Environmental Justice, Cumulative Environmental Risk, and Health Among Low- and Middle-Income Children in Upstate New York

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Objectives. We documented inequitable, cumulative environmental risk exposure and health between predominantly White low-income and middle-income children residing in rural areas in upstate New York.

Methods. Cross-sectional data for 216 third- through fifth-grade children included overnight urinary neuroendocrine levels, noise levels, residential crowding (people/room), and housing quality.

Results. After control for income, maternal education, family structure, age, and gender, cumulative environmental risk exposure (0–3) (risk > 1 SD above the mean for each singular risk factor [0, 1]) was substantially greater for low-income children. Cumulative environmental risk was positively correlated with elevated overnight epinephrine, norepinephrine, and cortisol in the low-income sample but not in the middle-income sample.

Conclusions. Cumulative environmental risk exposure among low-income families may contribute to bad health, beginning in early childhood. (*Am J Public Health.* 2004;94:1942–1944)

Environmental risks are not randomly distributed in the population; instead, they are inversely correlated to income.[1] Economically disadvantaged children live in noisier[2] and more crowded homes[3] and are exposed to more environmental toxins[1] than their middle-income counterparts. Housing quality is also inversely related to income.[4,5] Ethnic minorities also suffer disproportionate environmental risk,[6] and a few studies reveal no income–environmental quality link.[7] Although poor children are substantially more likely to confront singular environmental risks in their immediate environments, exposure to cumulative environmental risks may be a particularly important and unstudied aspect of environmental justice and health. If the ecology of childhood poverty is characterized by the confluence of environmental risks, examination of the health consequences of singular risks may underestimate the true environmental risk profile of low-income children.

We examined how exposure to residential crowding, interior noise levels, and housing problems, singularly and in combination, related to chronic physiological stress in a sample of low- and middle-income children in rural upstate New York.

METHODS

Participants

The income-to-needs ratio is a per capita index, adjusted annually for costs of living; a ratio equal to or less than 1 is the US Census Bureau's definition of poverty. One hundred fourteen third- through fifth-grade children (mean age=9.1 years, SD=1.10, 52% male) in this study lived in poverty (mean income-to-needs ratio=0.81). One hundred two third- through fifth-grade middle-income (mean income-to-needs ratio=2.79) children (mean age=9.2 years, SD=1.08, 52% male) also participated in the study. Sixty percent of the low-income sample lived with a single parent. Eighty-six percent of mothers in the low-income sample were high school graduates, with 3% also completing college. Twenty-four percent of the middle-income sample lived with a single parent. One hundred percent of mothers in the middle-income sample were high school graduates, with 44% also completing college. Representative of the rural northeast, the sample was predominantly White (96%). Our participants were drawn from a sample (n=270) of rural, low-income families (<1 income-to-needs ratio) and middle-income families (2–4 times the income-to-needs ratio) recruited from public schools, housing assistance programs, Head Start families, and cooperative extension programs in rural upstate New York. Our participants included the subset of the original sample in which 1 or more physiological stress measures were available.[8] Response rates could not be determined for the original sample because of voluntary responses to advertisements, announcements, and public presentations. The refusal rate of eligible families (income criterion and child age) was less than 5% and similar for both low- and middle-income families. The present sample of 216 children who have overnight neuroendocrine data does not differ regarding any background characteristic (age [$P<.35$], gender [$P<.15$], income [$P<.26$], single parent [$P<.78$], and mother's education [$P<.45$]) from the original sample of 270 children.

Procedure

One child per household and his or her mother participated in home interviews. Parental informed consent and child assent were obtained for all participants. Crowding was measured by dividing the number of people living in the household by the number of rooms (including bathrooms). Indoor noise was monitored for two 2-hour periods on different days with a Bruel & Kjaer Model 2236 Sound Level Meter (Bruel & Kjaer, Naerum, Denmark). Noise level during each period was assessed as Leq dbA (a measure of average sound pressure); means for each of the 2 periods were totaled and divided by 2 to produce an average. Housing quality assessment consisted of trained observer ratings with a standard instrument.[9] Raters trained to criterion (>90% independent agreement) conducted a walk-through evaluation of each residence with a 73-item rating scale (0- to 2-point scales) consisting of 5 subscales: structural quality (e.g., cracks in walls), clutter and cleanliness (e.g., materials on table/counters in kitchen), hazards (e.g., loose stair rail), indoor climate (e.g., ventilation), and chil-

dren's resources (e.g., designated play space). Extensive data on the reliability and validity of this standard housing quality index are available in Evans et al.[9]

Cumulative environmental risk exposure was estimated for each child. For the entire distribution of households, the mean and standard deviation for each environmental risk factor (i.e., crowding, noise, housing quality) were calculated. Risk exposure for each environmental risk factor was designated as greater than 1 SD above the mean for each respective factor and given a score of 1. The other exposure levels were considered lower risk and coded as 0. These 3 dichotomized environmental risk factors were then summed to form an index of cumulative environmental risk exposure (possible range=0–3).

Overnight (8:00 PM–8:00 AM) urine samples were collected, processed, and then deep frozen (–80°C) until subsequent biochemical assays could be conducted by technicians blind to the children's income status and environmental risk condition. Epinephrine and norepinephrine were assayed with high-performance liquid chromatography with electrochemical detection.[10] Free cortisol was measured with radioimmunoassay.[11] Creatinine was assessed to control for differences in body mass and incomplete urine voiding.[12] These neuroendocrine indices are reliable and valid indicators of chronic stress[13] associated with the development of cardiovascular disease and compromised immune functioning.[14]

RESULTS

Table 1 depicts descriptive information on each of the environmental risk factors. Low-income children were exposed to significantly more crowding ($t[214]=7.33$, $P<.01$), housing problems ($t[214]=8.31$, $P<.01$), and noise ($t[214]=3.21$, $P<.01$). A significant difference in the number of cumulative risks was detected for the 2 samples as well ($t[214]=5.62$, $P<.01$). Data for exposure to 2 or more cumulative environmental risks were collapsed because of the small number of children exposed to all 3 environmental risk factors.

Ordinary least-squares regression, statistically controlling for children's income-to-needs ratio, family structure (single-parent status), maternal educational attainment, and children's gender and age, was used to examine cumulative environmental risk exposure and chronic stress. The raw β coefficients are shown for the simultaneous equations (i.e., entered on the last step). Because most of the impact was associated with the category of 2 or more environmental risks, cumulative risk was fitted as a dichotomous categorical variable (0–1 risk=0; ≥2 risks=1).

Cumulative environmental risk exposure was significantly related to overnight urinary neuroendocrine levels in low- but not middle-income children (Table 2). For low-income children, all 3 indices of chronic physiological stress are significantly related to cumulative, environmental risk exposure. For middle-income children, cumulative environmental risk exposure was unrelated to any of the chronic stress indices.

We also examined with the same statistical controls in ordinary least-squares regression the relations between each singular risk factor (i.e., crowding, noise, housing quality) and chronic physiological stress. None of the singular risk factors was significantly related to any of the physiological stress outcomes within either the low- or middle-income samples.

TABLE 1—Descriptive Statistics for Environmental Risk Exposure Among Low- and Middle-Income Children in Rural Upstate New York

	Low Income		Middle Income	
Environmental Risk	Mean (SD)	Percent at Risk	Mean (SD)	Percent at Risk
Crowding (people/room)	.69 (.21)	18	.50 (.12)	8
Housing problems (0–2)	.71 (.30)	21	.42 (.20)	1
Noise (Leq dBA)	64.97 (7.18)	25	61.45 (7.41)	9
Cumulative environmental risks				
0		53		86
1		31		11
≥2		16		3
Level of cumulative risk exposure (SD)	.66 (.75)		.18 (.45)	

Note. Environmental risk is defined categorically (0, 1) as exposure to > 1 SD above the mean for the entire sample for each respective risk factor (crowding, noise, housing quality). Cumulative environmental risk is the sum (0–3) of these 3 environmental risk factors. Because of the small number of children exposed to 2 environmental risk factors, exposure to 2 and 3 environmental risks is collapsed into 1 group (≥2).

DISCUSSION

We investigated whether the natural covariation of environmental risk factors among children living in poverty has potential long-term health consequences for children. Considerable evidence indicates that low-income households face considerable environmental inequity, and suffer from greater exposure to a host of adverse physical conditions.[1] A unique, key feature of the environment of poverty with potentially far-reaching implications for children's health is the confluence of environmental risks confronting poor families.

Crowding, noise, and housing quality were assessed in a sample of low- and middle-income, rural elementary school children aged 8 to 10 years. Neuroendocrine indices of chronic stress increased in tandem with cumulative environmental risk exposure for the low-income children but not for the middle-income children. These significant increases in chronic physiological stress occurred independent of children's household income, family structure, gender, age, and maternal education. Low-income children exposed to a convergence of suboptimal living conditions suffered greater chronic stress compared with other indigent children who faced singular or no environmental risk. In contrast, middle-income children faced lower levels of environmental risk (only 3% faced 2 or more risks), which may explain why cumulative environmental risk was unrelated to chronic physiological stress in middle-income children. More than 5 times as many low-income children (16%) were exposed to 2 or more risks.

This study would benefit from a longitudinal investigation of cumulative risk exposure and children's health. Longitudinal research would offer stronger evidence, given the current

TABLE 2—Cumulative Environmental Risk, by Chronic Physiological Stress Index: Means (SD), β (SE) as if Last Entered, and ΔR^2 for Cumulative Environmental Risk After Control for Income-to-Needs Ratio, Single-Parent Status, Mother's Education, and Child Age and Gender

	Mean (SD)			
Income Level	0 (0–1 Risk)	1 (≥2 Risks)	β (SE)	ΔR^2
Cortisol (μg/mg creatinine)				
Poverty	.030 (.023)	.047 (.044)	.021* (.007)	.07
Middle-income	.023 (.013)	.025 (.012)	.000 (.010)	.00
Epinephrine (ng/mg creatinine)				
Poverty	4.71 (4.29)	8.07 (7.76)	3.61* (1.22)	.07
Middle-income	3.74 (3.17)	1.42 (0.54)	–2.08 (1.76)	.02
Norepinephrine (ng/mg creatinine)				
Poverty	29.01 (17.99)	48.75 (36.49)	22.05* (5.46)	.12
Middle-income	32.02 (15.67)	25.40 (8.28)	–8.14 (9.22)	.01

Note. Because most of the impact of cumulative risk was associated with the category of ≥2 environmental risks, cumulative risk was fitted as a dichotomous variable (0 = 0–1 risk; 1 = ≥2 risks) in the regression analyses.
*$P < .01$.

cross-sectional design, and enable us to study the duration and timing effects of cumulative environmental risk on children's health.[15] Other environmental risks (e.g., tobacco smoke) as well as psychosocial risk factors (e.g., family turmoil, parenting quality) that covary with poverty[16–18] should also be incorporated in future work. We also need research that includes families from low-income urban, inner-city settings, where the concentration of environmental risks may be even greater than those documented in the present rural population. Environmental injustice is a function of both race and income[1,6] and also could be influenced by urbanization. The generalizability of our results is constrained by our reliance on an opportunity sample of predominantly White elementary school children living in rural areas of upstate New York. Future work might include additional markers of morbidity such as hypertension or allostatic load.[14]

Our results have important implications for understanding the role of the environment in income-related health inequities. Foremost, they suggest that attention to singular environmental risk factors in isolation may obscure recognition of important health outcomes arising from cumulative risk exposure, especially among low-income populations. This study illustrates the value of conceptualizing cumulative, environmental risk exposure in a manner that begins to capture its natural, ecological covariation among some segments of the population. Cumulative environmental risk exposure within the home is associated with elevated neuroendocrine activity indicative of chronic stress among a sample of low-income White children living in a rural area; in contrast, cumulative environmental risk exposure appears unrelated to chronic stress levels among their middle-income counterparts. ■

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Contributors

G.W. Evans conceptualized and conducted the study. Both authors conducted the data analysis and wrote the article.

Human Participant Protection

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