CHAPTER - IV

DISCUSSION AND CONCLUDING REMARKS

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Poverty, unemployment and lack of development are the critical contributing factors to environmental hazards, that affect the health of millions of people world-wide. Industrialization is one of the important avenue that helps to reduce poverty and unemployment. It brings economical development of the country. So in the present era, the development of a country has become synonymous with its industrialization. During the earlier phases of industrialization, the environmental impact of industrial effluents did not pose much problem due to limited pace of development. Over the years, with increasing rate of industrial development and population explosion resulted in ambient environmental degradation and threats to human health as well.

Workplace hazards are all too common everywhere. The process of industrialization involves various occupational hazards, such as accidents and occupational diseases, which has detrimental effects on worker's health, leading to partial or total disability. This results into loss of man power, reduced productivity and ultimately hampers the economic development. Occupational health hazards in different industries have been a subject of research. There is great need of awareness, research and action to mitigate the ill effects of environmental degradation

due to industrialization. Hence the developing nations have now focussed their attention towards making the workplace safer to work. Various statutory provisions have been made in labour laws of nations of the world. Along with industrial progress various research works are carried out to provide ways and means of making the workplace safe, and free from occupational health hazards. Economic development can be compatible with obtaining clean and safe workplace environment.

The detrimental impact of industrialization is manifested to a higher degree in a developing country like India. In the present scenario of economic liberalisation, rapid industrialization is taking place in India. In India before the declaration of Environmental protection Act, 1986, industries were located mostly on the basis of availability of raw material, access to the market, transportation facility and other technoeconomic considerations, without paying adequate attention towards the environmental aspects. Recently environmental abuse in India has increased rapidly with almost 80% of industrial production being concentrated in few cities.

Among many industries, the textile industry especially in powerloom sector the workers are constantly exposed to adverse working environment, where there is prevalence of cotton dust,

noise, humidity, heat, vibration, inadequate ventilation and poor illumination. These occupational stresses make the working day not only uncomfortable but pose a threat to the health and safety of the workers.

It is significant to note that the textile industry is one of the biggest and oldest industry in India. Most of the research work on occupational health hazards in textile industry is carried out in foreign countries, whereas very little work is carried out in India. The powerloom sector offers ample research opportunities. Most of the research is carried out in the spinning unit of the powerloom sector.

The present work is carried out in powerloom sector at Ichalkaranji, where the business of weaving cotton textile yarn began in 1905. The 80 per cent powerlooms and handlooms are working in Ichalkaranji area in Kolhapur district (Hupare, 1979). Although Ichalkaranji town is popularly known as 'Manchester of Maharashtra', very little information is available on worker physiology. Whatever research work carried out, is directly on worker's physiological responses in relation with stresses in powerloom environment. Whether stresses can induce any histologic and biochemical alterations in workers is yet a subject to be fully explored. So to evaluate histologic alterations and

changes in total tissue protein content of powerloom workers, the present work has been carried out by developing animal model rat and exposing it to the powerloom environment at Ichalkaranji.

Another significant aspect of powerloom environment is the presence of stresses in workplace environment. Stress is a state of disharmony and/ or threatened homeostasis. Stress conditions vary and are found to range from physical to psychological, from mild to severe and from chronic to acute in powerloom environment particularly with reference to temperature, noise, humidity, vibration, light, dust and work load (Dubal *et al.*, 2001).

Stress research in the laboratory animals has assumed the significant role in the physiological sciences over the past decade, due to the fact that stressful stimuli influences the onset, and progression of a number of disorders in human beings leading to discomfort, headache, acidity, ulceration, hypertension and even stroke. There are large inter-individual differences in the temporal pattern of physiological and endocrinological stress responses. Experimental results indicate that "rapid adrenaline decreasers" tend to be physiologically better balanced and more efficient in achievement situations than "slow adrenaline decreasers" (Johnsson and Frankenhaeuser, (1973). An equally

important finding is that the time for unwinding varies predictably with the individual's state of general well-beings. Thus, in a group of industrial workers, the proportion of "rapid adrenaline decrease" was significantly higher after than before a vacation period, which had improved the workers physical and psychological well-being (Johansson, 1976). In the powerloom sector during powerloom operations the workers are constantly exposed to various occupational health hazards, and stresses, which are mainly arise due to heat, humidity, poor ventilation, cotton dust, noise vibrations and inadequate illumination. The powerloom generates heat, due to which workers are under high thermal stress. The thermal stress thus produced may manifests itself in the form of psychological as well as physiological fatigue. In powerloom sector, during weaving process, relative humidity maintained purposely. The heat load caused by such a microclimate, coupled with the climatic heat load and heavy work load, produce severe adverse effects on the health of the workers. These elevations in the temperature and humidity may cause the various disorders, like excessive sweating, heat cramps, discomfort and affect thermoregulatory mechanism. The physiological responses of various categories of workers in the cotton textile industry were significantly affected in relation to the thermal stress and work load (Sen *et al.*, 1970).

The weaving sheds of the powerloom sector were poorly ventilated, and the air inhaled by a powerloom worker was not always fresh. Poor ventilation prevents the air to circulate freely within the shed. This may causes hypoxia. The air was not only hot and damp but was also contaminated by large quantities of dirt and dust. The cotton dust produced during weaving process, when inhaled may often causes respiratory impairment in the powerloom workers. Cotton dust is considered to be responsible for the chronic respiratory disease known as Byssinosis, which is characterized by chest tightness. The expiratory stresses among the workers engaged in cotton spinning mills have been investigated by many research workers (Berry *et al.*, 1973; Parikh *et al.*, 1990 and Sawant and Kore, 1994). While the occurrence of Byssinosis among the powerloom workers has also been reported by some (Thiruvengadam *et al.*, 1970).

There are two separate research lines explaining the causative agent(s) for the subjective symptoms, that result from cotton dust inhalation. According to one, the causative agent(s) resides in the plant derived materials, mainly the chemical substances present in the cotton plant like histamine etc. (Bhatt *et al.*, 1988 and Rastogi *et al.*, 1986). While according to the other line of research, the causative agent(s) resides in the

microbial contamination of cotton fiber, particularly the bacterial endotoxins (Rylander, 1990).

Noise and vibration, are also the stress factors in the powerloom of which the workers are not aware. The noise produced in the powerlooms at Ichalkaranji ranged from 102 dB to 106 dB (Dubal, 1995) for which the maximum allowable exposure period under the Factories Act (1948) is upto 1 hour. But in the powerlooms workers are exposed to a much longer duration, which may cause various physiological and psychological disorders. The high level of noise in the powerlooms causes detrimental effect on the hearing, which manifest in the form of temporary and permanent losses in hearing sensitivity, physical and psychological disorders, hindrance to the inter-worker verbal communication, an increase in sweating and heart rate, hypertension and a most of other complications, including the cordio-vascular abnormalities.

There was very poor illumination levels ranging from 48 to 290 Lux (Dubal, 1995) in the powerloom sector at Ichalkaranji. According to the Factories Act (1987), the minimum permissible range of illumination to be 1,000 to 2,000 lux. Thus the light intensity in the powerloom was much below this. This may result into poor co-ordination of eyes and hands with brain and hence causes visual fatigue.

Laboratory animals have been used for studies in pharmacology, toxicology and psychology since long ago. It has been recently reported that in genetic set up there is seventy per cent similarity in rat and man. The hypothesis about disorders and spreading diseases in human body cannot be tasted directly, hence the various species of animals are required for the research in pathophysiological processes and evaluation. From the various laboratory animals, rat has been used extensively especially among the rodents group. It has been often stated that the rat is ideal animal model for toxicological studies is pneumoconiosis, bronchopneumonia and other related changes in the lung. The experimental animal model rat (*R. norvegicus*) used in the present study exposed to such a hazardous textile environment showed significant alterations in their behaviour. The experimental rats initially exhibit bizzare reaction and aggressive behaviour, probably due to the noise stress in powerloom. Earlier studies by Davis (1989) has shown that continuous exposure of noise results in a state of behavioural sensitization that is typically considered to be a non-associative form of fear or anxiety. Shankar *et al.*, (1999) also reported that noise is a stress or which has a significant effect on behaviour of an animal exposed to it. The rats smelled air continuously may be due to poor ventilation, and hot humid environment in the powerloom sector, which might

cause hypoxia. Previous reports indicate that, in hot humid environment oxygen transport mechanism suffers significantly due to fall in stroke volume (William *et al.*, 1962). Within first 15 minutes the experimental rats urinated and fecated might be due to fear caused by sound and vibration stress. After 15-20 minutes of exposure animals showed decreased locomotary activity and limp tail. This may be due to initiation of histological changes in rat organs due to stresses. Gehlot *et al.*, (1997) found that sound above 80 dB produces histological changes in rat organs. Similar observations were made by Flamenbaum (1974) in the maintenance phase of UN administration. The progression of cellular degeneration makes the animal clinically unhealthy or morbid and animal becomes inactive and responsiveless The experimental animal did not consume food and water. This highly significant suppression of food intake probably due to release of CRF. It is strongly supported by many research workers (Britton *et al.*, 1980; Coover *et al.*, 1971; Iton *et al.*, 1980; Krieger, 1974; Levine *et al.*, 1983 and Morley *et al.*, 1982).

During 2^{nd} and 3^{rd} exposure, the experimental rat did not show the behavioural changes as observed during first exposure. They behave normally, may be due to acclimatization to the powerloom environment. Exposure of experimental animal (rat)

the powerloom environment shows significant morphological, histologic and some biochemical alterations in the adrenal gland, kidney, heart and gastro-intestinal tract.

There was increase in the size and weight of the adrenal gland from Sets S_1 to S_3. This increase may be due to adrenal hyperplasia and hypertrophy. Stress induces adreno-medullary response in man (Workman *et al.*, 1984). Adrenaline in turn stimulates β_2 – receptors on the pituitary gland causing greater release of ACTH (Reisine *et al.*, 1983). ACTH can stimulate the adrenal medulla as well as cortex (Critchley, *et al.*, 1982; Ungar and Phillips, 1983). Various physical stresses cause greater release of adrenaline and greater increase in weight of adrenal gland. During stress there is uniform arousal of both the fight-flight sympatho-adrenal and the pituitary-adrenal-cortical systems. These two systems acting together participate in the stress response of an organism. The observed increase in the adrenal weight could be attributed to the well documented hypertrophy and hyperplasia of the adrenal cortical cells under the influence of stress (Kvetnansky *et al.*, 1995).

The experimental data of present study indicated that industrial stresses in powerloom sector induce hyperplasia and hypertrophy of adrenal cortex. These alterations could be due to

excessive secretion of ACTH. Sound stress induces hypothalamo-pituitary axis and sympathetic system stimulation, resulting in liberation of catecholamines and adrenocorticoides (Gehlot *et al.*, 1997).

Necrosis of cortical cells might be due to hyperactivity of these cells in order to secrete more and more cortisol to adapt with stress situation. Stress leading to a greatly increased requirement of adrenocortical hormones (Labhart, 1974).

Lesion observed in zonafasciculata region might be due to overactivity of this region and endotoxin(s) associated with cotton dust in powerloom environment. Bardakhch'yan *et al.*, (1986) reported ultrastructural lesions in adrenal gland due to injection of bacterial endotoxin (LD 80) which exceeded the adaptation capacity of adrenal cells leading to the cell death.

With increased exposure period there was increased damage of cortical cells. Medullary cells hypetrophid, in order to secrete more and more epinephrine and norepinephrine. Localized hemorrhage might be due to increased blood pressure. Epinephrine increases the pulse rate and cardiac output and thus the systolic blood pressure (Labhart, 1974). Granulated cytoplasm might be the indication of lipoid hyperplasia. Adrenal insufficiency causes lipoid hyperplasia (Siebenmann, 1957).

Although no work has been done related to the mechanism(s) involved in the histophysiological alterations in the animals exposed to various stresses. Present observations and the data of other investigators substantiate and laid support to observations made in present study. Thus the experimental data of the model in the present preliminary investigation perhaps, may provide support to the fact that industrial stresses induce histophysiological alterations leading to changes in the physiological responses of the concerned organ systems. There was increase in the total protein content in adrenal gland, might be due to increased secretion of ACTH; because ACTH promotes protein synthesis in adrenal cortex. ACTH induces a rapid increase of ribonucleic acid (RNA) concentration in adrenal tissue (Fiala *et al.*, 1956). All the cells of the adrenal cortex were able to synthesize DNA. Under stress not only production, but also a rapid utilization of DNA occurred (Truupyl'd, 1970). In Set S_3 the experimental rats S_3P_2 and S_3P_3 exhibit decreased amount of total protein as compared to that of control. This might be due to increased level of cortisol. With increase in the period of exposure to the stress, the plasma cortisol level may increases. Cortisol leads to a negative nitrogen balance as it promotes protein degradation (Labhort, 1974).

There was increase in the weight of the kidney of experimental rats, in Set S_1 as compared to that of control; probably due to swelling of renal tubules, accumulation of edematous fluid and increase in the size of the kidney.

There was swelling and flattening of tubular epithelium in Set S_1 could be due to increased load of reabsorption on tubular epithelium, under the influence of aldosteron. The various stresses might be causing hyper-functioning of adrenal cortex (Kvetnansky, Richard 1973, Kirillov *et al.*, 1971).

In Set S_1 accumulation of edematous fluid might be due to increased salt and water retention, under the control of ADH and aldosteron. In heat stress there is increased secretion of ADH (Marya *et al.*, 1988).

In Set S_2 necrosis of renal tubules and blockage of most of the renal tubules by necrotic mass was observed. It might be due to renal ischemia. In severe circulatory shock there is every possibility that heart simply fails to pump sufficient amount of blood to supply adequate nutrition and hence renal blood flow is likely to suffer because of strong sympathetic constriction of renal vessels. So lack of adequate nutrition often destroys many tubular epithelial cells (Guyton, 1986).

Fibroelastic hyperplasia was most significant in Set S_3. It may be due to hyperfunctioning of the adrenal itself. Presumably all above effects are due to activation of hypo-thalamo-sympathetic adrenal axis and resultant release of catecholamine from adrenal medulla due to the stresses existing in the powerloom environment including toxic stress of endotoxin(s), associated with cotton fibers in powerloom unit.

Kidney of experimental rat showed decreased in total protein. This may be due to necrosis of the renal tubules. Under the stress or endotoxin(s) influence, many kidney tubules undergo acute necrosis. The tubules loose their integrity and many cells of the tubules get damaged or severely injured and eventually became dead. Cytoplasmic debris accumulates in the lumen of the tubules and proteins from the kidney pass down to the urine. Thus, by the death of tubular cells there may be fall in the kidney protein level. Franklin (1963), Goldberg and Rabinowitz (1972), Journey and Goldstein (1961), Reich *et al.*, (1961) and Smuckler and Benditt (1963) have reported that actinomycin poisoning inhibits m-RNA synthesis and incorporation of amino acids in the protein resulting in the retardation of protein synthetic machinery. In the present investigation the decrease in the kidney protein content may possibly be partly related to the inhibition of protein synthetic machinery but it needs further investigation to

find out whether stress or endotoxin(s) in powerloom can induce similar inhibition of m-RNA synthesis.

Significant increase in the weight of heart might be due to hypertrophy of cardiac muscles. Stresses induce increase in the work load on the heart. Stresses also cause excess secretion of epinephrine and nor-epinephrine from adrenal gland (Axelrod and Reisine, 1984). These hormones acting on the heart and exert positive influence on the force of concentration which gradually might have caused the cardiac hypertrophy (Nagaraja and Jeganathan, 1999). With increase in exposure period there was no further significant increase in the weight of the heart. This type of response for the prolonged stress explains the partial adaptation of the animals to the stressful stimuli.

The size of heart increased in experimental rat could be due to cardiac hypertrophy, caused in response to the stress factors. The colour of heart was changed from fresh red to blackish red, might be due to hypoxia. Poor ventilation, humidity, heat these stresses affect oxygen transport mechanism.

Initially there was elongation of myocardial muscles and vacuolation. This might be due to increased workload in heart due to stresses like noise, hot humid environment in powerloom. This finding is in good agreement with various previous

investigations. Industrial noise causes significant elevation in diastolic blood pressure (Andren *et al.*, 1982). Hot humid environment causes increased heart rate (Sen *et al.*, 1981). Initially, there was hypertrophy of endocardium, might be due to the epinephrine and norepinephrine from adrenal gland, in response to the stress (Axelrol *et al.*, 1984), because these hormones acting on the heart and exert positive influence which gradually might have caused cardiac hypertrophy.

With increased exposure to the stress degenerative changes were observed in myocardium, as well as in endocardium. It might be due to hypoxia and increased amount of adrenaline caused by hyperfunctioning of adrenal medulla. These observations supported by Adonkin *et al.*, (1969). They found more numerous and more severe myocardial necrosis after combined administration of adrenaline and pituitrin. Similar observations were also made by Pokk (1967); in rabbits exposed to increased stress exhibited more numerous and more extensive myocardial destructive changes after receiving adrenaline. Due to hypoxia there is increased activity of lactate dehydrogenase (LDH) and decreased glycogen content and RNA content (Tsagareli, 1970). Administration of adrenaline in rats caused injurious action on contracting apparatus of the myocardium (Semenova *et al.*, 1967). Heat also causes a direct damage to the

myocardium as reported by Knochel *et al.*, (1961). Hemorrhage was also observed in endocardium, which may be acute manifestation of stress. It might also be due to increased heart rate, blood pressure caused due to noise stress. Noise stress causes increased sympathetic activity thereby leading to significant increase in blood pressure, heart rate (Saha *et al.*, 1996). In Set S_3 there was hyperplasia of epicardium, elongation of muscle cells of myocardium might be due to compensatory reaction to the stress. When heart muscles damaged the natural reparative processes of the body begin immediately to help restore normal cardiac function (Guyton, 1986).

There was significant decrease in the total protein content in the heart of experimental rat, might be due to the damage of cardiac muscles and due to stresses in powerloom. Stressful environment increases workload on the heart, which results into damage of heart muscles. In addition to cardiac damage, hyper-activity of adrenal cortex in response to the stress may also responsible for decrease in amount of protein in heart. Hyper active adrenal cortex may leads to more secretion of adreno-cortical hormones like glucocorticoids, cortisol, cortisone etc. Glucocorticoids by adrenal cortex decreases the quantity of protein in most of the tissues (Guyton, 1984). Cortisol causes negative nitrogen balance and degradation and mobilization of

proteins. These observations are supported by investigation by Dinu *et al.*, (1978). They found protein dystrophy in myocardium by administration of hydrocortisone during immobilization stress. Prolonged emotional stress induce pathological changes in myocardium, which may be associated with elevated glucocorticoids (Carncross and Bassett, 1975). Exposure to the heat stress causes marked increase in plasma cortisol (Colin *et al.*, 1968). In hypoxia there is decreased RNA in myocardium (Tsagarel *et al.*, 1970). Anoxia and defibrillation causes decreased protein synthesis in dog heart (Korotkina *et al.*, 1975).

The stomach weight of experimental rat was increased gradually as compared to the rat (control). This may be due to hypertrophy and hyperplasia of gastric epithelium. Similarly increase in proliferation of the cells, may lead to increase in the weight of the stomach. The significant increase in the weight of different organs suggests an increase in their activity as a result of the continued stress (Mionteiro *et al.*, 1989). The change in colour of the stomach, from fresh creamy white, to dull and dirty white might be due to hypoxia.

Hypertrophy of gastric gland epithelium during first exposure, might be due to the stress in powerloom, to secrete more amount of mucus, in order to compensate the stress

situation. Repeated overload induce, adaptation reaction in GIT (Derevyanko *et al.*, 1972). During first exposure of experimental rats of the stresses, in powerloom environment there was hypertrophy of adrenal cortex which may cause increased secretion of cortisol. This increased cortisol promotes the production of hydrochloric acid and pepsin by stomach (Labhart, 1974).

With increasing exposure period, there was damage of gastric gland epithelium. It might be due to autoimmunity against the gastric mucosa and adrenal insufficiency. Because of this, there may not be a secretion of corticosteroid and leads to gastrointestinal damage. Yano *et al.*, (1976), found gastric erosion in rats subjected to restraint and water imemrsion stress. Excessive HCl may also causes direct damage to the stomach mucosa. Repeated injection of histamine causes excessive secretion of gastric juice. Cotton dust induced histamine release from passive sensitized rat mast cells (Vangala *et al.*, 1982 and Douglas *et al.*, 1984).

In Set S_3 there was again hypertrophy and hyperplasia of gastric epithelium. Chief cells and parietal cells became tall and there were increased number of dividing cells. These changes might be meant for adaptation to stress situation. Cortisol appears

to stimulate the sensitivity of the gastric mucosa to histamine and to increase number and height of its parietal cells. These observations are also supported by Reid *et al.*, (1961). They found increase in the number of parietal cells of the gastric mucosa of dogs on administration of cortisone. Similar observations also reported by Bockus (1964).

The decrease in the amount of total proteins in the stomach of experimental rats was probably due to combined effect of stress, damage of gastric mucosa and hypertrophy of adrenal gland. The adreno-cortical hormones like glucocorticoids decrease the quantity of proteins in tissue. Glucocorticoids act by increasing the rate of breakdown of extra-hepatic proteins. Turnover of RNA and proteins decreased in digestive organs of rabbit due to vibration (Yie *et al.*, 1972).

Increase in the weight of the duodenum may be due to hypertrophy of muscularis externa, increased number of dividing cells, and large number of granulated goblet cells. These changes might be due to increased secretory activity of duodenal mucosa in response to the stress. These observations were supported by Mionteiro *et al.*, 1989, they found significant increase in the weight of different organs suggest an increase in their activity,

as a result of continued stress. The change in colour of duodenum from fresh white to dirty, dull white might be due to hypoxia.

The marked hypertrophy of muscularis externa of duodenum in Set S_1 might be due to compensation to stress reaction. Similar observations were found by Hill *et al.*, (1971) in additional stress produced in antiperistalisis. The lumen of intestinal gland showed large number of goblet cells filled with granules. This may be due to adrenal medullary hormones. Daily injection of epinephrine in adrenal ectomized rats show increased volume of neutral and acidic mucosubstances in intestinal goblet cells (Ahn *et al.*, 1972). There were number of dividing cells may be in order to allow rapid repair of duodenal mucosa.

Total protein content in duodenum decreased in sets S_1 and S_3 may be due to decrease in biosynthesis of proteins. Various stresses like vibration causes decreased turnover of RNA and protein (Yui, 1972). In Set S_2 the increase in the total proteins may be due to hypersecretion of adrenal medulla, which include epinephrine, cause increase in amount of sulfated muco-substances (Ahn *et al.*, 1992).

Presumably all above histophysiologic and total protein alterations in non pulmonary organs of rat exposed to stresses in

the powerloom environment are due to activation of hypothalamosympathetic-adrenal axis and resultant release of catecholamine from adrenal medulla due to the stresses existing in the textile environment including the toxic stress of endotoxin(s), associated with cotton fibers in powerloom units.

While concluding the present dissertation on "Effect of Stresses on Rat Organs Exposed To Textile Environment", it should be mentioned that, practically all the objectives with which the present work was taken up have been fulfilled. Thus, the present study describes the effects of industrial stresses on histophysiology and total protein changes in some of the non-pulmonary organs like adrenal kidney, heart stomach and duodenum of the male albino rat (*R. norvegicus*).

The present investigation opens up several avenues for further research on occupational stresses in the textile industry. Some ideas for such future work are in brief, listed below :

1. The present study has been carried out only in some selected powerloom sheds. A detailed study of textile environmental scenario of the powerloom industry with a view of the various occupational hazards will give a better picture regarding health and safety status and occupational hazards in powerloom industry.

2. In the present work only short term exposure of animal model has been tested, a long term exposure and the recovery changes after exposure will give a better understanding about physiological responses of exposed animal.

3. Recent trend in the study of occupational physiology concerns with investigation of combined effects of environmental stresses. Researchers mostly deal with the exposure of a experimental model to a complex unfavourable environment. The combine effect of these factors is determined by their interaction. Unfortunately, most of the researchers have not studied the physiological mechanism in the combination of factors like temperature, humidity, noise, dust and vibration etc. Only the final effect of this or that system or as the individual as a whole is studied. Therefore it will be interesting to study epidemiology of the long term effects of combined effects of noise, heat, humidity, vibration and cotton dust.

4. In the etiology and pathophysiology of organ system of rat after the exposure to textile environment several possible mechanisms may prove to be operative but yet, they remain to be evaluated. Among these are in-depth studies of

possible cellular and subcellular effects of cotton dust extracts, study of mechanism of release and effect of cell activating factor(s). Such studies will, however, are only possible when concomitant preparation of purified endotoxin(s) from cotton dust.

The improvement of layout design of powerloom sheds from ergonomic point of view, the improvement of hygienic norms on cotton dust, noise, high temperature, humidity and vibration as the most wide spread stressful factors of occupational environment is necessary for the maintenance of work efficiency and health status of workers and for creation of the environment for productive and qualitative labour.

The share of the powerloom sector in the national annual cloth output is more than 70 per cent. It is the largest employment generator sector with more than eighty lakhs persons directly employed. Its export performance is spectacular and it is a special class of rural entrepreneurs which mainly comes from scheduled class, scheduled tribes, nomadic tribes and other backward class. it is important to note that it is the only sector of textile industry, which is running without research or innovation of any kind. This sector can play a role in the economy of our country. The Powerloom Committee (1964) has stated that "The

powerloom is much more than an instrument of production. It is symbol of vast country wide process of economic transition and technological change. Behind it lie deep economic urges of millions of people to break through the barrier of poverty, to improve ever so little, their level of life line and to uplift themselves to a slightly higher social level." It seems that, a sweating toiling human mass, menaced by a grim struggle for existence, is in search of an anchor which would help them in escaping the drift of hunger and distress. Thousands of textile workers have abandoned their ancestral homes and have migrated to Ichalkaranji to fulfill the clothing requirement of the nation at the cost of their health and lives. Hence, it is necessary to take genuine measures for their health protection and this will be only possible, if one undertakes the extensive research in this important sector of the textile industry.