CHAPTER III
REVIEW OF LITERATURE
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In continuation with the previous chapter on theoretical framework, the present chapter is devoted to the research reviews related to the problem of the proposed research.

GLAUCOMA

Glaucoma is an eye disease which may have a psychic basis in its genesis because the aqueous humor present in the anterior chamber of the eye which is responsible for the normal intra-ocular pressure, its secretion and draining from the eye is controlled by the autonomic nervous systems.

The influence of stress as a predisposition of chronic simple glaucoma was studied by Thakore et al. (1985) working at Banaras Hindu University Eye Hospital at Varanasi. They selected 40 patients of established chronic simple glaucoma (Meetha Jhaamer) and 30 healthy individuals (control). Both the group of chronic simple glaucoma and controlled were analysed on life inventory modified after Mahendra et al. and Venkoba Rao. These patients were also subjected to estimation of:

1. Total Plasma Catecholamines
2. Plasma Adrenalin
3. Plasma Non-Adrenalin
4. Plasma Cortisone
5. 17 Hydroxy-Corticoestroides in urine
6. Urinary V.M.A. (venelilive, mendelic acid)
The analysis of frequency and severity (grade of stress) of various stresses recorded in control, and in patients of chronic simple glaucoma, reveal that glaucoma patients had significantly high index of stress on the basis of these observations they speculated, that there exists a possible correlation of stress and chronic simple Glaucoma. The frequently encountered stress in these patients was found to be, maladjusted in their families. From the biochemical studies conducted by the same authors they found evidence that chronic simple Glaucoma patients have elevated plasma levels of catecholamine adrenalin and non adrenalin which indicates a high stress index.

No research is done so far which might relate the psychological factors and the cataract condition, or the retinal detachment in the eye. But various cases have been observed by the investigator, which do indicate psychological association with Cataract. Glaucoma and Retinal detachment conditions, such cases are usually treated as purely organic ones.

Glaucoma

Factors that influence Intraocular Pressure

Many factors influence intraocular pressure.

AGE:

Most studies (Armaly M.F., 1965); (Draeger J., 1959); (Gaasterland D., Kupfer C., Milton R., Ross K., McLain L. and Maclellan H., 1978); (Hiller R., Sperduto R.D., and Krueger D.E., 1982); (Klein B.E., Klein R., 1981); (Levene R.Z., 1961) find a positive correlation between intraocular pressure and age. The
effect of increasing age on intraocular pressure is the result, at least in part, of increased blood pressure, increased pulse rate, and obesity (Bengtsson B., 1972); (Carrel R.S., Korczyer A.D., Rock M. and Goya I., Carrel R.S. 1984). It is unclear whether the rise in intraocular pressure with age represents an increase for all individuals or greater skewness of the data - that is a greater minority of the people having higher pressure while the majority shows no change. It should be pointed out that a number of studies find little correlation between intraocular pressure and age (Boles, Grenini B. and Cambiaggi A., 1960); (Carrel R.S., Korczyn A.D., Rock M. and Goya I., Carrel R.S., 1984). In addition one investigative team found that intraocular pressure declined with age in a group of Japanese workers (Shiose Y. 1984); (Shiose Y. and Kawase Y. 1986).

SEX:

It has been reported that women have higher intraocular pressure than men especially after age 40 (Armaly M.F., 1965). However, this finding was not confirmed in another study (Bulpitt C.J., Hodes C. and Everitt M.G. 1975).

RACE:

In the United States blacks have higher intraocular pressure than whites (Hiller R., Sperduto R.D. and Krueger D.E., 1982; Klein B.E. and Klein R., 1981). In part, this difference appears to be racial or genetic. However, this difference also reflects the increased iris pigmentation in blacks - that is
people with dark irides have higher levels of intraocular pressure regardless of race (Hiller R., Sperduto R.A. and Kreuger D.E., 1982). There is one report that the Tuni Indians of New Mexico have relatively low intraocular pressure (Kass M.A., Zimmerman T.J., Alton E., Lemon L. and Becker B., 1978). It is unclear whether this phenomenon is caused by genetic or environmental factors.

HEREDITY:

There appears to be a hereditary influence on intraocular pressure (Armaly M.F. 1965); (Armaly M.F., Montauro T. B.F. and Sayegh R.E., 1968); (Lenen R.J., Workman P.L., Broder S.W. and Hischhorn K., 1970) which is more likely polygenic in nature (Armaly M.F., 1967; Armaly M.F., Montavicius B.F., Sayegh R.E., 1968). A number of studies have indicated that first degree relatives of patients with open angle glaucoma have higher intraocular pressures than the general population (Armaly M.F., 1965); (Seddon J.M., Schwartz B. and Flowerdew G. 1983). In contrast one study found that spouses have similar levels of intraocular pressure, which suggests that there are important environmental influences as well (Bengston B., 1976).

HORMONAL:

Administration of corticosteroids topically periodically and systematically raises intraocular pressure.

Some researchers have questioned whether sex hormones have an influence on intraocular pressure. It has been noted that
Intraocular pressure varies with the menstrual cycle (Becker B. and Friedenwald J.S., 1952); (Dalton K., 1967); (Peterson G.D. and Miller S.J.H., 1963) and is low in the third trimester of pregnancy (Becker B. and Friedenward J.S. 1952); (Peterson C.D. and Miller S.J.H.; 1963); (Phillip C.I. and Gore S.M., 1985). However, other studies have not found good correlation between intraocular pressure and serum levels of progesterone and estrogen (Feldman F., Bain J., and Matuk A.R., 1978); (Green K., Cullen P.M. and Phillip C.I., 1984).


Diabetic individuals have higher intraocular pressure than the general population. The reason for this association is unclear. Other hormones including growth hormone, thyroxine, aldosterons, vasopressin and melanocyte stimulating hormones may influence intraocular pressure physiologically or when administered in pharmacologic doses (Kass M.A. and Sears M.L., 1977).

**Personality**

Some researchers have been focusing on coping strategies, others have been concerned with more stable individual resources for managing stress. Among the most important of these are personality characteristics. Some of the personality characteristics that have been studied are actually stable.
clusters of coping dispositions (Kobasa et al., 1982); (Rosenbaum 1984), while others are conceptualized as important largely because they influence the choice of coping strategies (Wheaton, 1982). Some personality characteristics are thought to be important because they tap the individual's vulnerability to stress. Neuroticism (Henderson et al., 1981), for example, is thought to modify the impact of stress because neurotic people are inherently more fragile emotionally. Others are thought to be important because they influence primary appraisal. The personality characteristic of interpersonal dependency is an example. It is thought to magnify the impact of interpersonal loss on health because dependent people appraise loss as particularly threatening (Hirschfeld et al., 1976).

A large number of personality characteristics, self feelings, and orientations to life that might be relevant to stress modification have been identified in the literature. Many attempts have been made to classify personality into core domains (McCrae et al., 1986). There have also been attempts to specify the central personality characteristics that amount for the effects of more global constructs such as the Type A behaviour pattern (Matthews, 1985). As noted earlier, there has also been considerable interest in determining whether any stable personality characteristics explain the putative effects of social support.

*Pathogenic Personalities*

One line of research involves the hypothesis that
particular personalities predispose people to particular illness. Examples include research on a cancer-prone personality, an arthritis-prone personality, and a personality type that predisposes to cardiovascular disease.

**Cancer:** Research on a cancer-prone personality began with a number of clinical reports pointing to an observed association between severe emotional loss and subsequent onset of cancer. More systematic epidemiologic studies verified this association in case-control designs and refined the argument into a two-part perspective: that cancer patients tend to have experienced a serious emotional loss and to have a premorbid personality which makes them prone to feeling of helplessness and hopelessness in the face of such a loss. Coping strategies were implicated indirectly, by suggesting that people with a cancer-prone personality used excessive denial and repression of emotions (Scurry and Levin, 1978-79).

There have been only a handful of truly prospective studies in which personality characteristics were assessed prior to the onset of cancer, yet they have been surprisingly consistent with the personality hypothesis. In a study of VA patients who were administered the MMPI, Dattore et al. (1980) found clear evidence of greater repression of negative emotion among men who subsequently developed cancer. Grossarth-Maticek (1980) reported evidence of the same pattern based on a community sample of respondents in Yugoslavia who were administered a personality inventory at baseline and then followed prospectively for ten
Research has also been done on the personality predictors of survival among people who have cancer. Prospective studies find evidence that helplessness/hopelessness in the face of initial diagnosis is associated with poor prognosis after controlling for objective predictors (e.g., Greer et al., 1979). One recent study suggested that personality is not a predictor of survival, though, among patients with advanced cancer (Cassileth et al., 1985).

The major speculation about these influences is that personality affects the immune system, thereby changing host resistance to malignant transformation of cells. Evidence consistent with this view includes the documentation of higher incidence of cancer in individuals with prior immunologic deficiencies (Gatti and Good, 1971) and evidence of an association between personality and immune competence (Jemmott and Locke, 1984; Heisel et al., 1986). No research to date, though, has presented data documenting an intervening influence of immunocompetence on the relationship between personality and cancer incidence or course.

Arthritis: Research on an arthritis-prone personality has much the same history as work on personality and cancer. Unlike the latter, however, there have not been prospective studies to document whether premorbid personality predicts onset of arthritis. Instead, a series of studies have been carried out to examine the personality characteristics of people with early
symptoms of arthritis and have shown that their personality profiles do not differ from those in the general population. On the basis of this finding, it has been argued that the personality characteristics found to characterize people with advanced arthritis are probably results of the illness rather than causes (Anderson et al., 1985). It is important to note, though, that there have not been prospective studies to determine whether personality predicts course of illness. Rheumatoid arthritis is an autoimmune disease; from what we know about the relationship between personality and immunity it is entirely plausible that evidence of a distinct personality among advanced cases of arthritis could reflect an influence of personality on the course of the disorder. In Rheumatoid Arthritis, the incidence rate is twice as common among females as males, common personality and stress patterns in this disease are chronic inhibited hostility, resentment and smoldering discontent, often allayed to some extent by self-sacrifice and serving of others. Strong dependency needs and further hostility when such activities thwarted. Often obsessive tidiness, over conscientiousness and adherence to routine.

Coronary Heart Disease: Numerous poorly controlled studies show that coronary heart disease and hypertension occur more frequently to people who are neurotic or characterologically depressed. However, prospective studies show that personality changes on these dimensions follow rather than proceed diagnosis for heart disease and this seems to explain the cross-sectional evidence.
At the same time, there is some evidence to suggest that helplessness/hopelessness is a predictor of survival of a heart attack (Siegel, 1985).

There is considerable research showing that a coronary-prone behavior pattern called Type A is a risk factor for coronary disease. Type A is seen as the interaction between a set of personality predispositions and situations that elicit responses of extreme competitiveness, hostility, and a sense of time urgency. Prospective studies have documented that this pattern is associated both with onset of coronary heart disease and with recurrence. Furthermore, an ongoing prospective intervention experiment has shown that recurrence rates in men who had previous myocardial infarctions and is significantly lower in an intervention group designed to alter Type A (Matthews, 1985).

Some recent prospective studies have failed to find Type A to be a significant risk factor. Matthews (1985) suggests that the controlling style of Type A people is likely to make them particularly likely to engage in the health-promoting behaviors that have been widely publicized over the past decade and might account for the failure to find an effect of Type A on CHD risk in these recent studies.

At the same time, considerable work is going on to unpack the complex conceptualization and measurement of Type A behavior. It is not clear, at present, whether hostility or an overcontrolling interpersonal style or some other personality characteristic represents the core component of the Type A pattern that is consequential for health (Matthews, 1982); (Kasi 1984).
It is likely that advancement here will require more careful analysis of explicit interactions between personality characteristics and the specific kinds of environmental situations that provoke Type A responses.

Other Illness Outcomes: Parallel literatures exist on the personality determinants of asthma, ulcerative colitis, and a wide range of autoimmune diseases (e.g., Stout and Bloom, 1986). There is also fairly consistent evidence that low scores on the ego strength scale of the MMPI are positively associated with susceptibility to experimentally exposed viral material and with hypersensitivity to a variety of vaccines (Jemmott and Locke, 1984).

Das and Shukla (1990) found evidences for the role of personality in psychosomatic illness. When sample of 30 patients suffering from peptic ulcers were compared with a controlled group of 30 normals and 30 surgical cases, who were not suffering from any psychosomatic problem, the first group was found to be showing distinct personality profile.

Personality and Stress-Reactivity

Most research on pathogenic personalities is based on the hypothesis that particular personality characteristics interact with stressful life experiences to decrease host resistance to illness. Early laboratory studies documented that an interaction of this sort exists between experimentally induced mild stresses and personality in predicting a variety of physiological outcomes.
Unfortunately, subsequent research on the effects of major stress has not attempted to replicate this kind of analysis. Data are available to study the relationship between personality and stress reactivity in the numerous studies that have examined the effects of psycho social factors on susceptibility to infectious disease (Jemmott and Locke, 1984).

Data from general population surveys that examine mental health outcomes or self-reports of overall physical health are consistent with the hypothesis that personality influences stress reactivity. (Kobasa et al. 1982), for example, documented that a variety of personality characteristics assessed at baseline modified the relationship between subsequent stress and self-reported physical illness. Personality characteristics associated with resistance to stress included a belief that one can control his environment, openness to novel experience, and a predisposition to appraise potentially stressful situations as challenges rather than threats.

Kobasa and her colleagues have hypothesized the existence of a "hardy" personality constituted by high scores on the three personality dimensions investigated in the above research. However, several other attempts to replicate the results of their investigations by other researchers have failed to show that hardiness protects against stress (Cohen and Edwardes, 1987). Consistent evidence exists that a belief in personal control is a resistance resource (Fisher, 1984); (Lefcourt, 1985), though, and it is likely that this is the main dimension of the hardy
personality which is consistently important for health.

The stress-buffering effects of other personality factors have not been as extensively examined, but there is nonetheless replicated evidence for the importance of self-esteem, introspectiveness, alienation, and neuroticism (Henderson et al., 1981; Cohen and Edwards, 1987).

The Effects of Stress on Health

Three types of experimental literatures provide indirect information about the effects of stress on humans. One involves the physiological effects of stress on animals. Shuttle-avoidance experiments with mice, for example, document a variety of immune system responses that increase susceptibility to such experimentally induced infections as herpes simplex, poliomyelitis, coxsackie B, and polyoma virus (Turkham et al., 1982).

A second type of experimental evidence comes from laboratory studies in which humans are exposed to mild forms of stress. Research of this sort has shown, for example, that experimentally induced stress affects corticosteroid levels in humans (Plaut and Friedman, 1981). Although these effects are too small to significantly impair health, it is plausible that more serious stresses would have more greater effects. This could be important because corticosteroids play a part in the onset of diabetes, peptic ulcers, and hypertension (Miller, 1980).

Human stress exposure experiments have also provided information about psychological mediators of stress-reactivity,
including situational cues and individual difference variables which modify the appraisal of stress and, in this way, influence the physiological effects of these situations (Lazarus and Launier, 1978).

Finally, field experiments have been conducted among people exposed to serious stresses like job loss or widowhood (Price et al., 1980). These experiments manipulate some of the presumed intervening variables in the stress-illness relationship to study the protective influence of resistance resources. While not designed to evaluate the effects of stress directly, these studies document the range within which an association between stress and illness can be modified.

Research on Aggregate Life Events

Measures of health outcomes are usually based on self-reports. In case control studies, patients are administered a life event inventory and asked to report on the events that occurred shortly before illness onset. Their reports are compared to those of healthy control respondents. Associations between illness and retrospective life event reports have been found for coronary heart disease (Wells, 1985), some types of cancer (Scalar and Animal, 1981), several kinds of autoimmune disease (Solomon, 1981), diabetes (Kimball, 1971), mononucleosis (Roark, 1971), self-reports of miscellaneous somatic complaints (Petrich and Holmes, 1977), and symptoms of psychiatric disorders (Thoits, 1983).
There are a number of methodological problems which suggest caution in interpreting the influence of life events on illness. One is that some of the events may be results of ill health rather than causes. Being fired from a job, for example, might result from a prior mental health problem. Failure to adjust for this self-selection leads to upward bias in research on the health-damaging effects of unemployment (Kessler et al, 1987).

Another methodological problem is that poor health might be associated with better recall of recent stressful events, which would bias estimates of life event effects. Recent research by cognitive psychologists suggests that this kind of bias could occur in studies of mental health outcomes because depressed mood increases recall of depressing events (Blaney, 1986). There is also evidence that such bias may occur among the physically ill, with illness increasing the number of negative events that are recalled (Schroeder and Costa, 1984).

Some researchers advocate asking respondents to rate the stressfulness of events that occurred to them (Sarason et al., 1978).

Research on Life Crises

There is a long history of research on how people react to specific life crises such as bereavement, chronic illness or rape. A substantial minority of people do not recover emotionally with the passage of time (Silver and Wortman, 1980). Death of a spouse leads to increased mortality risk, reduced immunocompetence, and increased morbidity for a variety of physical and psychological...
disorders (Osterweis et al., 1984). The evidence on retirement documents no adverse aggregate health effects (Ekerdt et al., 1983).

Research on Chronic Stress

People with chronic health problems commonly report that they have a long history of stress in one or more of their major life roles. An obvious question is whether these chronic stresses cause the illnesses. The most finely developed literature that addresses this question deals with job stress. Several different approaches have been adopted. One is to compare aggregate mortality and morbidity profiles of different occupations that are comparable in all known risk factors other than job stress. This approach has yielded striking evidence that indirectly implicates job stress in worker health (Kasl, 1978).

Other investigators have used a multivariate approach to study the effects of job characteristics. The most persuasive of these investigations are based on longitudinal designs in which job demands are used to predict subsequent changes in health. These studies provide compelling evidence that job pressures and conflicts can initiate and exacerbate coronary heart disease, peptic ulcers, diabetes, and psychological distress (Cobb and Rose, 1973; Kasl, 1978; House and Cottington, 1984).

Illustrative results include the finding that academic pressure among students is associated with subsequent onset of infectious mononucleosis (Kasl et al., 1979) and that chronic tension in a prison population is associated with subsequent onset
of upper respiratory infection (McClelland et al., 1982).

People with chronic role stresses are more likely than others to develop an acute upper respiratory infection when randomly exposed to a nasal spray containing viral material rather than a neutral solution (Jackson et al., 1960).

There are numerous methodological difficulties in research on the effects of chronic stress. The health problems and chronic stresses have typically been present for such a long time that it is difficult to separate cause and effect. The task of making a causal imputation is even more difficult because one cannot assume that stress exposure occurred for reasons that were random with respect to the respondent's prior health. The possibilities of dealing with these problems are too complex to consider here, but are discussed by Kasl (1978) and Kessler (1986).

Another difficulty is that chronic stress measures are largely based on subjective reports. This is particularly true in studies of interpersonal stresses, but it also occurs in studies of job stress. In the latter, it is almost always found that subjective reports about chronic stress are more strongly related than more objective measures to illness outcomes. This could reflect selective perception due to illness or an intervening influence of appraisal.

Research on vulnerability factors represents an important new direction in work on the relationship between psycho social variables and health (social support, coping strategies and personality).
Social Support

The term "social support" has been widely used to refer to the mechanisms by which interpersonal relationships presumably protect people from the deleterious effects of stress. Interest in these mechanisms was triggered by a series of influential papers published in the mid-1970s (Caplan, 1974; Cassel, 1976, Cobb, 1976), which reviewed literature demonstrating associations between illness and such factors as marital status, geographic mobility, and social disintegration. They argued that a theme present in all of these associations is the absence of adequate social ties or supports or the disruption of social networks. Although highly inferential in their arguments, these early reviews generated great interest in the possibility that social support can protect health.

In recent years, this initial enthusiasm has been replaced by a more critical examination of the issues (Cohen and Syme, 1985). Researchers have become increasingly sensitive to the methodological problems in the early research and a new generation of studies has begun. Some of these new studies examine the relationship between life stress, social support, and health in normal population surveys or in case-control studies. Others examine the part played by social support in adjustment to particular life crises. Finally, a number of recent studies involve the experimental manipulation of support. Each of these is considered below.
General Population Studies

One line of investigation has examined the effects of network characteristics and social support on subsequent mortality and morbidity in prospective surveys of the general population. The most influential study of this sort showed that a number of network and support indicators (marriage, contact with family and friends, church membership, other group affiliations) were associated with reduced mortality risk over a nine year follow up period in a large sample of respondents living in Alameda Country, California (Berkman and Syme, 1979). Subsequent reports by Blazer (1982) and by House, et al. (1982) showed similar results in other longitudinal community surveys. Reed et al. (1983) failed to find any such association, though, in an analysis of Japanese-Americans living in Hawaii.

Similar longitudinal studies have studied the association between support and onset of physical illness. The most rigorous of these focus on coronary heart disease. Despite broad consistency in finding some indicator of support associated with reduced morbidity risk, there are numerous inconsistencies in these studies. In some, support is associated with subsequent disease incidence but not prevalence while in others the only significant predictions are associated with prevalence. The effects are limited to blue collar women in one study, while they are found only among men in others. The kinds of support indicators that are most important vary from one study to the next. Clearly, results of this sort do not help specify the ways support may be important for health (Berkman, 1985). While
research on the relationship between support and physical illness has focused on direct effects, research on support and psychiatric disorder has concentrated on stress-buffering effects. In an influential program of research, Brown and Harris (1978) showed that the impact of life events on depression is reduced among people who have an intimate, confiding relationship with a friend or relative. In the largest of the studies conducted by Brown's group, nearly 40% of the stressed women without a confidant became depressed compared with only 4% of those with access to a confidant. This result has subsequently been replicated in several community surveys and case-control studies (Cohen and Wills, 1985).

A group of investigators from Australia have shown that trait neuroticism disrupts close supportive relationships and that this personality characteristic is also associated with the exacerbation of stress effects. When neuroticism was statistically controlled in their analyses, the buffering effect of social support was explained away (Henderson et al., 1981).

Studies of Specific Life Crises

In the past decade, numerous studies have been conducted to assess the impact of social support on adjustment to specific life crises such as widowhood (Vachon et al., 1982), unemployment (Gore, 1978), and criminal victimization (Burgess and Holstrom, 1979). Almost all of these studies have been concerned with mental health outcomes. Most of these studies find support shortly after the crisis to be a significant predictor of
subsequent emotional adjustment. Moreover, these studies provide information about the importance of particular kinds of supportive ties for particular problems. For example, it has been found that when one of the coping tasks is to obtain new information or adopt a new role, low density networks can promote adjustment more effectively than high density networks (Hirasch, 1979).

Life crisis studies also provide an opportunity to examine social support in relation to other aspects of the stress process -- such as cognitions, feelings about the self and coping strategies -- and thus help clarify the situations. Unfortunately, the life crisis studies carried out to date have not realized their potential in these ways. Most of these investigations have simply attempted to show that support is associated with subsequent adjustment without linking support to other variables that might help elucidate causal processes. For progress to be made, the advantages of this research design will have to be more fully exploited in the future.

**Experimental Support Interventions**

Most experimental support interventions have been designed and implemented in hospital settings and examine the impact of support on such outcomes as preoperative anxiety, recovery from surgery, or compliance with medical regimens (Levy, 1983); (Mumfrord, Schiesinger and Glass, 1982). There have also been several support interventions to facilitate coping with life crises such as widowhood, rape, job loss and life-threatening illness (Leavy, 1983).
These interventions operationalize support in many different ways, although all involve both emotional and informational interactions with support providers. Most have been provided by health care professionals and have been modest in scope. They have generally involved limited resources and a small number of sessions. Nonetheless, in the vast majority of cases, these manipulations have been effective in promoting both emotional adjustment and physical recovery. Unfortunately, the interventions carried out so far have not been designed to illuminate the mechanisms through which these influences occur. Furthermore, as most of these interventions have been multifaceted, it is impossible to determine which aspects of support are most effective (Dimatteo and Hays, 1981) and (Wortman and Conway, 1985).

Coping and defence

Coping has not been worked out as a theoretical construct in any detail within learning theory terms, but is related to the second factor in two-factor-theory of avoidance learning, where the essential feature is that the second, instrumental phase reduces the drive state induced by the first factor (Rescorla and Solomon, 1967; Coover, Ursin, and Levine, 1973). The relevance of this formulation of two-factor-theory of avoidance learning for the animal model has been supported by neuropsychological evidence for independent neural mechanisms for these two factors (Ursin, Coover, Kohler, DeRyck, Sagvolden, and Levine, 1975).

In a recent study on parachutist trainees activation was
found to be gradually reduced to basal levels, when the trainees learned to master the training situation (Ursin, Baade and Levine, 1978). All physiological variables except heart rate showed a fall as coping developed. However, epinephrine still showed a rise before and after trials even if the total level reached decreased with progressive trials. There was also some suggestion that a short-lasting testosterone fall showed the coping effect. The activation unaffected by coping may be referred to as phasic activation, while the coping sensitive mechanism may be reffered to a tonic (Ursin, 1978).

Rose, Poe, and Mason (1967) analysed circulating hormone levels in 46 men undergoing basic military training. They found five factors, two factors related to androgens and estrogens, one catecholamine factor, one cortisol factor, and one factor related to thyroid function. The suprarenal cortical activity was most consistently correlated with effectiveness of the defensive or coping operations. Low epinephrine levels were associated with the ability to express anger openly. Persky, Zuckerman, and Curtis (1968) analysed 54 male subjects, 29 mental patients and 25 hospital employees, with a battery of psychological tests and measurements of pituitary hormones and adrenocortical activity. In a multiple regression analysis they found that adrenocortical activity predicted anxiety and depression. Follicle-stimulating hormone, luteinizing hormone, and thyreotropic activity related to an affect expression factor which derived from hostility and anxiety scores in an ink-blot test.

In a recent study of hormones in 71 parachutist trainees,
and in 44 trainees in a longitudinal study, Ellertsen, Johnsen, and Ursin (1978) identified three consistent endocrine factors. Their analysis included plasma levels of growth hormone, testosterone, blood glucose, cortisol and free fatty acid (n = 71) and the urine levels of epinephrine and norepinephrine in parachutist trainees (N = 31). Factor analysis revealed three consistent factors: A catecholamine factor, a cortisol factor, and a testosterone-free fatty acid factor. Inspection of the unrotated factors also revealed no general activation factor. Oblique rotations gave the same result as orthogonal rotations. The factors are almost orthogonal or independent of each other (Johnson, 1979, unpublished). In spite of this, the group data from the longitudinal study showed a clear relationship between the way all hormon systems reacted to a fear situation and mastery of the task. Eyman and Ursin (1979) have factor analysed blood values of physiological variables in a study of American Navy company commanders (n = 31) under considerable physical and psychological work loads (Ward, Rahe, Vickers, Hervig, Conway, and Ryman, 1979). Again no general activation factor was apparent. This was true also for the unrotated factor matrix. Again, a cortisol factor and a testosterone factor were evident. Two factors appeared to be related to catecholamines: One factor involving uric acid, protein and occasionally systolic pressure. Systolic blood pressure, however, also related to cortisol. These data, therefore, collected for a different purpose, seem to confirm the existence of at least three independent endocrine activation factors.
The three endocrine activation factors relate differently to important psychological dimensions, and this makes it possible to arrive at a quite specific psychosomatic hypothesis, specifically directed at the great variance in psychosomatic data.

In the two early factor analyses (Rose et al., 1967; Persky et al., 1968) particular relationships were reported between endocrine factors and psychological variables. These studies were done on "joint matrices" where endocrine and psychological data were analysed at the same time. In the Ellertsen et al., (1978) study, and in the Ryman and Ursin (1979) study, the evidence for independent endocrine activation systems appeared in analyses involving only physiological variables. When this had been established, analyses were also run attempting to reveal any psychological specificity of these factors. In the Ryman and Ursin study of the company commanders, few if any, such relationships were found. The psychological variables tested may not have been the important variables for the individual variance in that sample in the parachutist trainee study, however, consistent relationships were found. The following discussion is based mainly on these findings (Baade et al., 1978). Specific relations between psychological and endocrine variables have also been suggested from studies of one or a few hormones. These will also be discussed, even if multivariate studies are required for demonstrating the specificity of such relationships.
The Cortisol Factor

The cortisol factor related to high defense as measured by the Kragh Test (1960), and low performance in parachutist jumps, and with low performance in other performance tests. The correlations between the cortisol level and performance were consistently negative. The largest negative correlations were found for any tests with time pressure. The increase in cortisol was also associated with fear.

Other studies have also suggested specific relationships between cortisol and defense (Rose et al., 1967) and depressions (Sachar, Hellman, Roffwarg, Halpern, Fukushima, and Gallagher, 1973). The regulation process of the adrenal cortex is disturbed in depressed patients (Carrol, Curtis, and Mendels 1976). Plasma hydrocortisone levels relate to ratings of anxiety (Persky et al., 1968). However Sachar et al. (1973) found that the adreno-cortical activity in depressed patients was related to emotional arousal and psychotic disorganization, not to depressive illness per se. For many of the papers suggesting particular relationships between cortisol and "helplessness" or "conservation-withdrawal" it should be noted that the increased activation has not been demonstrated to be specific for the pituitary adrenal axis. Even so, a specific relationship between cortisol and specific psychological and psychopathological processes seems acceptable, and requires further research.

The Catecholamine Factor

The catecholamine factor found in the parachutist trainees
(Ellertsen et al., 1978) was based on urine samples. This factor related to performance in intellectual tests and also to performance in airborne parachutist jumps. This was also true for plasma levels of free fatty acids, which is another indicator of catecholamine activity (Norum and Ursin, 1978). Positive correlations between performance and catecholamines have been demonstrated repeatedly in single variable studies by Frankenhaeuser and her group (see Frankenhaeuser this volume). In the parachutist trainees, catecholamines related to activity need and impatience, in particular with boring routines. The data agree with Roessler (1973), who found higher psychophysiological reactivity in coping individuals, or in people with high ego strength. It should also be noted that there is a suggestive similarity in the description of these catecholamine responders and what has been referred to as Type A behavior (Jenkins, 1976). Type A behavior is characterized by intense striving of achievement, competitiveness, easily provoked impatience, sense of time urgency, abruptiveness of gesture and speech, over commitment to vocation or profession together with excesses of drive and hostility. This Type A behavior is claimed to be related to heart infarctions. Glass (1977) has shown higher autonomic reactivity in such individuals, but offered a different explanation.

The psychological differences between cortisol responders and catecholamine responders open several interesting possibilities for differential psychosomatic involvement, which has also been suggested by Mason and his group (Mason, Maher, Hartler, Mougey, Perlow, and Jones, 1976).
The Testosterone Factor

In the parachutist trainees, testosterone was associated particularly with feminine identifications in the Kragh test and a particular "masculine" role factor evident from several psychological tests. Testosterone was also related to preference for thrill and adventure. It seems as if testosterone picked up particular types of threats to the individuals, mainly related to the masculine role in general, and the particular role of becoming a parachutist. Rose et al., (1967) found that androgens and estrogens correlated with particular assessments of the psychological state, but this was not characterised further. This is the least defined factor from a psychological point of view. The role for somatic pathology is also uncertain, and this factor will not be dealt with further.

SUSTAINED ACTIVATION PRODUCES SOMATIC PATHOLOGY

The strongest case for this theoretical position is the reciprocal set of data on the ulcerations in the non-coping rat (N. Miller), and the reduction of activation observed in the coping rat (Coover, Ursin, and Levine, 1973). This model is also the best model for psychosomatic disease from a psychological point of view. The psychological conditions for ulceration to occur are reasonably well established, and are accounted for by the concepts lack of control, and lack of coping (Weiss, 1972; Murison). According to the theory presented here, these psychological conditions lead to somatic changes through sustained or persistent
activation. Lack of coping, according to the definitions offered here, means that activation persists. (Murison) discusses further the application of this theory for the ulceration model.

Another well developed model is the spontaneous hypertensive rat, where the vascular changes have been described in detail (Folkow, 1975), but where less details are known about the psychological factors, except that sustained activation again seems to be a crucial mediator. Ulcerations and heart infarctions may occur even in individuals that objectively are performing well, when demands are very high, requiring constant, sustained activation, as in conflict situations, or under Sidman schedules (Murison, Corley, Shiel, Mauck, Clark, and Barber, 1977).

Sustained activation may be the possible mediator also for human psychosomatic disease. Life changes do produce disease (Rahe and Arthur, 1978), but only in a majority of individuals (Rabkin and Struening, 1976). When the coping mechanisms are insufficient for mastery of the situation, then health risks occur. Rahe and Arthur (1978) hold that all life events, including positive ones, constitute health risks, but only if the life event requires adjustment. Theorell found that distress was a crucial factor for disease occurring due to life events (Theorell, 1976). Lundberg and Theorell (1976) found that subjective scaling of life events improved the predictive value of life change indicators, health risk arises when activation is sustained for prolonged periods of time.

This seems also to be true for somatic problems related to work load. Work load related myocardial infarctions only occur
when there are problems related to the work situation (Theorell and Flioderus-Myrhed, 1977). The importance of job dissatisfaction as a risk factor has also been pointed out by Sales and House (1971). Gardell (1977) has reviewed recent Scandinavian data from working life research which clearly indicate that somatic and psychological health as well as general well being depends upon subjective control over the work situation. The highest disease-risk group is that which has the highest work load with least control over the work situation and the time pacing of the work operations. All these data agree well with the animal model of coping as the important dimension for somatic health, and the position of this paper that the link between the psychosocial factors and the somatic health is sustained activation.

The time course of activation may be a crucial factor in the pathogenesis of psychosomatic disease. In the coping experiments, trials or sessions are separated by 24 hours ("spaced learning"). In the ulceration and heart infarction studies, sessions are continuous, often for 24 hours ("massed trials"). The circadian rhythm is disturbed, the rats are sleep deprived, and in the ulceration studies also food deprived. Deprivation of food and sleep produce activation changes by themselves. These models, therefore, involve sustained activation, in differ from the coping experiments which involve acute and fairly short lasting activation sessions. The sustained activation seems a particularly good model for distressful events and chronic unsolvable conflicts in humans. Weiner (1977) has pointed to the possible role of disturbed circadian rhythms for producing
psychosomatic disease. This is particularly important that bereavement and distress are important for the development of psychosomatic disease.

Cortisol responders

In parachutist trainees there was a consistent relationship between cortisol, defense mechanisms and poor performance (Baade et al., 1978). The defense mechanisms interfered with an adequate and accurate evaluation of the threatening environment, which resulted in poor performance in dangerous occupations. The poor performance also affected standard performance tests. The coping level should be relatively low, and correspondingly the risk for psychosomatic disease should be high. However, the high defense mechanisms might defend the individual also against a realistic assessment of his objective performance. Such individuals might reach their incompetence level faster than others, but their psychological traits may also defend them against realization of their ill fate.

Psychosomatic pathology in this group should be related to cortisol. No specific models for such pathology exist, except for a suggestion by Henry and Stephens (1977) that cortisol pathology may be related to defective immune response mechanisms. Cortisol has also been related to gastric or duodenal ulcers, but this has been questioned recently by Weiner (1977). If cortisol is related to depression and distress, specific pathology may be expected from such states in cortisol responders. As mentioned previously, Theorell found distress to be an important psychosomatic factor.
Finally, high defense mechanisms are not always any handicap. It may be of particular importance in surgical emergency situations where body systems are due to collapse, and when even a moderate activation may be more than can be tolerated.

**Catecholamine Responders**

Catecholamine responders reacted strongly to the situation, they coped well, and showed the corresponding rapid fall in activation (Baade et al., 1978). The same characteristics are true for Roessler's high ego strength person (Roessler, 1973). They should, therefore, be well defended against psychosomatic pathology when coping is possible. However, their mode of physiological and psychological reactivity becomes a handicap if they experience a situation beyond their control. When they reach their "incompetence level" they may be worse off than their well defended cortisol counterpart. In all situations with sustained activation high reactivity may be a risk. Since catecholamine pathology obviously related to cardiovascular pathology, this may be the underlying mechanism for the claims of relationships between Type A behavior and heart infarctions.

As indiscriminate treatment of all Type A behavior or other high reactions as undesired seems inadequate. It makes little sense to reduce general coping potential and ego strength as a therapeutic endeavor. An effective and gifted performer is more likely to gain control over this environment, which is the most important aspect of coping (Frankenhaeuser and Rissler, 1970; Weiss, 1972). The catecholamine responders should be recommended
to accept challenges and their work situation, but only as long as their subjective estimate tells them that they master their situation. The pathological mechanisms involved have been illustrated in particular for cardiovascular pathology. It has been demonstrated that in genetically predisposed rats hypertension with pathological changes of the vascular bed is produced when rats are subjected to repeated and strong activation (Folkow, 1975). Similar changes have been produced in rats by sustained conflicts of territory (Henry and Stephens, 1977). Progressive increase in blood pressure and heart rate occurred together with atherosclerosis in mice, and renal damage also occurred.

Catecholamines are also involved in regulation of the plasma levels of free fatty acids (Norum and Ursin, 1978), and free fatty acids showed the same relationship to psychological traits as the catecholamines (Baade et al., 1978). Multifactorial analyses of heart infarctions identify serum cholesterol, blood pressure, cigarette smoking, diabetes and angina pectoris as risk factors (Goldbourt, Medalie and Neufeld, 1975). Serum cholesterol and blood pressure are obviously related to catecholamines. Cigarette smoking was higher in the Type A persons in the original reports on the relationship between Type A personality and heart infarctions (Friedman and Roseman, 1959). Psychological traits seem to be important links between environment and cardiovascular health, and further research should exploit this possibility of more specific psychosomatic hypotheses.

Not much material is available in this area for the present
research but various studies are done in the area of personality and activation which have served the purpose of framing the objectives and to develop the methodology. It also helped in understanding and interpreting the results and conclusions.