma in the year before enrollment; $P = .669$).

In multivariable analyses, the SEM showed that low SES (as a 3-measure latent variable) was directly associated with a lower ACT score ($\beta = 0.20$; 95% CI, 0.07-0.33). Specifically, a 1 SD change in the SES latent variable was associated with a 0.20 SD change in ACT scores. An indirect relationship between low SES and lower ACT scores mediated by stress was also observed ($\beta = 0.05$; 95% CI, 0.026-0.07) (Fig 2), after adjustment for other covariates. The contributions of direct and indirect effects of SES on ACT scores were 71%, and 29%, respectively. Similar direct and indirect associations between low SES and worse asthma control and preference-based quality of life were identified using the asthma APGAR scores and ASUI, respectively (see Figs E1 and E2 in this article's Online Repository at www.jacionline.org).

TABLE I. Demographic and clinical characteristics of PREPARE trial participants, categorized by poverty guideline thresholds

Characteristic	Overall cohort (N = 990)	Poverty*		P value
		No (n = 473)	Yes (n = 517)	
Demographic				
Age (y)	49 ± 13	49 ± 13	48 ± 13	.284
Sex: female	831 (83.9)	393 (83.1)	438 (84.7)	.4846
Race and ethnicity				
H/L	486 (49.1)	217 (45.9)	269 (52.0)	.053
AA/B	504 (50.9)	256 (54.1)	248 (48.0)	
Preferred language				
English	785 (79.3)	407 (86.0)	378 (73.1)	<.001
Spanish	205 (20.7)	66(14.0)	139 (26.9)	
Clinical features				
BMI				
Overweight (25-29.9)	207 (20.9)	105 (22.2)	102 (19.7)	.457
Obese (≥30)	689 (69.6)	323 (68.3)	366 (70.8)	
Medical comorbidities				
0	276 (27.9)	154 (32.6)	122 (23.6)	<.001
1	222 (22.4)	120 (25.4)	102 (19.7)	
2+	482 (48.6)	199 (42.1)	283 (54.7)	
Smoking status				
Current smoker	75 (7.6)	30 (6.3)	45 (8.7)	.079
Former smoker	239 (24.1)	104 (22.0)	135 (26.1)	
Nonsmoker	676 (68.3)	339 (71.7)	337 (65.2)	
Living in a smoking environment	216 (21.8)	91 (19.2)	125 (24.2)	.06
PHQ-2†	259 (26.2)	90 (19.0)	169 (32.7)	<.001
SES domains				
Educational attainment				
High school or more	850 (85.9)	448 (94.7)	402 (77.8)	<.001
Less than high school	140 (14.1)	25 (5.3)	115 (22.2)	
Employment				
Employed	416 (42.0)	280 (59.2)	136 (26.3)	<.001
Not employed	574 (58.0)	293 (40.8)	381 (73.7)	
SES mediators				
Inadequate health literacy‡	160 (16.2)	37 (7.8)	123 (23.8)	<.001
High stress§	303 (30.6)	112 (23.7)	191 (36.9)	<.001
High discrimination	297 (30.0)	141 (29.8)	156 (30.2)	.901

Significant values are bolded. Continuous variables are presented as means ± SD; categorical variables are presented as counts (proportions).

BMI, Body mass index.

*Participants whose household size-adjusted income is below the federal poverty threshold²⁸ were considered to be living in poverty.

†Patient Health Questionnaire-2 (PHQ-2) screens for depression with a 2-wk recall. Total score ranges from 0 to 6, and scores of ≥3 suggest depression.⁵³

‡Inadequate health literacy is based on participant responses to the Brief Health Literacy Scale.³²

§High stress was defined as having a score in the cohort's top quintile of the 4-item Perceived Stress Scale.³¹

||High discrimination was defined as having a score in the cohort's top quintile in the Everyday Discrimination Scale.³³

We did not find evidence for mediation of the effect of SES through health literacy or self-perceived discrimination on asthma control. Low SES was directly associated with asthma hospitalizations ($\beta = 0.24$; 95% CI, 0.11-0.38; Fig 3). A 1 SD change in the low SES latent variable was directly associated with 0.09 additional asthma hospitalizations. No direct association with asthma ER/UC visits was observed, however. No indirect association with asthma hospitalizations was observed through any mediator, yet low SES was indirectly associated with asthma ER/UC visits through an effect mediated by stress ($\beta = 0.026$; 95% CI, 0.003-0.048) (Fig 4). We did not detect an association between low SES and corticosteroid bursts for asthma exacerbations. All of the above models that evaluated the SES latent variable on multiple asthma morbidity measures were adequately fit, with adjusted goodness of fit more than 90% (χ^2 for the models $P < .001$). Finally, all of these results were robust to additional adjustment by smoking status, smoking environment, and depression.

We found that individual SES measures (ie, including only poverty, low educational attainment, or unemployment) had similar adjusted goodness of fit (>90%) and chi-square tests (P values < .001) for all asthma morbidity measures that were similar to those of the models with the SES latent variable. A 1 SD change in poverty, however, was directly associated with 0.19 additional asthma hospitalizations ($\beta = 0.48$; 95% CI, 0.15-0.80) and 1.28-point lower asthma control scores (ACT score $\beta = -0.29$; 95% CI, -0.004 to -0.59) but did not show any stress-mediated indirect associations with asthma morbidity outcomes (unadjusted associations between poverty and asthma morbidity measures appear in Table E1 in this article's Online Repository at www.jacionline.org). Both unemployment and education showed stress-mediated indirect associations with asthma control scores ($\beta = 0.06$, 95% CI, 0.03-0.09; $\beta = 0.05$; 95% CI, 0.02-0.07, respectively), but only unemployment maintained a stress-mediated indirect association with ER/UC visits ($\beta = 0.03$; 95% CI, 0.01-0.06). Analyses excluding Puerto Rican residents ($n = 101$) yielded results

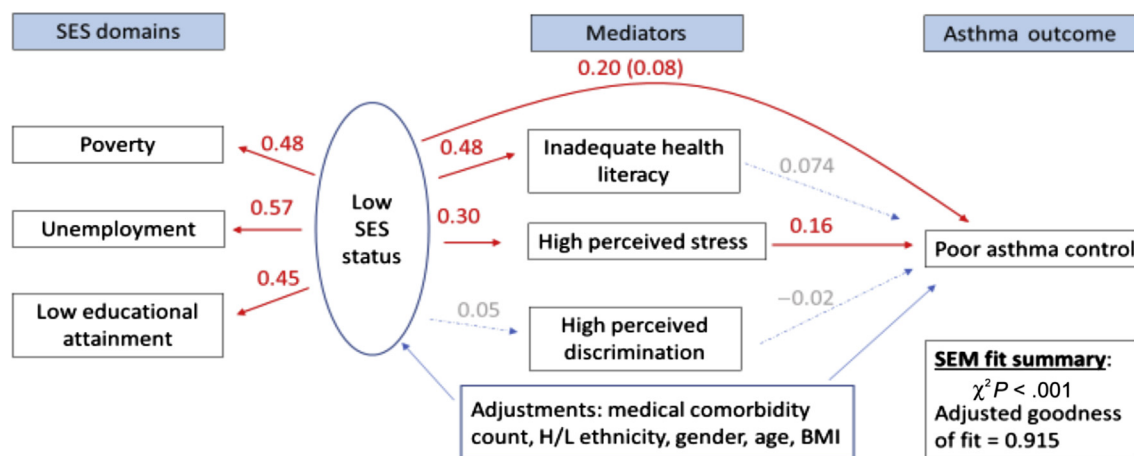


FIG 2. SEM depicting direct and indirect associations between low SES and poor asthma control mediated by high self-perceived stress. *BMI*, Body mass index. Numbers shown indicate pathway beta coefficients within the SEM. Those in red denote significant associations, whereas those in gray denote insignificant ones. The latent variable labeled “Low SES status” is composed of 3 SES domains: income- and household size–determined poverty status,^{42,43} unemployment, and low educational level attainment. Poor asthma control is indicated by lower ACT scores.⁴⁸

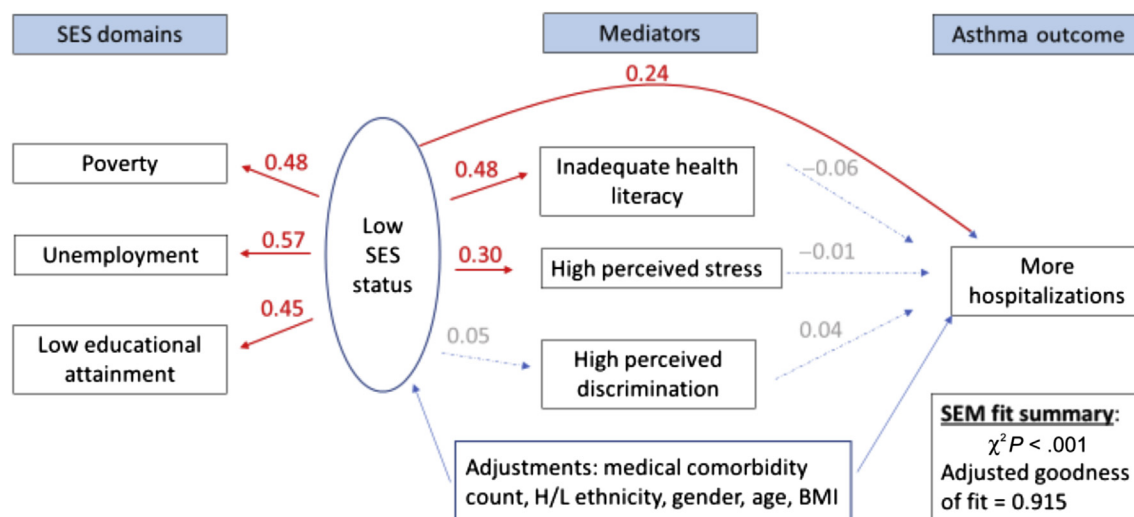


FIG 3. SEM depicting a direct association between low SES and asthma-related hospitalizations. *BMI*, Body mass index. Numbers shown indicate pathway beta coefficients within the SEM. Those in red denote significant associations, whereas those in gray denote insignificant ones. The latent variable labeled “Low SES status” is composed of 3 SES domains: income- and household size–determined poverty status,^{42,43} unemployment, and low educational level attainment. Hospitalizations are asthma related.

consistent with those that analyzed the complete cohort. All of these results were robust to additional adjustment by smoking status, smoking environment, and depression.

DISCUSSION

We identified associations between a multidomain model of SES that included poverty, education, and unemployment and several important asthma morbidity outcomes in a cohort of AA/B and H/L adults with moderate to severe asthma. To our knowledge, this is the first study to identify a relationship between SES and asthma morbidity outcomes using a multidomain latent variable in adults, and to identify perceived stress as an important mediator of the association between low SES and ER visits for asthma

exacerbations and several measures of worse asthma control in adults. Our findings are also novel in that they are based on data from 2 highly impacted racial/ethnic groups, thus implicating low SES as a risk factor for worse asthma morbidity outcomes independently of race/ethnicity and highlighting the need for policies that address these socioeconomic issues.

Using an SEM modeling approach, we uncovered direct and indirect pathways connecting SES with asthma morbidity outcomes. Both pathways affected asthma control as determined by ACT and asthma APGAR scores, as well as preference-based quality of life (ASUI). We found that most of the effect of SES on asthma control was due to direct effects, whereas high stress mediated 17% of the effect of SES on asthma control. This finding suggests that there are other exposures associated with low SES

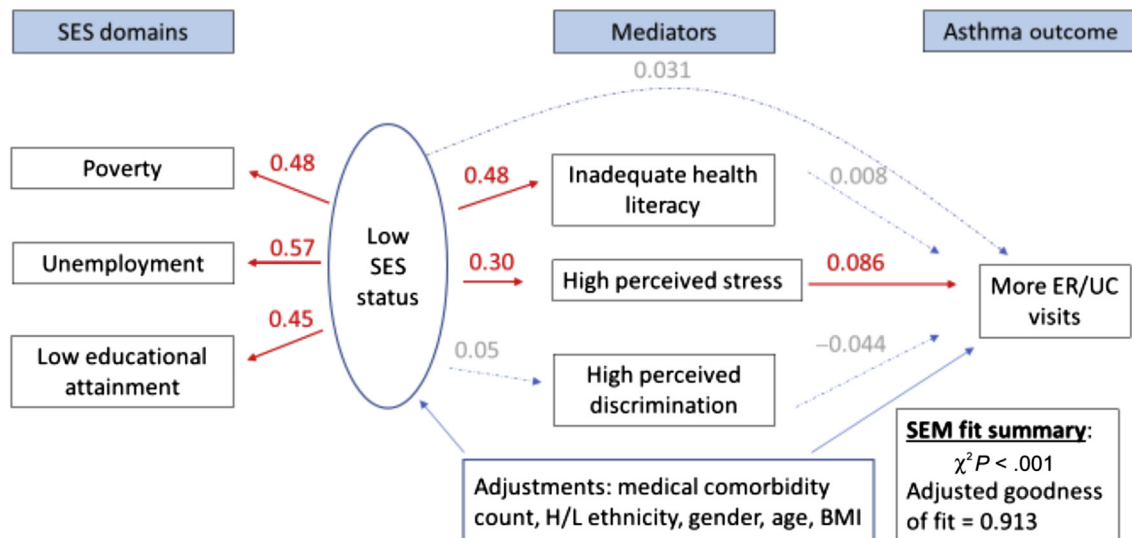


FIG 4. SEM depicting an indirect association between low SES and asthma-related ER/UC visits mediated by high self-perceived stress. *BMI*, Body mass index. Numbers shown indicate pathway beta coefficients within the SEM. Those in red denote significant associations, whereas those in gray denote insignificant ones. The latent variable labeled “Low SES status” is composed of 3 SES domains: income- and household size–determined poverty status,^{42,43} unemployment, and low educational level attainment. ER/UC visits are asthma related.

beyond stress, health literacy, and discrimination—for example, access to medical care, access to nutritious foods, pollution, and housing and built environment, among others—that could explain its association with asthma outcomes. The mediator effect of stress was particularly strong, contributing to roughly half of the overall effect of SES on asthma-related emergency department visits within the constraints of our model. Stress may result from psychological, behavioral (eg, unhealthy diet and poor sleep),⁵⁴ or environmental (eg, pollution and unsafe neighborhoods)^{30,55} factors, social isolation,⁵⁶ or systemic racism, all of which could exert an influence on asthma.^{19,57} We captured perceived stress using the 4-item version of the Perceived Stress Scale, which does not delve into specific stressors. However, single SES component analyses indicated that only unemployment had stress as a mediator on ER visits for asthma, suggesting that unemployment may be an important stressor. We cannot rule out the possibility that employed individuals may be reluctant to leave work for medical care and self-medicate asthma deteriorations at home, resulting in fewer ER/UC visits. Because of our cross-sectional study design, we cannot also assess the temporality between stress and unemployment, and thus cannot exclude the possibility that stress leads to unemployment instead of, or in addition to, unemployment leading to stress as a mediator on ER visits for asthma.

The magnitude of our results should be interpreted from a population-level and not a patient-level perspective. For example, we found that a 1-SD decrease in the low SES latent variable was associated with a 0.88-point decrease in the ACT score, which is below the minimal clinically important difference of 3 points.⁴⁸ However, value thresholds that are clinically meaningful at the patient level may be much larger than value thresholds at the population level. For example, decreases in salt consumption on average decrease systolic blood pressure by 4 mm Hg,⁵⁸ which is a clinically small change in systolic blood pressure at the individual patient level. But public health strategies to reduce salt consumption decrease all-cause mortality at the population level.⁵⁹ That being

stated, our results also show that individuals with the lowest SES latent variable score (10% of our cohort) on average had 2.7 lower ACT score points relative to those with the highest SES latent variable score (28% of our cohort), which is in the range of the individual-level minimal clinically important difference. Results in other asthma morbidity measures also further substantiate our findings connecting lower SES with worse asthma control.

The connection between low SES and worse asthma morbidity outcomes has long been established. For example, Schyllert et al³⁸ confirmed the association between individual SES components (ie, education and unemployment) and asthma morbidity. Yet, like most of the literature, this study used linear analytical models between single SES components and asthma outcomes and did not incorporate mediation. In contrast, latent variables are useful in approximating the complex nature of multidimensional concepts such as SES, whereas mediation analysis helps with understanding mechanism of action especially of abstract concepts such as SES. Although many studies on SES and asthma do not include AA/B and H/L participants, the 2019 study by Seibert et al³⁹ analyzed prospective data on 342 adults with asthma from Chicago where 85% of participants were AA/B or H/L, and looked at income, health literacy, and asthma outcomes using SEM and mediation. The authors found that AA/B and H/L patients, relative to Whites, had poorer asthma quality of life and higher rates of ER visits for asthma, and that these results were indirectly mediated by low health literacy and low income. Our findings complement these results showing that income, analyzed as poverty status, is directly associated with worse asthma morbidity, and expand upon them with our larger sample size representative of several regions of the United States and Puerto Rico. Seibert et al sought to understand SES as the mediator of racial/ethnic disparities, whereas ours sought to identify mediators of a multidomain SES variable on asthma outcomes within AA/B and H/L populations. Their study identified health literacy as a significant mediator between race/ethnicity and worse asthma

outcomes, whereas health literacy was not significant in our model. This discrepancy may relate to the different surveys used to capture health literacy (Rapid Estimate of Adult Literacy in Medicine⁶⁰ vs Brief Health Literacy Scale),⁴⁶ and single-city versus multicity cohort recruitment. We caution against concluding that inadequate health literacy is not associated with worse asthma outcomes in general, and may reflect our cohort and ascertainment methods, because most participants (84%) in the current study had adequate health literacy. Consistent with our results, Yakubovich et al⁴⁰ showed that South African children living in poverty had greater asthma prevalence and asthma “attack” rates and that this effect was mediated by greater psychological stress. Our results expand on theirs with our analysis of an adult American cohort, and validation through multiple asthma morbidity outcomes. A prominent role for stress as a mediator of the association between low SES and worse asthma outcomes in adults had not been shown.

Our study has some limitations. First, our results may not be generalizable to children. However, the literature has better addressed the dynamics of SES and asthma outcomes in children compared with adults. Second, the PREPARE trial only enrolled AA/B and H/L participants, and therefore our results may not be generalizable to other racial or ethnic groups. The lack of a White comparison group is a drawback that prevented comparing SES and asthma morbidity between racial/ethnic populations differentially affected by systemic racism, and should be addressed in future studies. However, even without a White comparator group, our study design allowed us to disentangle the effects of SES from those of race/ethnicity on asthma morbidity outcomes among a cohort of AA/B and H/L patients—a difficulty that had thus far limited much of the existing literature. By investigating a large cohort of AA/B and H/L individuals, we were able to determine that low SES, regardless of race/ethnicity, is an important driver of worse asthma morbidity outcomes. Third, our cohort was disproportionately burdened by asthma severity and comorbidities, partly the result of a selection bias from the PREPARE trial eligibility criteria. Our results on the relationship between SES and asthma morbidity may not be generalizable to other racial/ethnic adult minority populations. Fourth, we used a definition of poverty that was stipulated for the 48 continental states even if typically extended to US territories such as Puerto Rico. However, sensitivity analysis excluding Puerto Rican residents recapitulated findings from the larger cohort. Fifth, we had missing income data from 18% ($n = 211$) of our participants, as is common in many studies.⁶¹ These participants were significantly different from those with available income data in terms of demographic and clinical characteristics and asthma morbidity outcomes, and therefore multiple imputation analysis was not appropriate. Sixth, our findings are limited to the variables entered into the SEM. Although our study is the first to use a multidomain SES latent variable in adults with asthma, there are many other components to SES aside from poverty, education, and unemployment, and there are other mediators aside from stress, discrimination, and health literacy that could be important drivers of asthma morbidity. Indeed, the large direct contributions of SES to asthma morbidity may be indicative of unmeasured mediation pathways, which could potentially be due to other social or political determinants of health, such as structural racism, or unmeasured individual determinants, such as patient mental health (in domains not captured by the Patient Health Questionnaire-2 depression screen), access to primary care, or material hardship. Our findings, however, were robust to

adjustment by important covariates including age, sex, ethnicity, body mass index, medical comorbidities, smoking status, smoking environment, and depression, all of which have an impact on asthma morbidity. Seventh, this is a cross-sectional study and therefore cannot assess temporality or causality. Eighth, our surveys have not been validated in Spanish except for the ACT. Efforts should be dedicated to validating our other surveys in Spanish. Lastly, much of our data are self-reported, which may be subject to recall and other biases, though this concern is partly mitigated by our reliance on validated surveys, in addition to the multiple, complementary ways we measured asthma morbidity outcomes.

This study identified that a multidomain measure of SES associates with worse asthma morbidity among AA/B and H/L adults, and that this effect is partly mediated by stress. These findings support the notion that race/ethnicity alone does not impart worse asthma morbidity risk, and that differences in SES might account for a substantial portion of asthma morbidity in these highly impacted patient populations. Clinicians should be aware of the greater risk of asthma morbidity imparted by poverty, low educational attainment, unemployment, and stress and should seek to address these factors when and if possible. For example, clinicians should consider, where appropriate, counseling patients on stress reduction strategies and optimizing support and resilience as part of a comprehensive person-centered approach to asthma management. Investigators should identify stressors experienced by AA/B and H/L patients within these low SES groups and test stress management interventions optimized for these populations and inform population-level policy changes that will improve employment and education rates and minimize stressors.

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Key messages

- Low SES is directly and indirectly associated with worse asthma morbidity measures among Black and Latinx adults with moderate to severe persistent asthma.
- Stress is an important mediator of the association between low SES and asthma morbidity measures in these racial/ethnic groups.

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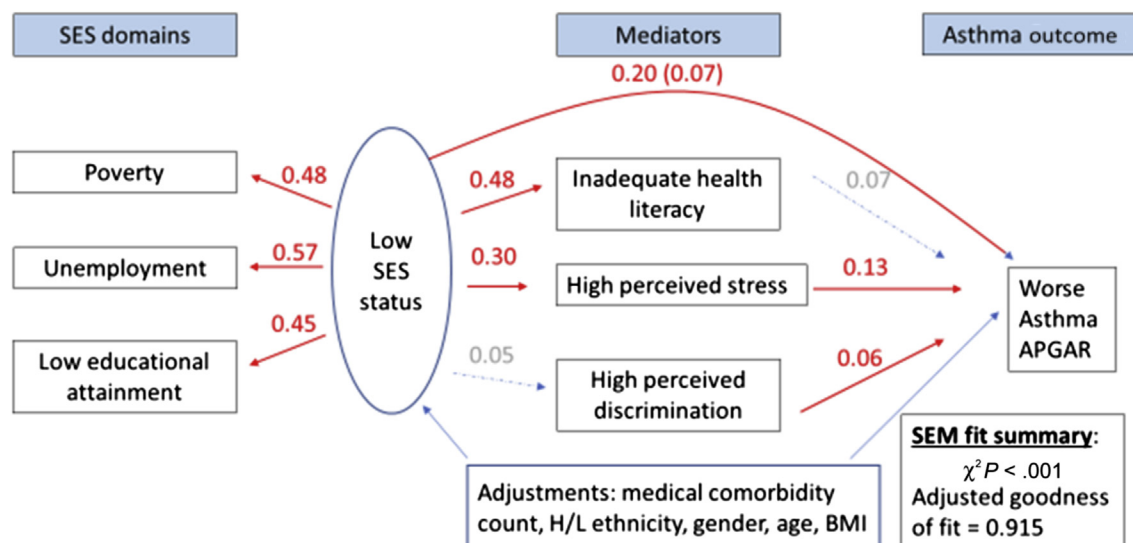


FIG E1. SEM depicting direct and indirect associations between low SES and poor asthma control (determined by the asthma APGAR scores),^{E1} mediated by high self-perceived stress. *BMI*, Body mass index. Numbers shown indicate pathway beta coefficients within the SEM. Those in red denote significant associations, whereas those in gray denote insignificant ones. The latent variable labeled “Low SES status” is composed of SES domains: income- and household size–determined poverty status,^{E2,E3} unemployment, and low educational level attainment.

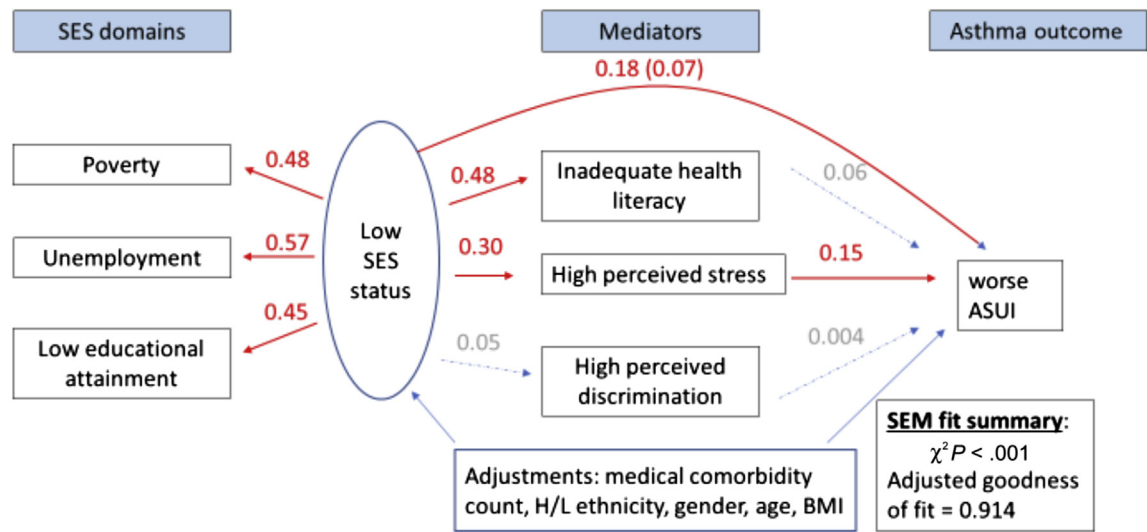


FIG E2. SEM depicting direct and indirect associations between low SES and poor asthma control (determined by the ASUI),^{E4} mediated by high self-perceived stress. *BMI*, Body mass index. Numbers shown indicate pathway beta coefficients within the SEM. Those in red denote significant associations, whereas those in gray denote insignificant ones. The latent variable labeled “Low SES status” is composed of 3 SES domains: income- and household size–determined poverty status,^{E2,E3} unemployment, and low educational level attainment.

TABLE E1. Asthma morbidity measures by poverty status among PREPARE trial participants

Asthma morbidity measures*	Overall cohort (N = 990)	Poverty†		P value
		No (n = 473)	Yes (n = 517)	
Steroid bursts				
0	354 (35.8)	174 (36.8)	180 (34.8)	.669
1	272 (27.5)	124 (26.2)	148 (28.6)	
2+	364 (36.8)	175 (19.7)	189 (36.6)	
ER/UC visits				
0	394 (39.8)	198 (41.9)	196 (37.9)	.405
1	224 (22.6)	106 (22.4)	118 (22.8)	
2+	372 (37.6)	169 (35.7)	203 (39.3)	
Hospitalizations				
0	802 (81.0)	407 (86.0)	395 (76.4)	<.001
1+	188 (19.0)	66 (14.0)	122 (23.6)	
ACT ^{E5} score	14.6 ± 4.4	15.3 ± 4.5	14.0 ± 4.3	<.001
ASUI ^{E6}	0.7 ± 0.2	0.7 ± 0.2	0.6 ± 0.2	<.001
APGAR ^{E7}	3.5 ± 1.8	3.2 ± 1.8	3.7 ± 1.8	<.001

Continuous variables are presented as means ± SD; categorical variables are presented as counts (proportions). Significant values are bolded.

*Asthma exacerbations in the 12 mo before enrollment were self-reported and categorized as outpatient corticosteroid bursts, ER visits, or hospitalizations.

†Participants whose household size-adjusted income is below the federal poverty threshold^{E8} were considered to be living in poverty.