INTRODUCTION
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Life is an adventure in a world where nothing is static where unpredictable and ill-understood events constitute dangers that must be overcome often blindly and at great cost; where man himself, like the sorcerer’s apprentice, has set in motion forces that are potentially destructive and may some day escape his control. Every manifestation of existence is a response to stimuli and challenges, each of which constitutes a threat if not adequately dealt with. The very process of living is a continual interplay between the individual and his environment, often taking the form of a struggle resulting in injury or disease.

[Rene Dubos, 1959]

Stress (physical, chemical, psychosocial) represents the reaction of the body to stimuli that disturb its normal physiological equilibrium or homeostasis, often with detrimental effects. The evocative stimuli are accordingly called as stressors. Earlier, the stress response was considered as non-specific and it largely involved the activation of pituitary-adrenal cortical axis (Hans Selye, 1946). However, as the field of neuroendocrinology evolved, the concept of non-specific stress response has been
Table - I

HOST DEFENSE AGAINST MICROORGANISMS

Phagocytes

Neutrophils
  Adherence, chemotaxis, engulfment, bactericidal action

Monocytes-Macrophages
  Phagocytosis, antigen presentation to lymphocytes, cytotoxic action

Lymphocytes

  T cells
    Cytotoxic action

  B cells
    Antibody production

Other sub-populations

  T helper, T suppressor, Natural killer cells

Humoral immunity

  Antibodies (IgG, IgM, IgA, etc.)

Miscellaneous

  Serum complement factors

  Interferon
refuted. Now, numerous studies have shown that the nature of the stress conditions determine the specific pattern of response (Mason, 1971; Elliott and Eisdorfer, 1982). For example, both public speaking and physical exercise produced increased plasma concentrations of norepinephrine and epinephrine. Norepinephrine, however, is higher than epinephrine during exercise, while epinephrine is considerably higher than norepinephrine during public speaking [Dimsdale and Moss, 1980].

The cognitive recognition of adverse stimuli evokes a series of neuroendocrine responses which disturb the physiological equilibrium or the homeostasis. This has a significant impact on the immune response in general. Conceptually, the status of the immune system (immunosuppression versus immunopotentiation) will depend upon the net effect of these changes. According to Golub and Gershwin (1985), the immune system may be modified by stress to produce both inappropriate (suppressive) and appropriate (enhanced) changes in immunocompetence.

Immunocompetence could briefly be explained as the capacity to identify and reject material foreign to the particular individual, whereas materials furnished with markers of self are accepted. This is accomplished by means of a complex system including many cellular and humoral factors as described in Table 1. Thus, according to this very simplified definition, infections are due to deficient
recognition, or mobilization of appropriate defense system against the invader, similarly, autoimmune disorders would be the consequence of a failure to recognize markers of self and an attack on a host's own tissues.

Unlike other stressors like heat, cold, shock, etc., which have been thoroughly studied for a considerable time, interest in noise as a stressor is comparatively new, and much still remains to be discovered about its effects. This may be due to the fact that our environment had always been a relatively quiet one until very recently.

Today, Technology has created many environmental pollutants, of which noise is an important and identifiable example. Many industrial processes since the industrial revolution, have generated noise of high sound level sufficient to cause deafness.

One of the salient international health problem, that is always high on the agenda at regular meetings of World Health Organisation is Noise! Whether you live in Madras or Manchester / Paris or Pittsburgh, there is no getting away from it. Noise is considered as any unwanted sound that may adversely affect the health and well being of individuals or population. Physically, sound is a mechanical disturbance, propagated as a wave motion in air and other elastic or mechanical media such as water or steel. Physiologically, sound is an auditory sensation evoked by this physical
phenomenon. However, not all sound waves evoke an auditory sensation. For example, ultrasound has a frequency too high to excite the sensation of hearing. The extent of the discomfort experienced by an individual subjected to noise will depend upon the frequency spectrum, intensity of the sound, duration, aural sensitivity of the listener, and upon the activities being undertaken at the time of noise exposure. The frequency is the physical characteristic of a sound that makes us feel it is low or high in pitch, it is measured in Hertz (Hz) which is the number of cycles of pressure fluctuations per second. Noise with a regular fluctuating pressure (sinusoidal) is called a sine wave noise. Most sounds have irregularly fluctuating pressures (white noise, eg. roar of a jet engine). Higher frequencies are usually more irritating and potentially damaging. Sound intensity is the amplitude of sound waves. Human hearing can detect a fantastic range of intensities. The maximum sound pressure that can be received without consequent damage are still a million times greater than those of minimum detectability. For this reason, a logarithmic scale is convenient and sound intensities usually are measured in decibels (dB). The duration of sound exposure can be of the utmost significance. A snap of the finger can generate a peak level of over 140 dB, but since the sound lasts only a fraction of a second, it is innocuous. However, the same intensity of noise for a longer duration can be critical.
The study of responses to noise stress is usually divided into two categories: effects on the auditory structures, and the systemic or the extra auditory effects on the body. Acoustic stress has been and is being studied in great deal with respect to its effects on the ear and it has been well established that chronic exposure is capable of promoting hearing loss. There are two distinct impairment mechanisms leading to hearing deficits. The conduction loss which is one type of impairment mechanism include rupture of the tympanic membrane, separation of the ossicular chain of the middle ear, or rupture of the fine membranes (sacs) of the cochlea and labyrinth. An exposure of intense noise with an intensity of 150 dB for about a minute could be sufficient to produce such damage. The other type of impairment mechanism is a form of sensorineural loss, stemming from damage of the sensory cells distributed along the basilar membrane of the cochlea. This type of deficit is associated with chronic exposure.

As human beings, are rarely exposed to an acoustical stress in isolation from other stresses, the relative importance of noise and its extra auditory effects have proved more difficult to delineate and to specify than its direct effects on hearing. Investigations on the non-auditory effects of noise stress has been neither as extensive nor as conclusive as those pertaining to hearing loss. But the available literature suggests that sound can be a significant stressor in respect of overall body physiology.
The level at which the physiological effects of noise begin to occur varies according to the physiological function measured. Jansen (1969) has proposed possible physiological reaction at 60 dB sound pressure level (SPL) and a possible beginning of injury at 95 dB. He has further shown some frequency dependence of the physiological effects of the noise with greatest effects produced by stimuli in the frequency range of 2000-3000 Hz.

In this study, an attempt has been made to evaluate the effect of noise stress of definite magnitude (97 dB at 3000 Hz) on certain immune functions in albino rats. The involvement of corticosteroid and central serotonin in the immunomodulation due to stress was also studied.

Moreover, in using animals to study the cumulative effects of noise, it has not been necessary to assume that the absolute sensitivity of animals and man to noise is the same, but merely that the relative sensitivity of animals to noise of specified temporal patterns is similar to that of man. Further, the frequency of noise used in this study is well within the range of audiofrequencies detectable by rats [Gay, 1973].