Almost five decades have elapsed since the effect of the so-called “urban factor” on lung cancer has been suggested.1,2 Air pollution has always been an attractive explanation for the 10–40% increase in lung cancer mortality observed in urban versus rural areas, but confounding from smoking and other factors has been a great limitation in interpreting geographical comparisons. Several attempts have been made to specifically evaluate the role of air pollution on lung cancer aetiology during the 1960s and 1970s.3

In 1976, in a review for the International Agency for Research on Cancer (IARC), Higgins5 concluded that the studies available:

“provide support for the view that air pollution is a factor in this disease. But the effect of pollution cannot be large. It is likely to be a small fraction (possibly a tenth) of the effect of cigarette smoking.”

When the results of the two large American cohort studies on air pollution, the Six Cities Study6 and the American Cancer Society (ACS),7 were published, a strong association between particulate matter (PM) concentrations and cardiorespiratory mortality was reported, and even though important individual confounders like smoking and occupational exposure were controlled for, a link with lung cancer was noted. Confirmation of the preliminary suspicions finally arrived, reinforced by the publication of the additional follow up of the ACS cohort in 2002.8 In this last report, the mortality of approximately 500 000 adult men and women was followed from 1982 to 1998. The study indicated a significantly increased mortality risk ratio for lung cancer (RR = 1.14, 95% CI 1.04 to 1.23) with an increase of PM2.5 (particulate matter with an aerodynamic diameter <2.5 μm). In the American studies, long term exposure was estimated from metropolitan annual average ambient concentrations; contrasts in air pollution exposure were based on inter-city concentration differences, yet no information on exposure to calculation; whether the lung cancer effect that has been found for exposure to PM in the large cohort studies can be explained on the basis of the ambient concentration of relevant lung carcinogens—arsenic, chromium, nickel, and PAH—or whether there is an overall effect of the PM itself. They combined the WHO unit risk factors with concentrations of cancer causing chemicals found in the atmosphere in typical US cities during the 1960s, and used these to predict annual cancer rates in the ACS study. They found that the cancer rates predicted and observed were rather similar; an indication that known chemical carcinogens are responsible for the lung cancers due to PM2.5. A rather tentative conclusion (in contrast with earlier findings from Röösli and colleagues9) given that there are several uncertainties regarding this issue that the authors point out.

In the meantime, research into the mechanisms linking PM and lung cancer progresses.10 In vitro studies suggest that particles impact genotoxicity as well as cell proliferation via their ability to generate reactive oxygen and nitrogen species. This may happen because of the physicochemical characteristics of the particle surface, or due to their ability to stimulate cellular oxidant generation via various mechanisms, including an inflammatory response. However, in vitro studies need confirmation by in vivo experiments.

Is particle matter responsible for causing lung cancer, regardless of its contents? The issue might have several implications for regulatory agencies in the future. For the time being we are left with an unresolved research question. However, international agencies may consider the accumulated evidence on air pollution and lung cancer available so far. Although the relative risks associated with complex, long term exposure are relatively small and difficult to detect, a large fraction of the population is exposed and the overall impact is not negligible.

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**Filtration technology arrests asthma allergen**

Measures to abolish the threat of asthma to public health from soybean dust have been so successful that they could underpin future control standards for allergens, according to an evaluation in Barcelona. Success was achieved by combining knowledge and skills across a wide range of sectors and agencies.

The initial alert was an asthma epidemic in June 1996 traced to dust from ships unloading soybeans in the port, near the city. A significant benefit was introducing dry filtration technology in late 1997 to reduce emissions. This took the form of micropore size filter sleeves or special filters in series with PTFE membrane filter cartridges to replace standard technology in late 1997 to reduce emissions. This took the form of micropore size filter sleeves or special filters in series with PTFE membrane filter cartridges to replace standard technology in late 1997 to reduce emissions. This took the form of micropore size filter sleeves or special filters in series with PTFE membrane filter cartridges to replace standard technology in late 1997 to reduce emissions.

Four indicators were used to evaluate effectiveness. Soybean dust emissions, available from 1997, dropped 96–98% between summers 1998 and 1999. Airborne dust load measured at one site in the port fell drastically (mean (median) 1089 (731) RAST U/m3 in June 1996 v 59 (35) U/m3 June-December 1998). No asthma epidemics were recorded in four city hospitals, and sentinel surveillance from the end of 1997 with a panel of high risk asthma sufferers uncovered just one cluster of symptoms, coinciding with unloading in adverse weather.

Similar problems with soybean dust have emerged elsewhere, including outside Spain, but no legislation has been developed to control the public health risk. That may change.