International prevalence rates of asthma and allergy are associated with income inequality

To the Editor:

The global burden of asthma and allergic diseases is rising, and decades of research on causal pathways has had limited impact on the development of effective prevention strategies.[1] It remains unclear what causes variations in prevalence rates between countries, and evidence on the role of poverty is mixed. Data from the World Health Survey show a U-shaped pattern, with the highest prevalence rates of asthma reported in low-income and high-income countries.[2] The International Study for Asthma and Allergy in Childhood survey data show some positive associations between per capita gross national product and atopy, depending on the age group, world region, and health outcome studied.[3,4] Although determinants linked to absolute poverty (such as calorific nutrition, lack of exercise, air pollution, poor quality housing, and tobacco smoking) have received attention, it is unclear to what extent causal pathways of asthma and allergies are influenced by the degree of socioeconomic inequality within countries. We use income inequality as a social characteristic that may shed light on poorly understood causal mechanisms of asthma and allergies. Despite income inequality being associated with a range of other diseases in adults and children, and different types of mortality,[5,6] a previous study on health outcomes and income inequality concluded that there was no association between asthma and income inequality in 60 US communities.[7]

We aimed to study the associations between income inequality and childhood asthma and allergies for countries represented in the International Study for Asthma and Allergy in Childhood Phase 3 survey. The survey was conducted from 2001 to 2003 in 55 countries and 104 centers, and provides data on prevalence rates of symptoms of asthma (wheeze in the last 12 months), atopic eczema, and allergic rhinoconjunctivitis for 6- to 7-year-olds and 13- to 14-year-olds.[8] Income inequality within countries was measured by the Gini coefficient, ranging from 0 (complete equality: everyone has the same income) to 100 (complete inequality: one person receives all the income). National estimates of the Gini coefficient closest to 2002/2003 were provided by the World Bank. Country income was measured by gross national income (GNI per capita) of 2003. Countries or territories for which no Gini coefficient or GNI per capita was provided were excluded from analyses (Barbados, Channel Islands, Isle of Man, Malta, Taiwan, Oman, and Kuwait).

Associations between income inequality or national income and symptoms of asthma and allergies were assessed with correlation tests and linear regression models in Stata 12. Because previous studies on health and income inequality have excluded low-income countries or focused on more affluent countries, we performed a sensitivity analysis with high- and middle-income countries only. According to the World Bank Atlas method, this excludes countries with a GNI below $735 in 2003.[9] As a result, 5 countries were removed from these analyses (India, Nigeria, Ethiopia, Pakistani, and Kenya).

A positive correlation between asthma symptoms and income inequality was statistically significant for 6- to 7-year-olds ($\beta = 0.38$; $r = 0.45$; $P = .009$) (Fig 1) and 13- to 14-year-olds

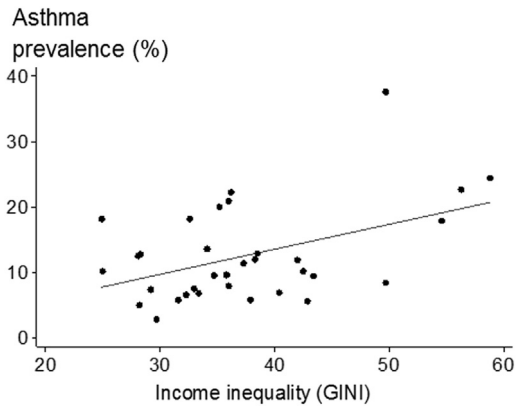

FIG 1. Asthma prevalence rates by income inequality (Gini) for 6- to 7-year-olds.

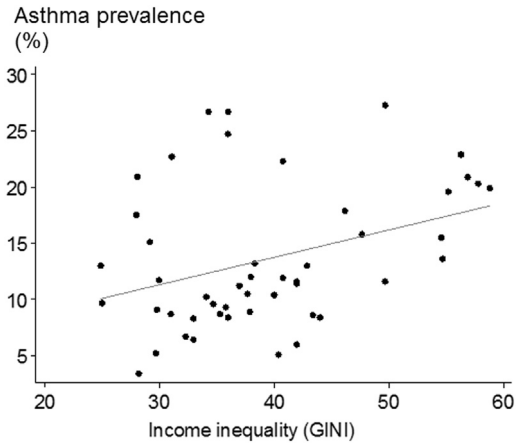

FIG 2. Asthma prevalence rates by income inequality (Gini) for 13- to 14-year-olds.

($\beta = 0.24$; $r = 0.36$; $P = .013$) (Fig 2). In both age groups, including GNI per capita as a covariate strengthened associations between income inequality and asthma (6-7-year-olds: $\beta = 0.49$; $P = .002$; 13-14-year-olds: $\beta = 0.33$; $P = .001$). However, prevalence rates of symptoms of asthma were not associated with GNI per capita for 6- to 7-year-olds ($P = .578$) or 13- to 14-year-olds ($P = .243$) (see Figs E1 and E2 in this article's Online Repository at www.jacionline.org). A sensitivity analysis with middle- and high-income countries only did not alter results.

In the 6- to 7-year-old age group, there was a correlation between rhinoconjunctivitis and higher income inequality ($\beta = 0.17$; $r = 0.39$; $P = .029$), which became stronger if GNI per capita was included in the model ($\beta = 0.26$; $P = .002$). Similar associations were observed in the 13- to 14-year-old age group in the bivariate analysis ($\beta = 0.31$; $r = 0.42$; $P = .003$) and in the model including GNI per capita ($\beta = 0.36$; $P = .001$). We found no evidence for associations between GNI per capita and symptoms of allergic rhinoconjunctivitis, nor did the data reveal associations between GNI per capita and eczema.

In the subsample of middle- and high-income countries, results generally did not differ from those of the complete sample.

However, there was some evidence for associations between eczema and income inequality in the 13- to 14-year-old age group of this subsample, both in the bivariate analysis ($\beta = 0.14$; $r = 0.30$; $P = .056$) and when GNI per capita was included ($\beta = 0.16$; $P = .045$).

In conclusion, income inequality rather than average income per capita is associated with symptoms of asthma and rhinoconjunctivitis. The same conclusion could not be drawn for eczema, which might be due to a difference in etiology, or because income inequality is associated with the prevalence of eczema in higher income but not lower income countries. Given that we found associations with income inequality but not absolute income, psychosocial rather than material factors may explain part of the difference in national prevalence rates of asthma and allergies. Income inequality may affect health through stress arising from status comparisons, unhealthy behaviors triggered by stress, and damaged social relationships.[6] Because the ecological design of this study provides evidence for associations but not causal relationships, potential causal mechanisms should be explored in future studies, taking into account individual- and area-level factors. This may improve our knowledge on the etiology of asthma and allergies.

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Atopic dermatitis disease control and age: A cohort study

To the Editor:

We report the results of an observational cohort study examining atopic dermatitis (AD) disease control by age. AD is the most common chronic inflammatory skin disease and is characterized by an episodic course. Conventionally thought to remit by adolescence, increasing data suggest heterogeneity in disease courses, though detailed prospective studies are lacking.[1]

To examine AD disease control by age, we used longitudinal data from the Pediatric Eczema Elective Registry. It is a cohort study designed to test whether there is an increased risk of malignancy associated with the use of pimecrolimus, and includes a patient-reported measure of disease control every 6 months.[2,3] To enroll, patients must have AD diagnosed by a physician and used 1% topical pimecrolimus cream for at least 42 of the last 180 days.

Our primary outcome, disease control, is a repeating composite variable based on self-reported control (complete, good, limited, or poor) and treatment use (any prescription treatments for AD over the same 6-month period). Those who reported complete control were subcategorized into 2 groups: complete control without treatment (ie, apparent remission) and complete control with treatment.

We examined the data graphically. Then, to account for the longitudinal nature of our data and repeated outcome, we created a separate binary generalized linear latent and mixed model for each level of disease control. This enabled us to calculate the subject-specific odds of better or worse control for each additional year of age while controlling for potential confounders specified *a priori* including age at onset, enrollment age, sex, race, family income, and history of atopic disease at enrollment. The number of subjects with at least 1 follow-up visit by October 2013 determined the sample size. For additional methodological details, see the Methods section in this article's Online Repository at www.jacionline.org. This study was approved by the institutional review board at the University of Pennsylvania.

There were 5,798 participants aged 2 to 26 years who returned 49,840 surveys. A total of 47% were males, 44% were white, the mean age of enrolment was 7.2 years, and the mean duration of follow-up was 4.2 years (for additional cohort characteristics, see Table E1 in this article's Online Repository at www.jacionline.org). Complete control without treatment was reported on 8% of all surveys, whereas good or limited disease control was reported 80% of the time. The largest change with age occurred among the complete control with no treatment group (Fig 1).

With multivariate models, we found an elevated subject-specific odds of complete control for each additional year of age and a reduced subject-specific odds of limited or poor control for each additional year of age (Table I). When we split those who reported complete control into treatment groups, we found that the subject-specific odds of complete control without treatment for each additional year of age compared with good, limited, or poor control were 1.78 (95% CI, 1.72-1.83; see Table I). This outcome was infrequent, however. Only 3967 (8% of all surveys), and 1205 (21% of all patients), *ever* reported a 6-month period of complete control without treatment. Of these patients, 546 (45%) subsequently reported medication use or less than complete control, suggesting that a minority of patients "outgrew" their