Life-course models of how the social environment affects childhood respiratory risk

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Understanding the social and physical contributors to asthma risk is an important undertaking, and the article by Sternthal et al¹ makes several very significant contributions toward this goal. In this study the authors recruited a cohort of women during pregnancy and followed the children through to age 2 years. Five hundred ten women participated in this study in which they were asked about their childhood socioeconomic status (SES), were administered psychosocial questionnaires about stress, and had dust samples taken to assess allergen exposure, and air pollution estimates were calculated based on residence. In children cord blood was used to assess IgE levels, and maternal reports of wheezing were obtained every 3 months from birth through 2 years. The authors found that low maternal childhood SES was related to childhood wheezing through adult SES, prenatal stress, and prenatal pollution exposure and that low maternal childhood SES was related directly to increased cord blood IgE levels.

One of the most novel aspects of this article is that it raises the notion that the factors that contribute to a child's health might not be limited to experiences during a child's life or even prenatally but that certain social experiences that far predate the child might nonetheless still exert effects on the child himself or herself. That is, the authors demonstrate that the family SES of a mother when she was a child is linked to her child's cord blood IgE levels and likelihood of wheezing. These findings are groundbreaking in raising the possibility that experiences or exposures in one generation (eg, parent) can be transmitted to another generation (offspring).

The findings from this study are consistent with other research that has documented that a parent's childhood SES also predicts the offspring's cardiovascular risk (blood pressure and C-reactive protein level) in a sample of healthy adolescents.² Taken together, these studies argue for the need to broaden our models about how SES across time can affect health outcomes. There are a number of models that have been proposed for how to understand the effects of SES across the life course. These include theories about how low SES has cumulative effects over a lifetime on health (accumulation models); how low SES during certain stages of life, such as early childhood, has particularly potent effects on later

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© 2011 American Academy of Allergy, Asthma & Immunology doi:10.1016/j.jaci.2011.06.020 health (critical period models); and how changes across the lifespan in SES can affect health (social mobility models).^{3,4} Yet all these models presume that it is the SES during a subject's lifetime that will affect his or her health. In contrast, the findings from this study suggest that life-course models will need to be revised to also consider SES from previous generations in understanding the effects on a subject's current health and that it is possible to have intergenerational transmission of the effects of low SES from parents to offspring.

How this intergenerational transmission occurs is an intriguing question that requires future research. One idea is that early childhood environments program physiological systems in the mother for life, which in turn have effects on the developing fetus during pregnancy. For example, early-life experiences can result in epigenetic modifications (stable changes in the activity, rather than sequence, of genes), which have been shown to be heritable and consequently are able to exert long-term biological influences.⁵ Another idea for which the authors found support is that early childhood SES leads to a greater likelihood of low adult SES), which in turn have more proximal effects on child health. More research is needed that delves into this question of how the effects of low SES can be transmitted from one generation to the next.

The article by Sternthal et al¹ is also important for highlighting the social environment's contributions to early childhood wheezing and related phenotypes. The authors assess a comprehensive battery of psychosocial factors potentially contributing to childhood asthma risk, including maternal experiences with interpersonal trauma, exposure to violence, discrimination, financial strain, and other negative life events. There is growing recognition that adverse exposures from the social environment can be detrimental to childhood asthma. This study nicely complements previous research that has identified stressful life events, violence exposure, stress appraisals, and family conflict as some of the key toxic social contributors to asthma.⁶⁻⁹ It will be important in future studies to categorize the full range of social exposures that can affect childhood asthma and to promote widespread acceptance of the message that adverse social exposures can affect childhood asthma in much the same way as physical-environment exposures.

In addition, this study provides a good model for designing comprehensive studies of the contributors to childhood asthma risk. For example, the study by Sternthal et al¹ assessed both social (eg, stress) and physical (eg, allergen exposure) contributors to childhood wheezing and cord blood IgE levels, and future studies should examine not only the simultaneous effects of social and physical exposures but also how they interact to affect asthma, as has been explored in several recent studies.¹⁰⁻¹² In addition, comprehensive studies of asthma risk factors should explore contributors at multiple levels, including neighborhood, family, and

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childhood characteristics, as has been done in this and previous studies,^{13,14} to properly acknowledge how children are embedded within broader environments that play critical roles in shaping how children develop.

Finally, a big strength of this study is the recruitment of a cohort of women during pregnancy to allow for prospective monitoring of children from birth onward. This sample will be a valuable asset for tracking the development of asthma and for assessing the factors predicting asthma onset without having to rely on retrospective reporting once asthma has been diagnosed. Birth cohort studies, such as the ambitious National Children's Study,¹⁵ will be the wave of the future for rigorous research designs to better understand the predictors of childhood respiratory and other diseases.

In sum, this study advances our understanding of the contribution of asthma risk factors across generations, the role that social factors play in childhood asthma, and broader multilevel approaches to understanding respiratory problems. More studies are needed that follow in the footsteps of this one.

REFERENCES

- Sternthal MJ, Coull BA, Chiu YHM, Cohen S, Wright RJ. Associations among maternal childhood socioeconomic status, cord immunoglobulin E, and repeated wheeze in urban children. J Allergy Clin Immunol 2011;128:337-45.
- Schreier HM, Chen E. Socioeconomic status in one's childhood predicts offspring cardiovascular risk. Brain Behav Immun 2010;24:1324-31.
- Hallqvist J, Lynch J, Bartley M, Lang T, Blane D. Can we disentangle life course processes of accumulation, critical period and social mobility? An analysis of disadvantaged socio-economic positions and myocardial infarction in the Stockholm Heart Epidemiology Program. Soc Sci Med 2004;58:1555-62.

- Pollitt RA, Rose KM, Kaufman JS. Evaluating the evidence for models of life course socioeconomic factors and cardiovascular outcomes: a systematic review. BMC Public Health 2005;5:7.
- Jirtle RL, Skinner MK. Environmental epigenomics and disease susceptibility. Nat Rev Genet 2007;8:253-62.
- Wright RJ, Mitchell H, Visness CM, Cohen S, Stout J, Evans R, et al. Community violence and asthma morbidity: the inner-city asthma study. Am J Public Health 2004;94:625-32.
- Sandberg S, Paton JY, Ahola S, McCann DC, McGuinness D, Hillary CR. The role of acute and chronic stress in asthma attacks in children. Lancet 2000; 356:982-7.
- Chen E, Hanson MD, Paterson LQ, Griffin MJ, Walker HA, Miller GE. Socioeconomic status and inflammatory processes in childhood asthma: the role of psychological stress. J Allergy Clin Immunol 2006;117:1014-20.
- Mrazek DA, Klinnert M, Mrazek PJ, Brower A, McCormick D, Rubin B, et al. Prediction of early-onset asthma in genetically at-risk children. Pediatr Pulmonol 1999;27:85-94.
- Shankardass K, McConnell R, Jerrett M, Milam J, Richardson J, Berhane K. Parental stress increases the effect of traffic-related air pollution on childhood asthma incidence. Proc Natl Acad Sci U S A 2009;106:12406-11.
- Clougherty JE, Levy JI, Kubzansky LD, Ryan PB, Suglia SF, Jacobson CM, et al. Synergistic effects of traffic-related air pollution and exposure to violence on urban asthma etiology. Environ Health Perspect 2007;115:1140-6.
- Chen E, Schreier HMC, Strunk RC, Brauer M. Chronic traffic-related air pollution and stress interact to predict biologic and clinical outcomes in asthma. Environ Health Perspect 2008;116:970-5.
- Sternthal MJ, Jun HJ, Earls F, Wright RJ. Community violence and urban childhood asthma: a multilevel analysis. Eur Respir J 2010;36:1400-9.
- Chen E, Chim LS, Strunk RC, Miller GE. The role of the social environment in children and adolescents with asthma. Am J Respir Crit Care Med 2007;176: 644-9.
- Landrigan PJ, Trasande L, Thorpe LE, Gwynn C, Lioy PJ, D'Alton ME, et al. The National Children's Study: a 21-year prospective study of 100,000 American children. Pediatrics 2006;118:2173-86.