Associations among maternal childhood socioeconomic status, cord blood IgE levels, and repeated wheeze in urban children

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Background: Independent of current socioeconomic status (SES), past maternal SES might influence asthma outcomes in children.

Objective: We examined associations among the mother's SES in the first 10 years of her life (maternal childhood SES), increased cord blood IgE levels (upper 20% [1.37 IU/mL]), and repeated wheeze (≥2 episodes by age 2 years) in an urban pregnancy cohort (n = 510).

Methods: Data on sociodemographics, discrimination, financial strain, community violence, interpersonal trauma, and other negative events were obtained prenatally. Prenatal household dust was assayed for cockroach and murine allergens, and traffic-related air pollution was estimated by using spatiotemporal land-use regression. Maternal childhood SES was defined by parental home ownership (birth to 10 years). Maternally reported child wheeze was ascertained at 3-month intervals from birth. Using structural equation models, we examined whether outcomes were dependent on maternal childhood SES directly versus indirect relationships operating through (1) cumulative SES-related adversities, (2) the mother's socioeconomic trajectory (adult SES), and (3) current prenatal environmental exposures.

Results: Mothers were largely Hispanic (60%) or black (28%), 37% had not completed high school, and 56% reported parental home ownership. When associations between low maternal childhood SES and repeated wheeze were examined, there were significant indirect effects operating through adult SES and prenatal cumulative stress ($\beta = 0.28$, $P = .003$) and pollution ($\beta = 0.24$, $P = .004$; P value for total indirect effects ≤ .04 for both pathways). Low maternal childhood SES was directly related to increased cord blood IgE levels ($\beta = 0.21$, $P = .003$). Maternal cumulative adversity (interpersonal trauma) was also associated with increased cord blood IgE levels ($\beta = 0.19$, $P = .01$), although this did not explain maternal childhood SES effects.

Conclusion: Lower maternal childhood SES was associated with increased cord blood IgE levels and repeated wheeze through both direct and indirect effects, providing new insights into the role of social inequalities as determinants of childhood respiratory risk. (J Allergy Clin Immunol 2011;128:337-45.)

Key words: *Childhood socioeconomic status, intergenerational, cord blood IgE, inner-city, childhood wheeze, structural equation models, life course*

Research links childhood factors to adult health, including respiratory disease.[1,2] In the context of women's health, childhood conditions might carry over to the next generation.[3] Although we recognize that childhood asthma has its roots prenatally[4-6] and that maternal socioeconomic status (SES) during pregnancy and the child's first years contribute to observed disparities, effects on the next generation might be influenced by maternal risk factors and health disparities rooted in a mother's own childhood SES. If early childhood experiences related to disadvantage can affect asthma risk in the next generation, then the need for interventions aimed at breaking the cycle of childhood poverty becomes heightened.

Although studies have begun to examine the relationship between maternal SES and correlated exposures assessed before pregnancy on her children's health,[3,7,8] none have examined the influence of maternal childhood SES on childhood wheeze and related phenotypes (eg, atopy). Intergenerational influences of maternal SES might be better understood if we consider how biological and social risks in the mother's childhood and subsequent life course can influence her perinatal health and behaviors, maternal-fetal exposures, and associated outcomes in her children.[9] Grounded in life-course epidemiology, this approach examines theoretical models linking exposures at different life periods and later health. The latent effects model proposes that early-life SES might determine maternal childhood environmental exposures (eg, pollutants, allergens, and stress) that prime her own immune system toward asthma and atopy, contributing to related disorders in her offspring.[10] A cumulative risk model suggests that psychosocial experiences related to childhood poverty and associated adversities accumulating over a mother's life (eg, family violence) might have lasting effects on maternal psychological functioning[11] and stress responses,[4] which, in turn, might affect the developing infant. Finally, the socioeconomic trajectory model posits that the mother's childhood SES might be related to her adult socioeconomic position (eg, educational attainment) and associated adverse social and physical

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Abbreviations used
IPT: Interpersonal trauma
MUP: Mouse urinary protein
SEM: Structural equation model
SES: Socioeconomic status

environmental exposures during her child's early development. This might manifest, for example, through perinatal health behaviors, such as maternal smoking,[12] contributing to childhood wheeze. Arguably, intergenerational effects of maternal childhood SES can be more fully explored through a life-course approach examining the direct relationship between maternal childhood SES and respiratory outcomes in the child, as well as considering potential pathways.[10,13]

We examine associations among maternal childhood SES, cord blood IgE levels (an index of early atopic risk[7]), and repeated wheeze in children enrolled in an urban pregnancy cohort: the Asthma Coalition on Community, Environment, and Social Stress (ACCESS) project. Using structural equation models (SEMs), we examine whether these childhood outcomes are dependent on (1) the direct effects of low maternal childhood SES versus indirect relationships operating through (2) the cumulative risk of SES-related social adversities throughout the mother's life course and/or (3) the socioeconomic trajectory of the mother (ie, adult SES) as related to environmental exposures and psychological stress experienced more proximate to the index pregnancy.

METHODS

Sample

This prospective cohort was originally funded to recruit 589 pregnant women and their children to study the effects of prenatal maternal and early-life stress on urban childhood asthma risk.[14] English- or Spanish-speaking pregnant women (≥18 years old) receiving prenatal care at Brigham & Women's Hospital, Boston Medical Center, and 3 community health centers were recruited between August 2002 and January 2006. Among 754 women approached who met the eligibility criteria, 78.1% agreed to enroll. There were no significant differences between participants and those who declined based on race/ethnicity, education, and income. Procedures were approved by the Brigham & Women's Hospital and Boston Medical Center human studies committees; written consent was obtained. Data on maternal childhood SES and childhood repeated wheeze were available for 510 mother-child pairs with follow-up to age 2 years; cord blood IgE measurements were available for 454. Baseline data on sociodemographics and prenatal exposures were obtained at 28.4 ± 7.9 weeks' gestation.

Maternal childhood SES

Housing status, a robust marker of economic circumstances over the life course, correlates with other conventional SES indicators (eg, income and assets) and is retrospectively reported with accuracy.[15,16] Parental home ownership in childhood is associated with later life outcomes, including physiological disruption.[15-17] Mothers reported whether their parents ever owned a home over the first 10 years of her childhood. A binary indicator of parental home ownership (ever between ages 0 and 10 years) indexed maternal childhood SES in the first 10 years of her life.

Outcome variables

Wheeze. At approximately 3-month intervals starting from birth, maternally reported wheeze was ascertained up to age 2 years. Caretakers were asked the following: "Since we last spoke with you on (date), has your infant/child had wheezing or whistling in the chest?" Two or more episodes constituted repeated wheeze.

Cord blood IgE. Cord blood total IgE levels (in international units per milliliter) were determined by using the CAP fluorescent enzyme immunoassay (Pharmacia [now Phadia], Uppsala, Sweden; lower limit of detection, 0.2 IU/mL).[18] IgE was log transformed and divided into quintiles, with serum levels at or greater than the upper 20% (1.37 IU/mL) considered high.[7]

Social pathway variables

Cumulative lifetime adversity. Interpersonal trauma (IPT) disproportionally burdens lower-income populations,[19] and trauma over the mother's life course has been associated with increased IgE levels in newborns.[7] IPT was assessed based on the mother's self-report on the Revised Conflict Tactics Scale[20] with documented reliability (range, 0.79-0.95) and validity in English and Spanish.[21] IPT was assessed during childhood (≤11 years of age), adolescence (12-17 years), adulthood before the pregnancy, and during the pregnancy asking whether anyone had ever pushed, grabbed, or shoved them; kicked, bit, or punched them; hit them with something; choked or burned them; forced them to engage in sexual activities; or physically attacked them in another way. Mothers were considered exposed in each period if they answered yes to any item. IPT was categorized as follows: (1) unexposed, (2) 1 time period (during childhood/teenage years only or adulthood and/or pregnancy only), or (3) chronic (both periods).

Educational attainment. Current maternal SES was indexed as less than high school, high school, and some college or more.

Cumulative prenatal stress. Lower maternal adult SES might be associated with increased chronic stressors experienced more proximate to the pregnancy.[22] Constructs assessed included discrimination, financial strain, community violence, and other negative life events.

The Experiences of Discrimination scale[23] ascertained maternal reports of ever experiencing racially motivated unfair treatment (eg, at school, getting a job, or from the police or courts). Items were summed, with higher scores indicating greater discrimination.

Financial strain was assessed through a 3-item index of economic difficulties in the past 3 months[24] scored on a 5-point scale and summed; higher scores indicated greater difficulties.

Community violence was obtained by using the My Exposure to Violence survey[25] assessing events over the past year, including hearing gunshots and witnessing or experiencing shoving or hitting, knife attacks, and shootings. Multi-item responses were summarized into a continuous score.[26]

Other negative life events in the past 6 months were assessed by using the Crisis in Family Systems survey,[27] encompassing 11 domains (ie, financial, legal, career, relationships, medical problems, safety in the community and home, other home issues, difficulty with authority, and discrimination), with multiple items in each domain. Women endorsed items as positive, negative, or neutral. The number of domains with 1 or more negative events were summed, creating a negative domains score, with higher scores indicating greater stress.

Physical pathway variables

Because lower-SES populations might be more likely to live in poor housing with increased indoor allergens[28] or communities with greater air pollution,[29] we also assessed the following.

Prenatal household allergens. Cockroach allergens (*Blattella germanica*: Bla g 1 and Bla g 2) and mouse urinary protein (MUP) were measured in settled dust collected within 2 weeks of the baseline questionnaire from the mother's bed and bedroom floor by using an mAb-based ELISA (Indoor Biotechnologies, Charlottesville, Va).[30] Increased cockroach levels were defined as Bla g 1, Bla g 2, or both levels of greater than 2 U/g,[30] whereas MUP levels of greater than the median were considered high.[31]

Housing deterioration. Because housing deterioration is predictive of household allergens,[28] an indicator of housing disrepair was defined by the number of adverse characteristics endorsed (eg, water damage, other evidence of leaks, and holes or cracks in the floor) categorized from 0 to 4.