Coarse Particles and Dust Storm Mortality

In a paper titled "Episodes of High Coarse Particle Concentrations Are Not Associated with Increased Mortality," Schwartz et al. (*1*) concluded that "coarse particles from windblown dust are not associated with mortality risk." The authors seem to have overlooked the medical literature on the health effects of the historic U.S. dust storms of the dust bowl in 1935 (*2–4*). Brown et al. (*2*) reported that

> the dust ... was exceedingly irritating to the respiratory tract and materially increased the number of deaths from pneumonia and other complications.... The storm that will be longest remembered came [to Dodge City, Kansas] on the afternoon of Sunday [April] the 14th at 2:40 p.m.... No fatalities are known.

They concluded that

> The "immediate" effects are shown in the [delayed] increase in morbidity and mortality from the acute infections of the respiratory tract.

Schwartz et al. (*1*) restricted their analysis to mortality occurring on "the day of the dust storm or the following day." Therefore, they may have not observed any delayed rise in mortality that could have occurred later because of the "immediate" exacerbation of acute respiratory infections in highly susceptible individuals, such as those with preexisting chronic obstructive pulmonary disease. Furthermore, improvements in modern medical treatments, such as the replacement of the sulfa drugs of the mid-1930s by penicillin and other drugs, could delay a mortal outcome beyond the lag observed in the 1930s.

David T. Mage
U.S. Environmental Protection Agency
National Center for Environmental Assessment/Environmental Media Assessment Group
Research Triangle Park, North Carolina
E-mail: Mage.David@epa.gov

References and Notes

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Dust Storms: Schwartz's Response

In response to our paper reporting no excess risk of death in Spokane following dust storms when compared to appropriately chosen controls (*1*), Mage cites an article from the 1930s that anecdotally reports high rates of respiratory illness during the dust bowl period in the Midwest. This period, which coincided with the Great Depression, was a period of extreme social and economic stress. Since our paper was published, a reanalysis of three diary studies by Neas and Schwartz (*2*) has reported that lung function and lower respiratory symptoms were associated with fine particles but not coarse particles. Gold et al. (*3*) reported that heart rate variability is associated with fine particles but not coarse particles. These new results confirm our findings, which are also supported by the toxicology studies cited in our paper. Based on this evidence, I find the anecdotal reports from the 1930s unconvincing.

Joel Schwartz
Harvard School of Public Health
Boston, Massachusetts
E-mail: jschwrtz@hsph.harvard.edu

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Uterotrophic Activity of a "Phytoestrogen-Free" Rat Diet

Thigpen et al. (*1*) recently discussed the phytoestrogen content of a range of commercially available rodent diets and the effect they could have on the outcome of reproductive toxicity and endocrine disruption studies. They suggested that careful attention should be given to the use of the casein-based and phytoestrogen-free diet AIN-76A (Research Diets, Inc., New Brunswick, NJ) in such studies. Their letter was primarily in response to a paper by Boettger-Tong et al. (*2*) in which the sensitivity of the rodent uterotrophic assay was reduced by the use of a diet containing phytoestrogens, leading to increased control uterine weights.

We previously described how we had changed our weanling rat diet from PCD to RM1 (both from Special Diet Services Ltd., Witham, Essex, UK) to decrease control uterine weights and thereby to increase the sensitivity of the uterotrophic assay (*3*). That change led to a reduction in the mean uterine weight of 24-day-old control rats from 33.7 ± 7.0 mg (mean ± SD; n = 155) to 28.3 ± 6.1 mg (n = 371). Treatment of these control rats with the antiestrogen Faslodex (AstraZeneca, Alderley Park, Macclesfield, UK) reduced uterine weights further to 18.3 ± 2.4 mg (n = 23), indicating low levels of phytoestrogens in the RM1 diet or the presence of prepubertal levels of estradiol.

Inspired by the letter from Thigpen et al. (*1*), we mounted a study in which pregnant rats are being fed one of four different diets throughout pregnancy and until weaning. We plan to observe sentinel developmental landmarks and reproductive organ weights. The diets under study are RM3/RM1 (*3*), AIN-76A (*1*), Purina 5001 (*1*), and a global diet containing no soy or alfalfa (Harlan UK, Bicester, Oxfordshire, UK). As a pilot study to the above comparative diet study, we exposed 21-day-old (weanling) female Alpk rats to our usual RM1 diet or to AIN-76A diet for 3 days, as in our standard usual uterotrophic assay (*3*). We also exposed a group of animals on the AIN-76A diet to the antiestrogen Faslodex. Animals were killed on postnatal day 24 and uterine weights were determined. The animals on the AIN-76A diet had heavier uteri than those on the standard diet, an increase that was abolished by concomitant treatment with the antiestrogen. The effect was confirmed in a larger repeat study (blotted uterine weights were 26.1 ± 6.7 mg for RM1 and 45.4 ± 22.3 mg for AIN-76A, with no statistically significant difference in the terminal body weights of the two groups; groups included 30 animals, and similar differences were also seen for dry uterine weights).

We have no explanation for the increases in uterine weight observed for the animals on the AIN-76A diet. These increases were substantial; one of the animals on the AIN-76A diet had a uterine weight typical for that of an estradiol-treated animal (136.8 mg blotted weight). There is no obvious source of estrogens in the AIN-76A diet from the specifications provided by the manufacturers. Thigpen et al. (*1*) noted that no phytoestrogens have been detected in this diet [the subsequently derived diets AIN-93G and AIN-93M have soybean oil added (*4*)]. An earlier paper by Thigpen et al. (*5*) showed that this same AIN-76A diet increased uterus to body weight ratios in weanling CD-1 mice as compared to animals maintained on Purina Chow 5002. The authors noted that it was unclear what