DDT and Breast Cancer Trends

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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., \( \geq 14\) 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 contribution 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 contribution 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.

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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

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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 resource-intensive Kobe beef production in Japan (36.4 kg of CO2-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 CO2-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 CO2-equivalent GHG emissions (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 CO2-equivalent GHG per pound of grain-fed beef from Johnson et al. (2003) and the 22.3 lb CO2-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 CO2-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 x 10,400 lb/acre/year, or 276.6 billion lb/year) results in the organic system totaling 770 billion lb of CO2-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.

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pasture soil organic matter (Boody et al. 2008; Rayburn 1993).

Dourmad et al. (2008) concurrently with our conclusion (Koneswaran and Nierenberg 2008) that more research is needed and noted that existing 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) concluded that environmental and public health benefits of 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.

Although Avery remains skeptical of 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.

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Traffic-Related Air Pollution and Stress: Effects on Asthma
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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 NO2 periods do not overlap, but they did not specify whether the stress measure preceded the 1998–2003 NO2 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 predominating over acute effects. If, however, the stress interval occurred after NO2 exposures, the interaction is potentially problematic, because we must then assume that stress levels measured after the 6-year NO2 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 NO2 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-NO2 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 NO2 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.