

Urban air pollution

India is facing a serious double burden of disease. Most of the old infectious diseases like malaria, filariasis and kala-azar have not yet disappeared; indeed they are bouncing back. At the same time, other chronic non-communicable diseases such as cancer, cardiovascular disease and respiratory disorders are becoming more dominant.

It is becoming clear that the pattern of economic growth that we are adopting is becoming increasingly associated with environmental pollution. A study comparing the rates of economic growth and the rates of growth of vehicular pollution and industrial pollution shows that during 1975–1995, the Indian economy grew by 2.5 times, but the industrial pollution load grew by 3.47 times and the vehicle pollution load by 7.5 times¹. Indeed, Indian cities are being exposed to high levels of air pollution and people living in these cities are paying a price for the deterioration in air quality. The World Bank has estimated that Indians are spending Rs 4550 crores every year on treatment of diseases caused by ambient air pollution².

Air quality in Delhi is deteriorating. Levels of primary pollutants – suspended particulate matter (SPM), nitrogen oxide and sulphur dioxide – have gone up significantly in the last decade³. The consistently high levels of total SPM are worrying. Limited data available from the Central Pollution Control Board (CPCB) indicate that the levels of small particles less than 10 micron (PM10) are very high. This size of particulates is known to cause severe damage to the lungs. In fact, the World Health Organisation (WHO) reports that there is no safe level for particulate matter emissions⁴. International studies have confirmed association between elevated levels of particulate air pollution and decline in lung function or increase in respiratory symptoms such as cough, shortness of breath, wheezing and asthma attacks. Studies have also found associations between particulate air pollution and rates of hospitalization, chronic obstructive pulmonary disease and restricted activity due to illness⁵. A World Bank study on the health effects of air pollution in Delhi revealed that SPM in Delhi alone led to premature death of

7491 persons² in 1991–1992. The study repeated for the year 1995 shows an increase to about 10,000 in just three years, which means a death rate of one person per hour due to air pollution⁶.

It is not known on what scientific database such as epidemiological studies, morbidity and mortality patterns that air quality standards published for India are arrived at. Given the knowledge of the harmful effects of particulate pollution and the high concentration of particulate matter in Delhi's air, we need to generate regular information on the ambient concentration levels of small particulates of diameter less than 10 micron and/or 2.5 micron and take urgent steps to control emissions of these particles.

It is well known that the combustion of diesel generates small particulate matter, nitrogen oxides (NO_x), sulphur dioxide (SO₂) and polycyclic aromatic hydrocarbons (PAH). Dieselization of private vehicles is accelerating in Delhi. The relatively low prices of diesel are promoting its use further, which is a factor that encourages automobile manufacturers to introduce diesel versions of their vehicles. This development is bound to worsen the air quality in Delhi, bringing in its wake a number of related health problems.

The new scientific information emerging from international studies indicates that the cancer-causing potential of diesel exhaust is very high. The Scientific Review Panel of the California Air Resources Board points out, based on human epidemiological data, that a chronic exposure to 1 µg/cum of diesel exhaust will lead to 300 additional cases of lung cancer per million people⁷. On this basis, for a population of ten million people in Delhi, this means 3000 extra cases of lung cancer for a chronic exposure to 1 µg/cum of diesel exhaust. A WHO study done on rats shows that chronic exposure to 1 µg/cum of diesel exhaust can lead to 16–71 additional cases of lung cancer per million exposed rats⁴. Recently, Japanese researchers have discovered a compound, 3-nitro-benzanthrone, in the exhaust fumes of diesel engines that may be the most carcinogenic compound ever analysed⁸.

Many studies have established that diesel exhaust causes mutations in

chromosomes and damage to DNA, triggering cancer. Diesel exhaust, rich in polycyclic aromatic hydrocarbons (PAH) and particulate matter, causes 10 times more mutation than leaded petrol, which in turn is 10 times more mutagenic than unleaded petrol, according to Swedish tests⁹. A 1993 US study that covered six cities found a significant association between air pollution and mortality due to lung cancer and cardiovascular disease. The study found that the probability of death increased significantly with an increase in exposure to fine particulates (PM10 and PM2.5) and sulfate particles than with an increase in total particulate pollution, aerosol acidity, sulphur dioxide or nitrogen dioxide. Specifically, the study found that when people were exposed to average PM10 levels of 47 µg/cum they suffered a mortality rate as much as 48 per cent higher than those exposed to lower levels. Similar death rates were associated with sulfate particles, average levels of which went up to 13 µg/cum in the cities⁵.

The results of the above studies are of concern even in the Indian context, as diesel exhaust accounts for a significant proportion of small particles including sulfates in the air. In fact, the first survey of PM10 in Delhi shows that they reach extremely high levels – as much as 500 µg/cum or 5 times higher than the standard prescribed by the CPCB^{10,11}.

Diesel also produces NO_x that is easily absorbed in the blood and then reduces the oxygen-carrying capacity of the blood. It makes the lung tissues brittle and leathery and can cause lung cancer and emphysema (severe breathing problems)¹². At the ITO Crossing, NO₂ is above the standards in one out of every five days¹³. Even more disturbing is the fact that NO_x from diesel once out in the air forms ozone, yet another harmful gas.

Ozone causes inflammation of the airways (bronchus and bronchioles), that leads to respiratory problems. Ozone may pose its worst health threat to those who already suffer from respiratory diseases, such as asthma, emphysema and chronic bronchitis^{14,15}. Ozone levels in Delhi are disturbing. A 1993 study by the Central Road Research Institute, New Delhi showed that average ozone levels were 10–40 per cent above WHO standards¹⁶.

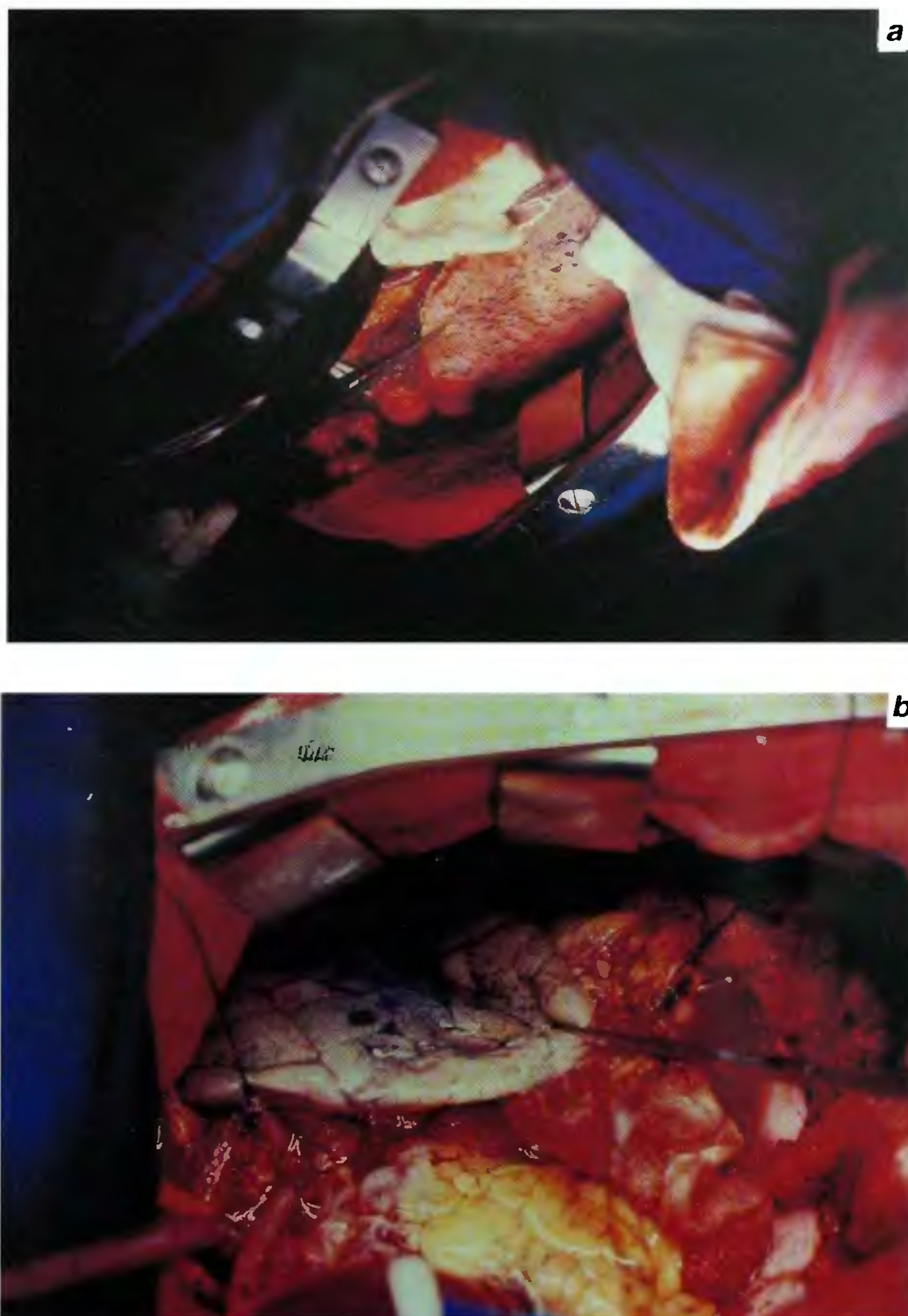


Figure 1. *a*, The clean lungs of a patient from Himachal Pradesh. *b*, The dirty lungs of a patient from Delhi. The black spots show deposition of carbon almost like a miner's lung. It is as if people of Delhi are living in a mine. It shows that the person has been regularly inhaling polluted air with a lot of carbon particles which can come from burning of coal or from vehicular exhaust. In Delhi, diesel vehicles put out a lot of carbon soot and diesel use is nearly three times that of petrol. Damage to lungs, however, comes not so much from the carbon as much as it comes from exposure to sulphur dioxide, nitrogen oxides and fine particles (less than 2.5 microns) which are present in diesel exhaust in a big way. If carbon from diesel exhaust is getting into the lungs as shown in this picture, then so are the deadly elements of diesel and petrol exhaust.

Diesel also produces carbon monoxide, which causes severe heart problems. A US study estimated that 6 per cent of the congestive heart failures and hospitalizations in the cities were related to an

increase in carbon monoxide in ambient air¹⁷. Carbon monoxide aggravates heart diseases by binding to the haemoglobin, thereby decreasing oxygen transport to the tissues.

There is an urgent need for comprehensive epidemiological studies to show how ambient air pollution is affecting people's health and quantify this information in order to provide policy tools for air quality planning. For instance, no nationwide survey on asthma sufferers has been conducted so far even though the cumulative prevalence rate of asthma in a place such as Delhi is estimated to be 1 to 2 children out of 10 (ref. 18). According to one study, 65 per cent of the people in Delhi are estimated to suffer from morning cough and phlegm and other respiratory symptoms¹⁹.

In other countries, governments have set up national level institutional mechanisms for medical research and monitoring in the area of air pollution with a view to influence policies. Medical associations have addressed the health issues related to air pollution and have pressurized governments to take corrective action. Most notable is the recent statement from the Australian Medical Association condemning the new reform package in Australia promoting the use of diesel by slashing taxes on diesel²⁰.

As a first step, we urge the government to stop the increase in levels of particles in the air by controlling the dieselization of the private vehicle fleet. Diesel commercial vehicles are already responsible for a significant portion of the tiny particles in Delhi's air. While it is important to conduct epidemiological studies to establish and enforce preventive policies in air pollution, there is already sufficient information available from international studies for us to start taking preventive action. Preventive action is critical for good public health management. Commercial profit and public good have to become mutually compatible and reinforcing.

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SCIENTIFIC CORRESPONDENCE

Susceptibility of brinjal shoot and fruit borer to the δ -endotoxins of *Bacillus thuringiensis*

Brinjal (egg plant) is one of the most important vegetable crops of India. It is widely consumed by all sections of the population and is relatively inexpensive and is available throughout the year¹. Brinjal is infested by a lepidopteran insect called brinjal shoot and fruit borer (BSFB *Lucinodes orbonalis* Guenee) which causes extensive damage to the growing shoot tips and fruits, thereby drastically reducing the marketable fruit yield². It is very difficult to control the pest because of its burrowing nature. Organic pesticides are widely used to control BSFB, which may adversely affect human health and environment³. Safe, effective and eco-friendly strategies to control insect pests include genetic engineering of brinjal using genes encoding insecticidal proteins⁴. *Bacillus thuringiensis* (Bt), a gram-positive soil bacterium, synthesizes insecticidal crystal proteins or δ -endotoxins during sporulation⁵. It was observed that many of the δ -endotoxins were lepidopteran-specific and were active at very low concen-

trations⁶. In the present study, we tested the efficacy of seven lepidopteran-specific δ -endotoxins of Bt towards the second instar larvae of BSFB. An artificial diet was formulated to rear BSFB larvae and perform insect bioassays.

Seven lepidopteran-specific Bt δ -endotoxin genes, viz. *cryIAa*, *cryIAb*, *cryIAc*, *cryIB*, *cryIC*, *cryIE*, and *cry2Aa* cloned in *Escherichia coli* expression vectors were obtained from Donald Dean (Ohio State University, Columbus). The *E. coli* cultures were grown for 48 h at 37°C in LB medium and δ -endotoxins were purified as described by Lee *et al.*⁷. The crystal proteins were solubilized in buffer containing 50 mM sodium carbonate (pH 9.5), and 10 mM dithiothreitol, at 37°C for 3 h. The proteins were electrophoresed by SDS-PAGE and the δ -endotoxin fraction of *E. coli* protein was quantified by laser densitometry⁸. Insect bioassays were done by coating the proteins on to BSFB artificial diet. A modified semisynthetic diet was used for bioassays⁹. The diet consisted of 120 g

black gram flour, 40 g wheat germ, 1.5 g ascorbic acid, 3 g sorbic acid, 3 g methyl-*p*-hydroxybenzoate, 10 g Wesson salt mixture¹⁰, 3 g aureomycin, 10 ml vitamin mixture¹¹, 5 ml formaldehyde, 32 g yeast and 16 g agar in one litre of distilled water. The mixture was poured into 24 well tissue culture plates (ICN, USA). The diet was allowed to solidify and different Bt toxins were coated on the diet. Six concentrations of each protein were tested. Two second instar larvae were released into each well of the tissue culture plate. The plates were incubated at 25°C under light : dark regime (12 : 12 h). Larval mortality was recorded after every 24 h and final mortality count was taken on the fourth day. Thirty larvae were tested for each protein and the experiment was repeated three times. The results of the bioassays were evaluated by probit analysis¹².

Table 1 shows the relative efficacy of Bt toxins against second instar larvae of BSFB. The protein Cry 2 Aa, was the most potent toxin tested followed by Cry