CHAPTER-III

Aims & Hypotheses
Many people know what the words Loyalty, Duty, Respect, Selfless Service, Honor, Integrity, and Personal Courage mean. But how often do you see someone actually living up to them? Soldiers learn these values in detail during Basic Combat Training (BCT), from then on they live them every day in everything they do- whether they're on the job or off.

The increased load on soldiers without any social support because of extreme regimentation can evoke some kind of diseases to which a person is prone at the time of birth. In today's world the psychosomatic diseases mainly like diabetes and hypertension have assumed menacing proportions. The present study deals with this aspect.

The interaction between genetics and environment in a person's life cause these psychosomatic diseases. According to Anxiety/ stress hypotheses, genotype provide vulnerability within a person that interacts with stress or anxiety or depression to produce the disorder.

Psychosomatic diseases are caused by interaction of multiple genetic and environmental influences. These diseases also cluster in families. Recent advances in molecular genetics are making it increasingly feasible to construct individual genetic profiles predicting susceptibility to heart disease, cancer and respiratory disorders. This paper reviews current knowledge about the social and cultural impact of providing people with information relating to their risk for future disease, focusing not only on currently available predictive genetic testing but also on hypertension, hyper lipidaemia and cancer screening. Researchers have highlighted the importance of issues of probability and uncertainty, and the tension between collective
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and individual goals in the assessment of medical risk. We conclude with a proposed research agenda for studies of the social and cultural impact of predictive genetic testing, and argue that there is a pressing need for rigorous, empirical, social research in this area (Davison, Macintyre & Smith, 1994).

Fifty percent of hypertensives have a positive parental history of hypertension (Waldstein, 1995). Peters, Munter, Bader, Hackenthal, Mullins & Ganten (1993) suggest that a type of rennin gene may be involved in development of hypertension.

Type-2 diabetes is a complex disease, which is also modified by a number of environmental and genetic factors that contribute at varying degrees to the final phenotype and possibly interact with each other. Deciphering the genetic background of the disease serves multiple goals ranging from expanding knowledge on the disease pathogenesis and identifying future targets for drug development to successfully personalizing clinical disease prediction and prognosis. In the present review Evangelia & Fotini (2012), aimed to systematically appraise the current evidence from genome-wide association studies (GWAS) on type-2 diabetes, identify the gene targets that have been assessed to-date, and discuss issues that stem from the rapid growth of this literature. The search identified more than 60 recently identified loci implicated with type-2 diabetes and related traits assessed in populations of European and Asian ancestry. A considerable number of the proposed genes seem to be related to beta-cell development and function, but there are several genes identified as "diabetes-gones" whose underlying pathway linked to diabetes remains poorly understood. Despite the increasing numbers of identified genetic markers, a large proportion of the observed type-2 diabetes heritability remains unexplained; larger GWAS on enhanced genotyping platforms, refined ascertainment of the characteristics of the populations under study and additional information from whole genome sequencing will contribute to a
more comprehensive view of the genetic architecture of the disease. This information is also anticipated to improve the predictive ability of multiple-loci genetic risk scores that will eventually be able to identify disease susceptibility over and above the traditional non-genetic risk factors.

There are various environmental factors also that act as causal factors for psychosomatic diseases, such as Occupational Stress (Rozanski, Blumenthal & Kaplan, 1999; Sanders & Lawler, 1992; Light, Turner & Hinderliter, 1992; Schnall, Pieper & Schwartz, 1990), Industrial/Traffic Noise exposure (Krantz, DeQuattro, Blackburn, Eaker, Haynes, James, Manuck, Myres, Shekelle, Syme, Tyrole & Wolf, 1987; Green, Schwartz, Harari & Najenson, 1991), Low Socio-economic Status (James, et. al., 1987; Rozanski, Blumenthal & Kaplan, 1999), Lack of Social Support (Rozanski et al., 1999), Modernization (Kaplan, 1994; James, et al., 1987), Personality Traits (Smith, 1958), Type-A Personality (Dutour, Boiteau, Dadoun & Feissel, 1996), Cultural Factors (Brown, 2002; Auslander, Thompson, Dreitzer & Santigo, 1997).

Even the psychological factors like Stress, Anxiety/Neuroticism and Depression in an individual are due to genetical as well as environmental components which has a great deal of effect on the Adjustment of an individual.

Recent studies have demonstrated that the free cortisol response to awakening can serve as a useful index of hypothalamus–pituitary–adrenal axis (HPA) activity. This endocrine marker is rather consistent, shows good intra individual stability across time and appears to be able to uncover subtle changes in HPA regulation. The present twin study of Wüst, Federenko, Hellhammer & Kirschbaum (2000), investigated genetic factors as sources of the inter individual variation of the cortisol awakening response. Furthermore, the relationship between psychological variables and morning cortisol levels was
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On two consecutive days saliva samples were collected 0, 30, 45 and 60 minutes after awakening in 52 monozygotic and 52 dizygotic twin pairs. Moreover, samples were obtained at 0800, 1100, 1500 and 2000 h. ('short day-time profile'). Additionally, the participants filled out questionnaires assessing chronic stress load, self-esteem and self-efficacy. Heritability estimates of $h^2=0.40$ for the mean increase and of $h^2=0.48$ for the area under the response curve indicate a significant impact of genetic factors on cortisol levels after awakening. However, no genetic influence on the short day-time profile could be observed. Furthermore, several aspects of perceived chronic stress, namely 'worries', 'social stress' and 'lack of social recognition' were significantly associated with the awakening cortisol response. The evidence for a medium-sized, yet distinct genetic influence on cortisol levels after awakening is discussed with regard to a potential clinical relevance of genetic determinants of HPA (re)activity. In line with several recent studies, the present findings further support the view that the cortisol awakening responses is consistently enhanced under chronic stress conditions.

Posttraumatic stress disorder (PTSD) develops in only a subset of persons exposed to traumatic stress, suggesting the existence of stressor and individual differences that influence risk. In another study by Stein, Jang, Taylor, Vernon & Livesley (2002), examined the heritability of trauma exposure and PTSD symptoms in male and female twin pairs of nonveteran volunteers. Scores on a traumatic events inventory and a DSM-IV PTSD symptom inventory were examined in 222 monozygotic and 184 dizygotic twin pairs. Biometrical model fitting was conducted by using standard statistical methods. Additive genetic, common environmental, and unique environmental effects best explained the variance in exposure to assaultive trauma (e.g., robbery, sexual assault), whereas exposure to non-assaultive trauma (e.g.,
motor vehicle accident, natural disaster) was best explained by common and unique environmental influences. PTSD symptoms were moderately heritable, and the remaining variance was accounted for by unique environmental experiences. Correlations between genetic effects on assaultive trauma exposure and on PTSD symptoms were high. Genetic factors can influence the risk of exposure to some forms of trauma, perhaps through individual differences in personality that influence environmental choices. Consistent with symptoms in combat veterans, PTSD symptoms after noncombat trauma are also moderately heritable. Moreover, many of the same genes that influence exposure to assaultive trauma appear to influence susceptibility to PTSD symptoms in their wake.

Anxiety/Neuroticism also have genetical and environmental effects which help to develop an individual's overall personality. Anxiety/neuroticism has about 70% to 80% genetic component, so are the diabetes and hypertension. Genes play a major role in developing Neuroticism in an individual. Environment also has a very important role to play (nearly 30%). The predisposing factors are genetic and the precipitating factors are environmental.

The authors Hettema, Prescott & Kendler (2004), examined the sources of co-variation between generalized anxiety disorder and the personality trait of neuroticism. Because women have higher levels of neuroticism and twice the risk of lifetime generalized anxiety disorder to men, gender-specific effects were also explored. Lifetime generalized anxiety disorder and neuroticism were assessed in more than 8,000 twins from male-male, female-female, and opposite-sex pairs through structured diagnostic interviews. Sex-limited Cholesky structural equation models were used to decompose the correlations between generalized anxiety disorder and neuroticism into genetic and environmental components, including sex-specific factors.
Genetic correlations between generalized anxiety disorder and neuroticism were high and differed (non-significantly) between men and women (1.00 and 0.58, respectively). When non-significant gender differences were removed from the models, correlations between generalized anxiety disorder and neuroticism were estimated at 0.80. The individual-specific environmental correlation between generalized anxiety disorder and neuroticism was estimated at 0.20 for both genders. There is substantial overlap between the genetic factors that influence individual variation in neuroticism and those that increase liability for generalized anxiety disorder, irrespective of gender. The life experiences that increase vulnerability to generalized anxiety disorder, however, have only modest overlap with those that contribute to an individual's level of neuroticism.

In another study by Martin, Jardine, Andrews & Heath (1988), data from 2,903 adult same-sex twin pairs were analysed to investigate whether the genetic determinants of symptoms of panic are different from those underlying the neuroticism personality trait. The results suggest that much of the genetic variation influencing the physical symptoms associated with panic is of the nonadditive type, perhaps due to dominance or epistasis. In both sexes these nonadditive genetic effects on physical symptoms influence the reporting of "feelings of panic". In males they also account for as much as half the genetic variance in neuroticism. The remainder is additive and also accounts for the balance of genetic variation in "feelings of panic". In females genetic variance in neuroticism is entirely additive but is not an important source of covariation with either panic symptom. Thus, symptoms of panic seem to be shaped in part by unique genetic influences which do not affect other anxiety symptoms. That a substantial part of the genetic variance in neuroticism in males may be due to the nonadditive effects on physical symptoms of panic may help to explain the rather low correlation between the
genetic influences found to affect neuroticism in males and their counterparts in females.

Anxiety/Neuroticism in psychosomatic patients like, hypertensive and diabetic patients rises and one of the major reasons of it is that they have to keep an account of the calorie intake in their daily diet, especially diabetics. They always have to be cautious of the food they are having so this food restriction acts as one of the precipitating factors that gives rise to the neuroticism in these patients. In hypertensive patients also everyone knows that it cluster in the families but even in this case food restrictions give rise to more Anxiety and Neuroticism.

In depression also both the factors, genetic and environmental have effect on an individual. According to the diathesis-stress theory of depression, an individual’s genetic makeup can predispose some people to be more susceptible to depression when they are confronted with negative life experiences. Traditionally, most studies testing these theories have focused on only one component of the stress model: either genetics or environment, but not their interaction. However, recent advances in genetics and genomics have stimulated a new arena of clinical research allowing scientists to test the interaction between genetic and environmental liabilities - the G x E design (Nauert, 2008).

In another study by Jardine, Martin, Henderson & Rao (1984), a genetic analysis of the trait of neuroticism and symptoms of anxiety and depression in 3,810 pairs of adult twins is reported. Differences between people in these measures can be explained simply by differences in their genes and in their individual environmental experiences. There is no evidence that environmental experiences that are shared by cotwins, such as common family environment or social influences, are important. There are differences between the sexes in gene action affecting neuroticism, and genetic effects become more pronounced with
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age in females. The lack of evidence for dominance variance affecting neuroticism contrasts well with the detection of considerable genetical non-additivity for extraversion in the same sample and reinforces the view that these two traits are not only statistically, but also genetically, quite independent. An analysis of the causes of covariation between anxiety, depression, and neuroticism shows that additive gene effects are more important causes of covariation than environmental factors. Genetic variation in symptoms of anxiety and depression is largely dependent on the same factors as affect the neuroticism trait. However, there is also evidence for genetic variation specific to depression.

There have been various studies which clearly state that chronic psychological factors like stress, anxiety/neuroticism and depression has negative effect on the health of an individual and give rise to the chronic illness or psychosomatic diseases like hypertension and diabetes and vice versa.

A review by Cohen & Herbert (1996), addressed the importance of studies of human psychoneuroimmunology in understanding the role of psychological factors in physical illness. First, it provides psychologically and biologically plausible explanations for how psychological factors might influence immunity and immune system–mediated disease. Second, it covers substantial evidence that factors such as stress, negative effect, clinical depression, social support, and repression/denial can influence both cellular and humoral indicators of immune status and function. Third, at least in the case of the less serious infectious diseases (colds, influenza, herpes), it considers consistent and convincing evidence of links between stress and negative affect and disease onset and progression. Although still in early days of its development, research also suggests a role of psychological factors in autoimmune diseases. Evidence for effects of stress, depression, and repression/denial on onset and
progression cancer is less consistent and inconclusive, possibly owing to methodological limitations inherent in studying these complex illnesses, or because psychological influences on immunity are not of the magnitude or type necessary to alter the body's response in these cases.

To review evidence that psychological factors affect the course of physical illness three areas are examined: epidemiological evidence showing the levels of psychiatric disturbance co-morbid with physical illness; health services research showing the burden of disease and care associated with this co-morbidity; randomised, controlled trials of psychological interventions in cancer, myocardial infarction and irritable bowel syndrome. There is substantial psychiatric co-morbidity with physical illness which is associated with increased disability, mortality and utilisation of health-care resources (primary care visits, hospitalization, length of hospital stay, cost). A small number of controlled intervention studies have shown the efficacy of psychological interventions to prolong survival in cancer and myocardial infarction, and to improve symptomatology in irritable bowel syndrome and other chronic somatizing conditions. Psychological factors do significantly affect outcomes of physical illness (Clarke, 1998).

To see the vica versa effect of the above a study was conducted by Cuthbertson, Hull, Strachan & Scott (2004), where the objective was To determine the incidence and severity of symptoms related to the diagnosis of post-traumatic stress disorder (PTSD) in a cohort of general ICU patients. They found a high incidence of symptoms consistent with PTSD 3 months after ICU discharge in this general ICU cohort. This was associated with younger patients and those who visited their GP or a mental health professional complaining of psychological symptoms.

These psychological and physical factors in an individual together effect his adjustment in daily life. In a study by
Sukantarat, Greer, Brett & Williamson (2007), the objective was to measure levels of anxiety, depression and post-traumatic stress among survivors of a critical illness and to relate these symptoms to general health parameters. A prospective study was conducted on patients who had spent a minimum 3 days (median 9 days) in a general intensive care unit (ICU). Of these patients, 51 were interviewed 3 months after discharge and 45 of them were reviewed at 9 months. General health was assessed by a physical symptom score, the EuroQol ‘thermometer’ and the Short Form 36 (SF-36) questionnaire. Physical and mental component summary measures (PCS, MCS) were calculated from the SF-36 data. Psychological health was assessed using both the Hospital Anxiety and Depression Scale and the Impact of Events Scale. At both 3 and 9 months after ICU discharge 24% of patients qualified as a ‘case’ of anxiety, while similar figures were seen for intrusion. The incidence of depression and avoidance was higher on each occasion. Four of the eight SF-36 domains improved with time, as did PCS, but there was no significant difference in physical symptom score, EuroQol value or MCS. Strong correlations were seen between the physical and psychological parameters at each time point. A substantial proportion of patients who survive a critical illness show evidence of anxiety and depression up to 9 months later, and most of them also have symptoms indicative of post-traumatic stress. Delayed physical recovery may contribute to this psychological morbidity. ICU follow-up clinics should be able to detect patients suitable for psychological intervention.

Patients with a chronic illness must continuously revise their lifestyle, adapting it to the behavioural limitations imposed by their state of health. These incessant adjustments of behaviour dictated by the patients’ need to adapt to their clinical condition also cause profound psychological changes. The experience of a patient with a chronic illness often leads to a
reformulation of self, which the patient may or may not be aware of, but which helps to facilitate successful behavioural adaptation. During the course of their disease, which spans from diagnosis to treatment, some patients have the opportunity to meet a psychologist, who has various tasks: understanding what stage of adaptation the chronically ill patients have reached, evaluating the patients' emotional state, facilitating their acceptance of their clinical condition, stimulating them to redefine their aims, if there are the presuppositions, and supporting their coping capacities and internal and external resources. This article is neither a review nor original research, but rather a "clinical exposition" with educational suggestions. The purpose of this article is to give a voice to the patients' internal dialogue, to what they say to themselves, to their narration of the illness, but also to explain the typical components of cognitive behavioural treatment in the setting of cardiological, respiratory and neuromotor rehabilitation (Pierobon, Giardini, Callegari & Majani, 2011).

So as we can see that the genes play a major role in physical and psychological factors. But at the same time environment also have a great impact in aggravating these factors. In present time these problems are coming more into notice. Chronic illness or psychosomatic problems, like diabetes and hypertension and psychological problems, like, anxiety, stress, depression etc. affect the individuals more often and the major reason in the life we are living. Now days these problems are termed as, "lifestyle diseases".

So keeping all the above factors in mind the present endeavor has been designed to fulfill certain aims for which the present study was committed at the time of initiating the present study.
Aims

Main aims of the present study are:

1. To compare the score on Neuroticism/Anxiety of the patients of Diabetes, Hypertension and other Psychosomatic problems with the normals. The reason being that the person will become more neurotic with the onset of the various psychosomatic problems and vice versa.

2. How the test scores which are related to the daily life hassles and family matters affect the soldier.

3. To know the effect of all the variables on the adjustment of the soldier in the various areas of the life.

4. To see if the diabetics in comparison to the hypertensive and patients suffering from other psychosomatic problems have high scores in neuroticism, depression and stress because of its greater complication.

5. To see if normal score high in adjustment.

Hypotheses

After keeping all the above factors and aims in mind, the hypotheses of the present study were formulated:

1. Stress scores will be higher in patients as compared to the normals.

2. Neuroticism / Anxiety scores will be higher in patients as compared to the normals.

3. Depression scores will also be higher in patients as compared to the normals.

4. Adjustment level will be less in patients as compared to the normals.
5. Diabetics will score higher in neuroticism and depression as compared to the other groups because of its greater complication.

6. Hypertensive will score higher in stress in comparison to the other group.