The correspondence section is a public forum and, as such, is not peer-reviewed. EHP *is not responsible for the accuracy, currency, or reliability of personal opinion expressed herein; it is the sole responsibility of the authors.* EHP *neither endorses nor disputes their published commentary.*

DDT and Breast Cancer Trends

doi:10.1289/ehp.11562

Cohn et al. (2008) suggested that birth cohort trends in breast cancer rates for women under 50 years of age are consistent with declining use of DDT (dichlorodiphenyltrichloroethane) after 1959. They cited Weiss (2007) in claiming that increased detection and treatment of *in situ* breast cancer must be considered when interpreting recent trends in breast cancer mortality rates in young women. The remarks of Weiss (2007) relate to women 40–49 years of age, and earlier detection and improved treatment of breast cancer has had a marked impact on breast cancer mortality rates in these women since 1990 (Berry et al. 2005; Chu et al. 1996). The birth cohort trends relevant to examining the possible impact of childhood DDT exposure on U.S. breast cancer rates, however, were firmly established well before 1990 in women < 40 years of age (Tarone 2007).

Cohn et al. (2007) reported a large increase in breast cancer risk estimates for *p,p′*-DDT [1,1,1-trichloro-2,2-bis(*p*-chlorophenyl)ethane] exposure with successive birth cohorts after 1930. Their reported odds ratio estimates by period of birth for the highest tertile of *p,p′*-DDT exposure were 0.6 for women born in 1931 or earlier (i.e., ≥ 14 years of age in 1945), 3.9 for women born in 1932–1937 (i.e., 8–13 years of age in 1945), 9.6 for women born in 1938–1941 (i.e., 4–7 years of age in 1945), and 11.5 for women born in 1942 or later (i.e., < 4 years of age in 1945) [Table 4, Cohn et al. (2007)]. In contrast, I have found no evidence of increasing breast cancer rates among young U.S. women born between 1930 and 1945 (Tarone 2007). I quantified trends in breast cancer mortality rates for U.S. white women 20–39 years of age (by 5-year age group) born during 1930–1945 using linear regression analyses with the logarithm of the age-specific rate as the dependent variable and year of birth as the independent variable (with two-sided *p*-values) [Surveillance, Epidemiology, and End Results (SEER) 2006; Tarone 2007]. The slope estimates did not differ significantly from zero for women in the three youngest age groups ($p > 0.25$), and there was a marginally significant decrease in rates for women 35–39 years of age ($p = 0.04$). Thus the trends in breast cancer mortality rates among women born in 1930–1945 are not consistent with the sharply increasing trend in odds ratios for childhood DDT exposure by birth period reported by Cohn et al. (2007). The most recent mortality rate contributing to the reported regression analyses (corresponding to women in the 35- to 39-year age group born in 1945) was for 1983, well before improvements in detection and treatment would have had any impact on breast cancer mortality rates.

Women born after 1945 were exposed to DDT for each of the first 13 years of life (and all years thereafter). In addition, DDT exposure increased from 1945 through 1959, when DDT use peaked (with dietary exposure peaking in 1965) (Wolff et al. 2005). If DDT exposure early in life markedly increases breast cancer risk, then some evidence of the increasing DDT use after 1945 might be expected in breast cancer mortality rate trends for young women born from 1946 through 1959 (Tarone 2007). Breast cancer mortality rates decreased significantly among women 20–24 years of age ($p = 0.009$) and 25–29 years of age ($p = 0.0002$) born between 1946 and 1959 (SEER 2006; Tarone 2007). The most recent rate contributing to these regression analyses was for 1987 (corresponding to women in the 25- to 29-year age group born in 1959). Breast cancer mortality rates decreased even more markedly ($p < 0.0001$) for women in the 30- to 34-year and 35- to 39-year age groups born from 1946 through 1959; some of the recent rates in these latter age groups were almost certainly affected by improved breast cancer detection and treatment, although decreasing trends were apparent in both age groups for rates well before 1990 (Tarone 2007). Thus, U.S. breast cancer mortality rates in women between the ages of 20 and 39 who were born between 1930 and 1959 show no evidence of an increase in breast cancer risk associated with their marked increase in DDT exposure during childhood.

The observed birth cohort trends in breast cancer rates do not refute a possible association between childhood DDT exposure and breast cancer risk, and contrary to the implication of Cohn et al. (2008), no such claim was made in my earlier letter (Tarone 2008). The regression analyses reported above suffer the weaknesses of all ecologic analyses, and in fact, the decreasing birth cohort risk of breast cancer in baby boomers has been observed in spite of trends in established risk factors (e.g., parity, age at first birth, and oral contraceptive use) that would predict increasing breast cancer rates among U.S. women born after 1945. If, as suggested by Cohn et al. (2007), the public health significance of DDT exposure early in life is large, then this would provide additional evidence that the factor or factors responsible for the paradoxical decrease in birth cohort risk of breast cancer observed among U.S. baby boomers must have a very powerful impact on breast cancer etiology, large enough to turn an expected increasing trend in breast cancer rates among baby boomers into a decreasing trend.

The author declares he has no competing financial interests.

Robert E. Tarone
International Epidemiology Institute
Rockville, Maryland
E-mail: bob@intepi.org

References

Berry DA, Cronin KA, Plevritis SK, Fryback DG, Clarke L, Zelen M, et al. 2005. Effect of screening and adjuvant therapy on mortality from breast cancer. N Engl J Med 353:1784–1792.

Chu KC, Tarone RE, Kessler LG, Ries LAG, Hankey BF, Miller BA, et al. 1996. Recent trends in U.S. breast cancer incidence, survival, and mortality rates. J Natl Cancer Inst 88:1571–1579.

Cohn BA, Cirillo PM, Sholtz RI. 2008. DDT and breast cancer: Cohn et al. respond [Letter]. Environ Health Perspect 116:A153–A154.

Cohn BA, Wolff MS, Cirillo PM, Sholtz RI. 2007. DDT and breast cancer in young women: new data on the significance of age at exposure. Environ Health Perspect 115:1406–1414.

SEER (Surveillance, Epidemiology, and End Results). 2006. SEER*Stat Software, Version 6.2.4. Mortality—Cancer, Total U.S. (1950–2002). Bethesda, MD:National Cancer Institute, Division of Cancer Control and Population Sciences, Surveillance Research Program, Cancer Statistics Branch.

Tarone RE. 2007. Breast cancer trends: the author responds [Letter]. Epidemiology 18:284–285.

Tarone RE. 2008. DDT and breast cancer [Letter]. Environ Health Perspect 116:A153.

Weiss NS. 2007. Breast cancer trends [Letter]. Epidemiology 18:284.

Wolff MS, Britton JA, Teitelbaum SL, Eng S, Deych E, Ireland K, et al. 2005. Improving organochlorine biomarker models for cancer research. Cancer Epidemiol Biomarkers Prev 14:2224–2236.

Editor's note: In accordance with journal policy, Cohn et al. were asked whether they wanted to respond to this letter, but they chose not to do so.

Beef Production and Greenhouse Gas Emissions

doi:10.1289/ehp.11716

In their article discussing the impacts of farm animal production on climate change, Koneswaran and Nierenberg (2008) called for "immediate and far-reaching changes in current animal agriculture practices" to mitigate greenhouse gas (GHG) emissions. One of their recommendations was to switch to organic livestock production, stating that

> Raising cattle for beef organically on grass, in contrast to fattening confined cattle on concentrated feed, may emit 40% less GHGs and consume 85% less energy than conventionally produced beef.

These claims are terribly misleading. Koneswaran and Nierenberg (2008) compared organic beef produced in Sweden (22.3 kg of carbon dioxide-equivalent GHG emissions per kilogram of beef) with unusual and resource-intensive Kobe beef production in Japan (36.4 kg of CO_2-equivalent GHG emissions per kilogram) (Cederberg and Stadig 2003; Ogino et al. 2007).

To achieve the ultra-high fat levels in meat preferred by Japanese consumers, Japan's wagyu cattle are raised and fattened for more than twice as long as typical U.S. beef cattle (Cattle Marketing Information Service Inc. 2007; Ogino et al. 2007). Moreover, all of the feed and forage for the Japanese animals (from birth through slaughter) must be shipped especially long distances—> 18,000 miles in the example cited. Hence, this beef has ultra-high GHG emissions and energy requirements.

According to several analyses, typical nonorganic beef production in the United States results in only 22 kg of CO_2-equivalent GHG emissions per kilogram of beef, which is 0.3 kg less than the Swedish organic beef system (Johnson et al. 2003; Subak 1999). These comprehensive life cycle analyses, which examined all aspects of beef production and all GHG emissions, seem to definitively rule out significant reductions in GHG emissions by switching to organic beef production.

In fact, if nitrous oxide and other emissions from land conversion are included in the analysis, a large-scale shift to organic, grass-based extensive livestock production methods would increase overall GHG emissions by nearly 60% per pound of beef produced.

According to Searchinger et al. (2008), each acre of cleared land results in 10,400 lb/acre/year of CO_2-equivalent GHG (over a 30-year period, based on estimated emissions from a proportion of each land type converted to cultivation in the 1990s). Our own analysis (Avery and Avery 2007) using conservative beef production parameters from Iowa State University's Leopold Center for Sustainable Agriculture shows that grain-finishing cattle is at least three times more land efficient per pound of finished beef compared to grass-finishing.

Cattle industry statistics [U.S. Department of Agriculture (USDA) 2008] show that, in 2007, the United States used 2 billion bushels of corn to produce 22.16 billion lb finished grain-fed beef (17.3 million head steers and 10.2 million head heifers at average dressed weights of 830.2 and 764.8 lb, respectively). At 150 bushels/acre corn, this means we used 13.3 million acres to produce the feed grains. Converting all beef production to grass-based finishing would require at least an additional 26.6 million acres of pasture/grass to produce 2007 U.S. beef output.

Using the 22 lb of CO_2-equivalent GHG per pound of grain-fed beef from Johnson et al. (2003) and the 22.3 lb CO_2-equivalent GHG per pound of beef for organic grass of Cederberg and Stadig (2003), each system producing 22.16 billion lb of beef would directly and indirectly result in 487.5 and 494.2 billion lb of CO_2-equivalent GHG emissions, respectively.

However, adding the "carbon debt" resulting from the additional cleared land required by the two-thirds less efficient grass finishing process (26.6 million acres × 10,400 lb/acre/year, or 276.6 billion lb/year) results in the organic system totaling 770 billion lb of CO_2-equivalent GHG emissions; or 58% higher than the conventional system's total of 487.5 billion lb.

In early 2007, the authors received funding from the GET IT (Growth Enhancement Technology Information Team) pharmaceutical companies that are members of the National Cattlemen's Beef Association, to conduct an analysis of the environmental impacts and costs of various beef production systems.

Alex Avery
Dennis Avery
Hudson Institute
Center for Global Food Issues
Churchville, Virginia
E-mail: aavery@hughes.net

References

Avery A, Avery D. 2007. The Environmental Safety and Benefits of Growth Enhancing Pharmaceutical Technologies in Beef Production. Churchville, VA:Hudson Institute, Center for Global Food Issues. Available: http://www.cgfi.org/pdfs/nofollow/beef-eco-benefits-paper.pdf [accessed 4 August 2008].

Cattle Marketing Information Service, Inc. 2007. Summary of Activity. Cattle Fax Update XXXIX(28):4.

Cederberg C, Stadig M. 2003. System expansion and allocation in life cycle assessment of milk and beef production. Int J Life Cycle Assess 8:350–356.

Johnson DE, Phetteplace HW, Seidl AF, Schneider UA, McCarl BA. 2003. Management variations for U.S. beef production systems: Effects on greenhouse gas emissions and profitability. In: Proceedings of the 3rd International Methane and Nitrous Oxide Mitigation Conference, 17–21 November 2003, Beijing, China. Beijing:China Coal Information Institute, 953–961.

Koneswaran G, Nierenberg D. 2008. Global farm animal production and global warming: impacting and mitigating climate change. Environ Health Perspect 116:578–582.

Ogino A, Orito H, Shimada K, Hirooka H. 2007. Evaluating environmental impacts of the Japanese beef cow-calf system by the life cycle assessment method. Animal Sci J 78:424–432.

Searchinger T, Heimlich R, Houghton RA, Dong F, Elobeid A, Fabiosa J, et al. 2008. Use of U.S. croplands for biofuels increases greenhouse gases through emissions from land-use change. Science 319(5867):1238–1240.

Subak S. 1999. Global environmental costs of beef production. Ecol Econ 30:79–91.

USDA (U.S. Department of Agriculture) 2008. Livestock Slaughter, July 2008. Available: http://usda.mannlib.cornell.edu/usda/current/LiveSlau/LiveSlau-07-25-2008.pdf [accessed 11 August 2008].

Beef Production: Koneswaran and Nierenberg Respond

doi:10.1289/ehp.11716R

Avery and Avery, who find comparing conventional Japanese and organic Swedish beef production misleading, propose relying on "comprehensive life cycle analyses" (LCAs) to quantify emissions from conventional U.S. beef production. However, neither study they cite (Johnson et al. 2003; Subak 1999) appears to be a comprehensive LCA, and it is unclear whether these studies considered emissions created by facets of beef production such as feed transport or pesticide manufacturing, as did Ogino et al. (2007). Additionally, contrary to Avery and Avery's conclusion, Subak (1999) stated that

> These results indicate that the intensification of beef production systems may be counterproductive because net emissions of carbon dioxide as well as nitrogen and other pollutants would increase.

For a more comprehensive analysis, additional production aspects must be considered. Ogino et al. (2007), for example, included the transportation of feed (> 18,000 km, not miles, as stated by Avery and Avery in their letter), which accounted for 8.3% of emissions.

A better comparison of conventional versus organic beef production may be an LCA of greenhouse gas (GHG) emissions from three Irish systems reported by Casey and Holden (2006). Conventional production generated the most GHGs, followed by agri-environmental, with the organic system producing the least GHGs.

In contrast to conventional production, organic farming can reduce nitrous oxide emissions by avoiding excessive amounts of manure, as stocking densities are limited to land available for manure application. Organic agriculture typically also uses less fossil-fuel energy, in part because thousands of feed transport miles may be reduced (Kotschi and Müller-Sämann 2004).

Pasture-based systems require less operational fuel and feed than do conventional systems, and they adeptly sequester GHGs in the soil, tying up 14–21 million metric tons of carbon dioxide and 5.2–7.8 million metric tons of N_2O in pasture soil organic matter (Boody et al. 2005; Rayburn 1993).

Dourmad et al. (2008) concurred with our conclusion (Koneswaran and Nierenberg 2008) that more research is needed and noted that existing LCAs often omit details such as land-use change information. Many LCAs—and other attempts to quantify GHGs from various systems (Avery and Avery 2007)—also lack data on pesticide use and animal transport from farms or feedlots to slaughter.

In our article (Koneswaran and Nierenberg 2008), we not only argued for refinement of agricultural practices but also for a concurrent reduction in animal product consumption in high-income nations, especially because the U.N. Food and Agriculture Organization has concluded that animal agriculture accounts for more GHGs than transport (Steinfeld et al. 2006). In addition to lowering GHG emissions, reducing animal product consumption could also decrease the incidence of cardiovascular disease, certain cancers, and obesity (McMichael et al. 2007). Given the developing global food crisis, it is important to note, as did Baroni et al. (2007) in the *European Journal of Clinical Nutrition*, that plant-based diets "could play an important role in preserving environmental resources and in reducing hunger and malnutrition in poorer nations."

Although Avery remains skeptical over the role of anthropogenic GHG emissions in global warming (2008), the Intergovernmental Panel on Climate Change (IPCC 2007) concluded that

> Most of the observed increase in global average temperatures since the mid-20th century is very likely due to the observed increase in anthropogenic GHG concentrations.

The link between GHG mitigation and organic or extensive animal agriculture systems is well established, as are the other environmental and public health benefits of less-intensive production systems. Understanding the efficacy of less technology-dependent mitigation strategies is critical as the effects of global warming become more evident.

Both authors are staff members of the Humane Society of the United States. D.N. also serves as a senior fellow with the Worldwatch Institute.

Gowri Koneswaran
Danielle Nierenberg
The Humane Society of the United States
Washington, D.C.
E-mail: gkoneswaran@humanesociety.org

References

Avery A, Avery D. 2007. The Environmental Safety and Benefits of Growth Enhancing Pharmaceutical Technologies in Beef Production. Churchville, VA:Hudson Institute, Center for Global Food Issues. Available: http://www.cgfi.org/pdfs/nofollow/beef-eco-benefits-paper.pdf [accessed 4 August 2008].

Avery DT. 2008. Nearly 32,000 scientists deny manmade global warming. Feedstuffs Newspaper (Minnetonka, MN) 16 June: 8.

Baroni L, Cenci L, Tettamanti M, Berati M. 2007. Evaluating the environmental impact of various dietary patterns combined with different food production systems. Eur J Clin Nutr 61:279–286.

Boody G, Vondracek B, Andow DA, Krinke M, Westra J, Zimmerman J, et al. 2005. Multifunctional agriculture in the United States. Biosci 55(1):27–38.

Casey JW, Holden NM. 2006. Greenhouse gas emissions from conventional, agri-environmental scheme, and organic Irish suckler-beef units. J Environ Qual 35:231–239.

Dourmad JY, Rigolot C, van der Werf H. 2008. Emission of greenhouse gas, developing management and animal farming systems to assist mitigation. In: Livestock and Global Climate Change: British Society of Animal Science, 17–20 May 2008, Hammamet, Tunisia. Cambridge, UK:Cambridge University Press, 36–39.

IPCC. 2007. Climate Change 2007: Synthesis Report. Geneva: Intergovernmental Panel on Climate Change. Available: http://www.ipcc.ch/pdf/assessment-report/ar4/syr/ar4_syr.pdf [accessed 4 August 2008].

Johnson DE, Phetteplace HW, Seidl AF, Schneider UA, McCarl BA. 2003. Management variations for U.S. beef production systems: Effects on greenhouse gas emissions and profitability. In: Proceedings of the 3rd International Methane and Nitrous Oxide Mitigation Conference, 17–21 November 2003, Beijing, China. Beijing:China Coal Information Institute, 953–961.

Koneswaran G, Nierenberg D. 2008. Global farm animal production and global warming: impacting and mitigating climate change. Environ Health Perspect 116:578–582.

Kotschi J, Müller-Sämann K. 2004. The Role of Organic Agriculture in Mitigating Climate Change: A Scoping Study. Bonn, Germany:International Federation of Organic Agriculture Movements.

McMichael AJ, Powles JW, Butler CD, Uauy R. 2007. Food, livestock production, energy, climate change, and health. Lancet 370:1253–1263.

Ogino A, Orito H, Shimada K, Hirooka H. 2007. Evaluating environmental impacts of the Japanese beef cow-calf system by the life cycle assessment method. Anim Sci J 78:424–432.

Rayburn EB. 1993. Potential ecological and environmental effects of pasture and BGH technology. In: The Dairy Debate: Consequences of Bovine Growth Hormone and Rotational Grazing Technologies (Liebhardt WC, ed). Davis, CA:University of California, 247–276

Steinfeld H, Gerber P, Wassenaar T, Castel V, Rosales M, de Haan C. 2006. Livestock's Long Shadow: Environmental Issues and Options. Rome:Food and Agriculture Organization of the United Nations.

Subak S. 1999. Global environmental costs of beef production. Ecol Econ 30:79–91.

Traffic-Related Air Pollution and Stress: Effects on Asthma

doi:10.1289/ehp.11863

Chen et al. (2008) examined the potential for social stressors to influence responsiveness to environmental pollution. Contrary to their initial hypothesis, and to results we reported previously (Clougherty et al. 2007), their findings indicated that chronic stress was associated with asthma symptoms and heightened inflammatory profiles only in low nitrogen dioxide areas. We would like to note several key issues in the emerging research on social susceptibility to environmental pollutants that should be considered as research on this work moves forward.

One key issue is that the relative timing of psychosocial stressors and physical exposures, which Chen et al. (2008) did not present, is critical for at least two reasons:

- Acute and chronic stress produce substantively different physiologic sequelae. Acute stress can induce bronchodilation with elevated cortisol (possibly masking short-term detrimental respiratory effects of pollution), whereas chronic stress can result in cumulative wear and tear (allostatic load) and suppressed immune function over time, increasing general susceptibility (McEwen and Seeman 1999).
- Temporal relationships between stress and pollution exposures matter. Depending on when measures are obtained, exposure misclassification is possible, which may influence the directionality of observed interactions. Chen et al. (2008) stated that the measured 6-month stress and NO_2 periods do not overlap, but they did not specify whether the stress measure preceded the 1998–2003 NO_2 exposure window or the amount of time that passed between exposures. If the stress interval occurred first, some increased susceptibility to subsequent pollution is plausible, provided that chronic stress effects predominate over acute effects. If, however, the stress interval occurred after NO_2 exposures, the interaction is potentially problematic, because we must then assume that stress levels measured after the 6-year NO_2 period (1998–2003) are relevant for the earlier time, which may not be the case. If, for example, respondents compared current stress to prior experience, an individual reporting high stress for one interval may have experienced lower stress previously, during those "reference" periods corresponding to the NO_2 window—potentially producing a negative interaction, as Chen et al. (2008) observed. More broadly, careful attention to relative timing and durations of stress and pollution exposures is critical in maintaining directionality and interpretability as we progress with this research.

Second, Chen et al.'s finding of significant effects of stress only in low-NO_2 areas (Chen et al. 2008) points to the possibility of nonlinear interactions and saturation effects at high exposures. Similarly, our group (Clougherty et al. 2006) reported that asthmatic children of families reporting higher fear of violence showed less symptom improvement in response to allergen-reducing indoor environmental interventions. Our results, counter to our initial hypotheses, suggested a saturation effect in our very high-exposure public housing cohort, where either high exposure alone may have been adequate to induce or maintain symptoms.

Third, Chen et al. (2008) did not address the spatial covariance among stress, socioeconomic status, and pollution, which can confound geographic information system–based air pollution epidemiology. In particular, communities near highways, with higher traffic-related pollution and lower property values, may be disproportionately composed of families having lower socioeconomic status. Because of this potential for spatial autocorrelation and thus confounding, accurate fine-scale exposure measurement is critical. However, Chen et al. (2008) did not present pollution or stress maps, the NO_2 model was not formally validated to this cohort's specific spatial characteristics, and spatial patterns in stress were not explored; thus we are left wondering whether, and how, spatial misclassification and confounding may be at play. Relatedly, social–physical correlations may vary by geographic scale (e.g., across vs. within neighborhoods); although a given neighborhood may have high mean pollution and stress, it is harder to argue that particular individuals (or residences) within these neighborhoods would be relatively more exposed to both (i.e., individuals living closer to highways are not necessarily more exposed to violence or family stress than are other community members).

Fourth, Chen et al. (2008) reported results for 73 asthmatic children. However, in the absence of information on disease chronicity, severity, or adequacy of medical treatment, it may be difficult to truly assess the influence of either stress or traffic-related pollution. Relatedly, it is important to distinguish between processes related to illness onset from those related to progression or exacerbation, and whether the negative interaction observed in their study could be expected in healthy adolescents.

Finally, the cohort studied by Chen et al. (2008) varied considerably in age (9–18 years), but the authors did not consider age-related asthma characteristics and responsiveness to family stressors and air pollution. Age stratification should have been used to compare the strength of individual and combined effects at multiple ages. It would also be interesting to know whether non–family-related stressors would produce similar interactions at all ages.

The issues we have highlighted—temporal relationships between stressors and pollution, nonlinearity and saturation effects, spatial correlations, age-related susceptibility, and distinctions between illness etiology and exacerbation—will be critical in the further study of social–environmental interactions. These effects may distort observed associations (e.g., saturation effects may reverse interactions at high exposures), but with sustained attention to these issues, we can better understand joint effects of social and physical environments on health.

The authors declare they have no competing financial interests.

Jane E. Clougherty
Laura D. Kubzansky
Harvard School of Public Health
Boston, Massachusetts
E-mail: jcloughe@hsph.harvard.edu

References

Chen E, Schreier HMC, Strunk R, Brauer M. 2008. Chronic traffic-related air pollution and stress interact to predict biologic and clinical outcomes in asthma. Environ Health Perspect 116:970–975.

Clougherty JE, Levy JI, Hynes HP, Spengler JD. 2006. A longitudinal analysis of the efficacy of environmental interventions on asthma-related quality of life and symptoms among children in urban public housing. J Asthma 43:335–343.

Clougherty JE, Levy JI, Kubzansky LD, Ryan PB, Suglia SF, Canner MJ, et al. 2007. Synergistic effects of traffic-related air pollution and exposure to violence on urban asthma etiology. Environ Health Perspect 115:1140–1146.

McEwen BS, Seeman TE. 1999. Protective and damaging effects of mediators of stress. Elaborating and testing the concepts of allostasis and allostatic load. Ann NY Acad Sci 896:30–47.

Traffic-Related Air Pollution and Stress: Chen and Brauer Respond

doi:10.1289/ehp.11863R

We thank Clougherty and Kubzansky for their thoughtful review of our article (Chen et al. 2008). We view our article, as well as their article on exposure to violence, air pollution, and asthma etiology (Clougherty et al. 2007), as suggestive regarding how the social and physical environments operate in asthma. Although the nature of the interaction effects were different in these two studies, the broader point—that there are interactive effects between the social and physical environments in asthma —is consistent and is the key message that we wish to emphasize.

We would like to address their specific comments. First, regarding temporal issues, Clougherty and Kubzansky raise the possibility that stress increases susceptibility to subsequent pollution. We agree that this is possible; we also recognize the possibility that chronic pollution exposure could heighten responses to subsequent stressors. As we stated in our "Discussion" (Chen et al. 2008), the time frame of assessments that were available to us for these analyses was not ideal, and future studies should more specifically coordinate the timing of exposures to both stress and air pollution.

Second, we agree it is possible that saturation effects may occur at high levels of pollution exposure. However, because pollution levels in Vancouver (British Columbia, Canada) are not extreme (the range in our sample was 10–30 ppb nitrogen dioxide), we think this is an unlikely explanation.

Third, regarding spatial covariance, in our study (Chen et al. 2008), family stress was measured at the individual level; thus, we do not have neighborhood-level stress maps or information on spatial patterns in stress. Although spatial covariance between socioeconomic status and air pollution has the potential to lead to confounding, the availability of individual measures of stress and air pollution exposure estimates at the resolution of individual addresses allowed us to evaluate interactions. Our longitudinal findings also diminish the likelihood of confounding. Further, previously published pollution maps (Henderson et al. 2007) have shown that, in our study area, air pollution levels are not spatially correlated with neighborhood socioeconomic status [e.g., see UBC (University of British Columbia) Centre for Health and Environment Research 2008].

Fourth, we presented information about disease characteristics in Table 1 (Chen et al. 2008). We also controlled for asthma severity and medication use in all analyses, as described in our article under "Potential confounders."

Finally, we agree that it would be interesting to know whether stress by air pollution effects vary by age. However, given the limited sample size in our study, we were unable to test this possibility.

The authors declare they have no competing financial interests.

Edith Chen
Michael Brauer
University of British Columbia
Vancouver, British Columbia, Canada
E-mail: echen@psych.ubc.ca

References

Chen E, Schreier HMC, Strunk R, Brauer M. 2008. Chronic traffic-related air pollution and stress interact to predict biologic and clinical outcomes in asthma. Environ Health Perspect 116:970–975.

Clougherty JE, Levy JI, Kubzansky LD, Ryan PB, Suglia SF, Canner MJ, et al. 2007. Synergistic effects of traffic-related air pollution and exposure to violence on urban asthma etiology. Environ Health Perspect 115:1140–1146.

Henderson SB, Beckerman B, Jerrett M, Brauer M. 2007. Application of land use regression to estimate long-term concentrations of traffic-related nitrogen oxides and fine particulate matter. Environ Sci Technol 41:2422–2428.

UBC (University of British Columbia) Centre for Health and Environment Research. 2008. Annual Average Air Pollution Levels, Asthma Rates and Neighbourhood Income Levels in Vancouver. Available: http://www.cher.ubc.ca/UBCBAQS/images/Asthma_Vancouver.gif [accessed 21 July 2008].