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THE IMPORTANCE OF RESEARCH ON THE ASSOCIATION BETWEEN SOCIOECONOMIC CONDITIONS AND ASTHMA

After your excellent issue on asthma (1), you will be aware that the prevalence of asthma has increased in several countries, especially in affluent Western countries and Latin America. In our opinion, studies of the relationship between asthma and socioeconomic differentials provide an opportunity to advance our knowledge of what causes asthma and may even lead to the possibility of preventing it. The speed of this increase suggests an environmental rather than genetic source. It has been hypothesized that this increase was a consequence of better living conditions and hence less exposure to infection, the so-called hygiene hypothesis. This hypothesis is supported by the high rates of asthma in affluent countries and a greater prevalence of atopic diseases in populations with higher socioeconomic status (SES). However, there is increasing evidence of a greater frequency of asthma in populations of lower SES in affluent societies, such as African Americans in the United States (2, 3) and subpopulations in Europe (4, 5), as well as in some Latin American countries where societies are characterized by a marked inequality in wealth, with large pockets of poverty. We believe there is a need for further studies on SES and asthma and that some major methodologic aspects must be addressed to find the right answer; we list them below and propose guidelines for research.

First, we must establish how and why the direction of the association with SES has changed over time, as has been observed in some places. For example, surveys in Sweden showed that the population of lower SES had lower prevalence of asthma in earlier cohorts, but later cohorts had the highest rates, a reversal of the effect with successive generations (6). In a study in Germany, higher prevalence of asthma was found in previous generations with high SES but not in recent ones (7).

Second, we need to assess the effect of SES at the collective as well as at the individual level. SES at the individual level is more closely related to factors such as genetic makeup, family resources, living standards, occupation, education, diet, and indoor environment. At the collective level, it may be related to, for example, community resources and outdoor environmental exposures. There is some evidence that SES at the collective level is associated with asthma morbidity; for instance, a study in the United States showed increased prevalence of asthma in children exposed to community violence (8). This evidence is particularly important in Latin America, where violence has become a major cause of morbidity and mortality (9). In addition, determination at the collective level can be seen as re-

sulting from two opposing forces (10): (1) poor people can benefit from living in richer areas because of better access to collective resources and less exposure to hazards (collective model), and (2) disparities in SES among individuals can lead to social barriers, less social cohesion, and ultimately health disparities (inequality model).

Third, it is important to discriminate atopic from nonatopic diseases and other phenotypes (11) in studying the social determination of asthma. Asthma has been conceived as a syndrome resulting from different biological pathways because of different combinations of causes (12). The most well-described factor is sensitization by allergens, which leads to the common classification of asthma as atopic and nonatopic. Asthma is also defined in terms of transient, persistent, and late-onset cases. Epidemiologic studies do not always present data according to these classifications. This diversity of causes of asthma raises the possibility of distinct etiologies and thus also distinct potential preventive measures.

Fourth is the potential artefact of better ascertainment of severe (untreated) asthma (13). The unexpected (from the hygiene hypothesis perspective) greater prevalence among those with lower SES could be an artefact reflecting differences not in the prevalence of asthma but in the prevalence of severe asthma, with severe disease being more likely to be diagnosed. It is now possible through well-planned asthma management programs and the use of preventive drugs like inhaled steroids to reduce the frequency and severity of asthma attacks. If poverty leads to worse control and consequently more severe asthma, and severe disease is more likely to be identified in surveys, a greater frequency of uncontrolled asthma among the poor (resulting from less access to good management programs) can be misinterpreted as an increase in the prevalence of asthma among the poor. It is important to separate clearly the presence of asthma from the presence of severe asthma.

Fifth, in an attempt to explain the SES differentials of asthma frequency and to develop preventive measures, it is important to develop conceptual models of causation, focusing on identifying potential "changeable causes" (14). Information in the literature suggests that changes in factors such as physical activity, sanitation, and some infections are related to the prevalence of asthma, and these factors are also related to SES. These changes strengthen the idea that SES is not a "cause" by itself, but this association with asthma is mediated by factors that differ according to SES and that have changed over time. For instance, it may not be enough to describe individuals as "poor" and "rich" or as "black" and "white." It is essential to identify the changeable factors that mediate the association between SES and health outcomes. Investigations of the causal pathway of asthma may require methodologic approaches beyond the traditional risk-factor analysis and a higher degree of theorization to test alternative hypotheses. For instance, hierarchical (15) or structural (16) equation modeling has been used to investigate the causality involving genetic and environmental factors as well as to evaluate control intervention.

In our opinion, a better understanding of the different moments when SES could have an impact on the progress from genetic predisposition to occurrence of severe disease (and the causal pathways) is key to formulating future programs for asthma prevention. It will determine whether prevention of asthma is possible and feasible and whether it should be done at the primary, secondary, or tertiary level. Attempts have been made at different levels of prevention (17–21). Primary prevention will probably require very earlier interventions to influence priming of the immune system in earlier life. Secondary intervention may be based on identifying mild (maybe even undiagnosed) cases for intervention to remove factors associated with progression of the disease (possible factors are obesity, diet, indoor smoking, allergen exposure, physical activity, and air pollution). Prevention at the tertiary level will include managing severe asthma.

Latin American societies may be a very useful setting for more in-depth epidemiologic studies on etiological factors and social context (22–24). The prevalence of asthma is high, an "anomaly" in the hygiene hypothesis that predicts lower prevalence in areas with high exposure to infection; the population in Latin America is highly heterogeneous in terms of living standards (where the "Europes" and "least-developed countries" share the same urban space) and lifestyles and environmental exposures are changing fast. If prevalence rates are high, it is reasonable to assume that populations in urban areas of Latin America are already exposed to the environmental changes behind the increase in asthma but without the living standard in the most Western affluent countries. We suggest more studies beyond the traditional "risk factor epidemiology," studies aimed at testing causal frameworks to assess disease burden in specific study sites, investigating the nature and intensity of current changeable exposures and which of these exposures explains the SES disparity. Moreover, studies should be conducted to assess exposures and prevalence between study sites at different stages of social development, which may shed light on the mechanisms behind the changes in prevalence in time and space and across generations.

Acknowledgments. All authors participate in the program Social Changes in Asthma and Allergy in Latin American, Wellcome Trust, Ref 072405/Z/03/Z. The authors have no competing interests.

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