Chapter 2

Review of Literature
The extrudates from industries cause serious concern in the people since it causes certain pathophysiological changes in them, especially in respiration. The intensity of these changes varies with the type of causative agents extruded from these industries. It is in this context the etiology of the respiratory diseases is to be considered, which mainly depends on these industrial pollutants. Kargel and Basel (1971) reported that the ventilatory functions of exposed workers are lower in the areas with higher levels of pollution. According to Ozhiganova and Ivanova (1990), the pulmonary diseases occur in workers exposed to industrial aerosols of complex composition including toxic substances and allergens along with fibrinogenic dust.

Lung function tests alone are not diagnostic to any particular disease but forms a part of diagnostic tool and provide guidance for prognosis in cases of asbestosis, chromium-beryllium disease and chronic obstructive bronchitis etc. (Ganguli, 1992). Chronic exposure of workers to harmful substances in the industrial environments were subjects more frequently to respiratory disorders (Lewin-Kowalik et al., 1994).

Baser et al. (1985) reported the effect of polyvinyl chloride (PVC) on PVC fabrication workers with duration and intensity of respiratory functions. Exposure to PVC, methyl ketone and other chemicals on respiration was reported by Oleru and Onyekwere (1992). They further suggested that the persons working in these plants are associated with obstructive air flow limitation. Sodium hydroxide mist
induces irritation and burns to the respiratory system leading to severe obstructive airway disease (Rubin et al., 1992). An excessive spastic reaction to respiratory tract has been caused by SO₂ and NO₃ leading to 20% decrease in FEV₁ and FEF₂⁵-⁷⁵% (Kolarzyk et al., 1990). But short term exposure to low concentrations of these chemicals fails to elicit any ill effect in respiratory functions (Huang et al., 1991).

Skalpe (1964), Enarson (1984) and Henneberger et al. (1989) have reported certain abnormalities in respiration due to exposure to certain gases viz., SO₂, H₂S, chlorine, chlorine dioxide and other organic sulphur containing gases and wood dust in pulp mill workers. Ferris et al. (1976, 1979) and Chan-Yeung et al. (1980) failed to observe any clinical observations relating to respiratory function impairment or increased levels of respiratory symptoms in pulp and paper mill workers. Kennedy et al. (1991) observed air flow obstruction in non-smokers as a result of chlorine dioxide, "gassing" incidents among pulp mill workers. Calvert et al. (1991) found no association between exposure to TCDD in the manufacture of 2,4,5-trichlorophenol and FEV₁, FVC or FEV₁/FVC%. It was also reported that the chemical industrial workers in a natural gas extraction industry were found to have their flow rates paradoxically worse even at low lung volume (Tessier et al., 1991). Workers in the same department showed restrictive and obstructive disturbances in ventilation revealing the adverse effect of smoking for a long period of time (Lewin-Kowalik et al., 1994). Caprolactum plant workers in a fertiliser industry having chronic exposure to a contaminated environment with chemicals like benzene, cyclohexane, NO, NO₂ and ammonia were reported not to have lost their lung function (Patel, 1990).

The impairment of respiratory function caused by industrial toxins can be complicated by the additional or subsequent exposure to other injurious agents. Cigarette smoking affects both the structural and functional ability of lungs (Sherman, 1992) and was considered to be the main cause of chronic air flow limitation (Bande et al., 1980; Lange et al., 1990). The influence of age, smoking
habits and the industrial exposure together makes the pulmonary function more miserable resulting in greater decline in FEV\textsubscript{1} (Phoon et al., 1984; Heierlic, 1984) while Nemery et al. (1992) were of the opinion that the significant difference in FVC and FEV\textsubscript{1} were not due to smoking habits.

Kamat et al. (1981), Larson et al. (1981), Beck et al. (1984), Christiani et al. (1986), Kawamoto et al. (1987), Parikh et al. (1990), Woldeyohannes et al. (1991), Hayes et al. (1994), Zuskin et al. (1994) and Abebe and Seboxa (1995) have reported a reduction in various respiratory functions in various stages with or without “byssinosis” with cotton dust while Jennison and Jacobs (1994) fails to note any relation between cotton dust and respiratory functions.

A reduction in lung function was reported in cigarette smokers than non-smokers (Buist and Ducic, 1979; Hammond et al., 1979; Beck et al., 1984; Sherman, 1992). This become all the more chronic or serious when cigarette smoking is associated with cotton dust or similar particles (Beck et al., 1984; Glindmeyer et al., 1991; Fishwick, et al., 1996). Kremer (1994) failed to get any significant association between lower levels of lung function and industrial exposure to airway irritants among synthetic fibre plant workers. Manganese dioxide dust has been reported to produce abnormal respiratory symptoms and ventilatory functions (Roel et al., 1992).

Graham et al. (1994) observed no significant change in FVC or FEV\textsubscript{1} in workers exposed to different levels of granite dust suggesting that they do not accelerate loss of pulmonary function, while an acute drop in FEV\textsubscript{1} with increasing exposure levels of aerosols of various cutting oils and coolant fluid was found among machine operators (Kennedy et al., 1989). Deng et al. (1991) found certain abnormalities in pulmonary function and respiratory symptoms associated with workers making sinistered magnets and suggested that this may be due to the exposure of respirable crystalline silica and asbestos. Exposure to dust or silica, wool and carbon may bring about significant impairment in VC and FEF\textsubscript{25-75%}, which was aggravated by the addition of smoking (Rao et al., 1992). Loss of lung
function with decrease in FEV$_1$/FVC%, MMEF, FEF$_{50}\%$, and FEF$_{75}\%$, associated with exposure to silica dust has been reported among smokers and non-smokers (Jones et al., 1975; Prowse et al., 1989; Hnizdo, 1992; Liou et al., 1996). Workers in ceramic and gold mining were at risk of developing chronic obstructive lung disease by the exposure to silica dust which was superimposed by the smoking habits (Bagatin et al., 1991; Hnizdo, 1992).

Smokers as well as non-smokers were found to have decrease in lung function in relation to increasing carbon black dust acid and its associated gaseous contaminants exposure (Gardiner et al., 1993), lowering of FEV$_1$, PEF, FEF$_{75}\%$, FEF$_{25}\%$, in stainless steel workers (Bogadi-Sare, 1990) and a significant decrease in VC, FEV$_1$/FVC% among solders in the electronic industry (Gupta et al., 1991).

Exposure to welding fumes causes risk of chronic bronchitis and impairment of pulmonary function, i.e., decrease in FVC, FEV$_1$, PEF and MMEF. This was aggravated by smoking (Ozdemir et al., 1995). Workers in rubber industry were exposed to various combinations of airborne contaminants. This may bring about decrease in all lung functions suggestive of the fact that exposure to noxious agents in rubber industry may be responsible for the development of chronic respiratory distress and decrease in lung functions (Governa et al., 1987; Zuskin et al., 1994). Dutton et al. (1993) suggested that elevated levels of phosphoric acid, phosphorus pentoxide, fluoride and coal tar pitch volatiles present in an industrial refinery contribute only weakly and inconsistently in lowering respiratory function.

Impairment of lung functions, i.e., lung volumes and flow rates associated with certain pathological changes in respiratory organs have been reported among workers with asbestos (Cohen et al., 1984; Oleru, 1984; Sue et al., 1985; Dujic et al., 1991; Hilt et al., 1992), with asbestos, cigarette smoke and air pollution (Wang and Lu, 1985), with mixed dust of asbestos and cement (Siracusa, 1984; Mukhtar and Mohan Rao, 1996), with iron (Fischbein et al., 1991) and with asbestos fibre (Osim et al., 1992). The respiratory effect of asbestos was further
super imposed by smoking (Cohen et al., 1984; Weiss, 1984; Sue et al., 1985; Wang and Lu, 1985; Landrine and Kazemi, 1991). An increase in mid expiratory flow rates in some asbestos exposed non-smoking workers was suggested as one of the earlier functional signs indicative of future development of paranchymal asbestosis (Dujic et al., 1991). A reduction in FVC and FEV$_1$, but a normal FEV$_{15}$, among asbestos workers were also reported by Osim et al. (1992).

Environmental analysis of some confinement buildings indicate CO$_2$, CO, NH$_3$, H$_2$S and aerosolised particles may usually exceed the threshold value limits or short term exposure limit values for these substances (Donham et al., 1977; Donham, 1978; Donham and Gustafson, 1982). Environmental exposure along with smoking has increased effect on decreasing lung function i.e., flow rates among swine confinement workers (Donham et al., 1984; Zuskin et al., 1992). Donham et al. (1984) reported that environmental exposure to CO$_2$ and H$_2$S causes irritation in the respiratory tract along with decrease in flow rates.

Workers with grains (Fonn et al., 1993) or animal feeds (Smid et al., 1994) industry are not only exposed to grains or feed dust but also several other materials like silica, fungi, rodent hairs, pesticide, etc. and many of which having the potential to induce respiratory diseases (Cotton and Dosman, 1978) or acute and chronic respiratory abnormalities (Huy et al., 1991).

Exposure to pollutants in aluminium industry may leads to the development of asthmatic symptoms as well as reduction in respiratory functions (Kongerud et al., 1990). Prevalence of respiratory symptoms is independent of the degree of dust exposure (Soyeth and Kongerud, 1992). Significant increase in FVC and maximum flow rates at 50% and 25% of FVC was observed among workers of glass blowing industry while workers in pickling industry had a reduction in respiratory functions with chronic respiratory symptoms (Zuskin et al., 1993).
Workers in paper industry were mildly exposed to high concentration of wood dust, chlorine, SO₂ and subsequently paper dust usually characterised as 'nuisance dust' (Ericsson et al., 1988). Impairment of lung functions has been reported with high exposure to paper dust for a longer period (Ericsson et al., 1988; Jarvholm et al., 1988; Dahlqvist, 1992). Workers in a production plant with heavy exposure to paper dust for a long period of time will have high incidence of impaired respiratory function compared with low or short time exposure (Ericsson et al., 1988, Jarvholm et al., 1988). The presence of low FEV₁, MMEF, MEF₅₀, and MEF₂₅₋₇₅ among workers of paper mill with a positive immediate intradermal reaction is suggestive of an obstructive airway reaction with immunological mechanism (Heederik et al., 1987).

Workers in wood industry had higher prevalence of respiratory impairment (Shamssain, 1992). Symptoms of work-related asthma in red cedar workers are more common after 10 years of exposure and FVC and FEV₁ are lower with higher wood dust exposure. But FEF₂₅₋₇₅ and FEV₁/FVC% were unrelated to dust concentration (Vedal et al., 1986). Respiratory consequences of exposure to wood dust and formaldehyde studied by Herbert et al. (1994) noted a significant difference in FEV₁/FVC%.

Based on the measurement of chemical pollutants and concentration of dust in tobacco industry a clear detrimental effect on lung function was reported (Popovic et al., 1992). However, the duration of exposure to tobacco dust fails to elicit any significant effect on ventilatory functions and no significant difference in lung function between smokers and non-smokers was evident (Mukhtar et al., 1991).