

THE BIGGEST ASTHMA TRIGGER OF THEM ALL?

New studies indicate how poverty itself Is inflammatory

The gas flares, crackers, distillers and steel towers of the giant Chevron oil refinery loom over North Richmond, California, the setting for much of the "Place Matters" episode of *Unnatural Causes*. The predominantly poor African American, Latino and Southeast Asian kids who live in Chevron's shadow are three times more likely be taken to the emergency room for asthma attacks than kids living a couple miles away in the upscale town of Lafayette. (Palaniaippan, 2006)

Ten miles south, 10,000 diesel trucks a day rumble through West Oakland, another poor and predominantly African American neighborhood, on their way to and from the Oakland port. Kids there are seven times more likely to be hospitalized for asthma than children in the rest of California. (Palaniaippan, 2006)

And just as poor people, especially poor people of color, are more likely to live in communities where the air quality is compromised by soot and chemicals, so too are low income families disproportionately exposed to asthma triggers inside their own homes, such as mold, mildew, cockroaches and dust mites, often because their landlords have not properly maintained their premises

Similar asthma crisis are faced by poor communities of color across the country. In Harlem, one in four children has asthma (Perez-Pena, 2003). In the black and Latino neighborhoods of Roxbury, Boston the asthma rates are 5.5 times that of the rest of the Massachusetts (Loh 2003)

Still, increased exposures to indoor and outdoor pollutants don't entirely explain the higher risks of asthma faced by low income populations. There is something else at work.

Now, scientists such as Edith Chen, Ph.D, are circling in on the missing link. They have found evidence that the very experience of poverty and the stress it induces might itself be an asthma "trigger."

Asthma, like most every disease, is marked by a continuous wealth-health gradient. On average, the wealthier you are the healthier you are and the longer you live. Dr. Chen co-founded the <u>Psychobiological Determinants of Health Lab</u> at the University of British Columbia to better

understand the pathways by which class gets under the skin and influences our heath. Rather than focus on how material pollutants, like soot, disrupt our physiology Chen and her colleagues are investigating how 'social pollutants' – that is, toxic social environments can become embedded in our bodies and increase susceptibility to disease.

Asthma is a chronic inflammatory disorder of the airways. Chen's SES (socio-economic status) and Childhood Asthma project has been tracking bio-markers in children, such as cortisol levels, lung function, and immune and inflammatory system function, and mapping that data against the children's life circumstances and child-family interactions. Their findings are already making news:

- Lower SES children have immune systems that respond more aggressively to stimuli by producing greater quantities of a key protein implicated in inflammation and asthma called Th-2 cytokines. Chen found that psychological stress explains part of this effect that is, that low SES children experience greater stress in their day-to-day lives, and in turn, these stressful experiences are linked to greater stimulated Th-2 cytokine production. This study, Chen says, is one of the first to describe a psychobiological explanation for the graded relationship between the health of children and their socio-economic status (Chen, 2006).
- Might the social environment even influence our genome? In another study, Chen and her researchers found that the genes regulating inflammation, stress responses, and even wound healing tend to be turned on more frequently (over-expressed) in children with asthma from a low SES background. These low SES children had genes that functioned differently from high SES children with asthma, with signaling pathways for inflammation increased. In turn, this signaling profile might make these children'sbodies more vulnerable to asthma attacks when exposed to triggers like soot or mold. According to Chen, this study provided the first evidence in a clinical patient population that the larger social environment can affect processes at the genomic level (Chen, 2009).
- It's long been known that inhalers don't bring much relief to a certain subset of asthma sufferers. But why? In this study, Chen looked at two hormone receptors that are the targets of the glucocorticoids and broncodilators doctors commonly prescribe for attacks. It turns out that kids with asthma who experienced both high chronic stress in their lives and a major acute life event (e.g. violence) had fewer of these receptors on their immune cells, a lot fewer: 1/5 the number of glucocorticoid receptors mRNA and 1/9 the number of β2-adrenergic receptors mRNA. Scientist believe this change, poverty-induced but biologically expressed, partly explains why treatment is more likely to fail kids with asthma who live in high poverty, high stress environments (Miller, 2006).

Scientists like Dr. Chen who are looking through the wide angle lens and not just the microscope are generating a better understanding of how family and neighborhood circumstances, including chaos, instability, violence and stress, contribute to asthma inflammatory processes. Their work is not only leading to a more accurate understanding of why health patterns along class and racial

lines, but why anti-poverty efforts, even more than drugs, offer the most promise for a healthier society.

Works Cited

Chen, E., Hanson, M. D., Paterson, L. Q., Griffin, M. J., Walker, H. A., & Miller, G.E. (2006). Socioeconomic status and inflammatory processes in childhood asthma: The role of psychological stress. *Journal of Allergy and Clinical Immunology*, *117*, 1014-1020.

Chen, E., Miller, G. E., Walker, H. A., Arevalo, J. M., Sung, C. Y., & Cole, S. W. (2009). Genome-wide transcriptional profiling linked to social class in asthma. *Thorax*, *64*, 38-43.

Chen, E. and Hannah M.C. Schreier. (2008). "Does the Social Environment Contribute to Asthma?" Immunology and Allergy Clinics of North America 28(3):649-664. Website for the Psychobiological Determinants of Health Lab at the University of British Columbia [available online 27 July 2010: <u>http://www2.psych.ubc.ca/~healthpsych/index.htm</u>].

Miller, G. E., & Chen, E. (2006). Life stress and diminished expression of genes encoding glucocorticoid receptor and D-adrenergic receptor in children with asthma. *Proceedings of the National Academy of Sciences, 103*, 5496-5501.

Palaniaippan, M, Prakash, S, Bailey, D. Paying with our Health: The Real Cost of Freight Transport in California. Pacific Institute. November 2006 <u>http://www.pacinst.org/reports/freight_transport/PayingWithOurHealth_Web.pdf</u>

Penn Loh & Jodi Sugerman-Brozan, *Environmental Justice Organizing for Environmental Health: Case Study on Asthma and Diesel Exhaust in Roxbury, Massachusetts*, 584 ANNALS AM. ACAD. POL. & SOC. SCI. 110, 116 (2002). 10. ACTION AGAINST ASTHMA, *supra* note

Richard Perez-Pena, *Study Finds Asthma in 25% of Children in Central Harlem*, N.Y. TIMES, Apr. 19, 2003, at A1.