DIESEL AUTOMOBILE EXHAUST AND LUNG SURFACTANT ACTIVITY : AN EXPERIMENTAL STUDY IN ALBINO RATS

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Summary: It has been observed that the lung surfactant activity decreased after exposure of the animals to diesel automobile exhaust. This decreased lung surfactant activity could be an important factor in the pathogenesis of pulmonary oedema and collapse seen in our experiments.

Key words: diesel automobile exhaust, surfactant activity, pulmonary oedema, surface tension

INTRODUCTION

Diesel automobile exhaust is a serious health hazard as it is emitted in close proximity to the breathing zone of the people (14). The main constituents of diesel exhaust are carbon monoxide, oxides of nitrogen, aldehydes, sulphur-dioxide, acrolein and hydrocarbons (9). The observations of Ash *et al.* (1) on the workers of diesel locomotive railway tunnel indicated that the incidence of respiratory disorders were more common in these workers. Studies on diesel smoke by other workers (10, 11) revealed pulmonary pathology like bronchitis, bronchiolitis with anthracosis, focal areas of collapse, focal compensatory emphysema, pneumonitis and pulmonary oedema. To understand the mechanism of action of different pollutants studies have been conducted with some of the components of diesel exhaust like nitrogen dioxide (3,4), carbon monoxide (13) and sulfur dioxide (2,5,8). But there is paucity of literature on the effect of these gases or of diesel smoke as such on lung surfactant activity which is well known to play an important role in maintaining the patency of the alveoli. Hence to characterize more closely the mechanism of action it was planned to study the effect of diesel automobile exhaust on the lung surfactant activity.

MATERIAL AND METHODS

Albino rats of either sex weighing 120 - 140 *g* were used in the present study. The rats were randomly divided into 2 groups, control and experimental, each comprising

of 10 animals. All the animals were fed a standard diet and water was provided *ad libitum*. The experimental group of animals were exposed to diesel automobile exhaust daily for 20 min for a period of 90 days. The exposure chamber in which the rats were exposed to diesel automobile exhaust measured 45 x 30 x 32 cms and was made up of a galvanized sheet with provisions for an inlet pipe and an outlet shutter with holes which also acted as a ventilator and maintained the atmosphere dynamic. It also had a provision of glass windows on either side for observation purposes. Diesel-exhaust was collected from the exhaust pipe of a diesel bus and collected in a Doughlas bag of 50 litres capacity. This was then connected to the exposure chamber through a respirator for a regulated supply of smoke into the chamber and fresh air was also pumped simultaneously to avoid asphyxia and to maintain the atmosphere dynamic. The animals were exposed to the mixture (2.5 litres of smoke and 2.5 litres of air per minute) daily for 20 minutes. The control group of animals were kept in the animal house but were not exposed to the diesel exhaust. The rats of both the groups were sacrificed at the end of 90 days and their lungs were dissected out. A portion of the lung tissue was used for measuring the lung surfactant activity and the rest was preserved in 10% formalin for histological processing. For the measurement of surfactant activity 0.5 g of lung tissue was minced in normal saline as reported earlier and its maximum surface tension (T_{max}) and minimum surface tension (T_{min}) were determined using a modified Wilhelmy type of balance (7). The surfactant activity indicated by extract stability index (E.S.I.) was calculated by the following formula.

$$E.S.I. = \frac{2\ (T_{max} - T_{min})}{T_{max} + T_{min}}$$

After exposure of the tissue, sections were at 7μ and stained with H & E for histopathological assessment.

RESULTS

(1) *Surfactant activity :* Surface tension (T_{max} and T_{min}) of the control and experimental group is given in Table I from which it is clear that in the experimental group maximum surface tension (T_{max}) and minimum surface tension (T_{min}) of the lung got increased significantly as compared to control group ($P<0.001$) thereby indicating a definite decrease in lung surfactant activity.

(2) *Histopathological studies :* The details are summarised in Table II.

(a) *Control group :* On macroscopic and microscopic examination the lung hardly showed any pathological changes and were found to be normal.

TABLE I : Effect of diesel automobile-exhaust on lung surfactant activity of rats.

Sl. No	Group	Surface tension (Dynes/cm) T_{max}	Surface tension (Dynes/cm) T_{min}	Extract stability index
1.	Control (10)	42.25 ±1.60	19.79 ±0.84	0.71 ±0.05
2.	Experimental (10)	48.94@ ±1.93	29.56@ ±0.94	0.49@ ±0.03

@P < 0.001
Values are mean ± SD
Figures in parenthesis refer to number of animals.

TABLE II : Histopathological findings in the lungs of diesel autoexhaust exposed albino rats (n = 10).

Macroscopic		
	1. Darkbrown colour	(10)
	2. Visible carbon particles	(10)
	3. Congestion	(7)
Microscopic		
	1. Bronchitis and bronchiolitis	(9)
	2. Interstitial pneumonitis	(8)
	3. Focal interstitial oedema and collapse	(8)
	4. Focal pulmonary haemorrhages	(6)
	5. Focal compensatory emphysema	(8)

Figures between parentheses refer to number of animals, in which the finding was positive.
n – denotes total number of animals.

(b) *Experimental group :* In this on macroscopic examination the colour of the lung was found to be brownish black and lungs appeared congested. On microscopic examination the most common findings were inflammatory changes leading to bronchitis, bronchiolitis and focal areas of collapse (Fig. 1). Further one could see compensatory emphysema (Fig. 2) and interstitial pulmonary oedema (Fig. 3) in most of the animals. The details of histopathological assessment as seen in different animals of the experimental group are summarized in Table II.

DISCUSSION

Diesel automobile exhaust is an important source of air pollution and acts as an irritant in most instances affecting the respiratory tract (15). Our findings of bronchitis,

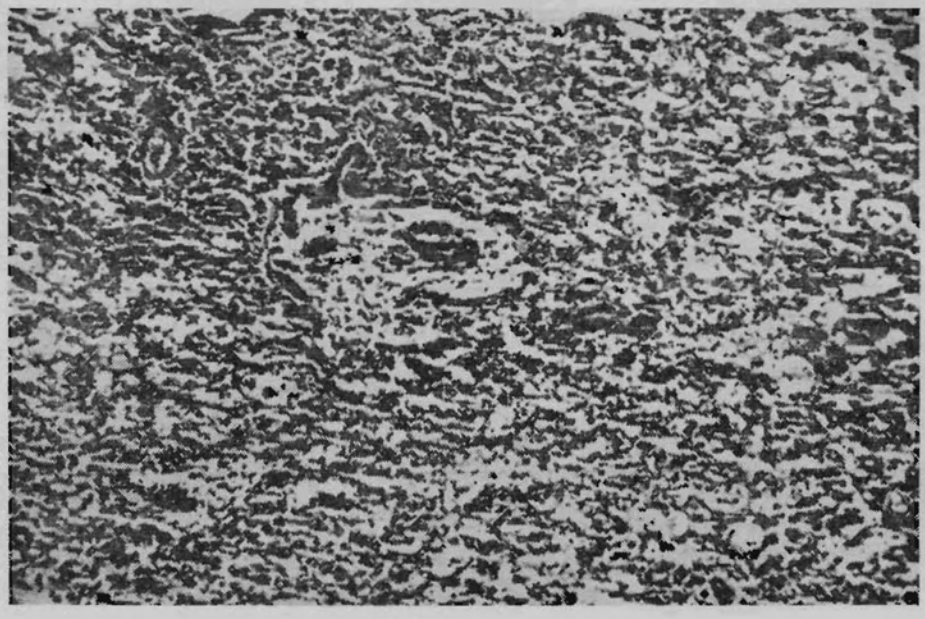

Fig. 1 : T.S. of rat lung (10 x 10 H & E) showing bronchiolitis and wide areas of collapse.

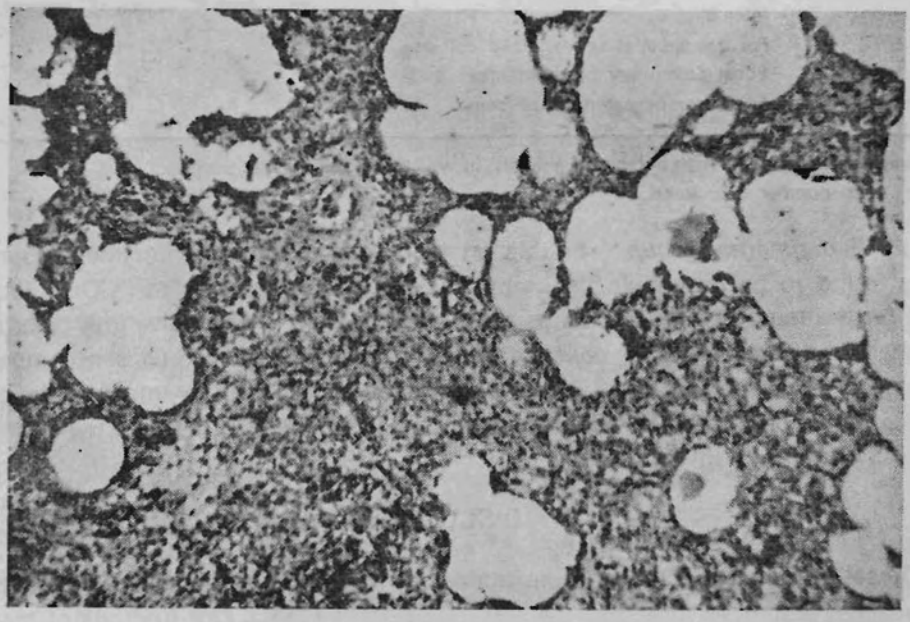

Fig. 2 : T.S. of rat lung (10 x 10 H & E) showing compensatory emphysema.

Fig. 3 : T.S. of rat lung (10 x 10 H & E) showing focal interstitial pulmonary oedema.

bronchiolitis, focal areas of collapse, compensatory emphysema and pulmonary oedema are similar to the earlier observaions of other workers (10,11) on dogs.

Battigelli (2) and Martin and Willoughby (8) found sulphur dioxide to have marked damaging effect on bronchi producing erosion of the lining epithelium of submucosa. On the other hand Fairchild (3) reported that Nitrogen dioxide may operate by a complex sequence of blockage of the sulphydryl enzymes associated with neurohumoral effects leading to permeability changes, release of histamine, bradykinin and slow reacting substance (SRS) which lead to congestion, hemorrhage, emphysema and eventually to oedema of lungs.

From the above reports it is clear that the triggering factor for the pulmonary pathology is the inflammatory reaction causing bronchitis and bronchiolitis which leads to airway obstruction and finally to a series of changes leading to compensatory emphysema. However, as regards the pathogenesis of collapse and pulmonary oedema it is felt that some other factors might be playing a greater role. On the basis of the results of our experiments it is quite clear that the diesel smoke exposure increased the T_{max} and T_{min} of the lung significantly ($P<0.001$, Table I). In other words, this means that the lung surfactant activity decreased. As the lung surfactant activity is very important for the maintenance of the patency of alveoli, a decrease in it facilitates the collapse of the alveoli and this explains our observation of pulmonary collapse.

Further it would be appreciated that since only 0.5μ tissue seperates the capillary blood from the air in the lung the problem of keeping the alveoli free from fluid is critical. The hydrostatic pressure and surface tension tend to drive the fluid from the capillary into the alveoli (6). These forces are opposed mostly by the colloid osmotic pressure of the blood. Under these conditions it is very evident that even a small increase in the alveolar surface tension might disrupt the critical balance and promote accumulation of fluid in the lung. As the surface tension is increased after the exposure of animals to diesel exhaust smoke, it is postulated that this might be an important contributing factor in the pathogenesis of pulmonary oedema observed in our experiments.

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