RHEUMATOID ARTHRITIS: A COGNITIVE-BEHAVIOURAL INTERVENTION

BY

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CONTENTS

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ABSTRACT

This study investigated both the mediating role of psychological adjustment in determining pain experience, disease status, and immune function in Rheumatoid Arthritis (RA), and the value of cognitive-behavioural intervention in improving the overall health status of such patients. Two related hypotheses were tested in a matched-random assigned two-groups design, with pre-, mid-, and post-intervention assessment. Fourteen (N=14) female RA out-patients, selected along established inclusion criteria, were allocated to either treatment (n=8) or control (n=6) groups after being matched on date of disease onset and ratings of coping efficacy. The treatment group received an eight week Stress Inoculation and Pain Management Training programme (sixteen 2-hour sessions) based on the conceptual approach of Meichenbaum (1985) and adopted from a program by O'Leary, Shoor, Lorig and Holman (1988). The program included educational material, instruction in palliative and cognitive pain management strategies and the application thereof in daily living, goal setting to improve activity function, and group discussion. The program was designed to nurture and develop existing coping skills, and to impart new strategies to cope with daily stress and pain.

Pre-intervention correlational analyses tested the extent to which mood disturbance, self-perceptions of coping efficacy, health locus of control and stressful life experience were related to intensity and quality of pain, disease activity, functional status and lymphocyte proliferation rate. Intra- and inter-group analyses were conducted to determine treatment effects in terms of change scores on the dependent measures, and case studies were conducted to evaluate individual response both to disease and cognitive-behavioural intervention.

RA was characterized more by poor psychological health status than physical disability. Such patients showing marked mood disturbance and poor perceptions of coping efficacy. Pain was found to be more a function of psychological adjustment than actual disease status, with the meaning of the pain, and the accompanying mood disturbance being the most salient factors of the pain experience. Lymphocyte proliferation rate was normal, and unrelated to disease or psychological variables. No significant treatment effects were found at intra- or inter-group levels of analysis. Case studies indicated the complex nature of the individual disease state, and the RA patient's response to psychological intervention. The value of cognitive-behavioural intervention in RA, and implications for future psychoimmunological research in RA are discussed in terms of such findings.

SUMMARY

The care of the Rheumatoid Arthritis (RA) patient reflects an important shift in our health care system's treatment of chronic disease patients. The role of the multidisciplinary team in considering the physical, psychological and social well-being of the RA patient is becoming a particularly important area of concern in traditional medical care. This thesis explores the role of health psychology in ameliorating the psychological impact of RA and its associated chronic pain.

The thesis consists of seven chapters, the first three providing a review of relevant literature pertaining to the involvement and inclusion of psychological theory and practice in health and disease. Chapter four outlines the central thesis of this work, and chapters five to seven are concerned with the nature of the study, and the outcome of the cognitive-behavioural intervention with a sample of RA patients.

In Chapter One, the background to the involvement of behavioural medicine and health psychology in the investigation and treatment of chronic disease is delineated. The chapter presents a broad overview of the field of behavioural medicine, and the historical and theoretical developments in models of disease contributing to the involvement of psychology in health maintenance and care. The historical changes in the epidemiology of disease and models of causality, the import of systems theory, and changing trends in psychology, has allowed behavioural medicine to challenge our concepts of disease and the treatment thereof. As such, behavioural medicine also challenges the demarcation of boundaries of professional responsibility in patient care. The new conceptualization of disease as a multidimensional experience and the result of the interplay among multiple factors, requires an expansion of professional boundaries in the care and treatment thereof (e.g., multidisciplinary involvement). Chapter One also introduces the synchronous systems model (Jasnoski & Schwarz, 1985) of health and disease. In this model, the individual is placed at the center of the interplay among environmental, physiological/biological, social, and psychological systems. Changes at any level of the human system are ultimately interpreted in terms of their impact on the individual person. The model, however, does not consider the person to be a passive object of these forces. Rather than merely adapting to the dynamic forces of these systems, the individual "optimizes" his/her environment to a greater or lesser degree. The influence of psychological and behavioural factors in governing this process of optimization becomes an important and legitimate area of research and practice.

To provide evidence of the role of psychological processes and experience in the onset and course of disease, Chapter Two explores the nature of the relationship between stress and disease. The cognitive-behavioural paradigm is identified as providing the most lucid conceptualization of the complex processes involved in the stress-disease relationship. It is argued that Everly's (1988) model of the human stress response, which utilizes a systems theory perspective, is able to delineate the mechanisms linking psychological experience and altered physical health status. Research documenting the relationship between stress and disease provides central support for these models.

The bulk of Chapter Two is devoted to a detailed review of the research exploring the links between psychosocial stressors and the incidence and progression of disease. The two main fields of investigation, psychosomatic medicine and psychoimmunology, are introduced and reviewed. Research findings from these two fields of

investigation demonstrate the adverse effect various psychological stressors have on health status and immune function. Such stressors include emotionally traumatic life events (bereavement, marital disruption, unemployment, academic examinations) and adverse psychological experience (depression, distress, anxiety, perceived loss of control, caring for chronically ill, and even exposure to environmental threat). The clinical significance of such stress-related immune system "dysregulation" is then reviewed in relation to the susceptibility, onset, and outcome of infectious and chronic disease. The chapter concludes with an examination of the value of psychological intervention, particularly cognitive-behavioural programs, in ameliorating the noxious effects of stress on health status in "high risk" patient populations (i.e., diseases associated with immunological dysfunction such as neoplastic and autoimmune disorders).

The prevalence of cognitive-behavioural techniques utilized in the psychoimmunological interventions with various patient populations, provides an indication the importance of this paradigm within health psychology. In Chapter Three, RA is identified as an important and appropriate disease condition to explore the value of psychological research and treatment interventions. RA is an ideal disease to investigate the assumptions of the psychoimmunological and cognitive-behavioural paradigms. RA being a complex disease phenomenon of unknown cause and unpredictable course, involving various physiological systems, autoimmune mechanisms, and patient characteristics. A central argument in this chapter is that the multidimensional nature of the disease demands a radical reformulation of its management. The chapter discusses the psychological impact of RA, and shows how psychological factors, particularly emotionally traumatic life events and maladaptive coping strategies in response to such events, probably play a contributing role to the onset and course of RA. A key underlying assumption held by the cognitive-behavioural approach to RA, is the central role played by cognitive factors (i.e., appraisal) governing the patient's response to his/her disease. As a result of the psychological impact of RA, it ia argued that the RA patient's appraisal of their efficacy to cope with their disease is severely undermined.

Perceptions of poor self-efficacy are shown to mediate the adverse effect of the stressors associated with RA on the patient's psychological and physical health status. Chapter Three concludes with a review of research investigating the value of cognitive-behavioural intervention in rheumatoid arthritis. These cognitive-behavioural interventions utilize strategies aimed at helping the RA patient to achieve greater self-control over their disease. By taking on more responsibility for its management, the RA patient becomes more actively involved in the treatment process and is encouraged to adopt a more adaptive response to the disease. Chapter Three thus attempts to incorporate the models introduced in Chapter One and the research evidence reviewed in Chapter Two, relate these to a particular disease, and provide a basis for the thesis presented in Chapter Four.

In Chapter Four a thesis is presented which links psychological adjustment, pain, disease activity, functional status, and immune function in RA. These variables are examined in the context of a cognitive-behavioural intervention based on Turk, Meichenbaum and Genest's (1983) and Meichenbaum's (1985) stress inoculation training treatment rationale. Cognitive pain management strategies are included in a "stress inoculation and pain management training" programme designed to: (a) improve the psychological adjustment and immune function, and (b) decrease the pain, functional disability and disease activity, of RA patients. It is argued that it is necessary to provide a model of the complex interrelationship among these variables in RA, delineating the central mediating role of psychological adjustment in the disease process. The bulk of the chapter is devoted to explaining the model and the variables to be operationalized and investigated. Two hypotheses underlie the thesis: (1) there is a

relationship between psychological adjustment and pain, disease status and immune function; and (b) cognitive-behavioural intervention would have significant benefit for RA patients.

In Chapter Five the method and procedures undertaken to test these hypotheses are reported. This chapter includes a description of the "Stress Inoculation and Pain Management Training" (SIPMT) programme conducted with a sample of chronic RA patients. The responses of these patients were compared to those of a matched control group at pre-, mid-, and post-intervention along the variables operationalized. Fourteen case studies are presented to demonstrate individual patient characteristics and responses to their disease and the intervention.

Results reported in Chapter Six and discussed in Chapter Seven indicate that RA has a significant psychological impact in terms of mood disturbance and poor perceptions of self-efficacy in coping responses to stressful events. An important finding is the extent to which the pain experience of the RA patients examined is shown to be more a function of psychological well-being than tissue damage or disease activity. The model depicting the interrelationship among psychological adjustment, pain, disease status and immune function is only partly supported. Furthermore, the SIPMT is not shown to provide significant improvement along the measures utilized. The case studies reveal interesting findings regarding the complexity of the individual RA patient's response both to her disease as well as the psychological intervention.

Chapter Seven provides a discussion of these findings in relation to previous research, the methodology utilized, and the patients responses during the intervention. The chapter concludes with recommendations for future research investigating the value of cognitive-behavioural interventions in RA. Of particular concern are subject characteristics (inclusion/exclusion criteria), methodological concerns (the nature of the intervention as well as the operationalization of key variables), and the value of examining case studies in such research.

CHAPTER ONE

PSYCHOLOGY AND HEALTH: AN INTRODUCTION

Overview

This chapter provides a broad overview of the field of behavioural medicine (a branch of health psychology), and the historical and theoretical developments in models of disease contributing to the involvement of psychology in health maintenance and care.

The purpose of such a discussion is twofold. Firstly, it is to provide a broad background to behavioural medicine against which the relevance of this thesis can be evaluated. Secondly, it is an overview of the historical development in our concepts of disease and health, delineating the three major models used to conceptualize and explain the causes and nature of disease. The Biomedical, Biopsychosocial (Engel, 1977), and Synchronous Systems (Jasnoski & Schwarz, 1985) models are considered in the context of other historical developments. These include: (1) the changes in the epidemiology of disease, (2) the increasing recognition of co-factors in the disease process following research investigating the role of stress and other psychosocial processes, (3) the contribution of General Systems Theory (von Bertalanffy, 1968), and (4) the changes occuring within psychology, such as interactionalism, which prepared psychology for the present collaboration with medicine in physical health care.

It is argued that the Synchronous Systems Model (Jasnoski & Schwarz, 1984) provides a framework to challenge the professional responsibility of psychology in health care practices. Having more than mere heuristic value, the model provides a means by which psychology may redefine its role in preventing, treating and managing human "dis-ease". By viewing disease as multifaceted and multidetermined, the influence of psychological factors and behaviour in all stages of the disease process become important and legitimate areas of concern, practice, and research.

1. Behavioural Medicine: Understanding Health and Disease

The epidemiology of mortality and morbidity is changing in industrialized countries (Matarrazo, Weiss, Herd, Miller & Weiss, 1984). As chronic disease has replaced infectious disease as a major medical challenge, "...behaviour and life-style have become more important as the context [own emphasis] of health and disease." (Jasnoski & Schwarz, 1985: 471) The burden of illness has shifted to deaths and disabilities in which behaviour plays an important role. Such behaviours include patterns of malnutrition, pathogenic inactivity, excessive use of drugs, noncompliance with medication regimens, and maladaptive responses to stressful experience (Matarrazo et al., 1984).

The "one-germ, one-disease, one-treatment" tenet upon which traditional disease theory is based, is undergoing a radical reformulation (Engel, 1977; Lipowski, 1977). There is, according to Turk, Meichenbaum, and Genest (1983), "...a shift in focus with regard to improving the quality of health --a shift from external factors, such as microorganisms and poor sanitation, to behavioural problems determined by the interaction among factors internal to individuals, the social environment, and the physical environment." (p.19, own emphasis)

Behavioural medicine (Agras, 1982; Pomerleau, 1982) represents this "shift in focus", where the notions of disease (onset, course, and outcome), illness, and illness behaviour are being re-examined and re-defined. "Illness" refers to the patient's "subjective experience of the objective disease", and "illness behaviour" refers to the processes resulting from the recognition of disease, how symptoms are defined, and the actions taken by the patient seeking help (Turk et al., 1983).

Disease onset may be associated with a number of factors, including "...[the] presence of stressful environmental conditions, perceptions by the individual that such conditions are stressful, the relative ability to cope with or adapt to these conditions, genetic predisposition to a disease, and the presence of a disease agent" (Rabkin & Struening, 1976: 1014). Ader (1980), for example, provides an extensive list of major risk factors that influence the maintenance, disruption and/or restoration of what he called the "homeostasis" of the individual.

The course and outcome of a disease would thus depend not only on the physical and psychological state of the patient, but also on the ways in which health care workers, significant others, and employers react to the situation. According to Turk et al. (1983), "the form and magnitude of illness and illness behaviours are products of both subjective experience and social definitions, as well as the severity and quality of actual symptoms and physical incapacity." (p.21)

Behavioural medicine, falling under the broad rubric of health psychology, has been defined as "...an interdisciplinary field concerned with the development and integration of behavioural and biomedical science, knowledge, and techniques relevant to health and illness and the application of this knowledge and these techniques to prevention, diagnosis, treatment, and rehabilitation." (Schwarz & Weiss cited in Matarrazo et al., 1984: 28)

The historical changes in the epidemiology of disease, and the development of new theoretical systems to accomodate for such changes, have thus created a context wherein novel approaches to the prevention and treatment of chronic disease have emerged (Matarrazo et al., 1984). Such approaches are grounded in an implicit understanding of the cause of the particular disease (i.e., the set of key aetiological factors), and the impact such disease has on the patient.

It is important to examine the development of causal models used to explain disease, and to determine the empirical validity of such generated models, prior to an examination of the actual application of behavioural medicine. Behavioural medicine is in fact a transgression of boundries (both professional and personal) which has been made possible by the development of new conceptualizations of what constitutes disease or health. In his landmark paper, Engel (1977) remarked that "...the importance of how physicians conceptualize disease derives

from how such concepts determine what are considered proper boundries of professional responsibility." (p. 129) Behavioural medicine questions our concepts of disease, thereby challenging the demarcation of these boundries of professional responsibility. As such, psychology will have an increasingly larger role to play in the treatment and prevention of disease and the care of illness.

1.1 Models of Causality

1.1.1 The Biomedical Model

The classic biomedical approach to understanding and conceptualizing human welfare within a dynamic ever-changing environment is increasingly being viewed as problematic (Jasnoski & Schwarz, 1985). Based on molecular biology, such a model reduces the disease experience to only physiological phenomena with linear causation (Engel, 1977). It can be seen as an inherently reductionistic conception of person-situation relational dynamics. Being a linear "cause-and-effect" model of illness and disease, that is, the search for an intruding noxious agent, it is an ineffectual model to apply to the complex nature of chronic disease processes (Jasnoski & Schwarz, 1985; Matarrazo et al., 1984; Engel, 1977).

The classic "germ theory" that person X exposed to pathogen Y will develop the disease caused by agent Y has thus almost universally been rejected (Ader, 1980). As Ader (1980) wrote: "One simple, universal observation underlies psychosomatic research. It is the observation that when a population of individuals is exposed to the same environmental pathogens, only some individuals manifest disease." (p. 307) Whilst one must acknowledge the existence of "germs" or viruses, the chain of causality from exposure to disease onset is neither simple nor unconditional (Jasnoski & Schwarz, 1985). A host of co-factors, both physiological/biological and psychosocial, co-determine the disease process .

The biomedical model is thus unable to truely explain the origins of chronic disease without considering variables beyond molecular biological dynamics, such as life-style and personality factors. In terms of causality then, according to Jasnoski and Schwarz (1985), "...a biomedical dysfunction appears necessary but not sufficient to explain crucial aspects of chronic disease, such as time of onset, severity and course, and acceptance of the sick role [own emphasis]." (p.471)

1.1.2 The Biopsychosocial Model

Engel's (1977) "biopsychosocial" model of health and disease subsumed the biomedical model as it took into account "stress" and environment-person transactions, as well as physical phenomena. Claiming to be a more inclusive scientific medical model (Engel, 1977), it represented an early break from a uni-directional, cause-and-effect analysis of disease. The model offered a concept of multiple causation and bi-directional interaction between factors internal to the individual, and between person and environment. The model thus challenged traditional biomedical approaches to disease, and constituted the apogee of behavioural medicine's research endeavour (Schwartz, 1982). Engel (1977) argued that the biomedical model did not suffice; that its framework was too exclusive.

> To provide a basis for understanding the determinants of disease and arriving at rational treatments and patterns of health care, a medical model must also take into account the patient, the social context in which he lives, and the contemporary system devised by society to deal with the disruptive effects of illness, that is, the physician's role and the health care system.
>
> (Engel, 1977: 132)

According to Jasnoski and Schwarz (1985), whilst this model was initially based on a dualistic understanding of mind and body, proponents of the model are moving towards a monistic understanding that "...the body and mind are simply different manifestations of a fundamental unity, the person." (p.472) Despite such changes, however, the term "biopsychosocial", as Jasnoski and Schwarz (1985) point out, "...still highlights the distinctions without focussing on the striking similarities." (p.472)

Whilst of heuristic value to the researcher, it is argued that the biopsychosocial model does not offer a completely satisfying account of the possible relationships between variables from anomalous systems. How can it begin to conceptualize the interplay between psychological experience and endocrine or immune system changes? It is not possible to begin to explain the interplay between biological and psychosocial systems without some explanation of how such systems are "connected" or interrelated (Jasnoski & Schwarz, 1985). Accordingly, it was necessary to construct a model which could present such interplay in a systematic and logical manner. The input from general systems theory provided a major impetus to our conceptualization of the relationships between such divergent "systems" and the development of workable models of human welfare and disease (Jasnoski & Schwarz, 1985).

The Contribution of General Systems Theory (GST)

General systems theory (von Bertalanffy, 1968) was developed as a response to the need for a reorientation in scientific perspectives, aiding investigations into life processes more amenable to scientific enquiry and conceptualization (Engel, 1977). By treating sets of related events collectively as hierarchical systems with their own particular functions and properties, it was possible to recognize "isomorphies across [the] different levels of organization." (Engel, 1977: 134) General Systems Theory holds that as all levels of organization are linked to each other in a hierarchical relationship, so changes in one system affects change in the others. To demonstrate this interrelationship, the theory utilizes a computer metaphor model of information processing and feedback loops (cybernetics). Systems theory's functional analysis is thus based on the organismic (synergism) principle that the whole determines the functions of the individual parts (Jasnoski & Schwarz, 1985). The system's function and structure in totality has greater import than the sum of the independent parts or components.

General systems theory thus "...conceptualized the means...whereby homeostasis, environment-physiology interaction, and change could occur." (Jasnoski & Schwarz, 1985: 470) It provides a coherent account of the possible relationhips between "anomalous" systems, and its underlying synergistic principle contributes significantly to preventing a reductive analysis of data. As such, its introduction provided the conceptual break from dualistic notions of the interplay between anomalous systems, to a monistic notion of the subservience of such systems to a greater totality.

Changes in Psychology

Psychology was also undergoing a series of paradigmatic shifts during this period of upheaval. Jasnoski and Schwarz (1985) provide an analysis of the traditional schools of thought, and discuss the two basic approaches to understanding human psychology. The "personalism" (i.e., having an individualistic bias, focussing on trait and personality theories) and the "situationalism" (i.e., neglecting the internal processes of the individual in favour of social forces and focussing on social learning theories and behaviourism) schools differed radically in their basic assumptions regarding human nature. As with medical science, psychology was limited by its dualistic approach to human nature, such an analysis being inherently reductionistic.

The advent of interactionalism and its meta-theoretical analysis of the interplay between these two extremes (e.g., the nature-nurture type debates) provided a conceptual leap in understanding relational dynamics within the field of psychology (see Riley, 1983). Psychology could now consider three factors underlying human psychological experience: the individual, society, and their interaction. Yet, as Henriques, Holloway, Urwin, Kenn and Walkerdine (1984) have argued, psychology is still locked in a fundamental dualism, despite the impact of interactionalism.

As with the "biopsychosocial" model, interactionalism continues to highlight the differences between the "individual" and "society", between "nature" and "nurture" without focussing on the striking similarities underlying such concepts. That is, psychology persists in considering the individual as somehow separate from the human system (the physical -both internal and external-, and social environments) in its totality, by assuming a process of "interaction" (Henriques et al., 1984). This dualism has an all pervasive effect on psychological knowledge in that it prohibits psychology from proceeding to a new level of analysis where the two poles could be subsumed as part of a greater totality. Henriques et al. (1984) attempted to introduce a new concept, "the social", which would somehow escape a dualistic understanding of human nature.

The difficulties facing psychology in attempting to transcend traditional conceptualizations, however, are reminiscent to the problems faced by medicine as indicated above by Engel (1977). Psychology's concepts determine the demarcation of the boundries of professional responsibility, and thus influence how psychologists conceptualize the well-being of their client/patient. Psychology, by virtue of its role in constructing both (1) itself as a profession, as well as (2) its objects (i.e., human subjectivities) of investigation (see Henriques et al., 1984), has remained relatively uncertain of the value of its contribution to medicine. Despite these limitations, interactionalism, according to Jasnoski and Schwarz (1985), "...prepared psychology for the present confluence with general systems theory from science and medicine." (p. 470)

1.1.3 The Synchronous Systems Model

It is from this point that a new model of understanding human health and disease is developed by Jasnoski and Schwarz (1985) in the form of the "synchronous systems model". The synchronous systems model (hereafter the SSM) is a further development in the conceptualization of person-context dynamics. Combining science, medicine

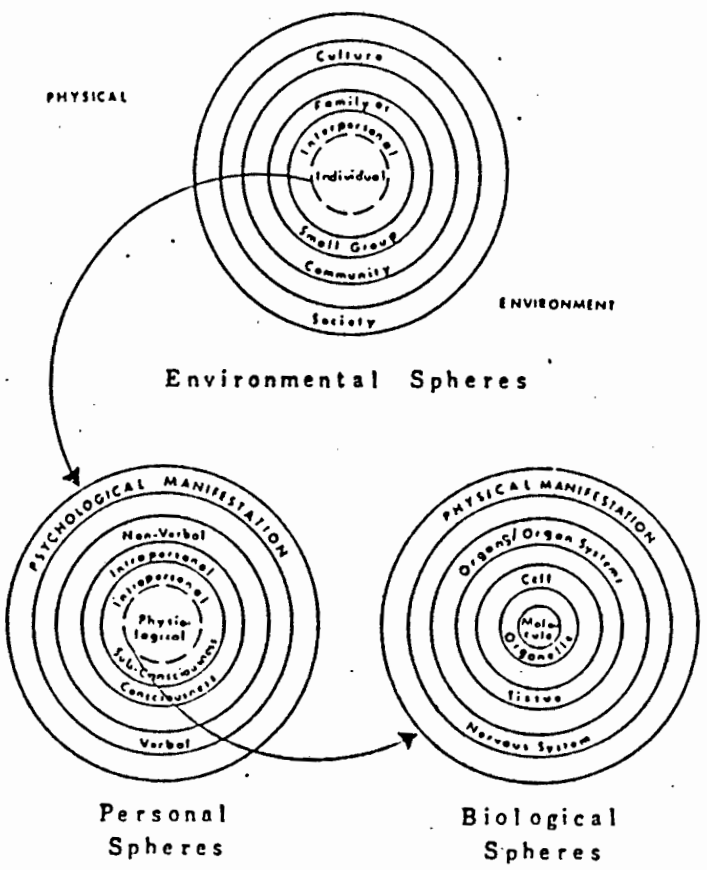
and psychology, it applies general systems theory to a biopsychosocial and ecological structure, depicting spheres of influence on human functioning (Jasnoski & Schwarz, 1985). Its authors claim to offer:

> ...a comprehensive conceptual framework for processing and utilizing a vast amount of information,...[increasing]...the explanatory power available to health service personnel...because conceptual boundries expand to include the wide range of data that different disciplines can furnish with their unique, highly developed technology.
>
> (Jasnoski & Schwarz, 1985: 468)

The structure of the model relies on human ecology's delineation of the "external" environment (both psychosocial and physical), and biomedicine's delineation of the "internal" environment (the descending levels of function in the body).

Figure 0.1 provides a two-dimensional representation of the structure and interplay between the biological, personal, and environmental spheres. The model argues that the individual has multiple levels of analysis, where at any given level, "each system is composed of parts that are themselves sub-systems, and each system is simultaneously part of at least one larger system, termed a suprasystem." (Jasnoski & Schwarz, 1985: 475)

Figure 0.1: The Synchronous Systems Model (adopted from: Jasnoski & Schwarz, 1985)



A fundamental implication of such a model is that when studying human phenomena at a given level/system, one should take into account relevant processes occuring at both lower (subsystem) and higher (suprasystem) levels, if one wishes to be succesful in fully understanding the particular system of interest. It is this aspect of the model that is of most value to the researcher working within the field of behavioural medicine.

The SSM provides a schema to justify the importance of the inclusion and comparison of the biological, personal and environmental aspects of the chronic disease patient. The model functions in terms of the regulation of stability between these dynamic spheres of influence (i.e., the homeostatic principle). Health and disease are defined systematically "...via the states of synchrony and dysynchrony [respectively], along with the processes of regulation and dysregulation." (Jasnoski & Schwarz, 1985: 477) "Dysregulation" is a new term introduced by Jasnoski and Schwarz (1985) and will be utilized throughout this thesis when describing immune system functioning in the individual. "Dysregulation" is thus a term specific to the SSM of health and disease. "Synchrony", in terms of this model, "...means 'timed together' or 'simultaneously', [referring to]...system functioning... characterized by contemporaneous rather than temporally linear patterns of process." (Jasnoski & Schwarz, 1985: 472) The model is thus based on a complex conceptualization of a structure of systems whose processes exist and function concurrently. This existence and function relies on the successful interplay between systems functioning synergistically. Synergistic meaning that "...the whole has an impact greater than the simple sum of its component spheres." (Jasnoski & Schwarz, 1985: 475) Whilst the model argues that each "sphere" requires analysis in evaluating health or disease, all events and experiences are ultimately interpreted as to their impact on the individual human (Jasnoski & Schwarz, 1985). As such, the individual occupies the fulcrum role (between environmental and biological spheres) in the synchronous model, as indicated in Figure 0.1.

Disease becomes "dis-ease", where the total system has lost its homeostatic equilibrium and where dysregulation in a particular system has affected the overall integrity of the total system, and ultimately the health of the individual. Disease, however, is still considered as an objective phenomenon, as it entails a dysregulation in the biological sphere (e.g., inflammation in joints). Such dysregulation has an influence (depending on the magnitude of the event) on the personal sphere, and is translated into a subjective experience of illness (e.g., pain and discomfort). Illness behaviour constitutes the actions taken by the individual in the environmental sphere of influence. Here, the constellation of the activities by the individual, his/her family, and medical services, constitute an attempt to reintroduce a state of regulation.

The health of the individual becomes a function of his/her successful adaptation to or adjustment within the system as a whole. It is important, however, to recognise that the concept of adaptation is limited. "Adaptation" entails the transaction between a person and environment, where the person "adapts" to changes at a higher (i.e., interpersonal) or lower (i.e., biological) level. Implicit to the concept of adaptation, is the perception of the person as a passive subject adapting to changes in his/her environment, and in no way affecting changes or making decisions regarding the nature of that environment. Individuals however, do have an impact on the nature of their environment through their actions or behaviour. Consider the effect that type-A behaviour has on creating an environment wherein the potential for stressful experience is increased. The impact that human actions have on the environment in terms of pollutants is yet another, though extreme, example of the active role played by the person within the total system.

Jasnoski and Schwarz (1985) introduced the idea of "optimization" to recognize the system effects of the person's actions upon the biological and/or environmental spheres. Optimization being "the cyclical, feedback process whereby people seek optimal environments for themselves." (Jasnoski & Schwarz, 1985: 477) Along with the concepts of disease, illness, and illness behaviour, the idea of optimization introduces the concept of "wellness". "Through wellness people display health [i.e., maintain homeostasis at all systemic levels] by continually functioning synchronously within their bioecosystem." (Jasnoski & Schwarz, 1985: 477)

The concept of optimization is reminiscent of Bandura's (1977) notion of "reciprocal determinism" as reported in the behavioural medicine literature on the stress-disease relation, and elaborated in Chapter Three. Reciprocal determinism concerns the reciprocal interaction in the transaction process between the individual and the environment. Just as the environment may impact on the person, so he/she plays a central role in determining or constructing the nature of that environment as well as the outcome of the transaction (Turk et al., 1983).

1.2. Summary

Health and disease are thus viewed as multifactorial states of being, where disease is multidetermined. The research (reviewed in Chapters Two and Three) emerging from behavioural medicine and the psychosomatic literature is evidence of the paradigmatic shift from the biomedical model to the biopsychosocial and synchronous models. Health research utilizing the synchronous model is involved in multidisciplinary projects on multivariate problems (Jasnoski & Schwarz, 1985). The contemporary rise of "multidisciplinary teams" within the health care system is a tacit recognition of the power, influence and efficacy of the new medical models. Psychology has become increasingly involved (see below) in the care of patients, and the management of illness (particularly related to chronic disease).

The emerging field of "Psychoimmunology", where the relationships between psychological experience and disease susceptibility, onset, and progression are explored, is a particularly important development (see Chapter Two). Research demonstrating links between psychological factors and immune system functioning can offer empirical validation of key aspects of the new medical models.

In Chapter Two, research is reviewed demonstrating the role of psychological experience in disease onset and progression. Chapter Two attempts to delineate past and current research falling under the two broad rubrics of psychosomatic medicine and psychoimmunology, and to provide a preliminary rationale for the interest of psychology in a specific chronic disease condition: Rheumatoid Arthritis (RA). Chapter Three will provide an account of this medical condition, associated with extreme pain and general disability, and demonstrate the significance of including psychological expertise, particularly cognitive-behavioural techniques, in the treatment and care of such patients.

CHAPTER TWO

STRESS, DISEASE AND HEALTH PSYCHOLOGY

Overview

This chapter explores the nature of the relationship between stress and disease, and the implications thereof for health psychology in particular. Following a discussion of the problems related to developing coherent definitions of the concepts involved, the cognitive-behavioural paradigm is identified as providing the most lucid conceptualization of the complex processes involved in the human stress-disease relationship. Everly's (1988) model of the human stress response, which utilizes a systems theory perspective to delineate the complex and dynamic processes involved, is introduced and related to Jasnoski and Schwarz's (1985) synchronous systems model presented in Chapter One. It is argued that the concepts utilized by the two models seem to be related in the sense that Everly's model is able to delineate the mechanisms involved linking psychological experience to altered physical health status.

The bulk of the chapter is devoted to a detailed review of the research exploring the links between psychosocial stressors and the incidence and progression of disease. Following an overview of the research linking stress, disease and immunity, the two main fields of investigation (psychosomatic medicine and psychoimmunology) are introduced. It is argued that the value of such research may only be recognized following a discussion of the underlying assumptions governing these two fields.

As psychoimmunology is seen as a development of psychosomatic research, research supporting the latter's attempt to identify and relate particular stressors (such as major life events, psychological states, and personality characteristics) to the onset, progression, and/or outcome of acute and chronic disease, is reviewed first.

Psychoimmunological research explores the mediating role of the immune system in the stress-disease relationship, and the dysregulatory (the term "dysregulate" is taken from Jasnoski & Schwarz, 1985) effect that particular psychosocial stressors have on immune system function. Following a brief overview of the structure, function and measurement of the human immune system, the effect of various psychosocial stressors on immune function are reviewed. These include naturally occurring stressors such as academic examinations, unemployment, marital disruption or divorce, and bereavement. Adverse psychological experience such as distress, perceived loss of control and feelings of helplessness, clinical depression, caring for the chronically ill, living under the threat of exposure to an environmental stressor (e.g., 3-mile island nuclear power station meltdown), and even chronic work stress, is also explored in relation to immune system dysregulation.

The clinical significance of such stress-related immune system dysregulation is then reviewed in relation to the susceptibility, onset, and outcome of infectious (e.g., herpes simplex virus, genital herpes) and chronic (e.g., malignancies, autoimmune disorders) disease.

The chapter concludes with an examination of the value of psychological intervention in ameliorating the noxious effects of stress on health status. It is argued that the lack of research in this area is unusual considering the evidence from the literature. Following a review of the small number of studies testing psychosomatic medicine and psychoimmunology's basic assumptions and research findings in applied settings, the section ends with a discussion of the role of health psychology in the treatment of immunologically resisted disease. The prevalence of cognitive-behavioural techniques utilized in the psychoimmunological interventions with various patient populations, provides an indication the importance of this paradigm within health psychology. Chapter Three focuses on rheumatoid arthritis, adopting a cognitive-behavioural perspective of the role of psychological factors in the onset, progression and treatment of this chronic auto-immune disorder.

1. Introduction

The popular notion that stress (or distress) is related to disease is not a recent one. Such popular wisdom is grounded historically in folk-lore and in classic medical philosophy prior to the dominant biomedical model of health and disease. When asked about the causes of or reasons for their illnesses or afflictions, people often make attributions to the stress of life (Jemmott III & Locke, 1984).

The contention that there is a relation between stress and disease can no longer be simply relegated to "naive theory", as two decades (Lipowski, 1977; Dorian & Garfinkel, 1987) of psychosomatic research has empirically validated this relationship. Research (reviewed in Rabkin & Streuning, 1976; Seyle, 1976; Lipowski, 1977; Engel, 1977; Engel, 1986; Ader, 1980; Dorian & Garfinkel, 1982; Lazarus, 1982; Jemmott III & Locke, 1984; Matarrazo et al., 1984; Stein, 1986; Vingerhoets & Marcellissen, 1988) has related the occurrence of both naturally occurring stressors (e.g., major life events such as life-style change, emotional disturbances, unemployment, marital disruption or divorce, bereavement, etc.), experimental stressors (e.g., overcrowding, physical restraint, exposure to predators), and failures of adaptation (i.e., maladptive behaviours, poor coping styles, etc.), to altered physical health status.

A coherent analysis of the empirical validity of the stress-disease relationship necessitates an explanation of the concepts involved. Whilst disease has been discussed in terms of the synchronous systems model of health and disease (see Chapter One, section 1.1.3), no explanatory model "stress" has been delineated.

2 Defining "Stress"

"Commonsense" would describe stress in terms of a host of different emotions or feelings, these generally being of a negative nature (e.g., to feel upset, distressed, angered, frustrated or uncomfortable). Stress would thus constitute an experience (i.e., a "response"?) during a designated period of time; it can be referred to as something

which is occurring in the present (e.g., "I am under a great deal of stress") or had something that had occurred in the past.

A number of problems exist with such commonsense understandings of the term. Firstly, the term itself is confusing. Over a decade ago, Rogers, Dubey and Reich (1979) wrote, "...the term stress has been used in so many different ways that it immediately presents a semantic nightmare." (p.153) Secondly, the experience of stress itself is not as salient as might be expected; it is possible for some individuals to deny the experience despite the presence of observed psychological and physiological indices. Finally, different individuals perceive different experiences as being "stressful".

A major problem with stress-related research is that researchers either take for granted their understanding of the experience, or use the term to denote numerous psychological and physiological variables. Taken that psychological experience is infinitely varied, research on the effects of stress would require both a coherent conceptualization of the nature of the processes involved, as well as specifying what the particular "stress" is which is under investigation. Research is thus burdened with problems of conceptualization and denotation of this significant component of psychological experience. These theoretical and methodological operationalizations of stress need to be considered prior to any evaluation of the stress-disease literature.

2.1 Constructing Models of Stress

Stress can be seen as a complex experience, and, as such, it is difficult to provide a standard, objective definition of this phenomenon. Various definitions of the term exist, each based upon a particular theoretical model or framework. In health psychology, the cognitive-behavioural theorists have dominated the field in terms of the construction of models to delineate the multifactorial and multi-directional nature of the stress-person relationship and the health consequences of such interaction. Even within the cognitive-behavioural paradigm, however, it is difficult to achieve a consensus of the meaning of the term, despite general agreement in the nature or characteristics of the stress-person relation.

"Stress" for example, has been defined as "a stimulus or situation that [is] generally appraised as aversive or unpleasant." (Vingerhoets & Marcellissen, 1988:279) This definition is in contrast to Seyle's (1950, 1976) original use of the term stress to refer to a "response", where stress entailed the organism's physiological and psychological response to threat (the "fight-or-flight" phenomenon). The confusion generated by stress being defined both as a stimulus and as a response can best be demonstrated in an early attempt to provide a broad definition of the experience. Lipowski (1977) thus defined "psychosocial stress" as referring "...to external and internal stimuli that are perceived by and are meaningful to the person, activate emotions, and elicit physiological changes that threaten health and survival." (p. 237)

According to Everly (1988), it has become necessary to employ a word to delineate the stimulus from the total experience, and the term "stressor" is frequently used to denote this stimulus. The term "stress response" is used to denote the psychological and physiological sequelae following exposure to the stressor.

"Stress" now becomes a compound term generally denoting the transaction (see Lazarus, 1982; Lazarus & Folkman, 1984) between a "stressor" (stimulus, event or situation) and an organism's response to that stressor, or "stress response". Stress, in terms of this transactional model, is not merely a linear or unidirectional cause-and-effect relation (i.e., exposure to stressor results in stress response). A process of appraisal has to occur, whereby the individual makes sense of the stressor. The process of appraisal determines the nature of the stress response. This model also takes cognizance of the timing, duration and context of the stressor, as well as the adaptive capacity, past experience, and resources of the individual, as co-factors in the stressor-stress response transaction (Dorian & Garfinkel, 1987). The transactional model also considers the origin of the stressor: whether it is exogenous (i.e., occurs independent or external of the individual) or endogamous (i.e., occurs within the individual or is constructed by that person).

In a more recent development of this transactional model, Everly (1988) utilized a systems theory perspective, "...one of interrelated multidimensionality...", arguing that the human stress response "...is best described within the context of the dynamic 'process' it represents." (p. 23) Everly's (1988) model presents the epiphenomenology of what he calls the "human stress response" to be that of a multidimensional interactive process possessing several key elements. These elements (Everly, 1988: 23) include:

(1) Stressor events (real or imagined)
(2) Cognitive appraisal and affective integration
(3) Neurologic triggering mechanisms (e.g., hypothalamus)
(4) The stress response (a physiological mechanism of mediation - e.g., the neuroendocrine axis)
(5) Target-organ activation (potential target-organ systems include the cardiovascular system, the gastrointestinal system, the skin, the immune system, and even the brain)
(6) Coping behaviour

These elements are organized into a model wherein the temporal sequencing of the stressor--stress response process is determined by a series of feedback loops governing each stage of the process. Whilst the cognitive-affective domain is the critical causal phase in most stress reactions (Everly, 1988), it is possible that environmental events may directly cause the activation of the stress response (e.g., injury).

In keeping with the cognitive-behavioural paradigm, Everly (1988) points out that "...one's interpretation of the environmental event is what creates most stressors and subsequent stress responses."(p.45) Following activation of neurologic triggering mechanisms and subsequent mobilization of the stress response (possessing at least three different efferent axes in the neuroendocrine network, Everly, 1988), "target organ activation" occurs. The ability of the person to act environmentally (i.e., against the stressor) or cognitively (e.g., reappraisal of the stressor), or both determines the intensity and duration of the activation of the target organs (Everly, 1988). The extent to which the stressor may result in disease or pathology.

Everly's (1988) model thus provides a detailed delineation of the stress-disease relationship, highlighting the pivotal role of cognitive-affective-behavioural processes. Based in a systems theory perspective, it accords well with Jasnoski & Schwarz's (1985) synchronous systems model of health and disease. Everly's model provides a detailed

and complex explanation of how excessive or chronic dysregulation caused by stressors (actual or imagined) would affect the whole person, culminating in dysfunction and/or pathology in one or more target-organ systems. Stressors, in relation to the synchronous systems model, may thus include ecological (e.g., pollution), social (e.g., overcrowding, violence, interpersonal conflict), psychological (e.g., emotional upset, anger, anxiety, frustration) or biological (e.g., physical exertion, injury, or virus intrusion) sources of disruption.

The synchronous model, in congruence with Everly's model of stress, holds that "...the dysfunctional effects...[caused by stressors on any one level]...can permeate to any spheres and sub-systems because of the interconnectedness and synergism of the system" (Jasnoski & Schwarz, 1985: 481). Everly (1988) merely explains the mechanism whereby stressors may or may not result in pathology. Furthermore, the synchronous model's concept of "optimization", accords well with the processes involved in the person's coping behaviours ("external" actions, and/or "internal" cognitive-affective processes) which seek to reduce the impact of the stressor, to mitigate the overall level of dysregulation or activation. As health now becomes a function of the individual's successful adaptation, or adjustment, within the human system in totality, the individual's ability to deal with stress becomes an important factor in the interplay of regulation and dysregulation.

A number of important co-factors, however, determine the extent to which the person's coping behaviours succeed in reducing the impact of stress. These would include the person's perceptions or appraisal, past-experiences, habitual behavioural patterns, coping styles, social supports, personality characteristics, ego defences and even illness behaviours such as substance misuse (Dorian & Garfinkel, 1987).

The use of of Everly's (1988) model to define and delineate the stress-person-disease process accords well with a cognitive-behavioural perspective of behavioural medicine. Its explanatory power lies in its ability to conceptualize and identify particular aspects of the complex processes involved, and to escape from the confusion generated by contradictory or vague conceptualizations. By adopting Everly's (1988) model, it is possible to avoid Rogers et al.'s (1979) semantic nightmare.

Unfortunately, the adoption of a particular model only makes sense in terms of the operationalization of variables and the explanation thereof for that particular study. When reviewing the literature, it is necessary to adhere to the particular researcher(s) use of the concept for critical appraisal of the value of his/her/their findings.

Authors of the major reviews in this area have adopted various approaches to this problem of ensuring a coherent account of the empirical validity of the stress-disease relation. Some reviewers (e.g., Kiecolt-Glaser & Glaser, 1986b) specify and focus on a particular kind of stress, such as life change events, to avoid ambiguity. Others, for example, Jemmott III and Locke (1984) resolve the term's semantic connotations through broad definition. Finally, Rogers et al. (1979) resolve this "semantic nightmare" by being explicit about the particular definition of stress used in the individual studies reviewed and critique them on methodological grounds.

The following review is eclectic in the sense that it attempts to incorporate the best of these approaches in evaluating the vast body of research. A simplified version of Everly's above model of stress will also be used in conjunction with Rogers et al.'s (1979) pragmatic approach, to ensure a coherent discussion of the literature.

"Stress" will therefore be used to denote a dysregulatory state following exposure to a psychosocial stressor that potentially has adverse effects on health. The stressor may be real or imagined, acute or chronic, excessive or mild. Its particular characteristics will also be noted in each individual study reviewed to avoid semantic confusion.

3. Stress, Disease and Immunity: An Overview

3.1 Introduction

The view put forward in Chapter One, that disease is multifaceted and multidetermined, finds strong support from research examining: (1) the effects of stress on health and well-being (i.e., psychosomatic medicine), (2) the effects of stress on immunity (i.e., psychoimmunology), and (3) the effects of psychological, particularly cognitive-behavioural, intervention with various patient populations. Chapter Three will examine this third body of research, with specific emphasis on the role of psychological factors as co-determinants in the etiology and treatment of a particular disease condition: rheumatoid arthritis (RA).

Prior to a detailed analysis of the research literature, it is necessary to introduce the two main fields of research. By examining the underlying assumptions and aims of psychosomatic medicine and particularly psychoimmunology, the nature of the relationship between stress, disease and immunity becomes clearer.

In Chapter One, mention was made of the emerging field of psychoimmunology wherein the relationships between stress and disease susceptibility, onset and progression are explored in relation to changes in immune system function. As psychoimmunology subsumes a large portion of psychosomatic research endeavour (i.e., that research seeking to correlate psychosocial stress and disease states), the research reviewed below will be considered under the broad rubric of "psychoimmunology". Psychoimmunological research is an exercise in determining the underlying mechanisms governing the relationship between stress and disease by examining the effect that stress has on immune system function.

Psychoimmunology has value in that its research findings may serve as empirical validation of the assumptions of both the synchronous systems model, and Everly's (1988) model of the human stress response. For the former, psychoimmunology may identify and record relations between anomalous data which would not, or could not, be accounted for by a strict biomedical model of human functioning. For the latter, psychoimmunology provides evidence to solve the problem of determining causal links between psychological experience and altered physical health status as a result of excessive or prolonged "target-organ activation" (in this case the immune and/or endocrine systems).

Historically, psychoimmunology can be seen as a development from the general findings in psychosomatic medicine, and its growth was influenced by both epidemiological and theoretical (i.e., the development of new medical models) changes. Renewed interest in the central role of the immune system in the maintenance of health,

and the incidence of infectious (e.g., viral), neoplastic (e.g., malignancies), autoimmune (e.g., rheumatic disease) and HIV-virus related disorders, was also an important factor underlying its development.

Psychosomatic medicine's contention that stress is an important co-factor in the disease process is taken a step further by psychoimmunology. The latter's basic premise being that stress may increase an person's vulnerability to disease onset or progression by means of exerting a dysregulatory effect on his/her immune system. Psychoimmunology is by definition (Solomon & Temoshok, 1987), a multidisciplinary field which seeks to document experiences and mechanisms (direct and indirect) which "...via central nervous system transduction, can alter resistance to disease."(p. 286)

A number of assumptions underlie this basic premise that psychosocial stressors can influence immune function and thereby alter physical health status. Firstly, it is assumed that the immune system acts in the interests of homeostasis; that it constitutes one of three regulatory systems in the body (see Ader, 1980; Antoni, 1987; and section 4 below). The other two physiological systems being the endocrine and central nervous systems. Secondly, it is assumed that these three regulatory systems can and do regulate one another. Antoni (1987) has listed points of convergence between these integrated systems, providing examples of such interaction. Thirdly, if psychosocial stressors dysregulate the immune system's functioning, then it is assumed that the individual's perception of his/her ability to respond to such experience and subsequent coping behaviours would be an important determinant of the health status of the individual.

The third assumption reveals the cognitive-behavioural perspective adopted by most psychoimmunological research, and underscores the value of Everly's (1988) model of stress. It also highlights the potential role that cognitive-behavioural intervention may play in the prevention and/or amelioration of disease.

Psychoimmunology has been concerned with a number of psychosocial stressors. These include acute naturally occurring stressors such as anxiety, anger, life-change events, bereavement, and inter- and intra-personal conflict. Chronic or long term stressors researched include overcrowding, lack of social support, loneliness, depression, psychiatric disorders, and personality characteristics. Research has examined the effects of such stressors on the incidence of disease as well as on various aspects of immune function.

3.2 Outline of the Literature

Following Jemmott III and Locke's (1984) method of conceptualizing the vast, multi-faceted body of research in psychoimmunology, this review draws on two literatures to depict evidence for the validity of psychoimmunology's implicit assumptions and underlying premises. Firstly, there are numerous studies linking psychosocial factors to altered susceptibility to infectious diseases (e.g., chronic tuberculosis, acute respiratory infections, necrotizing ulcerative gingivitis or "trenchmouth", recurrent herpes simplex), and/or proneness to chronic disease conditions (e.g., neoplacy and malignancies, autoimmune disorders, etc.,). This is evidenced in the recent psychosomatic literature.

The second body of literature relates such psychosocial factors to specific aspects of the human immune response. Within this body of literature, though not directly relevant to this thesis, is a growing research interest in examining the specific relations of particular, experimentally manipulated stress situations and functional and structural changes on biochemical levels. Using animals, this research is devoted to searching for, and identifying particular mediating mechanisms underlying the stress- immune dysfunction relation, viz. neurological and endocrinological (see Ader, 1980; Stein, Schleifer & Kelly, 1980; Jemmott III & Locke, 1984; Baker, 1987; and Dorian & Garfinkel, 1987; for detailed reviews of this body of research).

This review will start by examining these two literatures concerning stress and disease, evaluating the evidence suggesting that (1) psychosocial factors are associated with physical health status, and (2) can influence immune processes in humans. Following this, it will assess the clinical significance of such psychoimmunological findings, and focus on important methodological issues affecting the empirical validity of such research. Finally, it will discuss the value of cognitive-behavioural intervention with chronic disease states, on the basis of the findings in the literature.

4. Psychosocial Stress and Illness

4.1 Psychosomatic Research

Psychosomatic research has moved away from its own traditionally reductionistic "psychogenesis of disease" concept (Lipowski, 1977), to an approach which considers psychosocial stress in dynamic interaction with multiple factors predisposing, precipitating and perpetuating disease. Since the late 1960's, psychosomatic research has been devoted to examining the varying constellation of psychosocial stressors (qualitative, Engel, 1977; and quantitative, Holmes & Rahe, 1967), their time sequences (duration, frequency), and the modification (e.g., coping skills, adaptive capacities) made by individuals to maintain their health (Lipowski, 1977).

The psychosomatic literature can be categorized into three major areas of concern. These categories are not exclusive, and have heuristic value in terms of co-ordinating an overview of this area of research (for extensive reviews, see Lipowski, 1977; Rabkin & Streuning, 1976; Jemmott III & Locke, 1984; Matarrazzo et al., 1984). The overview of the research in this area is intended to provide a broad background to contextualize the psychoimmunological literature reviewed further on.

The three trends in psychosomatic research's examination of the relation between psychosocial stress and altered physical health status are as follows:

(1) The Illness Onset Model type research (pioneered by Holmes & Rahe, 1967) which correlates the existence of life events stress and disease onset. Such research is commonly descriptive and epidemiological, and attempts to demonstrate a temporal association between the onset of illness, and a recent increase in the number of stressful life events (Rabkin & Streuning, 1976).

(2) Etiological studies examining the role of specified social and psychological factors in the development and progression of a wide range of human diseases (chronic and acute).

(3) The study of mediating psychosocial factors and physiological mechanisms that clarify this stress-illness relationship.

4.1.1 Life Events and Disease Onset

The life events research has focused on the correspondence between life changes, stress and illness onset (Minter & Kimball, 1978). This research holds the underlying notion that "exposure to social stressors does not cause disease, but may alter the individual's susceptibility at a particular period of time and thereby serve as a precipitating factor." (Rabkin & Streuning, 1976: 1014) Accordingly, through both retrospective and prospective research, dependant measures were devised (e.g., the Social Readjustment Rating Scale, Holmes & Rahe, 1967) to establish the extent or relation between the number and degree of stressful life events and, the timing of disease onset.

Using these instruments as a measure of stressful life events, a number of elaborate and extensive life events research programs were conducted in the late sixties and seventies. Such studies used large samples (e.g., 1005 men on board a warship on combat duty off Vietnam: Rubin, Gunderson & Arthur, cited in Rabkin & Streuning, 1976) utilizing data obtained from medical records and self-report questionnaires. As Rabkin and Streuning (1976) wrote in their review, "...modest but statistically significant relationships have been found between mounting life change and the occurrence or onset of sudden cardiac death, myocardial infarctions, accidents, tuberculosis, leukemia, diabetes, and the entire gamut of minor medical complaints."(p.105)

The life events approach, though presenting impressive results, is not unproblematic. The empirical validity of its findings is questionable, and numerous debates have focussed on its methodological and theoretical aspects. For example, given the very large sample sizes, even small correlations of no practical or empirical utility may seem statistically significant and be interpreted as important findings (see Rabkin & Streuning, 1976). Furthermore, Wershow and Reinhart (cited in Rabkin & Streuning, 1976) pointed out that variability within group means were overlooked, and the practical significance of life events scores is questionable (see Vingerhoets & Marcellissen, 1988). It is beyond the scope of this thesis to provide an exhaustive account of the value, problems and implications of this research area. Vingerhoets and Marcellissen (1988) provide an extensive review of the life events literature.

This area of research may be regarded as providing interesting, though at times limited and questionable findings regarding the relationship between stress and disease.

4.1.2 Psychological States and Personality Determinants of Disease

Without getting entangled in the "specificity-generality" debate regarding the contributions of psychosocial variables to morbidity (see Lipowski, 1977), it is necessary to briefly discuss these two theoretical positions prior to an overview of research in this area. These theories differ with regard to the proposed nature of the causal links of stress and pathogenesis of disease.

The specificity approach, or "strong" psychosomatic model (Vingerhoets & Marcellissen, 1988), has increasingly been viewed as untenable as a valid explanatory model. Although research has correlated specific psychological and personality characteristics to specific physiological disorders (e.g., Type A behaviour patterns and the development of coronary heart disease, see Grossarth-Maticek, Bastiaans & Kanazir, 1985) the causal links remain unclear and the role of mediating variables are significant.

The generality approach, where various 'stressors' and personality characteristics are investigated as increasing the probability of a variety of chronic and infectious diseases, is more considered more tenable. This position avoids reductive simple cause-effect explanations of the relation between psychological states or traits and disease. The role of psychosocial factors are thus seen in this generality model, as constituting co-determinants, interacting with mediating variables such as life-style, dietary habits, biological and individual characteristics and social support.

Chronic "organic" diseases such as cancer and rheumatoid arthritis have been associated with personality and psychological antecedents. The role of psychological factors in the pathogenesis of cancer is both an interesting and controversial subject of inquiry (Bahnson, 1980; Sklar & Anisman, 1981). Depression, as measured by the Multiphasic Minnessota Personality Inventory (MMPI), has been related to increased death rate from malignancies in a long-term study conducted by Shekelle, Raynor, Ostfield, Garron, Bieliauskas, Liv, Maliza, and Paul (1981). What was significant about this study was that statistical association persisted even after adjustments were made for numerous confounding variables such as age, alcohol use, cigarette smoking and family history of malignancy. Temoshok, Heller, Sagebiek, Blois, Sweet, Diclemente and Gold (1985) findings support such research, where psychosocial factors constitued central indicators of the prognosis of malignant melanoma patients.

Studies of patients with breast and lung cancers (reviewed in Lipowski, 1977; Dorian & Garfinkel, 1987) show an association between a particular defensive stance involving the suppression of affect (e.g., the inability to express anger and avoidance of conflict) and the development of malignancies and prognosis of post-treatment intervention.

Recently, two studies conducted by Ploeg, Kleijn, Mook, and van Donge (1989) tentatively support this view that rationality and anti-emotionality may be important distinctive personality characteristics in patients with cancer. Through factor analysis of the psychometric properties of a rationality and anti-emotionality scale, the researchers found that these concepts were related to the control, suppression or repression of anger (Ploeg et al., 1989). Such coping styles have been significantly correlated with cancer incidence (see Cohen, Cullen & Martin, 1982). For a more complete discussion of the role of stress and the risk for and initiation of malignancies, the reader is referred to the reviews of Bahnson (1980) and Greer (1983).

In Chapter Three, the association between stress and the onset and/or progression of rheumatoid arthritis will be closely examined. At this point it is necessary to note that the research in this area provides support for the relation between stress and chronic disease.

A bulk of research has been dedicated to studying acute illness development (i.e., infectious disease) and their relation to psychosocial factors. Such studies range from controlled laboratory conditions with narrowly defined manipulations of stress (e.g., in animal research), to broad, prospective, longitudinal field studies.

The work of McClelland, Alexander and Marks (1982) investigating life stress and upper respiratory illness among male prisoners is exemplar of the research done on acute respiratory infections (see Jemmott III & Locke, 1984, for a review). They found a significant relation between severity of illness and degree of reported stress. Such work has been confirmed elsewhere (see Graham, Douglas, & Ryan, 1986), particularly psychoimmunological studies documenting periods of immunosuppression and acute respiratory infections with high levels of stress (see Jemmott III, Borysenko, Borysenko, McClellend, Chapman, Meyer, & Benson, 1983; Glaser, Rice, Sheridan, Stout, Speicher, Pinsky, Kotur, Post, Beck & Kiecolt-Glaser, 1987).

The herpes simplex virus is a convenient infectious disease to study (Luborsky, Mintz, Brightman & Katcher, 1976): it is common and the conditions under which the virus is activated are numerous. These conditions include colds, sunburns, fevers, menstruation and psychological factors such as 'stress', negative affects etc. (Dorian & Garfinkel, 1987). In an early study, Luborsky et al. (1976) demonstrated that recurrences of cold sores (recurrent herpes labialis) in student nurses were tentatively correlated with unhappiness on the Clyde Mood Scale. Their expectations that daily self ratings of mood (particularly negative affect) would show a build up of unhappiness on the days prior to actual cold sore episodes, were not confirmed. It is interesting that this negative result is ignored in a later review (see Dorian & Garfinkel, 1987: 396). This negative result is perhaps not surprising, as the evidence linking emotional stress (or distress) to development of cold sores is largely anecdotal (cited in Jemmott III & Locke, 1984). Patients seem to think that the incidence of their herpes simplex virus (labialis) is stress-related.

Kasl, Evans and Niederman's (1979) ambitious four year prospective study of infectious-mononucleosis, caused by the Epstein-Barr virus (EBV), in some 1400 military cadets, is an important early study of the research in this area of infectious disease. The researchers sought to identify psychological 'risk factors' predictive of the development of definite clinical infectious-mononucleosis. The researchers utilized two available sets of data drawn from a prospective epidemiological surveillance study of the class of 1973 of military cadets, and from various background academic and psychosocial records. Psychosocial risk factors which related to high demands for academic achievement and relatively poor results were predictive of the development of the clinical syndrome of infectious-mononucleosis.

More recently, Gannon, Banks, Shelton and Luchetta (1989) have critically assessed the correlations between psychosocial stressors, such as negative affect and stressful circumstances/events, and infectious disease. Although these relationships are statistically significant, the correlations tend to be small (Gannon et al., 1989). This suggesting that stress alone accounts for only a minor portion of the variance in infectious disease in human beings. Accordingly (Gannon et al., 1989), there has been a consequent shift in such research, which now focuses on investigating variables which may mediate the relationship between 'stress' and infectious (and chronic) disease.

Longo and Clum (1989) examined the psychosocial factors (including emotional distress, stress, health, locus of control and social support) and genital herpes activity (frequency, severity and duration) in 46 volunteers diagnosed with Herpes Simplex Virus infection. After completing the dependant measures (Profile of Mood States, UCLA Loneliness Scale, Multidimensional Health Locus of Control scales and the Hassels scale) retrospectively for the past 12 months, these researchers found that high rates of genital herpes recurrence among the subjects were associated with high stress frequency and emotional distress. Significantly, step-wise regression analyses revealed that this association was more apparent when the mediating effects of disease duration and "internality" were examined in an interactive model (Longo & Clum, 1989). Internal locus of control was powerfully associated with less severe outbreaks, and general social support did not play a significant role in this 'stress' episode severity relation.

Gannon et al. (1989) hypothesized that greater psychophysiological reactivity to and/or slower recovery from a laboratory stressor would be associated with more symptoms, either by a direct association with illness, or via an interactive relationship with environmental stress. The results indicated both a significant relationship between environmental stress and symptoms of illness, as well as support for the effects of psychophysiological activity in this relationship. Those subjects showing greater psychophysiological arousal to or slower recovery from the stressor, exhibited a stronger relationship between environmental stress and symptoms, than those who were less reactive and faster to recover (Gannon et al., 1989). This study demonstrates the complexity of this stress-illness relation, and highlights the mediating effect of psychological and physiological (biological) variables in determining symptoms of illness.

4.2 Summary

The psychosomatic stress research reviewed above provides interesting and important findings with regard to the stress-disease relationship. Such research laid down the foundations for the more complex psychoimmunological studies discussed below. It is unfortunate, however, that the applicability of the results of studies in this area are generally limited as a result of methodological inadequacies.

Problems with stress-related research include: poor operationalization of the concepts involved (such as "major life events" which seem to be based on culturally bound concepts); the correlational nature of the research preventing a clear delineation of causality; inadequate control of confounding variables; the use of self-report instruments; use of large samples where statistical significance may occur despite low correlation coefficients; and the presence of discrepant results in the literature.

In light of these inadequacies in the psychosomatic field of research, it is difficult to make any concrete assumptions regarding the effect of stress on physical well-being. It is possible that the research efforts of investigators involved in this area are coloured by their implicit interests in "discovering" causal links (i.e., there is a "hidden agenda" which researchers need to address in stress research wherein there is a tendency to over-report "significant" findings at the expense of possibly aknowledging failure of a study to meet its predictions). This can be demonstrated by Vingerhoets and Marcellissen's (1988) concluding comments that "the future of stress research is determined solely by the great efforts and creativity of investigators." (p.289) Such a conclusion does not

encourage confidence in the tentative findings of research in this area. An important factor which needs to be considered is the difficulty and perhaps reductive pursuit of attempting to identify causal relations between psychological and physical experience (i.e., trying to meet the requirements of a strict biomedical approach). The evidence from the research reviewed above does not provide confirmation, nor the indication, of how stress affected physical variables.

5. Psychosocial Stress and Immune Dysfunction

Prior to any review of the research documenting relations between psychosocial stressors and altered immune function, it is necessary to include a brief overview of the immune system. The central role that this system plays in the maintenance of health, and its relationship to other key organ systems needs to be clarified. As the development of more complex techniques or assays to assess various components of the immune system fostered changing notions of the nature of the immune system, so psychoimmunology grew in stature as a legitimate field of research.

5.1 The Immune System and Disease

A comprehensive description of human immune function is well beyond the scope of this thesis. A basic delineation of immune function, however, is sufficient to ensure comprehension of the research findings in psychoimmunology. The immune system (Stein et al., 1980; Bryant, 1986; Borysenko, 1987) is a complex surveillance system that protects the body from disease causing micro-organisms. It regulates our susceptibility to neoplasms (cancers), infectious diseases (viruses and bacterias), allergies, and autoimmune disorders (where immune cells attack the host's normal healthy tissue). The immune system therefore maintains the integrity of the organism; to accomplish this, an exceptionally complex immune response has evolved.

Rooted in what are called "lymphoid organs" (including the bone marrow, thymus, lymph nodes, spleen, tonsils, and appendix), a major function of the immune system is to distinguish "self" from "non-self" with extraordinary specificity (Calabrese, Kling & Gold, 1987). This capacity, according to Calabrese et al. (1987), "...must occur in the context of an ability to respond to invasion by foreign antigens without disrupting central homeostatic mechanisms." (p.1123) An antigen is usually defined as "any foreign molecular structure that is capable of causing the production of antibodies." (Bryant, 1986: 9) Antibodies are immunoglobulins that combine with and neutralize foreign substances (Jemmott III & Locke, 1984). Antigens include bacteria, viruses, parasites, and fungi. "Non-self" organisms also include cells that have undergone malignancy (i.e., neoplasms).

5.1.1 The Immune Response

The immune system's response to non-self material basically entails the release of highly specialized cells into the blood by these lymphoid organs. This response has traditionally been divided into two types: humoral and cell mediated immunity. These two responses are both dependant upon lymphocyte activity (Stein et al., 1980). Different lymphocytes (which produce antibodies) however, are involved in each response. For cell mediated

immunity, T-lymphocytes (T-cells) play the central role; whilst for humoral immunity, the production of B-lymphocytes (B-cells) is central. In addition to T- and B-lymphocytes, "...other immunological functions are ascribed to phagocytic cells...." (Calabrese et al., 1987: 1123) Natural killer cells (NK-cells), which form part of this complement system, attack and destroy tumour and virus-infected cells directly, without prior antigen interaction.

Humoral immunity thus refers to the synthesis and release of "free" antibody (which produces B-Lymphocytes and immunoglobulins such as IgG, IgM, IgA, IgE and IgD) into the blood and other fluids. This antibody is capable of direct antigen interaction. These immunoglobulins protect the host against encapsulated bacteria, neutralize toxins produced by such bacteria and carry out immediate allergic reactions such as asthma (Stein et al., 1980; Calabrese et al., 1987).

Cell-mediated immunity, in contrast, does not involve this antibody production. It (Bryant, 1986) refers to the production of "sensitized" T-lymphocytes (or T-cells). T-cells confer immunity directly by cell-antigen interaction; they protect against viral and fungal infections, reject alien grafts or organ transplants, cause delayed hypersensitivity reactions (inflammatory) and fight tumour growth (Rogers et al., 1979; Calabrese et al., 1987). Following contact with foreign cells, the T-cells proliferate, or undergo "lymphocyte transformation" (Stein et al., 1980), becoming immunologically active.

Two major subsets of T-lymphocytes can be identified (Bryant, 1986; Calabrese et al., 1987): "helper" and "suppressor" T-lymphocytes. The names "helper" and "suppressor" refer to their roles in cellular immunity. Helper T-cells (designated CD4+) help macrophages and other T- and B-lymphocytes. They "switch on" the immune response by produce lymphokines which stimulate the development of antibodies, and cytotoxic T-cells (which confer direct killing action). Suppressor T-cells (designated CD8+) act to "turn off" antibody mediated immunity (Calabrese et al., 1987) and serve as counteregulatory immune modulators by blocking off the differentiation of B-cells into plasma cells, as well as inhibiting helper T-cell activity.

Ader's (1980) description of the immune system being a "homeostatic defence mechanism" cannot be more clearly depicted here. "A proper balance between help and suppression appears necessary for the regulation of antibody synthesis (i.e., cell-mediated immunity)." (Calabrese et al., 1987: 1126) It is thus a regulatory system that relies upon a complex balance among its numerous components for optimal response to pathogens. It is thought that even minor fluctuations in this homeostasis (Dorian & Garfinkel, 1987) may have direct implications for the development of disease.

5.1.2 Functional Assessment of Immunity

Because of the complexity of the immune system, there is no single index of immunocompetence. Various functions of the immune system can thus be assessed, depending on the aims of the researcher, and the availability of expertise, reagents, and satisfactory funding.

According to O'Leary (in press), one of the oldest and most common methods to measure immune function is to expose lymphocytes (once separated from the blood) to relatively non-specific antigens called "mitogens". In such

in vitro laboratory "assays", three particular mitogens are most commonly used: Phytohaemmaglutinin (PHA), Concanavilin A (ConA), and Pokeweed Mitogen (PWM). The lymphocytes' proliferation rate to these mitogens constitutes a measure of the integrity of the immune system. This assay is problematic in that it tends to yield variable results (T. Dowdall, personal communication, June, 1990). It is affected by the reagents, the batch of mitogen, and the control serum (taken from a healthy subject) used.

It is also possible to measure the ratio between helper and suppressor T-cells, the levels of antibody (i.e., immunoglobulins) in serum or saliva, and natural killer cell function. The particular immune measures selected for a study should ideally reflect the specific goals of the investigation (O'Leary, personal communication, February, 1990).

5.1.3 Summary

The immune system is an enormously complex, multifaceted defence mechanism which can be called into action against invading antigens. It thus preserves the integrity of its host, preventing infections and malignancies and promoting the health of the individual on a physiological level. It is also a very sensitive regulatory system, where complex functions rely on proper balances between complementary systems. Autoimmune disorders demonstrate the potentially adverse effects it may have on the organism if the delicate homeostasis is threatened.

As the field of immunology is expanding at an extraordinary rapid rate (Rogers et al., 1979) it is becoming ever increasingly clear that the immune system is multifaceted, almost denying a reductive analysis of its functioning. The commonly held notion that the immune system is an autonomous physiological system, is rapidly being refuted by proponents of the psychoimmunology paradigm (see Ader, 1980, 1981; Dorian & Garfinkel, 1987; and Calabrese et al., 1987). Ader's (1980) description of the immune system as acting in the interests of homeostasis (cf. Cannon, 1935), concurs with recent assumptions that it constitutes one of three regulatory systems in the body which can, and do, influence one another (see Antoni, 1987; Korneva, Klimenco & Shkhinek, 1985; Locke, Ader, Besedovsky, Hall, Solomon & Strom, 1985; and section 2 above). The other two physiological systems being the endocrine and central nervous systems.

The above overview is by no means exhaustive, it aims at achieving a basic understanding of the immunological apparatus and the immune response. It is beyond the scope of this thesis to discuss the detailed biological antecedents and sequelae of the immune response, nor the complex assays used to measure immune function. There are many well documented interactions between immune processes and neuroendocrine functions (e.g., Rogers et al., 1979) described in recent reviews (see particularly Riley, 1981; Jemmott III & Locke, 1984; Plotnikoff, Faith, Murgo & Good, 1986; and Antoni, 1987).

The mediation of psychosocial influences on immune function by central nervous system and endocrine system mechanisms is increasingly becoming an important area of concern, and may provide explanations to promote insight into disease, susceptibility, onset and progression.

5.2 Psychoimmunological Research

This section is devoted to reviewing the evidence supporting psychoimmunology's contention that stress dysregulates aspects of immunity, thereby further explaining the relation stress has with altered physical health status.

A number of reviews exist which document the research done in this psychoimmunological paradigm (see Stein, Schiavi & Camerino, 1976; Rogers et al., 1979; Stein et al. 1980; Ader, 1980; Jemmott III & Locke, 1984; Temoshok & Heller, 1984; Tecoma & Huey, 1985; Jemmott III, 1985; Matler, 1985; Solomon, 1985; Stein, 1986; Kiecolt-Glaser & Glaser, 1986b; Baker, 1987; Dorian & Garfinkel, 1987; O'Leary, in press; Antoni, Scheiderman, Fletcher, Goldstein, Ironson & Laperriere, in press) and their authors have structured the enormous, expanding bulk of research by: (1) examining the major psychological stressors and their effects on immune function, (2) examining the role such factors have on chronic and infectious diseases by altering or dysregulating immune system function, and (3) looking at mediating variables such as coping styles, social support, personality characteristics and characteristics of the stressor itself.

A final significant approach to utilizing the growing body of psychoimmunological research and giving new directions for future investigation has been the work of Antoni and his colleagues (Antoni, 1987; Antoni et al., in press). Antoni (1987) and Antoni et al. (in press) have constructed models to synthesize the findings in psychoimmunological research. Such models incorporate the individual's characteristics (e.g., sense of control, adaptive capacities, social support systems) into a schematic depicting the psychosocial stress, neuro-endocrine and immunological chain of events. Such schemas, though speculative, have heuristic value in that there is an attempt to "tie-up" and incorporate all the divergent findings into a coherent model wherein these findings can be interpreted and analyzed. Furthermore, such models provide researchers with the means to plan and conduct psychological intervention programs to improve immune system regulation (see section 6). In Chapter Four, a similar attempt is made to delineate the key variables under investigation.

It is intended that the following review documents not only the progress in the field, but also the great complexity of relationships involved. Psychoimmunology, it is emphasized, is not involved in a naive reductionistic account of these possible relations. The complexity of psychoimmunological relations (Kasl et al., 1979) involves, among others:

> (1) The immune system itself is a complicated network, there is no single index of immunocompetence; (2) the relationship between psychosocial stressor and immunosuppression (either directly via central nervous system--immune system activity, or through neuroendocrine immunomodulation); (3) the nature, duration and timing of the stressor in relation to host characteristics; and (4) the clinical significance of changes in the various immune components to illness development.
>
> (p. 448)

Solomon and Temoshok (1987) devised a number of hypotheses that were most relevant to the field of psychoimmunology. These hypotheses, of which three are selected, are significant in that they offer a coherent conceptualization of the majority of psychoimmunological studies:

> (1) Emotional upset and distress ("state" characteristics) should alter immunocompetence and/or the incidence, severity and/or causes of diseases that are immunological resisted or are associated with aberrant immunological function.
>
> (2) Enduring coping style, capabilities, and personality factors ("trait" characteristics) should influence the susceptibility of an individual's immune system to alteration by exogenous events, including reactions to events.

On the basis of these two hypothesis and the evidence accumulated from such research, one may formulate a third hypothesis (Solomon & Temoshok, 1987). Namely,

> (3) Behavioural and/or psychological interventions aimed at countering the aversive effects of stress, should also be able to enhance or optimize immune function.

For health psychology, the third hypothesis points to the important part it may play in the future of psychoimmunology. It is necessary to review the evidence justifying such hypotheses.

5.3 Psychosocial Stress Factors and Immune Function

5.3.1 Bereavement

Conjugal bereavement is among the most potentially stressful events in life and has been associated with increased morbidity and mortality rates (Stein, 1986). Schleifer, Keller, Camerino, Thorton and Stein (1983) prospectively investigated the effect of bereavement on immunity in a longitudinal study of spouses of women with advanced breast carcinoma. Lymphocyte stimulation responses in 15 men to phytohaemmaglutinin (PHA), concanavalin (ConA) and pokeweed mitogen (PWM) were significantly lower during the first two months post-bereavement as compared with pre-bereavement responses. Such responses are an "in vitro" correlate or parameter of immunity, and indicate an impaired immune response. These investigators, however, recorded no difference in total lymphocyte or B- or T-cell numbers. That pre-bereavement responses did not differ from age and sex controls, demonstrates that the suppression of the lymphocyte responses is a direct consequence of the bereavement event. Although such suppression may be related to increased morbidity and mortality following the death of a spouse, Schleifer et al. (1983) caution that changes in nutrition activity, exercise levels, sleep and use of medication could influence lymphocyte responsively, and need to be accounted for. It is unfortunate that this study did not include measures to determine the clinical significance of such immune suppression.

This study supported the results of an earlier study by Bartop, Lockhurst, Lazarus, Kiloh and Penny (1977) on conjugal bereavement. Bartop et al. (1977) had also found that bereaved spouses compared with a non-bereaved

sample had significantly impaired lymphocyte proliferation in response to mitogenic stimulation (with PHA and ConA) approximately 8 weeks subsequent to the death of the spouse. In addition, these two groups did not differ in T- and B-cell counts or serum immunoglobulin.

To determine whether or not there were psychological mediating factors influencing the health impact of the bereavement event, Linn, Linn and Jensen (1982) studied 60 men who had experienced either family deaths or serious illnesses during the previous six months. Based on their scores on the depression scale of the Hopkins Symptom Checklist, the men were divided into high or low depression groups. The more depressed group had reduced lymphocyte responsiveness to PHA as compared to the low depression group. This study indicates that it is the distress, or depression, associated with the loss that is of significance, and not the event itself.

A more recent study by Jacobs, Mason, Kosten, Kasl, Ostfeld, Atkins, Gardner and Schneiber (1985) supports Linn et al.'s (1982) finding, suggesting that it is not the actual event (i.e., death of spouse or bereavement), but the degree of anxiety and distress accompanying a major life stressor. Jacobs et al. (1985) found no difference in immunological function and/or health status between 43 acutely bereaved widows and widowers compared to 24 non-bereaved married persons who experienced critical illness in a spouse.

5.3.2 Marital Disruption or Divorce

Marital disruption or divorce has been investigated in terms of its immunological consequences (Kiecolt-Glaser, Fisher, Ogrocki, Stout, Speicher & Glaser, 1987a; Kiecolt-Glaser, Kennedy, Malkoff, Fisher, Speicher & Glaser, 1988).

In a cross-sectional study of marital disruption, Kiecolt-Glaser et al. (1987a) examined the psychological and physiological mediators linking poor marital relations and stress-related physical illness as attested by epidemiologic studies (Somers, 1979). They found that women who had been separated 1 year or less had significantly poorer qualitative and quantitative immune function than demographically matched married women. The separated or divorced women had significantly poorer lymphocyte responsiveness to two mitogens (PHA, ConA) than married women, as well as lower percentages of helper T-lymphocytes. Furthermore, among married women, poorer marital quality was associated with greater depression and a poorer response on three qualitative measures of immune function. This particular study is significant in that it utilized a battery of six immunological assays and examined both within and between group differences along a variety of demographic, and psychological measures. Furthermore, the results were unchanged after adjusting for variables such as drug or alcohol abuse and nutritional habits. Finally, Kiecolt-Glaser et al. (1987a) presented evidence that qualitative indicators of immune functions were more sensitive to the psychological variables than the quantitative measures. The researchers concluded that the results demonstrated that one's interpersonal relationships may have health-related consequences.

In a related investigation, Kiecolt-Glaser et al. (1988) sought to determine whether there may be sex differences in response to marital disruption along such immunological and psychological variables. Kiecolt-Glaser et al. (1988) thus examined the possibility that there were also distress-related alterations in immune function in separated and

divorced men. Two hypotheses guided this more recent study: the first that separated-divorced males would be more distressed, lonelier and have poorer immunological functioning than their sociodemographically matched counterparts. Secondly, Kiecolt-Glaser et al. (1988) expected that higher marital satisfaction would be associated with better psychological and immunological functions. The prediction was based on the assumption that if stress affected immune function adversely, then positive psychological experience would enhance immunity.

Results indicated that there was a significant difference between the separated/divorced and married men groups (Kiecolt-Glaser et al., 1988). The former were found to be more distressed, lonelier and reported more recent illness than the latter. In addition, the separated/divorced men had poorer values on two functional indices of immunity (antibody titers to two herpesviruses: Epstein-Barr Virus and Herpes Simplex Virus), while not differing on quantitative indices (percentages of helper and suppressor cells and their ratio). Marital quality amongst married men was also associated with differences in antibody titers to Epstein-Barr virus (EBV). Finally, those men who had recently been separated, and initiated the separation/ divorce had better performance on the EBV assay than did non-initiators. Kiecolt-Glaser et al. (1988) concluded that the study demonstrated that an increase in psychological distress, sustained over time can lead to adversive immunological changes.

Unfortunately, neither of these two studies had any measure of illness per se, thus the clinical significance of such immune dysregulation is left to conjecture alone. Later, in what Jemmott III (1985) has called the "second generation" of psychoimmunological research, such measures were included to allow greater interpretation of the results.

5.3.3 Distress or Depression

Jemmott III and Locke (1984) reviewed a series of studies which examined the relationship between distress (and/or depression) and immune functioning. "Distress" was measured with life events questionnaires and measures of psychological distress (in particular the Profile of Mood States or POMS). In these studies (reviewed in Jemmott III & Locke, 1984), subjects were inoculated with a particular virus (e.g., A/Victoria/F5H3NZ or A/NJ/76 influenza immunization), completed psychological questionnaires, and had blood drawn for analysis of immunological functioning.

In one such study (Green, Betts, Ochitill, Iker & Douglas, cited in Jemmott III & Locke, 1984), the researchers reported that the more life change stress the subjects reported the lower their degree of lymphocyte cytotoxity (i.e., ability to destroy antigens) and diminished degree of lymphocyte response to mitogens. These impairments in immunological functioning were also related to higher POMS vigor scores, and total mood disturbance. In this study, it is not clear to what extent the incidence of stressful life events per se, rather than the psychological mood state of the subjects contributed to changes in immune function.

According to Baker (1987), bereavement reaction and morbid depression have much in common. Several studies have examined the effect of depression on immunity (Cappel, Gregoire, Thiry & Sprecher, 1978; Schleifer, Keller, Meyerson, Raskin, Davis & Stein, 1984; Murphy, Gardner, Grechon & Carroll, 1987).

Evaluating measures of lymphocyte function in depression in an early study, Cappel et al. (1978) found that psychotically depressed patients during the acute phase of their illness had impaired lymphocyte stimulation responses to PHA, than during clinical remission. Cappel et al. (1978) were unable to interpret these findings, due to a lack of significant difference with control subjects at their assessment points. The astonishing lack of any indication of the type of medication the patients were on, however, severely undermines the value of this study's findings. This methodological inadequacy is a significant feature of a number of studies in this area, and is repeated in the following study.

Schleifer et al. (1984) studied a hospitalized group of depressives and found significant reductions in lymphocyte proliferation responses to the mitogens PHA and PWM. In addition, those subjects suffering from depression had reductions in the numbers of T- and B-cells circulating systemically. These researchers also reported elevated cortisol levels in other patients with impaired lymphocyte function, a finding which is consistent with studies reporting the immunosuppressive effects of corticosteroids (see Stein et al., 1980; Stein, Keller & Schleifer, 1985; and Calabrese et al., 1987). The focus on corticosteroids is based on the knowledge that there is a direct correlation between degree of stress arousal (i.e., level of stress response) and concentration of circulating cortisol in the blood.

In a more recent study, Murphy et al. (1987) found that in a sample of 80 patients with depression (50 unipolar, 30 bipolar), many had reduced numbers of circulating lymphocytes. This abnormality was more common (approximately 57%) in the unipolar group. Although they had predicted that corticosteroid overactivity would produce a fall in the percentage circulating lymphocytes (this prediction being based on research indicating the immunosuppressive effects of increased concentrations of corticosteroids; see above paragraph), results demonstrated the opposite (Murphy et al., 1987). A paradoxical positive correlation between pre-dexamethasone cortisol and lymphocyte count in the bipolars emerged. In their discussion of their results, Murphy et al. (1987) argued that it was unjustifiable to provide a firm conclusion regarding the relation between cortisol levels and lymphocyte numbers for a number of methodological and theoretical reasons which were not accounted for (e.g., medication, nutrition, tests of other endocrine secretions, etc.).

These studies of bereavement, distress and clinical depression thus provide only limited evidence of the immunosuppressive effects of negative affect. The lack of control for the possible effects of medication regimens is a significant inadequacy of such research. However, the well documented literature of the hormonal/endocrinal correlates of mood and emotions, as well as the interregulation between the endocrinal and immune systems, does provide important support for psychoimmunology's assumptions.

5.3.4 Other Chronic Stressors

Other forms of chronic stressors such as long term unemployment (e.g., Arnetz, Wasserman, Petrini, Brenner, Levi, Eneroth, Salovaara, Hjelm, Salvaara, Theorell & Petterson, 1987), caring for the chronic ill (e.g., Kiecolt-Glaser, Glaser, Shuttleworth, Dyer, Ogrocki & Speicher, 1987b), loneliness (Kiecolt-Glaser, Ricker, George, Messick, Speicher, Gardner & Glaser, 1984b), chronic work stress (e.g., Dorian, Garfinkel, Keystone, Gorczynski, Garner, Darby & Shore, 1985), and long term exposure to an environmental stressor such as the threat of a nuclear

Power station meltdown, as in the case of 3-mile island (e.g., Baum, McKinnon & Silvia, cited in Antoni et al., in press) have been investigated in terms of their immunological consequences.

In a prospective study of unemployed Swedish women over 8 months, Arnetz et al. (1987) studied the immunological effects of the many advantages of having work (e.g., its social and economic benefits). The researchers were also interested in the benefits of psychological intervention to "buffer" the adversive effects of unemployment. Dividing the subjects into three groups: unemployed, unemployed plus psychosocial support programme, and securely employed; the women were assessed along a battery of immunological essays. These tests included lymphocyte response to PHA mitogen and reactivity to purified protein derivative (PPD) of tuberculin, percentage total T-cell and B-cell populations, and level of serum cortisol. After 9 months of unemployment, PHA reactivity of lymphocytes and reactivity to PDD decreased significantly in the first two groups of women. The psychosocial support programme did not counteract this decrease. No changes occurred in the securely employed group nor along the other parameters of immune function. The authors concluded that the data suggested the possible health consequences of long-term unemployment. Unfortunately no evaluation of the clinical significance of such changes in the immune system was included. The absence of data on diet, life-style factors, and health status undermine the value of such findings.

Obtaining psychological and immunological data from 34 family care givers of Alzheimers disease (AD) victims, and 34 sociodemographically matched comparison subjects, Kiecolt-Glaser et al. (1987b) noticed a significant difference between these two groups. The chronically stressed caregivers being more distressed, having significantly lower percentages of total T lymphocyte and helper T-cells, poorer helper-suppressor T-cell ratios, and higher antibody titers to Epstein-Barr virus. Percentages of natural killer cells and suppressor T lymphocytes did not differ significantly.

According to Kiecolt-Glaser et al. (1987b), the persistent alterations in cellular immunity associated with chronic stress evidenced poor psychological and immunological adaptation in the AD category, and may have important health consequences if the group had pre-existing immunological impairments (e.g., in older adults, AIDS and cancer patients). This conclusion is significant in that it identifies possible "high-risk" groups wherein the subtle stress-induced changes in immune function may have important health implications for the individual. In a sense, Kiecolt-Glaser et al. (1987b) were alluding to the current focus on such populations, and the health benefits of psychological intervention with these groups (particularly the work with HIV-infected individuals; see Antoni et al., in press).

The importance of loneliness (defined as the lack of social support), another naturally occurring stressor of possible long-term duration, has been investigated as a possible modifier of immunocompetence. In studies on non-psychotic in-patients, Kiecolt-Glaser et al. (1984b) found that those subjects who were loneliest (as rated on the UCLA Loneliness scale), had higher urinary cortisol levels, lower levels of natural killer cell activity, and poorer lymphocyte proliferation response to PHA stimulation. This study demonstrates the value of utilizing in-patient settings whereby life-style factors can be controlled to a certain extent.

5.3.5 Academic Stress

A number of studies have reported a similar relationship between academic stress and parameters of immunological functioning (Dorian, Garfinkel, Brown, Shore, Gladman & Keystone, 1982; Jemmott III et al., 1983; Kiecolt-Glaser, Gardner, Speicher, Penn, Holliday & Glaser, 1984a; Baker, Irani, Byram, Nagvekor, Wood, Hobbs & Brewerton, 1985; Glaser, Kiecolt-Glaser, Tarr, Speicher & Holliday, 1985b; Kiecolt-Glaser, Glaser, Strain, Stout, Tarr, Holliday & Speicher, 1986a; and Halvorsen & Vassen, 1987).

Dorian et al. (1982) collected data on 8 psychiatry residents undergoing their oral fellowship exam (considered an acute psychological stressor), and compared it to 16 sex- and age-matched psychiatrists not taking the exam. In a prospective pre- and post assessment design, the study aimed at measuring changes in immune (T-cell subpopulation, B-cell numbers, mitogen reactivity, natural killer cell activity, plaque forming cell responsiveness, antigen specific T-suppressor cell activity) and endocrine (hormone levels) function in relation to psychological experience. It aimed at relating such changes to psychometric measures of psychological stress, personality style and coping strategies. In the two weeks prior to the exam, the highly stressed group had higher B-, T-, and white blood cell counts, but lower lymphocyte responses to PHA and ConA as compared to the lower stressed group. Two weeks post-examination the only remaining immunological difference between the groups was an elevated lymphocyte response to PHA in the exam group. Such a result, according to the researchers, indicated that exposure to short term stressors could be beneficial if it was effectively dealt with (Dorian et al., 1982). Data on the coping strategies employed by the residents seemed to confirm such a conclusion. A further unexpected finding (in the sense that it was contradictory to other research findings) was lower cortisol levels both pre- and post-examination in the stress group. This study demonstrates the complexity of relationships involved in psychoimmunological research, emphasizing the need for multiple measures of psychological and immunological status.

Examining the possible interaction of personality type and immunocompetence as a function of academic stressors, Jemmott III et al. (1983) prospectively studied 64 dental students during their first year of study. Selecting salivary secretory IgA as an indicator of immune function, the researchers found that its mean secretion rate was significantly lower during periods of high stress than during those of low stress. Such a relation was maintained after including both within subject comparison (to control for seasonal rhythmicity of IgA secretion) and individual differences in the perceived stressfulness of their academic programme. In addition, Jemmott III et al. (1983) reported that those personalities characterizing the "inhibited power syndrome" (the combination of a great need for power and high activity inhibition) had lower secretion rates of IgA at all assessment-points than their fellow students who characterized the "relaxed affiliative syndrome" (Jemmott III et al., 1983: 1401). Such results demonstrate the important role that cognitive-affective appraisal of a stressor (in this case the pressure to complete academic requirements) has in determining the health outcome of such a relationship. In this case, some facets of the immune response were temporarily inhibited.

Investigating the effects of studying for final examinations (and the related feelings of loneliness and isolation) on immunocompetence, Kiecolt-Glaser et al. (1984a) assessed first year medical students pre- and post-examinations. Stress-produced immunosuppression over time, and differences in high and low scorers on the UCLA Loneliness

and Social-Readjustment Rating Scale were observed. High scorers on stressful life events and loneliness scales were found to have significantly lower levels of natural killer cell activity. Furthermore, total plasma IgA decreased significantly between assessment points for this group, whilst no changes were observed in Iga and Igm, and salivary Iga concentrations. Any extrapolation of the effects of such immunosuppression need to recognized as being speculative (Kiecolt-Glaser et al., 1984a) for two main reasons: firstly, lifestyle factors were not controlled for; and secondly, no data on health status was collected. This study, like many before, did not address the clinical significance of impaired immunity.

Baker et al. (1983) found that medical students during their first week in hospital experienced a high degree of psychological (raised subjective anxiety scores) and physiological (raised serum cortisol concentrations) arousal. Contradictory to expectations, these features were associated with a significant increase in the proportion of helper T-lymphocytes. The acute arousal was thus associated with improved immune function, possibly demonstrating the extent to which, in the short term, the stress response may benefit the individual in during critical periods. Baker et al. (1983) interpreted their findings in the context of diseases such as rheumatoid arthritis where the immune system is activated to the extent that it acts upon its host, attacking normal bodily cells and tissue (see Chapter Three). According to the researchers (Baker et al., 1983), a prolonged increase in helper T-cells could signify an inability in the immune system to de-activate or "switch-off" the immune response, due to an imbalance in the helper- and suppressor lymphocyte ratio.

A similar study by Glaser et al. (1985b) contradicted Baker et al.'s (1983) above suggestion that an increased percentage of helper T-lymphocytes was part of a physiological reaction to stress. Unlike Baker et al.'s (1985) single data collection point, Glaser et al. (1985b) examined longitudinal data collected over high- and low-stress periods for medical students undergoing examinations. Specifically, significant decreases in the percentage of lymphocytes, total T-lymphocytes, helper and suppressor cells, and decreased T-lymphocyte proliferation reactivity to PHA and ConA, were associated with high subjectively perceived distress.

The researchers reported evidence that such changes were not caused by relatively minor variations in nutrition or sleep. In addition, the students' self-reported good health suggests that these changes were not a function of other health problems. Glaser et al. (1985b) concluded that their findings are consistent others across diverse population groups, suggesting that the relation between greater distress and poorer immune function, though not immediately associated with an increase in illness, "...might have important consequences in individuals whose health is already impaired, ..are exposed to an infectious agent or carcinogen, [or] who already have latent viruses or undetected tumor cells, or in older population." (p. 238)

These findings were supported in a more recent study by Kiecolt-Glaser et al. (1986a) of the psychosocial modulation of cellular immunity in medical students one month before, and on the day of their examinations. Significant declines in the percentage helper T-lymphocytes, in the helper-suppressor cell ratio, and in natural killer-cell activity were observed between the two sample points. In order to counteract this predicted stress induced immunosuppression, the researchers (Kiecolt-Glaser et al., 1986a) also examined the extent to which a relaxation intervention programme (hypnotic relaxation was selected) would buffer such stress-related changes in cellular immunity.

Results indicated that the frequency of relaxation practice was a significant predictor of the percentage of helper cells in the examination sample (Kiecolt-Glaser et al., 1986a). Furthermore, a non-significant change in self-rated distress was also demonstrated in the relaxation group, as compared to significant increases in distress during examinations in the control groups. This study is important, in that it alludes to the possible enhancement of health, through the increase of positive emotions via relaxation training, as well as the psychological benefit of eliciting the relaxation response (see Benson, Greenwood & Klemchuck, 1975; Benson, Frankel, Apfel, Danields, Schniewind, Nemiah, Sifneos, Grassweller, Greenwood, Kotch, Arns & Rosner, 1978; Everly, 1988). Kiecolt-Glaser et al. (1986a) conclude that such speculation may have some preliminary empirical support.

More recently, Halvorsen and Vassen (1987) sought to critically evaluate the consistency of such research findings linking reduced immunocompetence and examination stress. As most of the earlier studies in this area assessed lymphocyte proliferative responses to mitogens such as PHA, ConA and PWM, these researchers sought to study antigen specific responses to alloantigens and a ubiquitous antigen (D. farinae). This study thus was more immunologically complicated, as the researchers examined more complex and specific indicators of minimum function. In a pre, mid and post longitudinal design, Halvorsen and Vassen (1987) demonstrated a decrease in the percentage of helper and suppressor cells, and an increase in circulating monocytes in phase II. Finally, the proliferative response of T-cells to antigens, mitogens and allogenic cells decreased from phase I through to phase III. The researchers concluded that acute examination stress has a detectable influence on certain cellular immunological functions.

5.3.6 Summary

The evidence provided in the above review, indicates a general consensus in the literature regarding the dysregulatory effect of various psychosocial stressors on immunocompetence. Qualitative changes in immune function with reduced lymphocyte responses to mitogens are commonly seen, whereas conflicting results surround the quantitative immunological changes such as percentage circulating lymphocytes (total, subsets), natural killer cells, monocyte cells, cytotoxic cells, and immunoglobulin levels. Whilst one may thus conclude that the immunomodulating potential of the different stressors are primarily qualitative, it is necessary to point out that a number of methodological inadequacies in the research in this area need to be identified and addressed.

(1) A problem with psychoimmunological research is the tendency to "oversimplify" the concepts involved (O'Leary, in press). By failing to measure different components of the immune system over successive assessment points in longitudinal designs, the complex processes involved in the psychoimmunological relationship are ignored (Dorian & Garfinkel, 1987). Furthermore, a large number of the studies reviewed above either fail to define and operationalize the particular stressor involved, or fail to include a battery of psychological measures to assess the effects of such stressors.

(2) The lack of appropriate control groups, and the poor utilization of the experimental design possibly undermine the validity of research findings (Jemmott III & Locke, 1984; Dorian & Garfinkel, 1987).

(3) The use of correlational studies (where it is attempted to assess the immunological sequelae during or following exposure to stressors) have the problem of controlling for a multitude of extraneous variables. These include: (i) changes in diet, sleeping patterns, exercise habits, minor changes in physical health status, and the effects of hospitalization and/or medication intake; (ii) timing, intensity and duration of the stressor; and (iii) the effect of mediating variables such as subjects' perception or appraisal of the stressor, their self-appraisal, coping behaviours and social support systems.

(4) A final inadequacy of a large number of studies investigating psychoimmunological relationships, is the failure to determine the clinical implications of stress-related immune dysregulation. As Jemmott III and Locke (1984) wrote, "...these [blunted immune] responses merely suggest that in an artificial, *in vitro* situation, lymphocytes are less capable of undergoing mitosis, a finding that need not translate into clinically relevant immunocompromise." (p. 1129) It is necessary to examine such immunological consequences in studies specifically assessing vulnerability to disease and progression of illness in order to specifically relate such immunocompromisation to disease states. In other words, to relate stress-induced immune dysfunction to disease onset or progression.

5.4 The Clinical Significance of Stress Related Impaired Immunity.

Epidemiological studies (reviewed in Kiecolt-Glaser et al., 1986b) of distressed populations (e.g., bereaved and divorced adults) suggest that these groups have significantly higher morbidity and mortality rates. Based on the evidence of the above studies demonstrating how stress (or distress) dysregulates immune system function, it is possible to suggest that stress-related immunocompromisation may be clinically significant. Yet, such a suggestion is based on deductive reasoning, and it is necessary to substantiate it on empirical grounds. What evidence is there that psychological factors, acting through the neuroendocrinal and immune system, influence the onset or course of a naturally occurring disease?

In this section, research concerning stress-related immune system dysregulation and disease susceptibility and progression is reviewed in relation to three categories of disease: infectious (i.e., through viral infection and/or recurrence of infection by latent viral activation as in the case of herpes simplex virus), malignancy and autoimmune disorders, and Acquired Immunodeficiency Syndrome (AIDS).

5.4.1 Infectious Disease

The association between acute disease states and psychosocial stressors has been well documented in the epidemiological and stress literature (see sections 3 and 4 above). As the timescale is likely to be relatively short, the connections are more apparent in comparison to chronic neoplastic and autoimmune diseases (Baker, 1987). Furthermore, "...clinically it has been noted...that infectious diseases are the result of host-micro-organism interaction." (Stein et al., 1980: 1965)

Earlier it was noted that Kiecolt-Glaser et al. (1987a) demonstrated that women experiencing the stress of marital disruption have higher antibody titres to EBV, reflecting poor cellular immune system control over the latent virus.

Similarly, Glaser, Kiecolt-Glaser, Speicher & Holliday (1985a) showed that periods of peak stress can effect changes in herpesvirus latency. Using a prospective design, the researchers examined the influence of examination stress and loneliness on herpesvirus latency as measured by changes in antibody levels to three herpesviruses: Eptein-Barr Virus, Herpes Simplex Virus-1 and cyto-megalovirus (CMV). 49 first year medical students were divided into high- and low-scoring loneliness groups (by a median split on the UCLA Loneliness Scale). The changes in antibody titres to EBV, HSV-1 and CMV were most marked in subjects scoring high levels of loneliness, with significant changes in both groups across the three sample points (1 month prior exam, on the day of the exam, and a week post-exam). Glaser et al. (1985a) concluded that Stress-related immunosuppression can significantly modulate herpesvirus latency.

Cohen-Cole, Cogen, Stevens, Kirk, Gaitan, Hain & Freeman (1981) studied the interrelationships between psychosocial, endocrine and immune factors to another infectious disease: acute necrotizing ulcerative gingivitus (ANVG or "trenchmouth"). Utilizing a retrospective, two-groups matched samples design, the trenchmouth patients reported more negative life events over the preceding year than the controls. Furthermore, the patients who were (1) more anxious and depressed at the time of presentation were (2) more likely to have significant emotional distress (General Health Questionnaire, MMPI), and (3) had depressed lymphocyte reactivity to CON-A. Cohen-Cole et al. (1981) found no statistically significant differences in the two groups in serum cortisol, prolactine, growth hormone, thyroid levels or urine catecholamines. The researchers concluded that their overall findings demonstrated that psychological and immune variables are significantly associated with trenchmouth.

In a more recent study, Kemeny, Cohen, Zegans and Conant (1989), investigated the effects of stress and coping on immune function and the outbreak of genital herpes. The relationships among stressful life experience, mood, helper-suppressor T-cells and genital herpes simplex virus (HSV) recurrence in thirty-six subjects with recurrent HSV were followed over a period of six months. Results averaging monthly scores indicated that high levels of stressful experience were related to lower proportions of both helper and suppressor T-cells. Subjects exhibiting high levels of depressive mood, anxiety or hostility (as measured on the Profile of Mood States) had a lower proportion of suppressor T-cells. Finally, those subjects with high levels of depressive mood had a higher rate of HSV recurrence. Kemeny et al. (1989) argue that it is possible to construct a model linking depressive mood, suppressor T-cells and HSV recurrence.

Such research demonstrates that the relationship between psychological factors and acute disease processes, particularly as a result of latent herpes virus, appears to be mediated by immunological processes. Psychoimmunological research in this area is invaluable, as it indicates the clinical significance of stress-related immune dysregulation. Research into neoplastic and auto-immune disease has also been significant in this respect, though notions of causality are more difficult to isolate in such chronic disease states.

5.4.2 Malignancies and Auto-immune Disorders

Altered immune system function is implicated in the pathogenesis of cancer and auto-immune disease such as rheumatoid arthritis (see review by Stein et al., 1980; and Chapter Three). A great deal of research has examined psychosocial factors in cancer incidence and progression, and has generally focussed on identifying psychosocial

factors related to disease outcome (see Cohen et al., 1982; Levy, 1985; O'Leary, in press). An increasing body of research, however, has begun to examine psychoimmunological mediation (see Pettingale, Philalithis, Tee & Greer, 1981; Levy, Herberman, Malvish, Schlien & Lippman, 1985; Kiecolt-Glaser, Stephens, Lipetz, Speicher & Glaser, 1985a).

Pettingale et al. (1981) examined immunoglobulin correlates of psychological responses to breast cancer diagnosis. The patient's reaction was significantly related to levels of IgM and IgG. Higher levels of the former in patients showing "denial" rather than "fighting spirit" or "stoic acceptance", and lower levels of IgG in those displaying "fighting spirit" than those showing "stoic acceptance". In their conclusion however, Pettingale et al. (1981) cautioned that "...the mechanisms by which such immunoglobulin changes could influence survival in cancer remain hypothetical" (Pettingale et al., 1981: 486).

Prompted by epidemiological data linking depression to mortality from malignancies (Shekelle et al., 1981, discussed earlier), Kiecolt-Glaser et al. (1985a) investigated the extent to which depression may impair ability of cells to repair damaged DNA, thereby increasing the vulnerability to damage by environmental carcinogens. Specifically, Kiecolt-Glaser et al. (1985a) assessed differences in DNA repair in lymphocytes obtained from high- and low-distressed psychiatric inpatients. In addition, comparisons were also made between this group and a control sample of demographically matched blood donors. The finding that a high level of distress is associated with significant dysfunctional differences at the cellular level provides evidence for a pathway through which distress could possibly influence the incidence of cancer.

The actual contribution of psychological factors to cancer incidence is minor in comparison to the existence of genetic predispositional factors, exposure to environmental carcinogens, and life-style risk factors. Furthermore. the influence of psychological coping traits to cancer progression is largely anecdotal (e.g., discussed in Cohen et al., 1982). Considering the high incidence of neoplastic disease in our society, and the stress-cancer hypothesis, this area constitutes an important area for future psychoimmunological investigation.

Greater attention will be devoted to the role of psychosocial factors in the etiology, progression, and treatment of rheumatoid arthritis in Chapter Three. The few studies which have investigated psychoimmunological relations in the onset and progression of this painful chronic disease will be reviewed following a detailed explanation of the characteristics of the disorder. It is sufficient, at this point, to comment that convincing associations between psychological experience and rheumatoid arthritis incidence and progression have been demonstrated in the literature.

5.4.3 HIV Virus Related Disease Progression

It is beyond the scope of this thesis to deal extensively with the psychoimmunological research being conducted in the area of Acquired Immunodeficiency Syndrome (AIDS) research in terms of the processes governing susceptibility, onset, and progression of infection by the Human Immunodeficiency Virus (HIV) virus. Solomon and Temoshok (1987) have critically evaluated the need for a psychoneuroimmunological perspective on AIDS and the reader is referred to their extensive review of related research.

The research in this area is typically of a longitudinal, prospective nature, where multiple analyses on psychological, immunological and symptom occurrence data are conducted (e.g., Solomon, Temoshok, O'Leary & Zich, in press). The AIDS phenomenon has been a forceful instigator of the search for co-factors, particularly psychosocial variables, that determine the progression of the disease (Todd, Burcham, Grant & Penny, 1987; Cohen & Wiseman, 1986).

Antoni and his collegues (personal communication, June, 1990) are involved in examining the influence of behavioural factors on immune functioning and disease progression among HIV infected individuals, and the possible benefits behavioural intervention such as exercise, training and relaxation may have on these patients. Finally, psychoimmunology has had other important contributions to AIDS research and treatment, promoting and substantiating more creative approaches to this disease, both by health professionals and the sufferers themselves. The introduction of multidisciplinary interventive strategies, and popularity of "alternative" healing practices such as relaxation, yogic practices, homeopathic and naturopathic, and spiritual support, is enthusiastically discussed in a paper by R.L. Cecchi (1984) of the Gay Men's Health Crisis Institute in New York.

6. Psychological Intervention and Immune Function: The Role of Health Psychology

6.1 Introduction

Despite the evidence accumulated by psychoimmunological research that stress and associated negative affect or distress is potentially threatening to one's physical health status, there is a surprising lack of research directed at investigating the possibilities of influencing immune function to promote health. This is even more surprising in the face of the recent evidence that hypnosis has been found to alter various components of the immune response (see Jemmott III & Locke, 1984), and the possibility of behavioural conditioning of the immune response (e.g., Smith & McDaniel, 1983). It is applicable to reiterate Solomon and Temoshok's (1987) third hypothesis governing the relationship between psychosocial stress and immune function. Namely:

> Behavioural and/or psychological interventions aimed at countering the adversive effects of stress, should also be able to enhance or optimize immune function.

The evidence presented in this chapter tends to support Solomon and Temoshok's (1987) first and second hypotheses that various psychological states and traits (or coping styles) seem to influence immune system regulation, thereby legitimizing a serious consideration of the third hypothesis. The role that health psychology may play in ameliorating the adversive effects of stress in high risk patient populations (i.e., those patients suffering from an immunologically resisted disease which has "known" associations with stress-related immune dysregulation) through the utilization of cognitive-behavioural techniques, is explored in this final section.

It is also apparent that efforts to counter stress in such patient populations, need to include a consideration of the psychological impact of their particular disease. Furthermore, cognitive-behavioural interventions also need to

ensure that patients adopt more adaptive coping behaviours in relation to their illness experience (i.e., the disease effects). As such, the area of pain management, particularly for the autoimmune disorders, becomes a central element of any cognitive-behavioural intervention.

6.2 Psychoimmunological Interventions

6.2.1 Behaviourally Conditioned Immunosuppression

The most extraordinary area of research demonstrating the possibility of psychological intervention to regulate immune function, has been studies demonstrating behaviourally conditioned immunosuppression. As early as the mid seventies, Ader and Cohen (1975) demonstrated the latter in rats using a taste aversion paradigm where saccharin (conditioned stimulus) was paired with cyclophophamide (unconditioned stimulus), a toxin which is immunosuppressive. After conditioning, subsequent exposure to saccharin on its own was found to exert an immunosuppresive effect as measured in lower antibody titres to an antigen.

Until recently, behavioural conditioning of the immune response was only demonstrated in animal research (Smith & McDaniel, 1983). Smith and McDaniel (1983) report data from their laboratory of a possible psychologically mediated effect on the delayed hypersensitivity reaction to tuberculin in humans. In this study the subjects expected to receive a tuberculin skin test (the Mantoux test) to each arm to compare the reproducibility of the tuberculin reaction (i.e., the size and quality of the resultant skin lesion). In actuality, a saline solution was consistently administered to the one arm (unbeknown to the subject). According to Smith and McDaniel (1983), results showed that the subjects' expectation alone, determined the delayed hypersensitivity response to the "tuberculin" antigen.

6.2.2 Social Support Programs

Very few studies have examined the extent to which psychological and behavioural interventions, aimed at alleviating stress, would promote immune system functioning. Social support, which has been linked to decreased morbidity and mortality (Matarrazo et al., 1984; Cohen, 1988), has been hypothesized to mitigate (the buffering hypothesis, Cohen, 1988) or mediate the harmful effects of stressful experience on the individual (Schwarzer & Leppin, 1989; Thomas, Goodwin & Goodwin, 1985). It is thus interesting that studies have not sought to investigate the possible immunological consequences following an improvement of social support for specific populations.

A problem facing such research is the difficulty of achieving a consensual definition of social support. According to Cohen (1988), "...the term has been assigned to a broad range of conceptualizations of social networks, and the functions they provide." (p. 270) A solution to the problem, however, is to limit one's definition of the construct to the social support measure utilized.

Thomas et al. (1985) recently examined the effect of social support (defined as the frequency and quality of social interaction) on stress-related (stress operationalized as symptoms of distress) changes in cholesterol levels, uric

acid level, and immune function in an elderly sample. Results from data obtained from 256 healthy elderly pensioners indicated a statistically significant inverse relationship between the degree of social support (based on an interview schedule for social interaction) and level of serum uric acid/cholesterol. In addition, the study found that social support was significantly positively correlated with total numbers of circulating lymphocytes.

The researchers (Thomas et al., 1985) concluded that social support systems may intervene between stressful stimulus and the physiological response to that stimulus, their findings concurring with the buffering hypothesis of social support. However, Thomas et al.'s (1985) results must be viewed with some caution for two main reasons: the design was retrospective; and only one aspect of social support was measured (i.e., cognizance was not taken of the existence, availability, and quality of social networks, relationships and resources). Such problems prevent a detailed analysis of which aspects of social support are the most important mediators in the stress-disease relation. Thomas et al.'s (1985) study highlights an important area of investigation into one possible avenue of psychological intervention with particular patient populations.

6.2.3 Stress Management Programs

Another area of investigation is the assessment of the immunological (and clinical) benefit of relaxation training to alleviate symptoms of the human stress response. Kiecolt-Glaser, Glaser, Williger, Stout, Messick, Sheppard, Ricker, Romisher, Briner, Bonnel & Donnerberg (1985b) examined whether reductions in distress might enhance immune function in 45 geriatric residents in an old-age home. The subjects were randomly assigned to one of three protocols: relaxation training, social contact, or no intervention. The interventions took place three times weekly for a month. The relaxation group, compared to the non-significant changes in the social and no-intervention groups, showed a significant enhancement on two parameters of cellular immune function (greater natural killer cell activity, and lower antibody titres to HSV Type 1 antigen). Furthermore, Kiecolt-Glaser et al. (1985b) found that such changes were also associated with decreases in distress-related symptomatology.

Antoni et al. (in press) are at the forefront of research investigating the "immunomodulatory capabilities" of behavioural interventions. Focussing on HIV infected individuals, they are investigating the benefits of aerobic exercise, stress-management, and relaxation training on various measures of immune status and endocrine secretions. The central role of mediating factors such as locus of control, perception of coping efficacy and social support, and self-esteem on stressors such as anxiety, depression and social isolation constitute a key element of their investigations (Antoni et al., in press). Results from work in progress show significant psychological (e.g., reductions in anxiety and depression, improved self perception of coping efficacy and esteem) and immunological (e.g., increase in helper T-cells, increase circulation of immunoglobulins) benefits (Antoni, personal communication, April, 1990).

6.2.4 Summary

Numerous opportunities exist to research the potentially beneficial consequences of interventions such as those by Kiecolt-Glaser et al. (1985b) and Antoni et al. (in press). Whilst numerous difficulties exist in psychoimmunological research in general (see summary section 5.3.6), the value of evaluating the effect of

psychological intervention in controlled, well-designed longitudinal studies (such as with Antoni et al., in press) is an important area for future research. An important feature of such psychological interventions is that the intervention has two aims: to improve the psychological experience of the patients involved, as well as attempt to produce meaningful change on an immunological level.

The small number of studies reviewed in this section are indicative of the need for research in this area. In Chapter Three, research investigating the effects of cognitive-behavioural interventions on the psychological, immunological and physical health status of rheumatoid arthritis patients will be examined closely. The role of health psychology in assisting the treatment and care of individuals suffering from chronic disease is clearly demonstrated in such research.

CHAPTER THREE

RHEUMATOID ARTHRITIS AND HEALTH PSYCHOLOGY: A COGNITIVE-BEHAVIOURAL APPROACH

Overview

The central aim of this chapter is to introduce rheumatoid arthritis as an appropriate and important disease to both explore the value of psychological intervention in a medical condition, and to test the assumptions of the psychoimmunological and cognitive-behavioural paradigms. Rheumatoid arthritis is also considered in the context of the synchronous systems model of health and disease (Jasnoski & Schwarz, 1985), and Everly's (1988) delineation of the human stress response.

Following an introduction to the chapter, an overview of the aetiology, pathology, and treatment of rheumatoid arthritis is provided. Rheumatoid arthritis is seen as a complex disease phenomenon of unknown cause and unpredictable course, involving various physiological systems, autoimmune mechanisms, and patient characteristics. The multidimensional nature of the disease, and the involvement of multidisciplinary health care demands a radical reformulation of its management.

The bulk of the chapter is dedicated to an examination of the research documenting the psychological factors related to the disease in terms of: (a) the impact of rheumatoid arthritis, (b) as determinants in disease onset and course, and (c) the benefits of cognitive-behavioural intervention. The review of research investigating the value of cognitive-behavioural intervention in rheumatoid arthritis is preceded by an overview to the underlying assumptions and strategies employed in such interventions.

The chapter thus attempts to incorporate the models introduced in Chapter One and the research evidence reviewed in Chapter Two, relate these to a particular disease, and provide a basis for the thesis presented in Chapter Four.

1. Introduction

The argument presented in Chapter One, that disease should be viewed as both multifaceted and multidetermined and not simply as a function of exposure to an external pathogen, is generally supported by the research reviewed in Chapter Two. An important implication of this argument, especially for psychology, is that how the individual responds to his/her disease should also influence the course thereof. Both Jasnoski and Schwarz's (1985) synchronous systems model, and Everly's (1988) delineation of the human stress response, underscore the crucial role of the individual in responding (i.e., coping behaviour) to the onset of disease (or dysregulation) or the course

thereof. Chronic disease demands a response from the individual with the aid of an intact health care system. The central aim of this Chapter is to explore the nature of this response in the context of a specific chronic disease condition, rheumatoid arthritis (hereafter RA). The Chapter also seeks to introduce a particular perspective that psychology may adopt in the analysis, assessment, and treatment of individuals suffering from this disease; namely, a cognitive-behavioural perspective.

A cognitive-behavioural perspective of behavioural medicine highlights the central role of cognitive factors (i.e., cognitions and actions governing a response to the disease) in the course of the disease. The onset of symptoms can be considered an acute stressor, whilst the incidence and progression of symptoms (e.g., pain, discomfort, disability or morbidity) over time can be viewed as chronic stressors for the patient (cf., Everly 1988)

According to Turk et al. (1983), the cognitive factors involved in the incidence and progression of disease are viewed as:

> (1) determining the ways in which individuals define health, disease and illness; (2) influencing decisions regarding the utility of engaging in either health-promoting...or risk related behaviours; (3) determining how individuals respond to symptoms and incapacities; (4) influencing how individuals utilize the health care system; and (5) contributing directly and indirectly [as in the case of psychoimmunology] to disease and illness.
>
> (p. 23)

Bandura's (1977) concept of "reciprocal determinism" (see Chapter One) demonstrates the role that cognitive factors would play in determining the interaction between the individual and their "internal" (i.e., physical or biological) and "external" (social or interpersonal) environments. Jasnoski and Schwarz's (1985) notion of "optimization" highlights the important role that the individual plays in determining and responding to these two environments. The response to symptoms is partly determined by cognitive factors, and as such these cognitive factors determine the efficacy of the optimization process. The excellent example given by Turk et al. (1983) provides an indication of the relevance of these similar concepts of "reciprocal determinism" or "optimization" in the disease process, and will be quoted in its entirety.

> A man awakens one morning and notes a dull throbbing in the frontal region of his head. The intensity of the stimulus may focus his attention on the sensations, and he may attempt to evaluate them, to decide what is the cause, probable course, and so on. He may, then, regard the pain as simply as a minor inconvenience resulting from his excessive alcohol intake the night before...; alternatively, he may regard it as a sign of a brain tumor, from which his father died. These different evaluations would likely lead to different emotions,...which in turn would partially determine behaviour....His choice [of action or behaviour] will depend in part upon his prior history [i.e., what actions he had taken before to alleviate the distress or symptoms]....Whichever course of action the man chooses, he will create a unique environment....The particular environment he shapes for himself in this way will, in turn, have effects on his thoughts, feelings, and actions....Each environmental route will have its own consequences....This complex chain of events may...be quite automatic and...have

> minimal impact on the man's life....On the other hand, it might involve a much longer period of time and have a powerful impact upon other aspects [e.g., vocation, family, etc.] of his life.
>
> (Turk et al., 1983:22-23)

The extent to which the health of the person is influenced, is dependent on the extent of the initial dysregulation and the individual's response to it. The nature of his/her response, in the context of his/her physical and social environments, would in turn determine the extent to which medical intervention or treatment is sought. Finally, the individual's response to treatment regimens would play a key role in his/her ultimate health status. Perhaps the pain is because of a tumor, hence early diagnosis could determine the prognosis. Depending on the stage of the disease, the individual is challenged to respond (i.e., to cope), and this response may influence subsequent actions taken by both patient and the health care system.

A cognitive-behavioural perspective would thus focus on the cognitions and actions taken or adopted by the patient during the course of his/her disease or illness. Illness may be viewed as being composed of five stages (Turk et al., 1983): (1) symptom perception, (2) medical contact, (3) acute illness, (4) convalescence and rehabilitation, and (5) chronic illness and/or disability. At all stages of the individual's illness, there is a constant process of appraisal and re-appraisal of his/her symptoms or condition, the treatment, and the impact of the disease on personal and social roles and activities. The meaning individuals thus ascribe to their illness is an important factor in the care and treatment of their condition.

Rheumatoid arthritis (RA), an autoimmune disorder (i.e., involving key components of the immune system), is considered an excellent example to use to test the concepts of Jasnoski and Schwarz's (1985) and Everly's (1988) models, the assumptions of the broad field of psychoimmunology, and the value of a cognitive-behavioural perspective in the treatment of the disease. Being perhaps the most common and best recognized of the rheumatic diseases (Anderson, Bradley, Young, McDaniel & Wise, 1985; Bennet, 1985), it is an easily studied disease phenomenon. This factor, as well as the significant psychological and social impact of the disease (Gardiner, 1980; Achterberg-Lawlis, 1982; Banwell & Ziebell, 1985; Anderson et al., 1985), makes it an important area of investigation for Health Psychology. RA underscores the value of research conducted under the broad rubric of behavioural medicine, as well as the prominence of the cognitive-behavioural paradigm in devising explanatory models and treatment strategies.

Prior to an examination of cognitive-behavioural interventions with RA patients, it is necessary to examine: (1) the cause and nature of the disease; (2) its psychological, behavioural, and social impact; (3) the evidence linking psychological factors to RA incidence and progression (e.g., psychoimmunological research findings); and (4), the basic assumptions governing cognitive-behavioural strategies and the value thereof.

2. Rheumatoid Arthritis

2.1 Aetiology and Pathology

RA is a chronic, degenerative systemic disease manifesting itself primarily in the joints , with primary symptoms of joint pain and stiffness, and fatigue (Harris, 1985; O'Leary, Shoor, Lorig & Holman, 1988). It occurs in about 1% of the general adult population between the ages of 20 and 50 (Kelly, Harris, Ruddy, & Sledge, 1985), with women being more commonly affected than men: the overall ratio being 3 to 1 (Anderson et al., 1985). It is believed that a similar epidemiological pattern is present in South Africa (Meyers, personal communication, March, 1990).

Despite intensive research into the aetiopathogenesis of RA, there is to date no known specific cause nor cure for this condition (Anderson et al., 1985). According to Bennett (1985), several aetiologic mechanisms have been proposed as having some influence on disease manifestations, including autoimmune, endocrine and metabolic processes. Other "causal" agents include viral infections and genetic and nutritional factors (Harris, 1985). Similarly, geographic, occupational and other demographic variables have also been studied with similar conclusions of having possible influence on disease manifestations (Anderson et al., 1985). These latter influences are not necessarily causative (Anderson et al., 1985).

The pathology of RA has its basis in an inflammatory response in the membrane surrounding joints, involving autoimmune processes (Harris, 1985). It is for this reason that it is known as an "autoimmune disorder". Very briefly, this immune response involves the production of local antibodies and antigens, increased vascular inflammation, and proliferation of lymphocytes in the synovium lining (Anderson et al., 1985). These substances damage the synovium and cartilage between the joints (generally the hands, wrists, and knees), cause soft tissue swelling and warmth, and result in pain and deformity. As this proliferation continues, the lesions within the synovium lining can lead to joint destruction (Harris, 1985).

According to Harris (1985), "the rate of progression of the disease depends upon both the intensity and the chronicity of the disease." (p. 886) Thus, the disease of a patient who experiences intermittent flares of joint inflammation will not progress as rapidly to joint destruction as the disease of a patient who has continuous manifestations of joint inflammation (Harris, 1985). All the aetiologic mechanisms, causal agents and demographic variables mentioned above, play an important role in determining the intensity and chronicity of an individual patient's disease.

The onset of RA is largely insidious, with only a minority of patients experiencing an acute onset of symptoms (Harris, 1985). The course thereof is chronic and unpredictable (Weiner, 1975; Bradley, Young, Anderson, McDaniel, Turner & Agudelo, 1984). The "...waxing and waning course of these painful periods of inflammation is often accompanied by the progressive and irreversible deterioration of the joint itself, causing additional pain and limitations in mobility." (Smith, Peck & Ward, 1990) As Weiner (1975) wrote, "rheumatoid arthritis patients learn, along with their diagnoses, that the disease is not only incurable, but its specific manifestations are unpredictable." (p. 98)

2.2 Medical Treatment Regimens

Biomedical treatment of this disease is possible, yet only partly successful (Ruddy, 1985). Reasons for this lack of success (Ruddy, 1985; Harris, 1985; Anderson et al., 1985; Achterberg-Lawlis, 1982; Meyers, personal communication, March, 1990) include:

(1) difficulties in identifying a causal agent (see above);
(2) inadequate tailoring of treatment regimen to each patient's unique condition and characteristics;
(3) poor patient compliance (i.e., nonadherence with medication) with treatment recommendations (reviewed in Anderson et al., 1985);
(4) poor communication between patient and clinician in the explanation of the nature of the disease, and regarding treatment goals;
(5) damage to the joints through overactivity or the lack thereof;
(6) costs (in terms of treatment expense as well as a result of vocational disability); and
(7) unclear or inadequate definition of "treatment outcome".

Despite such limitations, according to Ruddy (1985), a pessimistic approach is not warranted for "...most patients have gratifying responses to treatment...allowing them to continue to function [relatively] harmoniously with their environment." (p. 979) Explicit goals of the management of RA include the relief of pain, the prevention of joint destruction, and the preservation or improvement of the patient's functioning (Ruddy, 1985)

Such goals, according to Hunder and Bunch (1982, cited in Anderson et al., 1985), need to be seen in the context of a combination of efforts to minimize tissue injury and loss of function, balance favourable and adverse drug effects, and repair damage that cannot be prevented. Furthermore, the highly variable nature of the disease and the differing life-styles of patients require an individualized treatment regimen (Anderson et al., 1985). A basis for a satisfactory outcome of treatment, according to Harris (1985), is the understanding by the patient and members of the health care team of realistic therapeutic goals. Such therapeutic goals are based on the needs and priorities of the individual patient and weighed against the risks of various therapeutic options (Harris, 1985). Members of an arthritis health care team, according to Harris (1985), include (in order of priority):

(1) patient, (2) family physician, (3) rheumatologist, (4) orthopedist, (5) nurse specialist or practitioner, (6) occupational therapist, (7) physiotherapist, (8) rehabilitation or vocational advisor, (9) psychologist, (10) social worker, (11) pharmacist, (12) nutritionist, (13) clergy, and (14) community agencies.

This description of the people responsible in the treatment and care of a RA patient is revealing for three main reasons. Firstly, it places the individual patient as the primary member of the team thereby emphasizing: (a) the key role played by his/her response to the disease, and (b) the extent to which all activities of the team are centered around the patient. Secondly, this description demonstrates the complex nature of the disease as well as the need for multidisciplinary involvement in arthritis health care. And finally, taken that the description is from an

important medical textbook on rheumatology, the inclusion of a psychologist in the team is noteworthy of the current revolution or "heresy" in medicine. The important assumptions of Engel's (1977) biopsychosocial model are clearly being applied in Harris's (1985) description. The important role that psychology may play in the treatment of RA patient(s), specifically the utilization of cognitive-behavioural stress and pain management techniques, will be discussed in detail below (see section 4).

Therapy is thus a combination of four major areas of intervention (see Harris, 1985). These are discussed briefly.

2.2.1 Supportive Measures

Such measures, according to Harris (1985), include patient education on the nature of the disease process and the treatment thereof, emotional support, rest, exercise and physiotherapy (to strengthen and maintain joint movement), and occupational therapy and rehabilitation (to assist the patient in improving his/her functional status).

2.2.2 Pain Relief

The pain relief strategies to be utilized, "...requires a decision about the cause of the pain." (Harris, 1985; Meyers, personal communication, March, 1990). Treatment includes reducing disease activity (e.g., Non-steroidal Anti-inflammatory Drugs, or NSAID's), use of analgesics, immobilization of joint/limb, surgery, and even hospitalization (Harris, 1985). It is important to note that one's understanding of the nature of the pain experience governs both the decision and type of pain relief strategy adopted.

The measures discussed by Harris (1985) do not take into account the role of psychological factors in the experience of pain, particularly chronic pain. The cognitive, affective, and behavioural responses of the patient need to be addressed (Melzack & Perry, 1975; Melzack, 1983; Melzack & Wall, 1965, 1988; Turk et al., 1983; Karoly & Jensen, 1987; Felton & Revenson, 1984; Bradly et al., 1984; Ciccone & Grzesiak, 1984; Smith, Peck, Milano, & Ward, 1988; Arntz & Schmidt, 1989; Ingram, Atkinson, Slater, Saccuzzo, & Garfin, 1990). The cognitive-behavioural dimensions of pain will be discussed in section 3 below.

2.2.3 Anti-Inflammatory Drugs

The aim of anti-inflammatory medication is to reduce the inflammation, ameliorating the warmth, swelling, and stiffness between and around the joints (Harris, 1985). Such medication (discussed in Anderson et al., 1985; Harris, 1985) include the Non-steroidal Anti-inflammatory Drugs (NSAID), systemic corticosteroids, and local injections of corticosteroids.

2.2.4 Remission-Inducing Agents

Such medication includes "long-acting agents [which] do not provide analgesic or anti-inflammatory relief but tend to act slowly, usually over a period of 2 to 6 months." (Anderson et al., 1985) Agents which have been shown to induce remission include: antimalarials, gold salts, and penicillamine (Harris, 1985).

3. Psychological factors and RA

Psychological factors associated with RA are important both in terms of the adverse effects the disease has on patients, as well as the possible causal role personality traits and/or psychological states may have in disease onset and progression (cf., the psychoimmunological literature). The link between psychology and RA is supported both by anecdotal accounts and research findings. Patients generally attribute their disease and its later exacerbation to life experiences (e.g., stressful life events), and emphasize the marked changes RA has effected on their lives.

Studies investigating the link between psychological factors and RA, have attempted to (a) assess the psychological impact of RA (e.g., Gardiner, 1980; Meenan, Yelin, Nevitt, & Epstein, 1981; Vollhardt, Ackerman, Grayzel, & Barland, 1982; Kazis, Meenan, & Anderson, 1983; Liang, Rogers, Larson, Eaton, Murawski, Taylor, Swafford, & Schur, 1984; Banwell & Ziebell, 1985; Reisine, Goodenow, & Grady, 1987; Hawley & Wolfe, 1988; Frank, Beck, Parker, Kashani, Elliott, Haut, Smith, Atwood, Brownlee-Duffeck, & Kay, 1988; and Smith et al., 1990), (b) examine the psychological and behavioural determinants of disease onset and progression (e.g., Hendrie, Paraskevas, Baragar, & Adamson, 1970; Moldofsky & Rothman, 1971; Crown, Crown, & Fleming, 1975; Rimon & Laakso, 1985; McFarlane & Brooks, 1988), and (c) identify co-factors mediating the effects of the disease on the psychological well being of the patient (e.g., Parker, McRae, Smarr, Beck, Frank, & Walker, 1988b).

The psychological literature on RA will be reviewed according to the above three categories of research interest.

3.1 The Impact of RA

Psychological reaction to RA, according to Banwell and Ziebell (1985), "...is an interaction between the disease, its treatment, and the patient's perception of their illness [including their pain]." (p. 498) Whilst some features of the disease are more likely to have psychological effects (Banwell & Ziebell, 1985), it is important to note that the nature of the patient's response is more a function of their perception or appraisal (see Ciccone & Grzesiak, 1984; Everly, 1988) of the symptom, than the symptom itself. The changes in physical appearance (e.g., as a result of joint deformities), the chronic pain and fatigue, the loss of energy and functional ability (Banwell & Ziebell, 1985), and the unpredictable course of the disease (Weiner, 1975) can have significant adverse effects on a patient's affective, behavioural and social functioning (Gardiner, 1980; Ciccone & Grzesiak, 1984; Anderson et al., 1985). Such factors can be considered as significant stressors in the RA patient's daily life, and require some form of coping behaviour. The nature of such coping behaviour depends upon the patient's perception of the stressor and determines the ultimate impact of the disease.

3.1.1 Chronic Pain

Chronic pain, defined as intractable benign pain which is present most of the time or longer than 6 months (Turk et al., 1983: 75), is of primary concern for both RA patient and health-care worker (Kazis et al., 1983). Kazis et al. (1983) demonstrated the importance of pain levels in both physician and patient's evaluation of overall health. The Arthritis Impact Measurement Scales (AIMS) were used to assess physical disability, psychological status, and pain in a sample of 729 RA patients. Results showed that pain was a significant indicator of health status, medication usage, and predictor of future pain level and disability. Kazis et al. (1983) concluded that "...pain is an important contributor to health status and health behaviour of the rheumatic disease patient." (p.1021)

According to Hart (1974, cited in Anderson et al., 1985), patients with RA can experience pain from multiple sources. These include "...local pain in joints due to inflammation, chronic changes in articular tissue, complications of the arthritis, systemic illness, [and] medication side-effects...." (Anderson et al., 1985: 370) As Banwell and Ziebell (1985) wrote, "people with arthritis must learn to live and function with chronic pain while still attending to the signals of acute pain." (p. 502)

Symptomatic relief of joint inflammation and the "judicious use of remittive drugs" (as discussed above in section 1.2) are only partly effective as strategies for managing arthritic pain (Parker, Frank, Beck, Smarr, Buescher, Phillips, Smith, Anderson, & Walker, 1988a: 593). Coupled with the unpredictable course of the disease (Weiner, 1975), and the seemingly hopeless prognosis, the RA patient's perception of the possibility of pain relief (both present and future) is seriously undermined.

This lack of effective control over pain may have a profound psychological effect on the patient (Garber & Seligman, cited in Bradley et al., 1984; Felton & Revenson, 1984; Arntz et al., 1989; Smith et al., 1990). These include (Ciccone & Grzesiak, 1984): (1) adverse mood/affect (e.g., anger and depression), (2) behavioural disorders such as inactivity, (3) psychophysiologic disorders such as elevated muscle tension, and (4) poor psychosocial functioning (e.g., reduced social interaction).

3.1.2 Affective, Cognitive and Behavioural Disturbance

Changes in mood include: (1) depression (Frank et al., 1988; Liang et al., 1984) and the related experience of "learned helplessness" (Smith et al., 1990) or "perceived lack of control" (Arntz et al., 1989); (2) anxiety (Moldofsky & Rothman, 1971; Volhardt et al., 1982) and associated denial (Banwell & Ziebell, 1985); and (3) anger (Levitan, 1981, cited in Anderson et al., 1985).

In an early prospective study on the relationship between mood and RA pain, Moldofsky and Chester (1970) found a significant relationship between fluctuations in joint tenderness and mood changes. Moldofsky and Chester (1970) identified two pain-mood patterns among hospitalized patients: a "synchronous state", characterized by mood changes within an anxiety or hostility spectrum; and a "paradoxical state" in which pain intensity was inversely related to a sense of hopelessness. Mood changes in the former were either preceded or accompanied by changes in pain.

Liang et al. (1984) examined the psychological experience of a sample of patients with RA or systemic lupus erythematosus (SLE). Administering the Schedule of Recent Events (SRE), the Minnesota Multiphasic Personality Inventory (MMPI), and the Health Locus of Control scale (HLC), the researchers found that both diseases had a profound impact on the patients' psychological and social lives. Both groups experienced mild to moderate life crises in relation to family, spouse (arguments and sexual problems), and social activity.

Results from the MMPI were most revealing, with marked elevations in the Depression, Hypochondriasis, and Hysteria scales. According to the researchers, "...the magnitude of present and feared future loss account for the abnormal Depression scores." (Liang et al., 1984: 18) The abnormal scores on the Hypochondriasis scale was interpreted as being more a function of the multiplicity of symptoms and the continuous need for self-monitoring, than actual dysfunction. Finally, the researchers argue that hysterical symptoms in such patients "...may help rechannel anxiety released by the uncertain, potentially disabling, life-threatening aspects of these diseases." (Liang et al., 1983: 18)

In a 3 year prospective study of 400 RA patients, Hawley and Wolfe (1988) investigated the relationship between psychological (depression and anxiety) and clinical factors using the Arthritis Impact Measurement Scales (AIMS). On entry into the study, Anxiety was found to be more common than Depression in this sample. Furthermore, clinical and demographic variables explained 25% of the variance in psychological scores, indicating that an important proportion of total anxiety and depression is related to the disease itself (Hawley & Wolfe, 1988). Such findings, however, do indicate that most of the psychological factors are due to the patient's characteristics and life experience. The longitudinal data tend to support this finding (Hawley & Wolfe, 1988), where the development of depression over the course of the study was unrelated to clinical features (including pain and disease activity variables). Such development was strongly related to socioeconomic characteristics (i.e., age, sex, race, marital status and family income). Hawley and Wolfe (1988) conclude that their findings do not suggest that disease factors are unimportant, but rather that their effect on psychological scores are buffered by internal coping mechanisms.

Hawley and Wolfe's (1988) findings are somewhat supported in the study by Frank et al. (1988). The latter sought to determine whether the presence of depressive symptoms in RA could be attributed to underlying depressive disorders rather than clinical factors. Frank et al. (1988) applied DSM-III diagnostic criteria for depressive disorders to a large sample (137) of RA out-patients. Furthermore, the relationship between pain and disease activity and depression (both current depressive symptoms and history of depressive disorders) was also examined. A large percentage (42%) of the sample met the criteria for a depressive disorder. This overall rate of depressive disorders supported those found by Rimon & Laakso (1985) in a 15-year follow-up study (see below, section 2.2.3). Frank et al. (1988) caution that the endorsement of symptoms of RA such as fatigue and insomnia by the DSM-III, may have inflated the level of depressive disorders. This difficulty of using instruments with "criterion contamination" (i.e., responding to features associated with a chronic illness rather than to psychological status) in RA research, was noted by Hawley and Wolfe (1988). The latter endorse the use of the AIMS, which, they argue "...do not appear to have differential sensitivity to specific diseases...." (p. 940)

All depressed patients in Frank et al.'s (1988) study reported significantly more pain (McGill Pain Questionnaire, Visual Analogue Scale, and pain questionnaire) than nondepressed patients. An important implication of this result was the finding (using discriminant function analyses) that the best predictor of depression diagnosis was the number of words chosen on the MPQ. Frank et al. (1988) point out that such findings indicate the need for clinicians to "...recognize that depression is predicted by the patient's appraisal of his/her disease, including pain and satisfaction." (p.942)

A variety of cognitive-behavioural approaches to RA have identified several psychological factors as mediating the relationship between RA and depression or psychological status. Parker et al. (1988b) investigated the importance of coping strategies in determining the health status (psychological, functional, pain, and disease activity) for 84 out-patients with RA. The researchers utilized an extensive battery of psychological (including the Ways of Coping, Symptoms Checklist-90-Revised, Beck Depression Inventory, Hassels Scale, and Arthritis Helplessness Index), a functional status (the AIMS), pain (Visual Analogue Scale and McGill Pain Questionnaire), and disease activity measures.

Results indicated a significant relationship between coping process and psychological and functional status. The researchers reported that patients who cope with their disease by restructuring life goals, rather than hoping for unrealistic solutions and engaging in self-blame, are less likely to be depressed and have better functional status (Parker et al., 1988b). According to Parker et al. (1988b), there was a tendency among patients in their sample (having limited income and low education level) to use "wish fulfilling fantasy" as a coping strategy. This strategy was associated with higher levels of depression, "helplessness", more daily stress, and greater psychological distress (Parker et al., 1988b).

In their discussion of their findings, Parker et al. (1988b) argue that the study demonstrates that "...patients who are unable to think (i.e., cognition) realistically about their situation and to restructure their life goals, encounter substantial psychological difficulties as a consequence of their RA." (p. 1382) This study, furthermore, provides support for the clinical notion that helping patients to develop realistic plans and goals may promote better psychological adjustment and improved functional capacity (Parker et al., 1988b). This will be explored in greater detail below (see section 4).

The results of Parker et al.'s (1988b) study support those of an earlier study by Felton and Revenson (1984) which examined the emotional consequences of two coping strategies, information seeking and wish-fulfilling fantasy, in the context of the "controllability" of four different chronic illnesses. The Ways of Coping Scale (WOC) was administered to a sample of 151 hypertensive, diabetic, RA and systemic blood cancer out-patients. It was found that the value of the coping strategy adopted was related to the degree of "realism" and accuracy of perceptions they involve (Felton & Revenson, 1984). According to Felton and Revenson (1984), "...in our data, information seeking, representing the active...confrontational approach to illness, is linked to decreased negative affect, whereas wish-fulfilling fantasy, the avoidant strategy of diverting attention from the realities at hand, is linked to indicators of poor [psychological] adjustment." (p. 350)

An interesting finding of Felton and Revenson (1984), was the fact that the consequences of coping were not related to the controllability of the individual's disease. This highlights the importance of the individual's appraisal of their disease over and above the actual nature of the disease itself, in terms of the psychological impact of the disease. Hence, the individual's perception of controllability determined the coping strategies utilized and the ultimate outcome in psychological health status or adjustment. The perceived lack of control over the disease and its symptoms, especially pain, thus becomes an important mediator in the disease--psychological morbidity equation.

In a recent study, Smith et al. (1990) investigated this equation more closely, exploring the important mediating role of the RA patient's appraisal of their disease, and the psychological impact thereof (i.e., depression). The researchers hypothesized that depression resulted more from patients' beliefs that they could not control their disease or its impact (i.e.,helplessness), than the actual severity of disease state. Whilst severity of depression was found to be a function of disease severity, the association was mediated by patients' views of their ability to control or cope (i.e., scores on the Arthritis Helplessness Index) with their disease (Smith et al., 1990). Smith et al. (1990) conclude that cognitive-behavioural interventions focussing on helplessness may be particularly useful in ameliorating depression among RA patients. Abramson, Metalsky and Alloy's (1989) delineation of the cognitive factors facilitating the development of helplessness is an important paper in this regard.

As pain is central concern for the RA patient, the effect that this notion of helplessness and the coping strategies adopted have on the experience thereof, is an important area of research. The unpredictable course of RA and the waxing and waning of periods of joint inflammation and acute pain (Smith et al., 1990) possibly has great effects on the patient's perception of his/her ability to cope (i.e., control) with pain. The importance of this observation is best elaborated in Arntz and Schmidt's (1989) comment that "...some chronic pain patients seem to suffer in a manner that is disproportionate to medical findings and display very helpless behaviour, whereas others lead fairly undisturbed lives." (p.131) In section 4, this will be discussed in more detail.

The research reviewed above lends strong support to Everly's (1988) model of the human stress response. There is evidence of the central role that appraisal processes play in determining the coping behaviour adopted by the person, and the ultimate impact that stressors (e.g., disease condition and/or symptoms such as pain) have on the health status of the individual RA patient.

There is a clear trend in psychological research on RA where studies are moving towards an examination of mediating factors governing the impact of RA. An important implication of such research interest is the development of a knowledge base whereby psychological intervention, particularly cognitive-behavioural approaches, may successfully treat and/or reduce the negative impact that RA has on patient's psychological and functional status. Such interventions will be reviewed in section 4. It is necessary, at this point, to briefly examine the psychosocial impact that RA has on the patient, keeping in mind the role of mediating cognitions and coping strategies.

3.1.3 Psychosocial Function

RA patients also experience major changes in their psychosocial functioning as a result of their chronic disease. These changes include reductions in self-esteem, life-satisfaction, social activities, and vocational and/or family functioning (Banwell & Ziebell, 1985; Anderson et al., 1985).

Meenan et al., (1981) attempted to delineate the sociomedical problems experienced by RA patients. A detailed questionnaire and interview was conducted with 245 patients, eliciting information on: demographic characteristics; disease and treatment history; work (i.e., occupation and estimates of physical demands and autonomy) and income effects (i.e., lost earnings, and income sources); and psychosocial changes (i.e., frequency of changes in work disability, divorce, family employment, residence, and leisure activities). Meenan et al. (1981) found that major losses in the areas of work, finances, and family structure were extremely common. The majority of workers were totally disabled, and 63% of the total sample experienced a major change in their psychosocial status as a result of RA (Meenan et al., 1981). Meenan et al. (1981) argue that their findings raise questions of the emphasis and approach of arthritis health care workers:

> The ultimate goal of medical care is to improve the well-being of the patient. It is clear from this study that the prevention and amelioration of socioeconomic problems should be a major aspect of the care of an individual with rheumatoid arthritis.
>
> (Meenan et al., 1981: 548)

The psychosocial impact of RA as a result of physical disability has been investigated by Deyo, Inui, Leiniger and Overman (1982, cited in Anderson et al., 1985). Utilizing the Sickness Impact Profile (SIP), it was found that between 43% and 52% of RA patients reported dysfunction on the psychosocial dimension subcategories of social interaction, communication, or emotional behaviour.

These findings are supported by more recent research by Resine et al. (1987) and Reisine, Grady, Goodenow, & Firfield (1989), who examined the impact of RA on women as "homemakers" in the former, and as workers in the latter. Reisine et al. (1987) developed a measure of homemaker functioning based on conceptualizing the homemaker role on two levels: "...the instrumental functions associated with meeting the physical needs of the household, and the nurturant dimension concerned with meeting the expressive needs of the household." (p.90) Reisine et al. (1987) administered the questionnaire to a sample of 142 female RA out-patients, and found that RA significantly limited both instrumental and nurturing functions associated with managing a household. In their discussion of their findings, Reisine et al. (1987) argue that it is imperative to examine disease impact on the homemaker when evaluating the effects of RA on women.

Based on the evidence of the work disability being a major result of RA, Reisine et al. (1989) explored the added effects of women's home responsibilities on the ability to remain employed. It was found that greater family responsibilities were associated with lower risk of work disability. According to Reisine et al. (1989), "having more responsibilities in the homemaker role may contribute to a sense of self-worth that enhances, rather than undermines, social functioning." (p.542)

3.1.4 Summary

The psychological and social impact of RA is an important area of concern for both researchers and clinicians working with RA patients. The psychological reaction to RA can be seen as an interaction between the disease itself, its treatment, and the patient's perception or appraisal thereof. The response of the patient (i.e., the coping strategies utilized to adjust to the effects of the disease), is mediated by his/her appraisal of the nature, extent, impact, and treatment of the disease, and his/her ability to respond effectively.

The physical impact of RA includes changes in physical appearance, chronic pain and fatigue, and an unpredictable course of joint inflammation and disability. The psychological impact of these symptoms include adverse affective (depression, anxiety, and anger), cognitive (perceived lack of control or helplessness, maladaptive coping strategies such as wish-fulfilling fantasy or catastrophizing), and behavioural changes (e.g., inactivity, reduced social interaction, preoccupation with symptoms). RA and its associated physical disability has a major effect on psychosocial or socioeconomic status. The resultant vocational incapacity, impaired ability to successfully continue the role of homemaker, and reduced frequency of social interaction and communication, constitute an important area of concern.

The evidence from research investigating the impact of RA highlights the importance of determining the effects of the disease on the patient's psychological health status. It is clear from this literature, that cognitive-behavioural interventions may play a central role in the treatment and care of the RA patient. Prior to a detailed analysis of the benefits of such psychological intervention, it is essential that research investigating psychological determinants in RA onset and progression is reviewed.

Any psychological intervention needs to recognize the possible role played by psychological components as co-factors in the aetiology and subsequent pathology of RA. Furthermore, the recent research investigating psychoimmunological relations in RA, provides important information on causal links between adverse psychological experience and the disease.

3.2 Psychological Determinants

3.2.1 An "Arthritic Personality"?

Despite the long term interest in the possible influence of psychological factors in the onset of RA dating from 1909 (Jones, 1909, cited in Anderson et al, 1985), it was only in the mid-1960s that studies began to investigate personality traits as co-factors in the aetiology of RA (Anderson et al., 1985). Hypothesized characteristics of such "arthritic personalities" (Polley, Swenson, & Steinhilber, 1970; Anderson et al, 1985) include: proneness to depression; being dependent, conscientious and perfectionistic; and an inability to express emotions such as feelings of anger.

Research into the personality attributes of RA patients, however, is fraught with methodological and conceptual problems (Moos, 1964, Achterberg-Lawlis, 1982, Anderson et al., 1985)). As early as 1964, Moos (1964) reviewed

this literature, and highlighted some of the problems effecting the empirical validity of such research findings. These include:

(1) Lack of consensus in theoretical orientation and assessment procedures in terms of RA diagnosis and the construct of psychological stress;
(2) Lack of control for subjects' general medical information (e.g., duration and severity of illness, prognosis, degree of functional disability, type and amount of medication used, etc) and sociodemographic (e.g., age, sex, education, SES) characteristics;
(3) Heterogeneity within the RA population;
(4) Excessive attention to negative personality and behavioural factors (i.e., a pathogenic bias);
(5) Lack of appropriate control groups; and
(6) Retrospective research designs relying on self-report procedures/evaluation.

The psychological tests utilized contributed to the inadequacy of such research findings. According to Achterberg-Lawlis (1982), "...many [tests] had questionable psychometric properties, others...have highly subjective interpretations, and most of the standardized tests used were designed to detect pathology, which is typically not present."(p. 985) Another significant problem associated with testing such patient populations is that of "criterion contamination" (Hawley & Wolfe, 1988; Frank et al., 1988) of the test instrument used (i.e., the instrument responding to features associated with the chronic disease rather than psychological status or attributes).

The research on the personality attributes of RA patients will not be reviewed for two main reasons: firstly, it is beyond the scope of this thesis; and secondly, extensive reviews elsewhere (see Moos, 1964; Anderson et al., 1985) have negated the value of such investigation. The problems of this early research have led some reviewers (e.g., Anderson et al., 1985) to conclude that:

> ...there is little or no support for the evidence of an arthritic personality that predates the disease, and in some way leads to disease onset. Negative personality characteristics noted among arthritis patients are more feasibly explained as reactions to this chronic disease rather than as causal factors.
>
> (p.362)

The recent research (see above section 3.1) investigating the mediating role of the individual's appraisal of their disease and the cognitive strategies adopted by that person, is possibly a more fruitful area of enquiry into the determining role of psychological factors.

3.2.2 Stress and RA Onset and Progression

Psychological stress has been identified as having an important role in RA onset and progression. Evidence (Achterberg-Lawlis, 1982; Anderson et al., 1985) suggests that many RA patients experience significant psychological stress prior to symptom manifestation. Such psychological stress being the result of emotionally traumatic life events (Rimon & Laakso, 1985) such as the loss of significant persons by death or separation, marital crises, financial difficulties, and interpersonal conflicts. RA patients also experience a great deal of stress following

disease onset, as a result of both the chronic and acute pain associated with the condition, as well as the distress related to its functional impairment (see above section 3.1).

Whilst early research (working within Holmes & Rahe's Illness Onset model) reported the effects of stressors on the onset of RA (e.g., Heisel, 1972, cited in Anderson et al., 1985), the effect thereof, according to Rimon (1969, cited in Achterberg-Lawlis, 1982), has been noted to be independent of the nature of the specific stressor (e.g., anger, depression, financial strain). This observation was an early step towards recognizing the complexity of the stress-disease relationship, and provides support for the important health implications following elicitation of the "stress response" (Everly, 1988).

Life stress has been associated to disease onset, course and severity in RA sufferers. In a retrospective design, Heisel (1972) administered a modified form of the Social Readjustment Rating Scale to newly diagnosed juvenile RA out-patients and their matched controls. Results indicated that the mean Life Change Unit score of the RA group for the year prior to testing, was twice that of the controls. Key events for the RA group included major life changes such as moving to a new school and parental divorce or marital disruption (Heisel, cited in Anderson et al., 1985). Despite the methodological inadequacies of Heisel's (1972) study (e.g., unknown validity of measure, retrospective design), the results did concur with other research findings (reviewed in Anderson et al., 1985) on the role of major life events as co-factors in disease onset.

In an attempt to avoid such methodological problems, Baker (1982) investigated the causal role of major life events over a year preceding onset (or actual diagnosis according to criteria established by the American Rheumatoid Association) of RA. In a prospective design, patients attending a rheumatology clinic who were believed to be developing early RA, were assessed by means of a psychiatric interview upon entry into the study and one year later. Only those patients with a definite diagnosis of RA completed the second interview. The interview examined the incidence of life events during the previous year for the RA group and their age-matched controls. According to Baker (1982), significantly more RA subjects experienced adversive life events during the year prior to symptom onset, than the controls.

According to Anderson et al. (1985), "...high levels of marital dysfunction have emerged in several studies as significant stressors amongst RA patients." (p. 364) In their review of the literature, Anderson et al. (1985) cite examples of marital dysfunction, including: physical abuse by a spouse, divorce, and decisions to remain in negative marriages. Anderson et al. (1985) however, question the extent to which the samples used in such investigations were representative of the RA population.

In a 15-year follow-up study, Rimon and Laakso (1985) examined the association between life stress and the clinical course of RA over the course of one year. An earlier study (Rimon, 1969, cited in Rimon & Laakso, 1985) had differentiated two groups of rheumatoid arthritis sufferers: (a) those where the disease was less connected with genetic factors than major psychodynamic conflict situations (called the "major conflict group"); and (b) those where hereditary predispositions overwhelmed psychosocial changes (the "non-conflict group"). Results from Rimon's (1969) study indicated that the onset of RA was sudden in the major conflict group, having unequivocal and severe symptoms. The non-conflict group showed an insidious onset of symptoms in the context of a

hereditary predisposition. Over the course of a year, exacerbations of RA in the major conflict group continued to be associated with "emotionally traumatic life events"

The 15-year follow up study (Rimon & Laakso, 1985), re-examined the original patients, relying on an interview collect data on major life events during the interim period, and the patient's reaction to them. Clinical assessment of disease condition, consideration of available medical data, and information from psychiatric clinics was also included in the study (Rimon & Laakso, 985). The researchers found that later exacerbations of the disease of the patients from the major conflict group were significantly related to increases in traumatic life experiences.

There is limited value, however, in merely investigating the association between stressors such as major life events and the incidence of RA. *Firstly*, early research into the role of stressors in RA onset and progression, was based on a simple model of the stress-disease relationship. Despite Rimon's (1969) observation that the effects of stress on RA was independent of the nature of the stressor, such research did not include an analysis of the individual's appraisal thereof, nor the coping strategies adopted in response to it. Everly's (1988) cognitive-behavioural model of the stress-disease relationship highlights the important mediating role of the individual's cognitive and affective response to the stressor. *Secondly*, numerous methodological problems such as controlling for the timing, duration, and intensity of the stressor, and the use of self-report measures in retrospective designs undermine the empirical validity of such research findings. And *thirdly*, apart from the difficulty of controlling for a host of psychological (e.g., coping styles, personality characteristics, social support systems) and physical (e.g., the insidious nature of disease onset, diet, early symptoms, disease condition, etc.) factors, the research does not offer an explanation of causality. A significant correlation between incidence of stressors and RA onset or progression does not imply a causal relation.

As Anderson et al. (1985) point out, very few of the investigations into the effects of stress on RA have attempted to specify the causal pathways between stress and the development of this disorder. This is very significant because stress may (Anderson et al., 1985):

> (a) predispose a person to greater susceptibility to pathogenic challenge; (b) directly initiate the pathophysiological chain of events, or (c) exacerbate the initial pathophysiology.
>
> (p.363)

The recent growth in psychoimmunological research (see Chapter Two) offers particularly exciting possibilities in identifying the above causal pathways. Given the evidence of interaction among the central nervous, endocrine and immune systems, there is the possibility of documenting more precise relations between psychological stress and the immune system responses of RA patients. The few studies that have included immunological measures in their investigation into the relationship between stress and RA are reviewed in the next section.

3.2.3 Psychoimmunology and RA

Initial research available on RA in this area reveals promising prospects for more intensive psychoimmunological investigations with RA patients. In an early study, Hendrie et al. (1971) examined the relationship between

immune status and incidence of life stress for polyarthritis patients. In a retrospective design, Hendrie et al. (1971) administered the Social Readjustment Scale (SRS) to, and estimated immunoglobulin levels of, 74 RA patients with less than a six-month history of objective joint disease, and matched controls. Results were not conclusive, with there being no significant difference in mean SRS scores between the polyarthritic and controls groups. Potentially stressful situations did not occur more often for polyarthritics than the general population (Hendrie et al., 1971). However, the researchers reported a relationship between high life change scores and elevated immunoglobulin (Ig) levels, which concurred with previous research findings (Hendrie et al., 1971). The exact nature of this relationship between stress-related elevated immunoglobulin levels and RA remains unclear (see Barden, Mullinax, & Waller, 1967, cited in Hendrie et al., 1971).

In a landmark study, Zuatra, Okun, Robinson, Lee, Roth & Emmanual (1989) continued this interest in the relation between life stress and immune parameters in RA patients. The relationship between life stress, perceived coping efficacy and lymphocyte alterations were examined among patients with rheumatoid arthritis. The researchers (Zuatra et al., 1989) were interested in whether the effects of life stress on the body's capacity to maintain well-being in chronic disease sufferers, was mediated by individual differences in coping and adaptation. Zautra et al. (1989) hypothesized that immune parameters would be affected by the occurrence of major and daily life events and by psychological distress. Furthermore, Zautra et al. (1989) investigated whether coping efficacy would mediate the relations obtained between life-stress events, psychological distress, and changes in immune parameters.

Thirty-three female RA patients between the ages of 30 and 70 years old were assessed in a prospective design. Life-stress events, psychological distress, perceived coping efficacy, health status, and percentages of circulating lymphocytes were assessed over three sample points. Results from the study indicated two direct paths between life stress processes and immune functioning: (1) major life events were linked to T-helper/T-suppressor cell ratios; and (2) small stressors were related to percentages of T- and B-cells. In addition, psychological distress was inversely related to the proportion of circulating B-cells. Finally, the patients' appraisals of themselves and their perceived coping efficacy ratings, were related to lymphocyte counts.

In their discussion of their results, Zuatra et al. (1989) suggested a causal chain whereby stressful events affect coping efficacy, which influences the level of psychological distress, which in turn affects relative proportions of T- and B-cells (Zautra et al, 1989). According to Zuatra et al. (1989), "...although these pathways have different implications for theory building, all support the conclusion that immune parameters are linked with experiences of life stress for RA patients." (p. 12) An important finding was the relationship between the patients' appraisal of themselves and their ability to handle life-stress events, and lymphocyte counts. Coping efficacy, furthermore, seemed to mediate the relationship between stressful events and psychological adjustment. The finding that coping and distress did not correlate with variations in helper/suppressor T-cell ratio, may suggest (Zuatra et al., 1989) that psychological adjustment to events may be irrelevant to some important indicators of immune functioning.

An important conclusion by Zautra et al. (1989) was that their initial expectation that stressors associated with RA would lead to a vicious circle of illness recurrence was overly simplistic. Hence, "our data underscore the importance of studying all components of the life-stress process: each appears to play a unique role in the immune

system response." (Zautra et al., 1989: 13) Finally, Zautra et al. (1989) suggested that future research should examine the correlation between coping efficacy and immune parameters in RA, including more ratings in prospective designs, in order to clarify the role of individual coping and adaptive capacities in the maintenance of health and well-being.

This emphasis on the importance of observing individual RA patient responses over time concurs with Jasnoski and Schwarz's (1985) argument that it is necessary to consider all changes (or dysregulation) in the human system in terms of their impact on the individual person. By occupying the fulcrum role in the synchronous systems model of health and illness, the individual patient needs to be more closely examined when investigating the complex interaction between psychological, physiological, and immunological variables (see Chapter Four). Zautra et al.'s (1989) study underscores this need.

Other research on the psychoimmunological relations in RA patients, have investigated the psychological, immunological and physical benefits of psychological interventions utilizing cognitive-behavioural techniques (e.g., O'Leary et al., 1988). The value of such research may only be appreciated following a brief delineation of the basic assumptions and strategies employed in cognitive-behavioural interventions. Section 4 of this chapter will discuss the cognitive-behavioural perspective on RA in greater detail, particularly in relation to the strategies adopted in the various pain and stress management programs conducted for RA patients.

3.2.4 Summary

The role of psychological factors as co-determinants in RA onset and progression remains an important area of research. The extent to which psychological factors either predispose individuals to, directly initiate, or exacerbate the course of RA remains unclear. The current interest in psychoimmunology may provide insight into unravelling this mystery.

The problems of early research (methodological and theoretical) into the existence of an "arthritic personality" led reviewers to conclude that the personality characteristics identified (such as "proneness to depression", "being dependent", an "inability to express emotions") were reactions to the disease rather than causal factors. Such research demonstrated the need to assess mediating factors such as the individual's appraisal of their disease and associated stressors, as well as their cognitive and/or behavioural coping strategies utilized.

The role of major life events (i.e., emotionally traumatic life events) in RA onset and progression has been a significant area of research. Marital crises, bereavement, or periods of interpersonal conflict have all been identified as common life events preceding or exacerbating symptoms of RA. An important finding was the extent to which the effect of stress on the RA patient was independent of the particular nature of the stressor involved. Rimon and Laakso (1985) differentiated between two types of RA patients: (a) the "major conflict group" where the disease was less connected with genetic factors than major psychodynamic conflict situations; and (b) the "non-conflict group" where hereditary predispositions overwhelmed psychosocial changes. Such research recognizes the heterogeneity of the RA population, indicating the need to avoid the use of simplistic theoretical models and

research designs (e.g., self-report measures, retrospective designs, poor control of confounding variables) in this area of investigation.

Despite the demonstrated association between life events and RA, such research fails to provide an account of causality in the stress-RA onset and/or progression relation. Recent research relying on the basic assumptions and evidence of psychoimmunology may provide important information regarding the role of psychological factors as co-determinants in RA. Life stress has been associated with elevated immunoglobulin levels in early polyarthritics. Recently, the magnitude of the life event was significantly correlated with the extent of change in immune status in female RA patients. Patient perceptions of coping efficacy were also related to immune parameters, and were identified as an important mediator in the life events--immune status relationship.

Whilst researchers caution against the use of simplistic models to depict the stress-RA disease relation, it is also clear that research in this area needs to consider the individual patient's response to life events and/or other stressors. The complexity of the interrelationship among psychological, immunological, and disease status variables needs to be examined in the context of the individual patient. Such an argument is supportive of the synchronous systems model (see Jasnoski & Schwarz, 1985) presented in Chapter One, which considers all changes (i.e., dysregulation or dysynchrony) in the human system in terms of their effect on the individual person.

4. Psychological Intervention in RA: A Cognitive-Behavioural Perspective

4.1 Introduction

The various strategies adopted by researchers when assisting RA patients to manage stress, pain, and other symptoms of the disease, fall under the broad rubric of a cognitive-behavioural approach to chronic disease. The central role of cognitive and behavioural factors (i.e., cognitions and actions governing a response to the disease) in the course of the disease, thus constitutes an important area of concern for any psychological intervention in RA.

4.1.1 Applications

Cognitive-behavioural approaches have been applied in a variety of settings (Turk et al., 1983). These include:

(1) disease prevention (or health promotion) programs to modify behavioural risk factors such as smoking, obesity, diet, alcohol abuse, and "maladaptive life-styles" (e.g., lack of exercise, poor stress management, etc.);

(2) interventions to alleviate distress related to aversive medical and/or dental procedures and techniques;

(3) to improve stress/anxiety management in patients suffering from stress-related disorders (e.g., tension headaches, ulcers); and

(4) to help patients to adapt to and cope more efficiously with their chronic medical condition by reducing the impact of their symptoms (i.e., pain and discomfort).

It is beyond the scope of this thesis to provide a review of the extensive body of research documenting the effect of cognitive-behavioural procedures in all the above-mentioned settings (see Turk et al., 1983, for a comprehensive account of the efficacy of such interventions). However, the role of psychological techniques as applied in the fourth setting (i.e., with patients suffering from chronic medical conditions) constitutes a central interest of this investigation.

4.1.2 Cognitive-Behavioural Modification

Cognitive-behavioural modification (Turk et al., 1983) has been shown to improve RA patients' experience of pain, reduce the adverse impact of stressors related to life events and disease condition, and assist the patients in developing a more adaptive response to their disease (Bradley et al., 1984; Bradley, Young, Anderson, Turner, Agudelo, McDaniel, Pisko, Semble, & Morgan, 1987; Parker et al., 1988; and O'Leary et al., 1988).

It is important to recognize that "cognitive-behaviour modification" is a term that is applied to a variety of therapeutic techniques or strategies (Turk et al., 1983), each based on slightly different conceptualizations of the underlying theoretical basis for expected change. Such theories are usually behaviouristic in orientation, ranging from conditioning (i.e., changing reinforcement contingencies), to information processing models as well as social learning conceptualizations (Turk et al., 1983).

The therapeutic techniques or strategies thus originate from diverse approaches to cognitive and behavioural change, including: cognitive therapy, rational-emotive therapy, stress inoculation training (Meichenbaum, 1985; see Chapter Four), anxiety management training (Suinn, 1976), coping skills therapies, and training in self-management techniques (e.g., cognitive restructuring, re-appraisal). Cognitive-behavioural programs (Bradley et al., 1984; Everly, 1988; Turk et al., 1983; O'Leary, personal communication, March, 1990) thus include interventions such as:

(a) education on the nature and cause of pain and stress;
(b) biofeedback training;
(c) relaxation training (including Progressive Muscular Relaxation, Autogenic Training, Hypnotic Relaxation, Neuromuscular Relaxation, Visualization);
(d) instruction in coping skills particular to the condition and modelling of appropriate coping behaviour;
(e) individual and/or group discussions and the facilitation of a supportive environment wherein change can occur;
(f) goal setting and related reinforcement contingencies;
(g) cognitive pain management strategies such as dissociation, relabelling, attention refocussing, vivid imagery, and distraction; and,
(h) the provision of homework assignments to test out cognitions and beliefs.

Whilst different aspects of cognitive experience may be emphasized, and different prescriptions, strategies or treatment rationales for intervention are offered to the patient, these approaches do share some common elements (Turk et al., 1983, Bradley et al., 1984). Treatment is an active, time limited, and structured process. The

underlying assumption is that the patient's feelings and actions or behaviour are largely determined by their appraisal of their experience. As such, according to Turk et al. (1983), "...the therapist is concerned with the contribution of cognitions, affect, and behavioural patterns to the maintenance of psychological and physical problems." (p.5) By helping the patient to "identify, reality-test, and correct maladaptive, distorted conceptualizations and dysfunctional beliefs" (Turk et al., 1983:4), he/she is assisted in reducing the negative impact of his/her disease.

A central objective is to "empower" the patient to adopt an active and more adaptive coping response to their condition. Cognitive-behavioural interventions can thus be considered as "self-control" programs, where "...the responsibility for the reduction of [stress], pain and disability most appropriately belongs to the patient." (Bradley et al., 1984: 1354) These interventions explicitly seek to increase the patient's appraisal of his/her self-efficacy, or his/her perceived ability to control pain and disability. According to Turk et al. (1983), "...a common feature of cognitive-behavioural treatments is that the patient is viewed as an important agent in guiding, directing, and controlling his or her own health, disease, and illness." (p.33) Through education and information, instruction in coping skills, and training in the successful application thereof, the cognitive-behavioural perspective seeks to implement the underlying principles of behavioural medicine.

Such a perspective is in accordance with the practical implications of Antonovsky's (1984) "salutogenic" paradigm. Rather than assuming sole responsibility in treating the patient and focussing only on pathology, cognitive-behavioural interventions seek to enable the patient to begin to take on more responsibility for the management of their health. These therapies, according to Turk et al. (1983), have adopted a view of change similar to that Bandura's (1977) "reciprocal determinism":

> Behavioural change is a reflection of the intimate interrelationships among the patient's cognitive structures (schemata, beliefs), cognitive processes (automatic thoughts, internal dialogue, images), interpersonal behaviours, and resulting intrapersonal and interpersonal consequences
>
> (Turk et al., 1983: 5).

Cognitive-behavioural intervention seeks to promote behavioural change by helping patients to become more aware of their beliefs regarding their condition, the images they hold of their disease and its progression, their internal dialogue which influences their perception of themselves and their ability to cope, as well as their interaction with significant others (Turk et al., 1983). By recognizing the connections among cognition, affect and behaviour, the patient is encouraged to minimize the impact of maladaptive behaviour on their health and well being. Such change accords well with Jasnoski and Schwarz's (1985) concept of "wellness", where through a process of optimization, the individual can achieve a state of relative synchrony within his/her system (see Chapter One).

4.2 Cognitive-Behavioural Intervention in RA

Cognitive-behavioural interventions with RA patients focus primarily on chronic pain management. Given that pain (both acute and chronic) is the most important index of health status in the RA patient (see section 3), the

reduction thereof constitutes a central concern for both patient and psychologist. Apart from the disability, possible deformity, and social disruption as a result of RA onset and progression, pain is seen as a significant stressor for the RA patient. The patient's appraisal thereof is influenced by a number of factors; including the intensity and duration of the pain, and the extent to which the pain is controllable (i.e., through medical and/or psychological strategies) or perceived as such.

Given the unpredictable nature of the RA disease process, the waxing and waning periods of joint inflammation and pain, and the sometimes obscure treatment regimens, the RA patient's appraisal of their ability to control their pain and related disability is severely undermined. The periods of exacerbation and remission in pain and disease activity may cause the RA patient to view RA as beyond his/her effective control. With the psychological impact of the disease (see section 3), this perceived lack of control, or learned helplessness, may "...produce increased perceptions of pain and pain behaviour, as well as reduced attempts to develop new coping behaviours or engage in activities of daily living." (Bradley et al., 1984: 1354)

In their review of psychological approaches to the management of arthritis pain, Bandura et al. (1984) argue that:

> It appears reasonable to assume...that [cognitive-behavioural] interventions may be beneficial to RA patients because these interventions may provide them with (a) realistic information concerning their disease; (b) strategies for anticipating and coping with pain and related emotional distress; and (c) increased appraisals of self-efficacy in coping with pain.
>
> (p. 1354)

Everly's (1988) model of the human stress response provides an important conceptual framework within which to investigate the importance of cognitive-behavioural interventions with RA patients. The central role of appraisal in determining an individual's response to a stressor (in this case the pain, disability, and deformity) highlights the possible adverse effect of learned helplessness in the RA patient. Everly's (1988) model concurs with Bandura's self-efficacy theory (cited in O'Leary et al., 1988), which postulates that a person's appraisal of his/her capabilities affects his/her behaviour, cognitions and emotional reactions (O'Leary et al., 1988). Several studies have investigated the benefits of cognitive-behavioural intervention to promote self-regulation and self-management of the pain and stress of RA.

Randich (1982) compared the effects of a stress inoculation training programme (SIT) developed by Turk et al. (1983) to that of a social support intervention and a no-treatment control, condition. Randich (1982) predicted that the cognitive-behavioural programme would reduce functional disability and pain in a sample of RA patients. The patients were assessed pre- and post-treatment, and at an 8-week follow-up. The SIT group showed significant changes in patients' reports of functional, social, leisure and vocational activities compared to that of the other two experimental conditions. No significant changes were found in patients' self-reports of pain intensity. Despite the adequate methodology employed by Randich (1982) in terms of experimental design, the use of solely self-report measures to assess treatment outcome is an unfortunate limitation. The accuracy of patients' self-reports of outcome is questionable.

In a more comprehensive and longitudinal study, Bradley et al. (1984) investigated the efficacy of a biofeedback assisted, cognitive-behavioural group therapy programme for RA patients. The three-group design study included a structured social support programme and a no-treatment control group. The cognitive-behavioural programme consisted of 15 sessions, 5 of which were allocated to individual instruction in thermal biofeedback training to promote increased peripheral skin temperature at the most affected joints. The other 10 group sessions were structured similarly to that of Turk et al.'s (1983) stress inoculation training model. The sessions encompassed four phases (Bradley et al., 1984): an educational component, a skills acquisition phase (i.e., instruction in palliative coping strategies and setting and achieving behavioural goals), self-instructional training (i.e., practice in the generation and use of self-rewards), and an application phase. The social support group attended a two phase programme over 15 sessions consisting of an educational and a support component. According to Bradley et al. (1984), "the factor that differentiates the [social support] from the [cognitive-behavioural] subjects is that the former receive encouragement to develop their own coping strategies while the latter receive direct instruction in various coping strategies." (p. 1356)

Assessment was at pre- and post-treatment, and at a 6 and 12 month follow-up utilizing a number of dependent measures for physiological (i.e., peripheral skin temperature and disease status), demographic, behavioural (i.e., a timed 50-ft walk and video-recorded pain behaviour), pain (i.e., intensity, quality) and psychological (i.e., anxiety, depression, locus of control, and arthritis helplessness) variables. Bradley et al. (1984) reported preliminary results for an initial sample of 38 patients. It was found that only the cognitive-behavioural group produced consistent decreases in pain behaviour, pain intensity, morning stiffness and functional disability. An important finding was an associated increase in perceived self-efficacy to manage their RA symptoms as well as an increase in peripheral skin temperature. Both the cognitive-behavioural and support groups showed a decrease in anxiety and depression scores on the respective measures.

In their discussion of their findings, however, Bradley et al. (1984) cautioned that the small sample size limited the reliability of the observed change, and suggested that future studies used multiple outcome measures as well as analyses of individual patient's covert experiences to determine the mediating effect of cognitions on disease and coping abilities.

In a more recent paper, Bradley et al. (1987) reported the final results of their study having a sample size of 53 patients. A more comprehensive analysis of the data supported the earlier preliminary findings. The cognitive-behavioural programme produced significant reductions in pain behaviour and RA disease status scores at post-treatment relative to the other two groups. Important disease status findings were reductions in scores on the Articular Index (which measures disease activity and pain in the joints) and the disease activity ratings in the cognitive-behavioural group. Bradley et al. (1987) reported that these treatment effects were independent of systemic differences among the three groups in age, duration of disease, socioeconomic status, functional class, medication changes, or appraisal of treatment value.

In an evaluation of the programme components by the patients, relaxation and imagery training was considered the most popular and effective strategy. Bradley et al. (1987) argued that such findings suggest that "...relaxation training may have been the treatment component primarily responsible for the reductions in pain and disease

activity." (p.1112) Of the changes observed in the cognitive-behavioural group, only trait anxiety scores showed significant reductions at 6-month follow-up. Bradley et al. (1987) pointed out that a possible explanation for this group's failure to maintain their treatment gains, was the great deal of variation in their application of their coping skills post-treatment. This study highlights the importance of examining individual patient's response to the intervention to allow a more detailed explanation of the effects of the treatment programme.

In a similar but more recent study, Parker et al. (1988a) also investigated the effectiveness of a cognitive-behavioural pain management programme for reducing RA pain in a prospective (12 month), randomized three-groups design with comprehensive dependent measures. 80 RA patients were randomly assigned to a cognitive-behavioural, attention-placebo, or a control group. Baseline assessment included psychological (coping strategies and perceptions of coping effectiveness, depression, daily hassels, and psychological symptoms), pain (quality and intensity), functional (arthritis impact) and disease status (Westergren ethrocyte sedimentation rate, grip strength, walking speed, morning stiffness, joint counts) measures. Measures were repeated at 6- and 12-months for the whole sample.

According to Parker et al. (1988a), their cognitive-behavioural pain management programme was based on the conceptual approach of Karol, Doerfler, Parker, and Armentrout (cited in Parker at al., 1988a), "...with minor changes that were based on the findings of Turk et al. (1983)." (p. 595) Following a week long in-patient programme of education on the nature of pain and the treatment of RA, and instruction in coping strategies, the cognitive-behavioural group met monthly in an extensive support group programme to ensure continued application of the cognitive-behavioural principles to everyday life. The attention control group attended a week long in-patient education programme, as well as monthly support group meetings for continued education on aspects of their disease. The difference between these two groups was based solely on the instruction in coping strategies for the former.

Parker et al.'s (1988a) findings supported those of Bradley et al. (1984, 1987). The cognitive-behavioural group showed a significant improvement in their ability to cope with the pain of RA. Parker et al.'s (1988a) use of the Coping Strategies Questionnaire (Rosenstiel & Keefe, cited in Parker et al., 1988a) provided valuable information on the nature of this improvement in coping strategies for pain. Thus, this group were more able to avoid catastrophizing about their condition than the other two groups, and showed enhanced perceptions of self-efficacy to control pain. A significant finding was the maintenance of such treatment effects at 12-month follow-up. According to Parker et al. (1988a), "[the] improved coping skills and enhanced self-efficacy were quite impressive in light of the disease progression...and the limited socioeconomic resources of this sample." (p.599) Parker et al. (1988a) also reported a greater improvement in those patients who demonstrated high adherence to treatment strategies during the 12-month period. The efficacy of the intervention was thus a function of the patient's compliance with the programme and their utilization of the coping strategies taught.

Unlike Bradley et al. (1984, 1987), Parker et al. (1988a) did not find significant changes along disease, functional, or psychological status variables, despite the improved coping and enhanced self-efficacy. Only the small high adherence subgroup of the cognitive-behavioural group demonstrated improvement along these variables. The researchers concluded that whilst the benefits of the cognitive-behavioural intervention were highly specific to the

coping process, the gains that the group made, in spite of progressive joint involvement, are "...extremely important from a clinical standpoint." (Parker et al., 1988a: 600)

A more complex study by O'Leary et al. (1988) provides further support for the inclusion of cognitive-behavioural pain management programs in the multidisciplinary care of patients with RA. O'Leary et al (1988) highlight the central role of appraisal or self-efficacy beliefs in effective management of RA. O'Leary et al. (1988) investigated the extent to which stronger self-efficacy beliefs may affect both the behaviour and emotional responses to the effects of RA. O'Leary et al.'s (1988) study is significant in that it sought to examine the interrelationships between a multitude of psychological, physical, and immunological variables. Unlike Zautra et al.'s (1989) psychoimmunological study which merely correlated psychological and immunological variables in RA patients, O'Leary et al. (1988) sought to determine the psychoimmunological benefit following the manipulation a psychological variable whilst controlling for a host of extraneous variables.

30 RA patients participated in a prospective study to test the hypothesis that enhanced perceptions of self-efficacy to manage pain and disease effects would result in an improvement in pain, disease, and immunological status. Utilizing a matched-pairs two group design, 15 patients were assigned to a cognitive-behavioural treatment condition, which provided instruction in skills relevant to self-management of RA, and 15 to a control group. Assessment included arthritis outcome measures (i.e., pain analogue scales; Health Assessment Questionnaire Disability Scale; and clinical assessment of disease activity), a self-efficacy questionnaire (includes three efficacy components: Arthritis Self-Efficacy Scale, self-efficacy to manage pain; and self-efficacy to function), psychological measures (Zung Depression Scale, UCLA Loneliness Scale, Perceived Stress Scale), and immunological assays (Westergren erythrocyte sedimentation rate; T-lymphocyte subsets; and lymphocyte proliferation rates).

The cognitive-behavioural treatment group met once a week for two hour sessions for five weeks. Treatment included education on the nature and impact of RA, instruction and training in cognitive and behavioural pain-management strategies, and goal setting. The findings of the study supported those of Bradley et al. (1984, 1987) that cognitive-behavioural treatment of RA improves the psychological health status of such patients. Results indicated that the intervention enhanced perceptions of self-efficacy, reduced pain and joint inflammation, and improved psychosocial functioning in the treatment group (O'Leary et al., 1988). Despite these improvements however, no significant change was demonstrated in ethrocyte sedimentation rate and serum measures of immune function (i.e., percentage circulating T-cells and lymphocyte proliferation rates). O'Leary et al. (1988; 1990: personal communication) explained the failure of the treatment to have the predicted effect on immunological functioning in terms of the heterogeneity and variance in RA immunology.

At baseline assessment, perceived self-efficacy was positively correlated with disease status and levels of suppressor T-cells, and negatively related to helper T-cells. This finding is significant in the sense that RA is associated with increased lymphocyte proliferation and activation between and around the synovium membrane of the joints (Bennet, 1985). The helper T-cells "turn on" the immune response, and it is therefore possible that perceptions of self-efficacy in coping with the effects of the disease may in some way mediate the relationship between psychological distress and altered immune function demonstrated in the psychoimmunological literature. O'Leary et al.'s (1988) finding that perceived coping-efficacy was accompanied by reductions in negative affect in their

sample would support such an interpretation. The intervention, however, failed to have any effect on immune function variables.

O'Leary et al. (1988; and O'Leary, personal communication, June, 1990) are currently attempting to determine which components of the cognitive-behavioural intervention are significant contributors to the observed changes in the research literature. Finally, the work of O'Leary and her colleagues (personal communication, June, 1990) in consolidating and developing non-medicinal pain management techniques, and assessing their experiential and immunological benefits for RA patients, constitutes a solid foundation for continuing work in this area.

4.3 Summary

The variety of strategies or techniques used in interventions with RA patients to assist them in the management of stress, pain, and disease symptoms fall under the broad rubric of "cognitive-behavioural modification". A cognitive-behavioural perspective highlights the central role of cognitive and behavioural factors (i.e., cognitions and actions) in the course of RA. Of central interest, thus, are those factors governing the person's response to their disease.

This section has sought to demonstrate the extent to which cognitive factors are extensively involved in the incidence and course of RA. By governing the person's response to their disease, such factors effect ways in which patients perceive their disease, the decisions they make regarding the effectiveness of particular responses (i.e., coping behaviour) to the disease, and their utilization of the health care system and compliance to treatment regimens. Recent research has indicated the role such cognitive factors, particularly perceptions of self-efficacy, may play in directly or indirectly contributing to the course of RA.

Perceived self-efficacy influences the individual's appraisal of the related stress, pain, and disease effects of RA. The (a) unpredictable onset, course, and nature of RA; (b) waxing and waning periods of joint inflammation, pain and disability; and (c) sometimes obscure and long-term treatment regimens; have a significant psychological impact on the RA patient (see section 3). These characteristics of RA, furthermore, have an adverse effect on the patients' appraisal of their ability to control and cope with the disease. This learned helplessness, or poor perceived self-efficacy, has been shown to mediate the extent to which disease characteristics determine the psychological and physical health status of the patient.

Therapeutic strategies employed in cognitive-behavioural interventions are directed at helping the RA patient to become more aware of the relationship between their appraisal of and concomitant response to the disease. Through education and instruction, the patient is taught to recognize the role played by them in determining the impact of disease effects on their psychological and physical well-being. By assisting the patient to take on more responsibility in the management of their disease through self-control techniques, the patient is empowered to adopt an active and more adaptive coping response to their condition. Cognitive-behavioural interventions in RA thus seek to increase the patient's appraisal of his/her self-efficacy in controlling and coping with the disease.

The strategies employed include: (a) education; (b) instruction in palliative (i.e., relaxation training techniques) and instrumental (i.e., problem solving or management strategies) coping strategies, and cognitive pain management

strategies; (c) goal setting and using reinforcement contingencies; (d) biofeedback training; and (e) facilitation in group discussion and the creation of a supportive environment wherein change can occur. A common feature of the various cognitive-behavioural interventions is that treatment is an active, time-limited, and structured process.

The studies reviewed in this section provide evidence of the psychological (i.e., cognitive and affective) and physical (i.e., functional status, disease activity, and immune function) benefits of cognitive-behavioural intervention with RA patients. The studies utilized a multitude of dependent measures in longitudinal two- or three-groups designs, assessing patients pre- and post-treatment, and at follow-up points ranging from a few weeks to a full year. Significant features of such research being the use of adequate experimental designs, the inclusion of control groups, and the control of patient and disease characteristics. The reliance on self-report measures of some psychological and pain variables remains a problem in this area of research.

Randich (1982) demonstrated reductions in patient's self-reports of the functional, social, leisure, and work impact of their disease. Bradley et al. (1984) and Bradley et al. (1987) found significant decreases in patients' scores on measures of anxiety, depression, pain behaviour and intensity, morning stiffness, functional disability, as well as associated increases in their perceptions of self-efficacy in the management of RA symptoms. A significant finding of Bradley et al.'s (1987) research was that such changes were independent of patient characteristics, including sociodemographic, medication, or disease variables. Patient's also rated relaxation training as the most effective strategy in minimizing the adverse effects of RA.

Parker et al. (1988a) demonstrated enhanced perceptions in self-efficacy associated with a greater ability to avoid negative appraisals (e.g., catastrophizing) in patients in their study. They did not, however, find significant changes along disease, functional, or psychological status variables. This discrepancy in findings (in terms of other research) was interpreted on the basis of the sample characteristics and disease progression over the course of the study.

Finally, O'Leary et al.'s (1988) study supported the findings of Randich (1982) and Bradley et al. (1984; 1987) and confirmed earlier findings (see Zautra et al., 1987, reported above) regarding the association between perceptions of self-efficacy and immunological function. O'Leary et al. (1988) found reductions in pain and joint inflammation, and enhanced perceptions of self-efficacy and psychosocial functioning. Whilst the cognitive-behavioural intervention did not significantly effect immunological functioning, perceived self-efficacy was positively correlated with disease status and suppressor T-cells, and negatively related to helper T-cells at baseline analyses. The heterogeneity and variance in RA immunology was considered a confounding variable.

The findings of such research indicate the need for further research into the complex relations among psychological factors, disease activity, pain and immunological functioning in RA. In Chapter Four, a model of the relations among psychological adjustment (perceived coping efficacy, mood and locus of control), pain (quality and intensity), disease (joint inflammation, disease activity, and functional status), and immunological function (lymphocyte proliferation responsivity) in RA patients is proposed.

The thesis presented in the next chapter is thus based on the theoretical models delineated, and the evidence of the research reviewed in these first three chapters.

CHAPTER FOUR

PSYCHOLOGICAL ADJUSTMENT, PAIN, DISEASE STATUS AND IMMUNE FUNCTION IN RHEUMATOID ARTHRITIS

Overview

The thesis presented in this chapter is that: (a) there is a relationship between psychological adjustment and pain, disease status and immune function; and, that (b) cognitive-behavioural intervention would have significant benefit for RA patients. The introductory section provides an overview of the logical steps followed in the first three chapters, to the point wherein it is possible to provide a rationale for this particular study.

It is argued that it is necessary to provide a model of the complex interrelationship among these variables in RA, delineating the central mediating role of psychological adjustment in the disease process. The bulk of this chapter is devoted to explaining this model, the key variables to be investigated, and how these variables are operationalized in this study. A number of predictions are made regarding the nature of the interrelations among these variables. Following a brief description of the focus of the study, the key research questions investigated and the related predictions and hypotheses to be tested are outlined.

1. Introduction

In Chapter One, the background to the involvement of behavioural medicine in the investigation and treatment of chronic disease was delineated. The historical changes in the epidemiology of disease and models of causality, the import of systems theory, and changing trends in psychology, allowed behavioural medicine to challenge our concepts of disease and the treatment thereof. Behavioural medicine thus also challenges the demarcation of boundaries of professional responsibility. The new conceptualization of disease as a multidimensional experience and the result of the interplay among multiple factors, requires an expansion of professional boundaries in the care and treatment thereof. The current popularity of a multidisciplinary team approach to chronic disease in particular, is evidence of the shift in focus in health and disease.

The synchronous systems model was introduced, placing the individual at the center of the interplay among these complex factors or systems (i.e., environmental, physiological or biological, social, and psychological). Changes at any level of the human system are ultimately interpreted in terms of their impact on the individual person. The model, however, does not consider the person to be a passive object of environmental, social, interpersonal, personal and/or biological forces. Rather than merely adapting to the dynamic forces of these systems, the individual "optimizes" his/her environment to a greater or lesser degree. The influence of psychological and behavioural factors in governing this process of optimization becomes an important and legitimate area of research and practice.

To provide evidence of the role of psychological processes and experience in the onset and course of disease, Chapter Two explored the nature of the relationship between stress and disease. The cognitive-behavioural paradigm was identified as providing the most coherent conceptualization of the complex processes involved in the stress-disease relationship. It was argued that Everly's (1988) model of the human stress response, which utilizes a systems theory perspective, is able to delineate the mechanisms linking psychological experience and altered physical health status.

Research findings from the two main fields of investigation, psychosomatic and psychoimmunological research, demonstrated the adverse effect various psychological stressors have on health status and immune function. Such stressors include emotionally traumatic life events (bereavement, marital disruption, unemployment, academic examinations) and adverse psychological experience (depression, distress, anxiety, perceived loss of control, caring for chronically ill, and even exposure to environmental threat). Initial evidence of the clinical significance of stress-related immune dysregulation indicates the need for psychological intervention with "high risk" patient populations (i.e., diseases associated with immunological dysfunction such as neoplastic and autoimmune disorders). The chapter concluded with an examination of the value of cognitive-behavioural intervention in ameliorating the noxious effects of stress on individuals and/or altering immune function.

In Chapter Three, RA was identified as an important and appropriate disease condition to explore the value of psychological research and treatment interventions. It was shown the extent to which RA has a significant psychological impact on the patient. It was also shown how psychological factors, particularly emotionally traumatic life events and maladaptive coping strategies in response to such events, play a contributing role to the onset and course of RA. A key underlying assumption held by the cognitive-behavioural approach to RA, is the central role played by cognitive factors (i.e., appraisal) governing the patient's response to his/her disease. As a result of the psychological impact of RA, the RA patient's appraisal of their efficacy to cope with their disease is severely undermined.

Perceptions of poor self-efficacy seem to mediate the adverse effect of the stressors associated with RA on the patient's psychological and physical health status. The cognitive-behavioural interventions examined in the previous chapter utilized strategies aimed at helping the RA patient to achieve greater self-control over their disease. By taking on more responsibility for its management, the RA patient becomes more actively involved in the treatment process and is encouraged to adopt a more adaptive response to the disease.

In this chapter, a thesis is presented which links psychological adjustment, pain, disease activity, functional status, and immune function in RA. These variables are examined in the context of a cognitive-behavioural intervention based on Turk et al.'s (1983) and Meichenbaum's (1985) stress inoculation training treatment rationale. Cognitive pain management strategies are included in a "stress inoculation and pain management training programme" (SIPMT) designed to: (a) improve the psychological adjustment and immune function, and (b) decrease the pain, functional disability and disease activity, of RA patients.

2. Rationale of Thesis

There is a need for further research into the role of psychological factors, particularly the role of appraisal (cognitive and affective), in RA. This need is based on the evidence: (a) linking psychological factors to the onset and course of RA, and the severity of the pain experience; (b) the relationship between adverse psychological experience and immune function; and (c) the benefits of cognitive-behavioural intervention with such patients.

The development of the research in this area indicates an improvement both in research methodology and the components of the cognitive-behavioural intervention programs. Furthermore, such research shows an increasing complexity in theoretical foundations, demonstrating the extent to which psychological research in this area is attempting to delineate the complexity of the relationships involved.

It is essential for continued investigation into the relationships among particular psychological factors and aspects of the immunological and disease processes underlying RA. Such investigation needs to incorporate the recommendations of earlier research into more adequate and empirical research designs. The variables examined need to be effectively operationalized in the context of a clear theoretical orientation, and delineation, of the nature of the relationships among these variables. Models depicting the interaction among psychological, physiological and immunological processes thus have important heuristic value for research in this area.

This study, therefore, must being seen in the context of the evidence reviewed in the previous two chapters, as well as the needs delineated above. By constructing a model to depict the interrelationships among the psychological, pain, disease activity and immunological variables, and testing that model through cognitive-behavioural intervention, this thesis aims to contribute to this area of investigation.

3. Constructing a Model

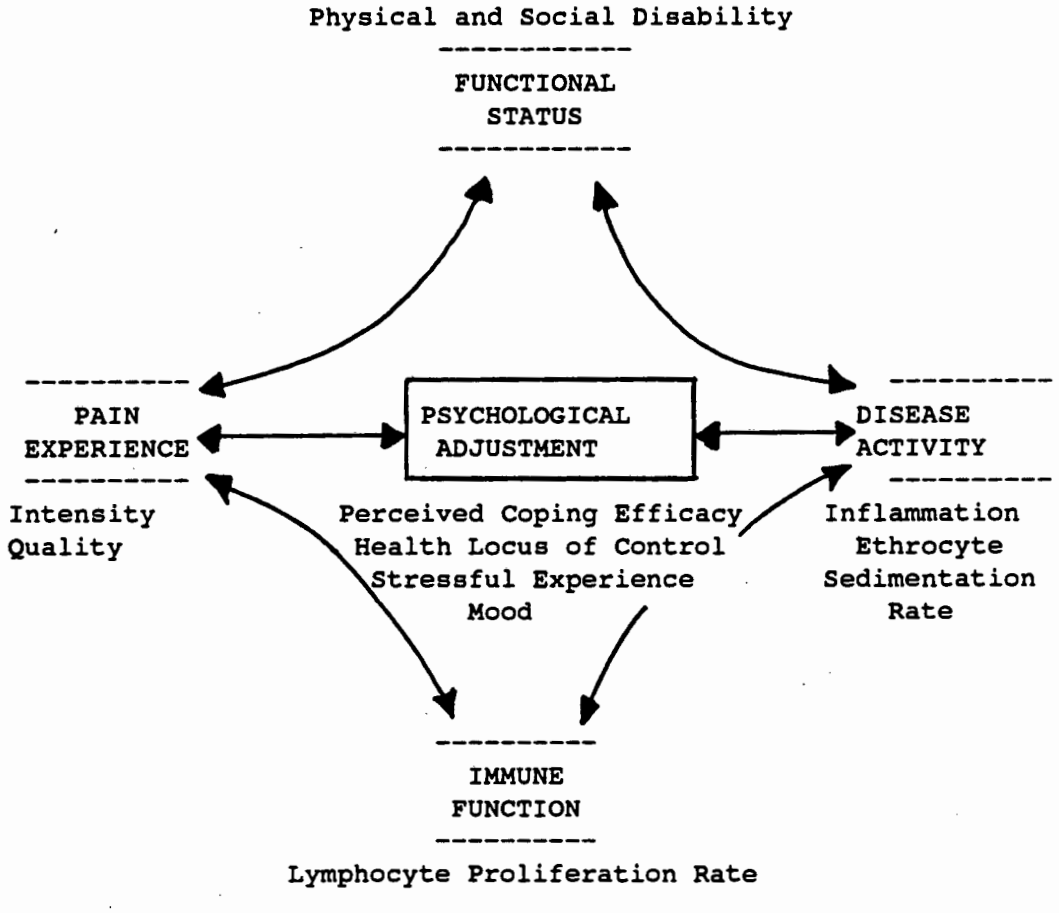
The synchronous systems model (Jasnoski & Schwarz, 1985) and Everly's (1988) model of the human stress response, both attempt to demonstrate the complex interaction among psychological and physical variables utilizing a systems theory perspective. The former offers a compelling account of the fulcrum role of the individual person within a dynamic, complex, synergistic system of synchronous interrelation (the homeostatic principle) among environmental, personal, and biological forces. The latter offers a more detailed account of the causal mechanisms linking psychological experience and altered health status. The value of these models lie in their ability to "demystify" the area of investigation, providing some theoretical orientation from which research in this area may be evaluated and contextualized.

It is necessary, however, to construct a simple model of the interaction among such "systems" in the context of research in RA. Clearly, such a model should also include variables which research has already identified as playing an important role in RA onset and progression. Research utilizing this model would then be limited only in terms of the feasibility of any particular study (i.e., constraints include available resources, expertise, appropriate patient populations, and methodological limitations).

It is proposed that psychological adjustment constitutes an important mediating variable in the relationship between disease activity and pain experience. Psychological adjustment also mediates the relationship between pain and functional status, and pain and immune function. Similarly, psychological adjustment mediates the relationship between disease activity and functional status, and disease activity and immune function.

Given the cognitive-behavioural paradigm within which this study is located, psychological adjustment thus governs the RA patient's response to their disease, thereby playing a central role in the course thereof. Psychological adjustment itself, however, is also affected by pain, disease activity and functional status. If the psychological impact of RA on the patient is ignored, or the patient is unable to cope with his/her disease, a vicious cycle could develop wherein decreased psychological adjustment would further aggravate pain, disease activity, functional status, and possibly immune function. Figure 0.2 illustrates the interplay among these key variables, demonstrating the important role played by psychological adjustment in the disease process.

Figure 0.2. A Model of the Interplay Among Psychological, Physical, and Immune Variables



This study proposes to test the model in two ways: (1) by examining the associations among these variables in a sample of RA patients; and (2) by manipulating the psychological adjustment variable through cognitive-

behavioural intervention with these patients. At this point, it is necessary to examine these variables more closely, and consider their operationalizations.

3.1 Psychological Adjustment

Psychological adjustment, it is argued, entails the interplay between (a) an individual's exposure to stressors, (b) their appraisal both of their ability to cope with these stressors (i.e., self-appraisal) and the stressors themselves, (c) their response (i.e., cognitions, affect, and actions) to the stressor, and (d) level of emotional distress.

In this study, four particular psychological factors are identified as playing an important role in determining psychological adjustment: perceived coping efficacy, health locus of control, mood state, and extent of stressful life experience. Optimal psychological adjustment is thus operationalized as elevated ratings of perceived coping efficacy (as assessed through a semi-structured interview), a more internalized locus of control (as indicated on the Multidimensional Health Locus of Control scale), little mood disturbance (as assessed by the Profile of Mood States), and reduced frequency of stressful life events (as indicated on the Stress Evaluation Inventory).

3.1.1 Perceived Coping Efficacy

This construct, located between coping efforts and general adjustment (Zautra et al., 1989), is influenced by central features thought to regulate adaptation to stress. Coping is understood as a transactional component of the life-stress process (Lazarus et al., 1984). The nature of the events experienced, the person's appraisal of both the event and their ability to respond, as well as his/her capacity to respond effectively, are thought to determine coping strategies and their ultimate success in aiding adjustment. Coping efficacy thus defines a judgement process "interposed between coping efforts and adjustment" (Zuatra et al., 1989).

Perceived coping efficacy is thus operationalized as the extent to which the person appraises the efficacy of his/her coping response to a stressful event (including an acute, painful disease episode). This efficacy is in terms of: the individual's satisfaction with his/her response and the response of others; and his/her expectations of future coping efficacy in similar events. According to Zautra et al. (1989), this construct is closely linked to actual current events, and the proximal outcomes of coping efforts in relation to these events. Research has demonstrated the central role that patients' perceptions of self-efficacy in coping with stressors play in mediating the impact of RA and pain. It thus provides some subjective evaluation of the patient's appraisal of and response to stressful events .

3.1.2 Health Locus of Control

Health locus of control, as with the coping construct, is thought to play a central role in determining the extent to which the patient plays an active role in maintaining and promoting his/her health (Wallston, Wallston, & DeVellis, 1978; Wallston, Smith, King, Forsbery, Wallston, & Nagy, 1983). This construct is operationalized as the degree to which an individual believes his/her health is determined by "internal", "chance" factors, as well as the role of "powerful others" in contributing to their well-being.

The patient's appraisal of the extent to which his/her health is dependent on "internal" (i.e., his/her actions) factors, determines the extent to which the patient takes on more responsibility for his/her health. A concomitant belief that health is also dependent on "powerful others" (i.e., significant persons in the patient's life, including health care professionals) determines the extent to which the RA patient is compliant with the treatment strategies. If the patient believes his/her health is a function of "chance" events (i.e., events beyond personal control), his/her resultant actions or response to the disease would be undermined. Research has demonstrated the extent to which an "externalized locus of control" is related to depression (Burger, 1984), is a mediator between stressful life events and depression (Ganellen & Blaney, 1984), and increases pain experience (Arntz et al., 1989). It is argued that there is a close relationship between a patient's perception of their lack of control over their health, and their poor perceptions of coping efficacy (i.e., the "learned helplessness" syndrome).

Perceived coping efficacy and health locus of control are two constructs which provide insight into factors governing a patient's cognitive response to their disease.

3.1.3 Mood State

Mood is operationalized as the extent to which the individual experiences feelings relating to tension, depression, anger, fatigue, confusion, and vigor (McNair, Lorr & Droppleman, 1976). The more tense, depressed, angry, fatigued and confused he/she feels, the greater his/her mood disturbance. Vigor, on the otherhand, "lifts" or buffers this mood disturbance.

Mood states, it is argued, are closely linked to the patient's response to stressors in three ways. Firstly, the level of mood disturbance provides an indication of the level of distress experienced by the RA patient. Secondly, the level of mood disturbance provides some indication of the extent to which coping responses are effective (i.e., adaptive) in ameliorating the adverse effects of stressors and pain. Thus mood is more closely related to the patient's reaction to their disease, than to underlying personality characteristics.

Finally, the level of mood disturbance influences the RA patient's response to their disease. A high level of mood disturbance would adversely affect perceptions of coping efficacy and locus of control, reinforcing the syndrome of learned helplessness. A stable mood profile, as indicated on the Profile of Mood States (McNair et al., 1976) would thus contribute to the RA patient's psychological adjustment, thereby playing an important role in the disease process.

3.1.4 Stressful Life Experience

Stressful life experience is operationalized as the individual's perception of the frequency of stressful events or experience in the three important areas of daily life: career, family, and personal-social interaction (Madsen, 1981). The frequency of such experience plays an important role in contributing to the RA patient's general psychological well-being. Over and above the impact of RA, such patients still have to cope with daily life hassles which may place added demand on their coping resources. The lower the frequency of daily hassles, the less the coping

resources of the RA patient are taxed, and the less his/her psychological adjustment is threatened. This would also indicate, to some extent, whether or not these areas of daily existence are supportive environments.

3.2 Pain

Pain is considered a complex experience, involving the interplay among numerous factors (Melzack & Wall, 1965, 1988; and see Karoly & Jenson's, 1987, "multiple contexts of pain"). These include:

(1) physical (i.e., degree of tissue damage, inflammation, and disability);
(2) psychological (i.e., cognitive and affective factors governing appraisal in terms of the cause, meaning, quality, intensity, impact, and self-efficacy to manage the pain); and
(3) social (i.e., the response and support of significant others, including health care workers) factors.

Pain is thus only partly dependent on the extent of disease activity, with psychological adjustment playing an important mediating role in determining the overall impact of pain related to RA and the experience thereof.

Pain is operationalized as both the intensity and the quality of pain sensations experienced. The intensity is in terms of the individual's rating of the severity of his/her pain. The quality of the pain experience entails the extent to which the individual rates his/her pain along three dimensions: sensory, affective, and evaluative (Melzack, 1975). The RA patient's affective and evaluative response to their pain (i.e., the meaning of their pain sensations) is central to understanding the psychological and functional impact thereof. The sensory dimension provides an indication of the nature of the pain experienced by the individual patient. The lower the ratings of severity of pain, and the less the individual rates their pain along the three dimensions, the better their pain experience.

3.3 Disease Activity

Disease activity in RA includes both the clinical (i.e., the degree and extent of joint inflammation and destruction, frequency of acute disease episodes, functional disability, and the presence of extra-articular characteristics such as nodules, etc.) and haematological (i.e., ethrocyte sedimentation rate levels of key elements) aspects of the disease. In this study, disease activity is operationalized as the clinical evaluation of the extent and associated pain of actual joint inflammation (Articular Index), and the haematological estimation of ethrocyte sedimentation rate (ESR).

3.4 Functional Status

This is consists of a combination of the physical and psychosocial disability caused by RA (Meenan, Gertman & Mason, 1980). Functional status is operationalized as the individual's performance in terms of these two areas of disability. Physical factors include ratings of physical and household activities, dexterity, and activities of daily living. An important psychosocial factor is the extent to which RA effects social activities (i.e., the extent to which the disease effects prevent the patient from maintaining adequate social interaction).

3.5 Immune Function

Immunological function in RA is operationalized as the rate of *in vitro* lymphocyte proliferation following exposure to mitogens. Ideally, immunological function should include an analysis of the levels of circulating T- and B-lymphocytes, and the ratio between helper and suppressor T-cells. It is however, not possible to include such analyses in the present study for financial and practical reasons.

As RA is considered an autoimmune disorder characterized by an excessive inflammatory response in the joints, it is important to gauge the extent to which the RA patient's immune function is dysregulated (e.g., such as poor lymphocyte proliferation response rate) through psychosocial factors. It is argued that whilst immune function (operationalized as rate of lymphocyte proliferation response) is related to disease activity and pain, psychological adjustment plays an important role in mediating the possible dysregulatory influence of adverse psychological experience (as a result of disease effects).

4. Focus and Predictions of this Study

The focus of this study is thus on examining the extent to which the above model is a valid delineation of the role of psychological factors (particularly the patient's cognitive, affective and behavioural response to their disease) in RA. The cognitive-behavioural intervention programme (see Chapter Five) is proposed as a further means of testing both the model, as well as the value of psychological intervention in RA.

The study therefore addresses the:

(a) The chronic and acute pain experience of the RA patient, and the psychosocial impact of the disease;

(b) The patient's perceptions of coping efficacy, their belief in their ability to control their health, and their mood state in relation to important disease, pain, and immunological variables; and

(c) The benefit of a cognitive-behavioural intervention to improve the patient's ability to manage the pain and other disease effects along psychological, physical and immunological variables.

Based on the evidence in the research literature reviewed in the preceeding chapters, and the model delineated above, a number of predictions can be made:

(1) RA would have a significant impact on the psychological well being of such patients.

(2) Optimal psychological adjustment (i.e., good perceptions of coping efficacy, little mood disturbance, an internalized health locus of control, and little stressful experience) would be related to: low pain intensity and quality ratings; little disease activity; good functional status; and optimal immune function. Similarly, poor psychological adjustment would be associated with poor performance along these variables.

(3) Pain experience and functional status would be more a function of psychological adjustment than disease activity (i.e., psychological adjustment would mediate the effects of disease activity on pain experience and functional status).

(4) Change in psychological adjustment would be related to change along pain and disease activity variables, and functional status. Such change being dependent on the predicted (i.e., first prediction) nature of the interrelationships among these variables.

5. Research Questions and Hypotheses

A number of important questions are posed in this thesis. Firstly, do cognitive processes play a central role in mediating the dysregulatory effects of stress (associated with the disease) on psychological and physical health status and immune function in RA? Secondly, would cognitive-behavioural intervention, as an adjunct therapy, provide any benefit for RA patients in terms of psychological and physical well-being? Finally, on the basis of the first two questions, would change in psychological adjustment be associated with significant change in pain, disease status and immune function in RA?

Two hypotheses are tested in this study:

(1) A significant relationship exists between psychological adjustment and: pain, functional status, disease activity, and immune function in RA.

(2) Cognitive-behavioural intervention will produce significant change in psychological adjustment, pain, functional status, disease activity and immune function in RA patients.

The method utilized to test these hypotheses is outlined in the following chapter.

CHAPTER FIVE

METHOD

1. Design

To test the two hypotheses, a matched-random assigned two-groups design, with pre-, mid- and post- intervention assessment was set up. Pre-intervention relationships between key variables, and individual and inter- and intra-group changes over time, were analyzed.

2. Subjects

Twenty-seven patients who attended the rheumatoid arthritis out-patient clinics of Princes Alice Orthopedic Hospital and Groote Schuur Hospital in Cape Town, and who fulfilled the American Rheumatoid Association (ARA) criteria for a diagnosis of definite or classic rheumatoid arthritis (RA) were asked to participate as subjects. These patients were referred to the researcher by senior consultants at the two clinics according to pre-established inclusion criteria. These criteria included: (1) diagnosis of RA; (2) seropositive to the Rheumatoid Factor; (3) female; (4) active phase of the disease; (5) great deal of reported pain; (6) ages 35-60; (7) functional status of II or III (ARA); and (8) ability to participate in the study.

Twenty-four patients provided informed consent (Appendix C) and expressed interest in participating in the study. Patients were made aware of the commitment required in order for the study to be of practical benefit to them and the researcher. Transport problems became a major obstacle to the initiation and running of the investigation, with patients having to utilize public transport (paid by the researcher), which was both unreliable and expensive, to get to the meetings. Untimely State cuts in the subsidization of hospital transport resulted in the lack of transport for the patients participating in this study.

Seventeen patients; 8 in treatment group (TG) and 9 in control group (CG) arrived for their first session and completed the pre-treatment assessment questionnaires, clinical and immunological measures. Of these, 3 (CG) failed to arrive for their second session, and hence no bloods could be drawn for clinical and immunological assessment of these patients.

A total of fourteen (N=14) patients (8 in TG, 6 in CG) completed all of the treatments as well as the mid-, and post-assessments. The high attrition rate (10 out of 24) of subjects in the two groups was due to a number of factors. Two were suddenly prevented by their husbands (who felt that their wives received enough treatment for their arthritis) from participating; two could not be traced prior to initiation of the study because of changes in their home addresses; one decided that the programme would be of no use to her and thus decided to not attend

the introductory session; two patients were unable to utilize public transport for practical reasons (e.g., for the one patient it would have required catching two busses, the train and a taxi to get to the venue); one patient gained part-time employment prior to initiation of the study; and finally, two patients (CG) experienced unexpected difficulty in taking time off from their work to get to the four assessment sessions for their group.

The mean age, onset/duration of the disease, socio-economic status, educational, and demographic characteristics of the 14 subjects who completed all of the treatments are presented in Chapter Six, Section 2.1.

3. Procedure

3.1 Selection

A protocol of the research was sent to the Ethics and Research Committee of the Faculty of Medicine for mandatory approval (see Appendix A) for the inclusion of hospital patients in the study. Following acceptance by the Committee, senior consultants at the two out-patient clinics provided prospective patients with a brief description of the study (Appendix B), and referred them to the researcher. These patients had been selected out of a substantial patient population along stipulated inclusion criteria, with the assistance of the Head of the Arthritis Unit of the Department of Medicine, University of Cape Town.

The patients were seen individually during their monthly appointment to the clinic. They were given a brief description of the project as being "an investigation into the psychological (mental and emotional) aspects of RA, especially the pain associated with RA". Patients were also informed of the 8-week duration of the study, and that some committment was expected from them should they decide to participate in the programme. Furthermore, it was explained that the information obtained from such a study would be of benefit to both themselves and the medical profession. For the former, the programme aims at providing and instructing participants in various strategies to manage pain and the effects of the disease. For the latter, the information would assist the medical profession in their understanding of the impact of RA and the development of further pain management techniques and improved patient care.

It was stipulated that a limited number of patients were being selected and that those who were unable to participate in the first programme, would get the opportunity to do so at a later date. This was to anticipate possible inclusion of the patient into the control group. At all times, it was made clear to the patient that this was a study of how RA was affecting their life, and that all information gathered would be utilized to develop an effective psychological and physical therapy for persons suffering from RA.

Once informed consent (Appendix C) was obtained, subjects were given a written description of the programme, provisional starting dates, times and venues, an addressed envelope and a Pre-Interview Questionnaire (Appendix D) to complete and return to the researcher. The interview concluded with a discussion of any anticipated difficulties in participating in the study (such as transport, employment, family/spouse, time, venue, finance, etc.).

3.2 Allocation to Condition

Upon receiving the Pre-Intervention Questionnaire from the initial pool of patients (N=24), subjects were matched in pairs according to two criteria: date of onset of RA, and rating of ability to cope with present pain and disability. Members of each pair were randomly assigned to the treatment (TG) or control (CG) condition.

Subjects were then contacted telephonically and starting dates, times and venue (a seminar room in the arthritis department of Groote Schuur Hospital) for their "programs" were confirmed for the two groups respectively (see Appendices E, F, and G).

3.3 Assessment

Following description and discussion of the various questionnaires at the introductory session, subjects each received a battery of measures (see Section 5, and Appendices H to M) to complete and return on their next session. Strict instructions of how this should be done were given and subjects were asked to contact the researcher should they experience any difficulty with the questionnaires. Home appointments were made for the researcher to visit each subject to complete the Perceived Coping Efficacy interview. Subjects in the treatment group were given a Treatment Perception Questionnaire (Appendix N) to complete to determine their expectations regarding the value of the programme. Bloods were drawn (3 x 15ml vacutainers) by an experienced nursing Sister, who also completed the Ritchie Articular Index (Appendix O) on each subject (the same nursing sister completed all subsequent clinical assesments). Bloods were transported to Groote Schuur Hospital and the University of Cape Town Haematology laboratories for immunological assessment.

Matched subjects thus underwent identical assessment at the same time of day (8.30-9.30; TG Monday, CG Tuesday), at the same venue, and by the same clinic Sister. Subjects received a Programme Outline (Appendix G), detailing dates, time, venue, session number, and assessment points. Pre-assessment occurred during the first week (Sessions 1, 2, and 3), mid-assessment in the fourth week (Sessions 8 and 9), and post-assessment in the eighth week (Session 16).

Pre-assessment included the full battery of psychological, pain, and health status measures, administration of the Ritchie Articular Index (RAI), and withdrawal of blood for haematological analysis. Only two psychological measures (for mood disturbance and perception of coping efficacy), a pain measure, and the Ritchie Articular Index (RAI) were administered at mid-assessment. Post-assessment was exactly the same as pre-assessment.

4. Treatment Conditions

4.1 Treatment Group (TG)

The treatment group (TG) received a comprehensive "Stress Inoculation and Pain Management Training" (SIPMT) programme based on the conceptual approach of Turk et al. (1983) and Meichenbaum (1985), and adopted from a programme utilized by O'Leary et al. (1988). The SIPMT programme included the content of

O'Leary et al.'s (1988) treatment programme, adapted it to Meichenbaum's (1985) "Stress Inoculation Training" (SIT) approach, and incorporated the conceptual and practical strategies or techniques discussed by Turk et al. (1983) in their influential book on cognitive-behavioural therapy for chronic pain.

The object of the programme was to not merely remove stress and pain, but also to encourage subjects to view stressful or painful situations as problems to be solved rather than as threats to themselves and their well-being. The emphasis was on allowing the subjects to become more aware, and objective, of the impact of RA and the pain experience on their lives. The aim being to assist them in identifying possible and existing areas for effective intervention. The programme was designed to nurture and develop existing coping skills and to instruct and demonstrate "new" strategies or skills to cope with daily stress and pain.

By providing conceptual models of pain and stress, and explaining the rationale underlying the different techniques, the programme sought to promote the use of palliative (such as progressive muscular relaxation training, and breathing exercises) and cognitive (such as distraction, use of vivid imagery, dissociation, relabelling, self-appraisal and self-encouragement, communication skills, and goal setting) stress and pain coping techniques as active coping skills. It further sought to ensure that subjects developed a "proactive" defence against possible stressors in their lives and achieved a more adaptive self-appraisal of their ability to manage pain and disease effects.

The SIMPT programme consisted of sixteen 2-hour group sessions (9.00 - 11.00am), twice-weekly, for a duration of 8-weeks. The sessions were conducted by the researcher, with each session including both didactic input and group discussion. The subjects were encouraged to participate actively in the session, contributing their knowledge and experience for the benefit of the group and the researcher. Their collaboration was thus sought after and encouraged, and the subjects were at all times aware of the rationale behind the programme and the techniques demonstrated.

The programme was highly structured, with each session having a specific aim, but was kept flexible enough to accommodate needs expressed by the subjects on any particular day. Due to the "clinical" nature of intervention, the programme had to be flexible enough to deal with feelings/needs which a support group of this nature would engender. With the group discussions focussing on the effects RA had on the subjects' lives and their self-perceptions, powerful feelings of loss were aknowleged for the first time in some of the participants. The programme had to accomodate for these feelings, as well as the reaction of the participants to the researcher. The researcher had to ensure that the programme followed the highly structured treatment manual, yet at the same time respond to the dynamic group processes underlying such an intervention strategy. This will be discussed in greater detail in Chapter Seven.

The programme was designed around Stress Inoculation Training's three therapeutic phases: conceptual, skills application and training, and application and follow-through (see Appendix P for overview of programme). A treatment manual (Appendix Q) was drawn up by the author to ensure that the programme could be reproduced in later research. Below is a brief overview and description of the aims of each phase, and the pain management strategies utilized. The Treatment Manual (Appendix Q) provides a detailed explanation of both the content and procedures employed in the SIMPT programme.

4.1.1 Conceptual Phase

The introductory phase to the programme. In these sessions (1 to 4), the subjects were educated in terms of: (a) the rationale for the psychological (or non-medical) techniques for managing pain; (b) the effects of chronic pain; (c) the nature of stress and its effect on the body; (d) the two pain cycles that have been identified (the "depression/inactivity - pain", and the "anxiety/tension - pain" cycles); and (e) the process and importance of goal setting. The alternative goal system was utilized to assist subjects in developing more practical and satisfactory alternative activities to replace those which were lost due to the disease effects of RA.

The central emphasis in this phase was to ensure the active collaboration of the subjects in order to maximize the benefits of instruction in self-help skills. By providing information, and ensuring comprehension of the rationale behind the non-medical treatment of pain and collaboration, it was assumed that compliance and utilization of the skills would be nurtured. A biopsychosocial model of pain was introduced, explaining the role of psychological and behavioural processes contributing to the pain experience. A model of stress (stressor-appraisal-stress response) was also introduced, explaining the role that stressful experience and coping techniques play in adapting to the pain and disability as a result of RA. A model of RA was presented and discussed in terms of the subjects' understanding and reasons for having or developing RA. Pain diaries (Appendix R) and goal sheets (Appendix S) were used to further these aims. Finally, the treatment programme was outlined, to ensure that patients had a clear perception of the context of the strategies that were to be employed.

4.1.2 Skills Acquisition and Training

In these sessions (5 to 12) the subjects were introduced to, and trained in, effective strategies to manage stress and pain (reviewed in Turk et al., 1983). Two major forms of coping were identified: palliative (emotion focussed) and cognitive (problem focussed). The former utilizing relaxation exercises and the latter utilizing cognitive pain coping strategies.

Subjects received the rationale for each technique, were given instruction in the technique, and shown when and how the technique could best be used. Worksheets for each strategy facilitated this instruction (Appendix T). Following O'Leary's (personal communication, April, 1990) suggestion, the strategies were tailored for use during specific painful activities such as climbing stairs, walking, using transport, carrying groceries, doing housework and general daily activities. It was emphasized that it was necessary to practice the technique for optimal efficacy. A "buddy system" was also set up whereby subjects were encouraged to keep in contact with each other telephonically to promote goal setting and the reinforcement thereof.

4.1.3 Application and Follow-Through

The final phase focussed on consolidating the skills and knowledge acquired in the first two phases. Emphasis was thus placed on communication and support within the group, on reinforcement, and encouragement in utilizing the coping skills learned.

Group discussions focussed on the impact of RA and pain on their lives and the role that the cognitive-behavioural skills could play in improving quality of life. The syndrome of "learned helplessness" or passive coping was addressed in both didactic and group discussions, and appraisal of coping efficacy by self, group and researcher occured. The role of effective communication of one's feelings and pain experience to significant others was discussed and instruction was given in effective ways of communicating needs. This final phase of the SIMPT focussed on problem solving in terms of any difficulties encountered in utilizing the coping skills taught in the programme, the need to anticipate set-backs and obstacles, and the means to devise appropriate solutions to such difficulties.

4.2 Control Group (CG)

The control group (or no adjunct therapy group) met for four sessions (1 to 2 hour duration) where the researcher provided them with some information of the role of psychological experience on pain. The meetings were kept informal with the sole purpose of the subjects receiving the questionnaires, having their blood drawn, and discussing any difficulties encountered with the self-report measures. It was necessary, however, to provide some discussion and information during those sessions to ensure increased compliance to the study. This also included an element of the social inducement of change in both groups.

5. Dependent Measures

5.1 Pre-Interview Questionnaire (PIQ)

This questionnaire was designed specifically for this study. It is divided into three sections: Personal and Biographical details; Description of your pain; and Pain-coping strategies. Sections two and three (13-items) were adapted from Karoly and Jenson'sa (1987) suggested pre-interview questionnaire in their discussion of the "clinical pain interview". The PIQ assessed the degree of the subject's perceived or subjective pain experience; their use of coping strategies (including medication, exercise, relaxation, bedrest, and physiotherapy); and their perceptions of their ability to control or manage their pain. The latter data was used as one of the variables in matching pairs of subjects prior to random allocation to condition (onset of illness was the other variable).

5.2 Measures of Internal Validity

The Treatment Perception Questionnaire (TPQ) constituted the first measure of the internal validity of the investigation. The TPQ, taken from Bradley et al. (1987), consisted of three 7-point rating scales of subjects' expectations for improvement, and beliefs about the credibility of the cognitive-behavioural intervention. This questionnaire was administered to the TG group following their introductory session.

The second measure of the internal validity of the investigation, consisted of monitoring all subjects' analgesic and arthritis-related medication intake over the duration of the study. Data was obtained from medical folders pre-,

mid-, and post-intervention to assess differences between the two groups with regard to changes in medication usage between assessment points.

5.3 Perceived Coping Efficacy (PCE)

This semi-structured interview questionnaire was based on Zuatra et al.'s (1989) measurement strategy to assess self-efficacy in coping (see earlier discussion, Chapter Four). Following Zuatra et al.'s suggestions (personal communication, August, 1989), the subject was first asked to select the four most stressful events from those that had occurred during the month prior to the interview. The events included anything that made the subject anxious, upset, concerned, angry, frustrated or distressed. "Arthritis flare-up" was also included as a stressful event. The subject was then asked to explain how she responded to each event (such as how she felt, what she did to resolve the crises, who she spoke to, etc.). Immediately following the inquiry about specific coping responses to the event, the researcher asked the subject to rate the following using a 5-point rating scale:

(1) The level of satisfaction she felt with her own response;
(2) The level of satisfaction she felt with the response of others;
(3) The degree to which she would be able to cope successfully with the event should it recur in the future.

These coping efficacy ratings (grounded in specific events, specific responses made to events, and expectations about similar future events) were summed across the four events to create a 12-item coping efficacy scale. Zuatra et al. (1989) judged internal consistency reliability to be adequate for this experimental scale (Cronbach = 0.64); and found that it demonstrated a moderate level of stability over time (10 months) in a separate study (personal communication, August, 1989).

5.4 Multidimensional Health Locus of Control Scales (MHLC)

Form-B of the MHLC (Wallston et al., 1978), an 18-item questionnaire, yields scores on three independent dimensions of health locus of control beliefs. The "Internal Health Locus of Control" (IHLC) scale measures "health internality", the extent to which a person believes health is a function of his/her behaviour (Wallston et al., 1983). The "Chance Health Locus of Control" (CHLC) scale assesses "chance externality", the degree to which a person believes that his/her health is unpredictable, a matter of fate, luck, or chance (Wallston et al., 1983). Finally, the "Powerful others Health Locus of Control" (PHLC) scale measures "powerful others externality", tapping the person's beliefs that health is largely determined by the actions of powerful others, either family members, friends, or health professionals (Wallston et al., 1983).

Wallston et al. (1983) have reviewed the research using the MHLC scales and are presently developing Form-C (personal communication, May, 1990) which can be easily be made to be more specific to any existing medically-related condition (including arthritis, cancer, diabetes, high blood pressure, migraine headaches, etc.). The psychometric properties of the MHLC scales are well researched with large samples, including: chronic subjects, college students, healthy adults and persons engaged in preventative health behaviours (Wallston, personal communication, May, 1990). Snow and Thurber (1983) investigated the factorial validity of the scales, supporting

the contention that the health locus of control construct is multidimensional (see Stanley, Hyman & Sharp, 1984), and concluding that the MHLC is sensitive in the assessment of differing health-related attitudes and beliefs.

5.5 Stress Evaluation Inventory (SEI)

The SEI, a 30-item questionnaire, was developed by Madsen (1983), and yields scores on three subscales, each assessing a major stress-related area: Career, Family, and Personal-Social. These scores are based on the frequency of occurance of potentially stressful events or experiences. Factorial analyses of the SEI (Madsen, 1983) show these subscales to be largely unidimensional and homogeneous. The reliability (Madsen, 1983) of the overall scale for women is excellent (0.82), and reliabilities of the individual 10-item subscales were considered acceptable (Career, 0.66; Family, 0.89; Personal-Social, 0.75). The scale's validity was assessed by correlating it with faking scales empirically developed from the 16 PF Form A item pool (Madson, 1983). The results confirmed the expectation that when people are motivated to fake good, they tend to deny the applicability of items in the SEI, the converse also being true. Research utilizing this measure would thus have to account for the implicit motivations of subjects involved in the study for accurate assessment of stressful life experience. The SEI has been effectively used as an assessment tool for developing applicable stress management programs (Madsen, personal communication, 1990).

5.6 Profile of Mood States (POMS)

The POMS is a factor analytically derived 65-item inventory (McNair et al., 1976) which measures six identifiable mood or affective states. These include: Tension-Anxiety, Depression-Dejection, Anger-Hostility, Vigor-Activity, Fatigue-Inertia, and Confusion-Bewilderment. The POMS has proved to be a sensitive measure of the effects of various experimental manipulations upon normal and psychiatric out-subject subjects. In research conducted by McNair et al. (1976), reliability and validity studies proved the POMS to be a highly satisfactory measure. Internal consistency ratings of 0.9 and above have been recorded using large samples.

Test-retest reliability data (McNair et al., 1976) from a sample of 100 out-patient subjects demonstrate stability coefficients considerably lower than the 0.8 - 0.9 levels expected of stable personality characteristics. This was expected, as stability of a fluctuating state like mood should hardly be expected to reach the levels of stability required of personality traits (McNair et al., 1975). The factorial validity of the POMS has been found to be excellent (McNair et al., 1976), considering the results for different subject and normal samples in 6 factor analytic replications. Face validity of the scale is good, and McNair et al. (1976) report on four areas of research providing evidence for the predictive and construct validity of the POMS.

5.7 McGill Pain Questionnaire (MPQ)

The MPQ (Melzack, 1975; Melzack, 1983; Melzack & Wall, 1988), a 20-item questionnaire, is a frequently reported dependent measure in both clinical evaluations and treatment trials with pain subjects (Reading, 1983; Melzack, personal communication, 1990). It consists of 3 major classes of word descriptors: Sensory, Affective, and Evaluative; that are used by subjects to specify subjective pain experience. The MPQ also includes a pain intensity

scale. The scale thus yields three major measures: (1) the pain rating index (PRI); (2) the number of words chosen; and (3) the present pain intensity (PPI). These provide quantitative data of the subjects pain experience that can be treated statistically.

Reading (1983), reviewing the research using the MPQ, reports high consistency index ratings of 70% to 75% for various subject samples. Increased use of the MPQ in clinical settings testifies to its face validity, and Reading (1983) and Prieto and Geisinger (1983) report on factor analytic studies confirming Melzack's (1975) original postulation that the MPQ reflected three main dimensions of the subjective pain experience. Furthermore scores on the MPQ, particularly the affective scale, correlate consistently with scores on depression inventories and on the Minnesota Multiphasic Personalty Inventory (MMPI) (Reading, 1983; Bradley, 1983). Finally the scale has high concurrent and predictive validity, as well as demonstrable discriminant validity (Reading, 1983) using different subject groups.

5.8 Arthritis Impact Measurement Scales (AIMS)

The AIMS (Meenan et al., 1980) is a 66-item evaluation instrument developed to measure patient outcome in the rheumatic diseases (Meenan, personal communication, March, 1990). It is designed to measure the health status component (health status as defined by the World Health Organization) of outcome in a multidimensional fashion using 9 specific scales, 3 summary component scales and one overall impact item. These scales include (Meenan, 1982): Mobility, the ability to move about the community; Physical Activity, or lower extremity function; Dexterity, or hand function; Household Activities, or routine household tasks; Activities of Daily Living, basic self-care tasks; Social Activity, interaction with friends and family; Anxiety; Depression; and Pain. The AIMS also includes items related to Health Status (co-morbidity), Health Perception and the Impact of RA.

The AIMS yields scores expressed in the 0 to 10 range for each subscale, with 0 representing good health status, and 10 poor health status. Furthermore, these scores can be combined into three health status components: Physical function, Psychological status, and Pain (Meenan, personal communication, March, 1990). Reliability and validity indices of the scale have been extensively researched and reported on (Meenan et al., 1980; Meenan, 1982; Meenan, Gertman, Mason & Dunaif, 1982; Kazis et al., 1983; and Fries, 1983). Meenan (1982) has reviewed the measurement properties (reliability, validity, generalizability, and sensitivity) of the AIMS from the responses of 625 arthritis patients, and found that all 9 scales exceeded generally accepted criteria for these properties using a variety of statistical approaches.

5.9 Ritchie Articular Index (RAI)

The Articular Index was devised by D. M. Ritchie in 1964 (Bird, Le Gallez & Hill, 1985), and is used extensively in the clinical assessment of arthritic patient populations. The RAI comprises a simple scoring system for joint tenderness, and it assesses the severity of disease activity by relating it to the amount of pain felt. A scoring chart is used (see Appendix N), and the Index attempts to quantify the patients experience of pain.

The assessment has to be performed by the same person, as it involves a subjective interpretation of the patient's joint tenderness by observing their response (mild pain = 1, moderate pain = 2, and severe pain = 3) to the pressure applied to the specified joints. As such, its intra-rater reliability is dependent on the skill and consistency (i.e., the amount of pressure or "squeeze" exerted on the joint) of the person administering the assessment. The Index yields a maximum possible score of 78. Scores usually range from 0 to ±40 (Bird et al., 1985). An extremely high score (i.e., 30) would indicate extreme disease activity, a score of over 15 could indicate widespread but mild disease activity, a score of less than 15 indicating little activity. The first RAI performed on a particular patient produces a baseline measure for later reference and comparison (Bird et al., 1985). It is thus important that the same person administers the measure, monitoring the patient's progress over time.

5.10 Ethrocyte Sedimentation Rate (ESR)

Ethrocyte sedimentation rate (ESR) is an haematological index of disease activity. Peripheral blood was drawn from an ante-cubital vein into a 15ml vacutainer (Radem Labs, Santon, South Africa) and sent to Groote Schuur Hospital's laboratory for haematological analysis.

5.11 Lymphocyte Proliferation Rate

Immunocompetence was assessed by the proliferative response of the subject's lymphocytes following mitogenic stimulation. Peripheral blood was drawn from an ante-cubital vein into 2x15ml vacutainers (Radem Labs, Santon, South Africa) containing EDTA. The blood specimen was then double-diluted with RMPI-1640 (Gibco, Europe Ltd., Scotland), separated on Lymphoprep (Nycomed, Oslo, Norway) and washed twice in RPMI-1640. After a second wash, the cells were resuspended in RMPI-1640 and 20% Fetal Calf Serum (Gibco, Europe Ltd., Scotland) and counted on a Coulter Counter (Coulter Electronics, Hialeah, FL). The cell count was then adjusted to $5x10^5$ /ml. The control cells, obtained from a normal donar and stored frozen, were thawed, diluted in 20ml 20% FCS in RMPI-1640 and washed twice. After counting, these were also resuspended in complete medium at $5x10^5$ /ml.

100ul of each specimen and the control was pipetted into a microtitre plate (Sterilin Ltd., Hounslaw, United Kingdom), such that there were 12 wells for each specimen. 50ul of Conconavilin A (Calbiochem Co., La Jolla, California), Pokeweed Mitogen (Flow Laboratories, Norway), Phytohaemmaglutinin (Wellcome Diagnostics, England) and complete medium were added in triplicate (3 wells per specimen). The microtitre plate was then wrapped in cellophane and incubated at 37 deg/C with 5% CO_2 for three days (72 hours). After pulsing with 1ul 3H-thymidine (3H-T2r) (Amersham PLC, Amersham, United Kingdom), cells were incubated for a further 18 hours at 37deg/C, before harvesting with a cell harvester (Titertek, Skatron, Norway). Dried discs were placed in scintillation vials, dissolved with 2ml toulene plus PPo plus bis-MSB, and counted in a beta counter (Beckman LS 1801: Beckman Instruments, Fullerton, CA).

The results are expressed as a percentage of the control count for each mitogen by dividing the subject's result by the control used for that particular assay.

CHAPTER SIX

RESULTS

1. Data Analysis

1.1 Data Capturing

All questionnaires administered were collected from the subjects, scored, and punched into data files (Data Capturing Department, UCT) for each assessment point. All subsequent statistical analyses were carried out using SAS software (SAS Institute Inc, Cary, U.S.A: VMS SAS Production Release 5.18, 1988). A SAS programme was drawn up to read the data, score the subject's responses, and to create the key variables necessary for subsequent data analysis.

1.2 Statistical Analyses

In order to test the hypotheses that:

(a) a significant relationship exists between psychological: and pain, rheumatoid arthritis, and immune response variables; and that

(b) stress inoculation and pain management training will improve psychological adjustment and thereby effect changes along these variables;

the following stages were used in the data analysis.

(1) Pre-intervention sample mean $\pm\underline{SD}$ scores were calculated for all variables to determine the extent to which the sample differed from expected norms. These were tabulated (see Table 1 and Table 3), and compared to norms and maximum ranges of response.

(2) A test for normality (SAS Procedure, Univariate Analysis) for each variable was conducted to determine whether or not the data satisfied the assumption justifying parametric statistical analysis. The variables were then grouped accordingly for later statistical procedures to ensure that the appropriate tests were performed for the parametric and non-parametric data.

(3) A correlation matrix (Pearson Product-Moment correlation coefficient) was set up to test the first hypothesis at a 5% significance level. The matrix was then broken down, to allow for meaningful examination of the correlations between key variables and tabulated accordingly (see Tables 5 to 10).

(4) To determine whether or not pre-intervention differences were a factor predictive of later change over time, baseline comparisons (t-tests and Wilcoxon Rank Sum tests) between the experimental and control groups were made on all measures (see Tables 11.1 and 11.2).

(5) To test the second hypothesis (to examine treatment effects), change scores were calculated for all key variables. The following calculations were performed:

MID - PRE = Change 1

POST - PRE = Change 2

These change scores were used in a series of univariate analyses (paired-difference t-test and Wilcoxon Signed Rank test for paired groups/observations) to determine inter- and intra-group differences over time (see Tables 12.1 to 13.2 for summaries of these results). These analyses tested the extent to which mean change was significantly greater or less than 0 (where 0 indicates that change was non existent). The lymphocyte data was not converted to change scores, but expressed as a percentage response compared to the healthy control serum used in the assay (see Tables 14.1 and 14.2).

(6) To examine observed differences between the two groups' distributions of change scores along key variables, a number of side-by-side graphs were plotted. These graphs provided summary information regarding the nature, pattern and extent of observed differences. This facilitated a discussion of the results of the analyses of the intra- and inter group changes over the duration of the intervention.

(7) Based on the small sample size, the heterogeneity of the subjects within each group, the clinical nature of the immunological data, and the results of the findings in the above mentioned analyses, a case-study approach was adopted. A further justification of the need to examine individual change, is the importance placed on the central role played by the individual in health and disease. Each individual has a unique response to changes within the human system, and this response cannot be determined through standard statistical procedures.

This procedure provided valuable and meaningful information pertaining to the two hypotheses, in that it was able to offer more insight into the changes observed. In order to compare the variables in a meaningful way, it was necessary to reduce all variables to the same scale of measurement. This allowed for an analysis of the relationships between the key variables over time for the individual subject.

2. Pre-Intervention Analyses

2.1 Sample Characteristics

Sample characteristics and mean $\pm$SD scores on key variables are provided in Table 1. The sample (N=15) was primarily (60%, n=10) composed of middle-age "coloured" women with a mean $\pm$SD RA onset date of 1983 $\pm$3.75 (mean duration = 7 years). This onset date is the date of actual diagnosis of classic RA, and thus may not be indicative of actual duration of morbidity. Figures 1.1 and 1.2 demonstrate the distribution of the sample along age

Table 1: Sample characteristics and mean scores on Major Variables (Pre-intervention)

Variable	N	Mean/ or Percentage	±SD
Sample Characteristic			
Age	15	49	9.44
Population Group			
Coloured	10	60%	
White	5	30%	
Moslem	2	14%	
Married	7	41%	
Divorced	2	12%	
Widowed	5	29%	
Other	3	18%	
Income Bracket	15	≤R1000 p/m	
Onset of disease	17	1983.29	3.75
Psychological Adjustment			
Total Mood Disturbance	16	200.44	40.29
Perceived Coping Efficacy	16	32.19	9.77
Health Locus of Control			
Internal	16	24.38	5.51
Powerful Others	16	21.38	6.21
Chance	16	20.63	8.72
Stress Evaluation Inventory	16	12.56	8.21
Pain Experience			
Total Pain Rating Index (PRIT)	16	25.31	13.88
Present Pain Intensity	16	2.25	0.77
Ritchie Articular Index	15	13.26	6.17
Pain (range = 0-10)	16	6.50	2.02
Impact of Arthritis (AIMS)			
Psychological (range = 0-10)	16	4.16	1.84
Physical (range = 0-10)	16	3.80	1.16
Inflammatory Index			
Ethrocyte Sedimenation Rate	11	37.27	28.72
Baseline Immune Response*			
Treatment:			
BPHA	7	204.7%	75.06
BPWM	7	210.6%	65.09
BCON	7	403.8%	197.54
Control:			
BPHA	5	126.8%	23.79
BPWM	5	210.2%	30.67
BCON	5	585.7%	936.18

*BPHA= Baseline Phytohaemaglutinin percentage response rate, BPWM= Baseline Pokeweed Mitogen percentage response rate, BCON= Baseline Conconavilin A percentage response rate.

Figure 1.1: Distribution of Age
Sample at Pre-intervention

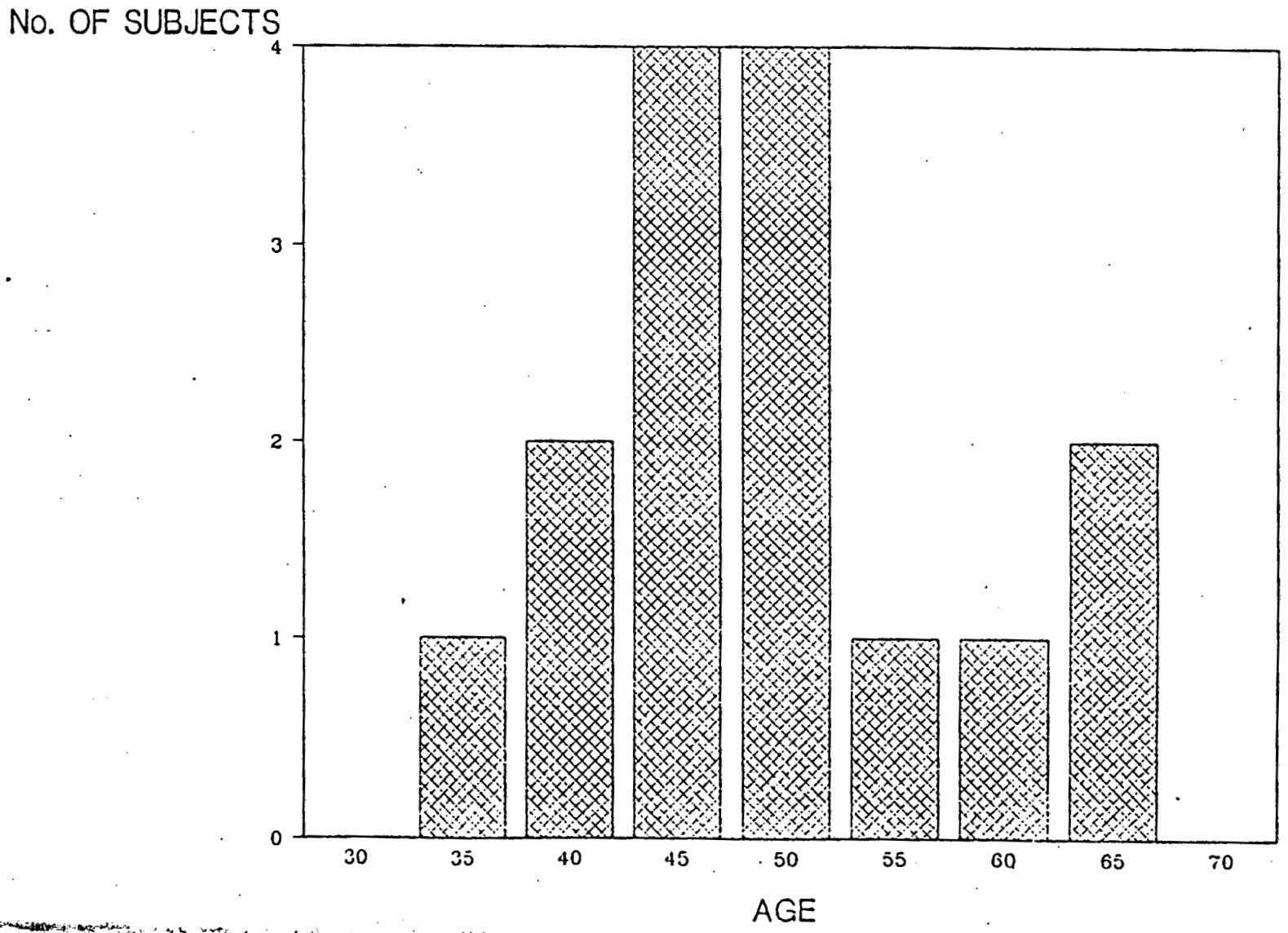
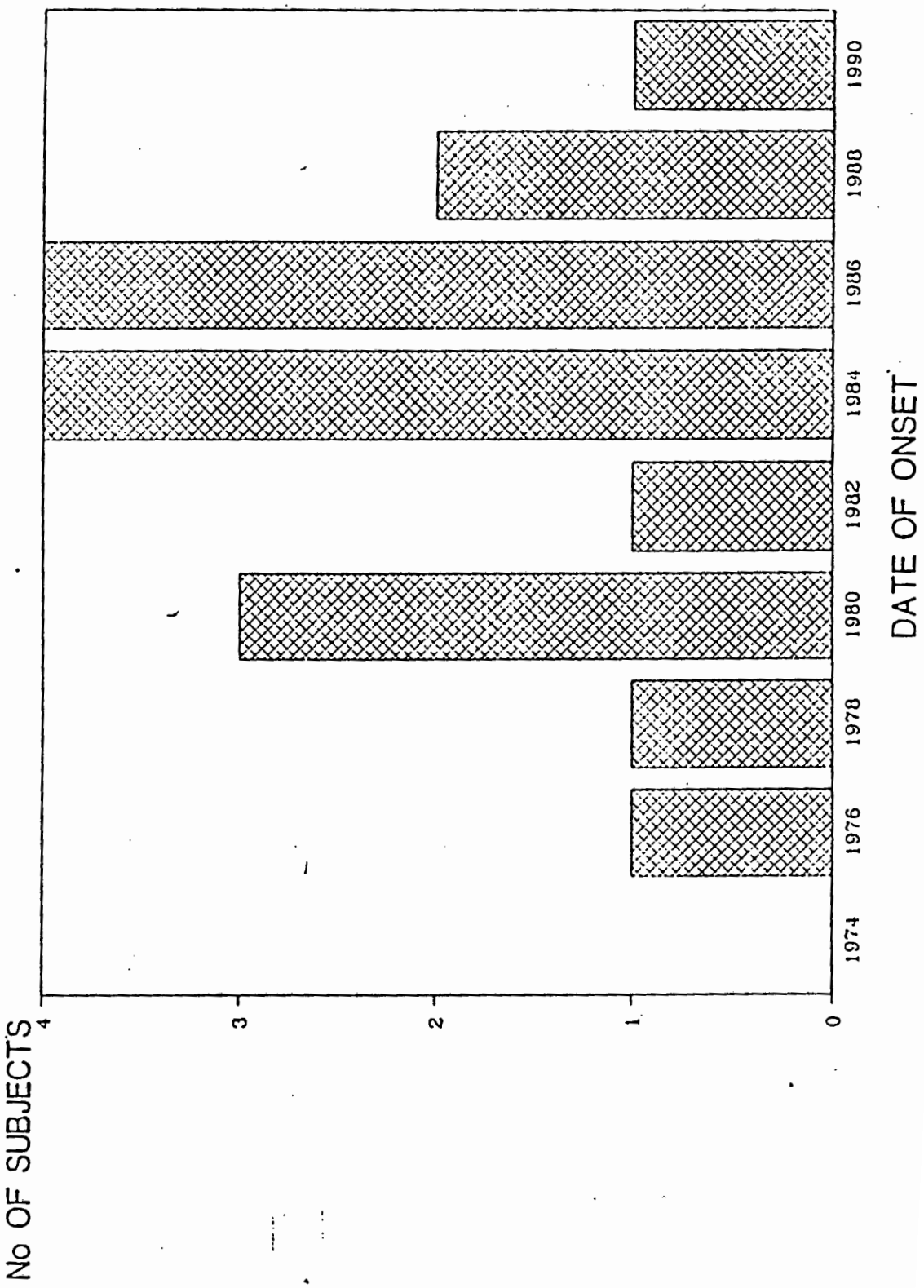


Figure 1.2: Distribution of Date of Onset
Sample at Pre-intervention



onset. Almost half (44%, n=7) the sample were married, the remainder being either widowed (32%, n=5), divorced (10%, n=2), separated or single (14%, n=3).

The sample had a relatively moderate level of education, with 60% (n=9) having had a standard nine or lower education, and 40% (n=6) a matric or further training. Only a fifth of the sample was employed (20%, n=3), the rest being either medically unfit for work (25%, n=4), house-wives (31%, n=5), pensioners (13%, n=2), or unemployed.

The mean income of the group was particularly low, with 85% (n=13) of the subjects receiving less than R1000.00 per month. This indicates that the sample is comprised mainly of working class women from low income (it was necessary to remunerate the subjects' transport costs to ensure attendance to the sessions) urban areas in the greater Cape Town municipality. The cultural and class differences within the sample did not seem to hinder the subjects relationships with each other. The fact that they all suffered from RA was seen as a common denominator.

2.2 Baseline Psychological Adjustment, Pain and Disease State

The sample (see Table 1) had a relatively poor pre-intervention level of psychological adjustment (high total mood disturbance, and low perceived coping efficacy); high levels of reported pain (total pain rating index of words describing qualities of pain experienced, verbal analogue of pain intensity, and impact of pain); a mean articular index indicating widespread but mild disease activity; and a mean ethrocyte sedimentation rate that was well above the normal range (0 to 7) for women. The impact of arthritis on health status was moderate, with higher scores on the psychological dimension.

2.2.1 Psychological Adjustment

The mean ±SD total mood disturbance (TMD) of the sample (200.44 ±40.29) was well above average (normal = 136 ±8.00), supporting the prediction that RA has a significant impact on the psychological well-being of these patients (See Figure 1.3). A break down of the mean ±SD TMD score revealed the extent to which the elevated levels of the individual mood factors contributed equally to the overall mood disturbance (Tension-anxiety 52.3 ±12.23; Depression-dejection 50.88 ±8.44; Anger-hostility 50.56 ±8.90; Vigor-energy 52.31 ±12.23 Fatigue-inertia 51.13 ±12.76; and Confusion-bewilderment 48.69 ±8.93). The sample was considered to be particularly "unstable" on the tension, anger, depression, and fatigue mood factors.

Self-report ratings of perceived coping efficacy tended to support this finding of poor psychological adjustment. Moderately low (±54% of ideal level of coping efficacy) perceptions of coping efficacy in response to stressful events were found. Figure 1.4 shows the distribution of the sample on this variable, where only two subjects had very good perceptions of coping efficacy. No real differences existed between the sample's mean ±SD scores indicating satisfaction with self (56%, or 11.2 ±3.9) or with others (53%, or 10.6 ±3.4) as a result of their response to the events. This was also true for their belief in their ability to cope with similar events in the future (52%, or 10.4 ±3.8).

Figure 1.3: Distribution of Total Mood Disturbance scores Sample at Pre-intervention

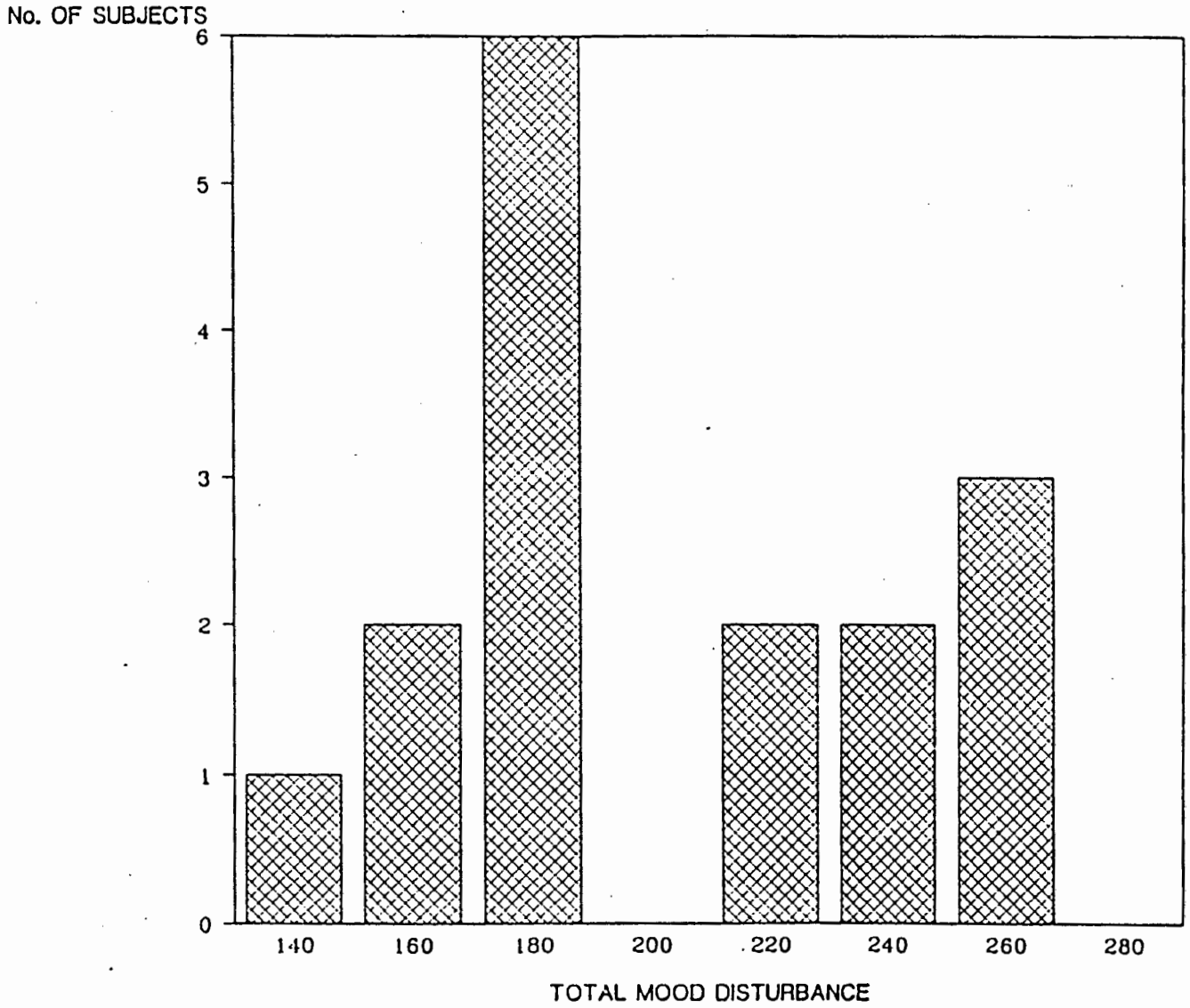


Figure 1.4: Distribution of Perceived Coping Efficacy scores Sample at Pre-intervention

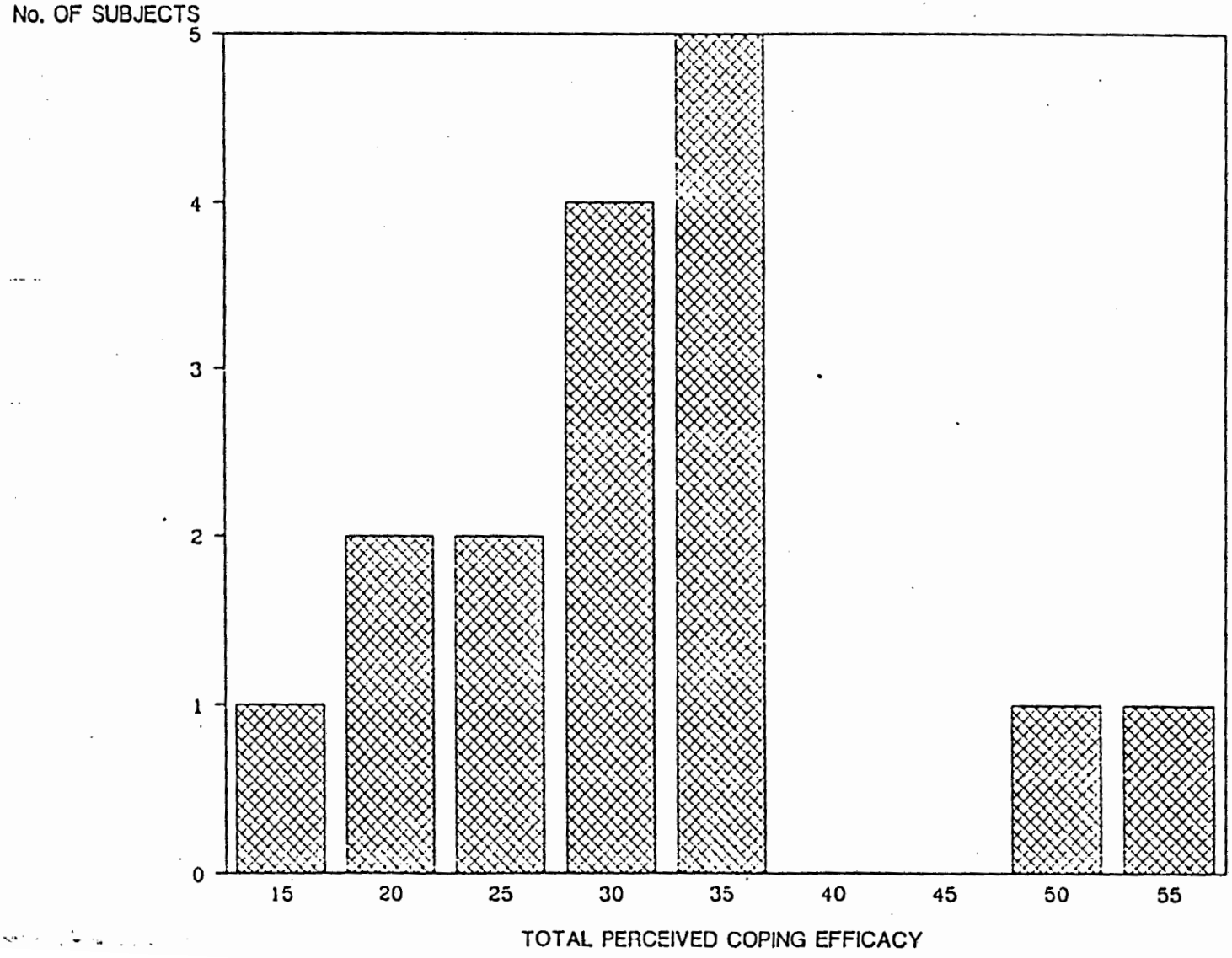


Table 2: Typical stressful events and responses to them as recorded in the Pre-intervention Coping Efficacy interview.

1.	Children and Husband did'nt want to help clean the house.
1.1	I did it myself, and moaned about it. Felt very upset and let down.
2.	Having to look after my daughter's child during the day.
2.1	Could'nt cope with her, she is too active, and I got frustrated.
3.	Being alone at night when my husband does shift work.
3.1	Felt terrible; having arthritic pain and being on my own, I felt helpless.
4.	Having to keep the peace in the house.
4.1	I was scared the arguments would lead to a fight, I cannot control them.
5.	Having no time to do things for myself (always for the family).
5.1	I always seem to be running about for my family, but I cannot refuse even when I am in pain.
6.	Shopping for the groceries.
6.1	I get frustrated as I got caught in the rain and could'nt run home because ofthe arthritis.
7.	Having to walk far, and to rush to get on and off the taxis/busses.
7.1	I just hope I can manage because I have to do what I want to do despite the pain.
8.	Worried about our financial problems.
8.1	First kept quite and felt upset, then spoke to son about his work problems.
9.	Severe pain and went to the clinic for some pills but the doctor said I was imagining it.
9.1	Felt angry and upset, and told him I don't need his medicine, and wen without it for the whole month.
10.	Fell off chair after standing on it to reach for something.
10.1	First woried about the chair, but then realized that I had hurt my knees and lay down for a bit.

Table 2: Continued....

11.	My ankle needs to be operated on, but they have not yet sorted it out for me yet which is very frustrating.
11.1	I felt they were'nt listening, was angry that they took so long to see what the trouble was; I could'nt talk about it to anyone.
12.	My eldest son stayed out late.
12.1	I was worried (we have had a lot of trouble recently), stayed up all night and scolded him when he got back.
13.	Having to cope with my housework, shopping, and work and get everything done on time.
13.1	I felt exhausted and very stressed, felt as if everything was getting out of control.
14.	Tense living circumstances where my daughter always complains about me.
14.1	I try to please her, but never seem to do the right thing as she always accuses me of messing things up.

The events (see Table 2) identified by the subjects as being particularly stressful gave some indication of the possible causes for such elevated mood disturbance scores. Closer analysis of the themes that emerged from these semi-structured interviews (see Table 2), revealed that the stressful events were predominantly inter-personal in nature, relating to significant others. The subjects considered domestic events (such as familial disputes, finances, loneliness, household tasks) and difficulties related to RA (such as pain, dissatisfaction with treatment or medication, disability) as particularly stressful.

Mean $\pm\underline{SD}$ scores on the SEI (see Table 1) did not support these findings, where low levels of total stress experience (12.56 $\pm$8.21, compared to a norm of 19.51 $\pm$9.32 for healthy women) were recorded. This result is unusual considering the mean TMD score, and should thus be interpreted with caution. Extremely low job stress scores (1.81 $\pm$3.64), however, affected the total stress score. Scores on the Family and Personal stress (5.38 $\pm$3.93 and 5.33 $\pm$2.78 respectively) subscales were consistent with expected values (7.59 $\pm$4.16 and 4.96 $\pm$3.72 respectively) for healthy women (Madsen, personal communication, 1984).

Closer analysis of the response rates on the items of the Stress Evaluation Inventory revealed that subjects were not acknowledging the true extent of their stress experience (i.e., these scores did not concur with the stressful events listed in the semi-structured interview to assess perceived coping efficacy). It is not clear whether or not these conflicting results were due to inadequacies in the measure or failure on the part of the respondent to recognize/admit the true nature of their stressful experience.

The sample displayed an expected health locus of control for a chronic illness population. One-sample t-tests (two-tailed) were conducted to calculate the extent to which this sample's mean scores on the three locus of control dimensions differed from the norms available. The following calculation was used:

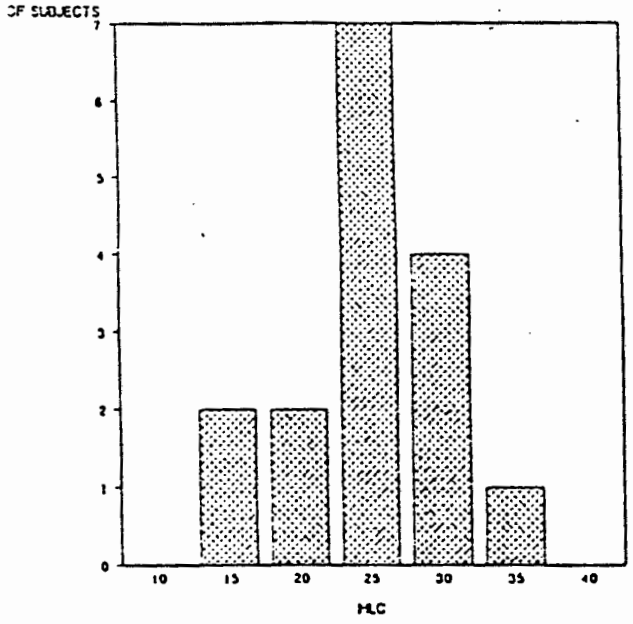
$$\underline{t} = \frac{\underline{M} - \underline{u}}{(\sqrt{\underline{s}^2/\underline{N}})}$$

It was found that the sample did not differ significantly from the expected means for such a group (IHLC, $\underline{t}$ = -1.02; PHLC, $\underline{t}$ = -.75; and CHLC, $\underline{t}$ = 1.37; $\underline{df}$=15, $\underline{p}\leq 0.15$).

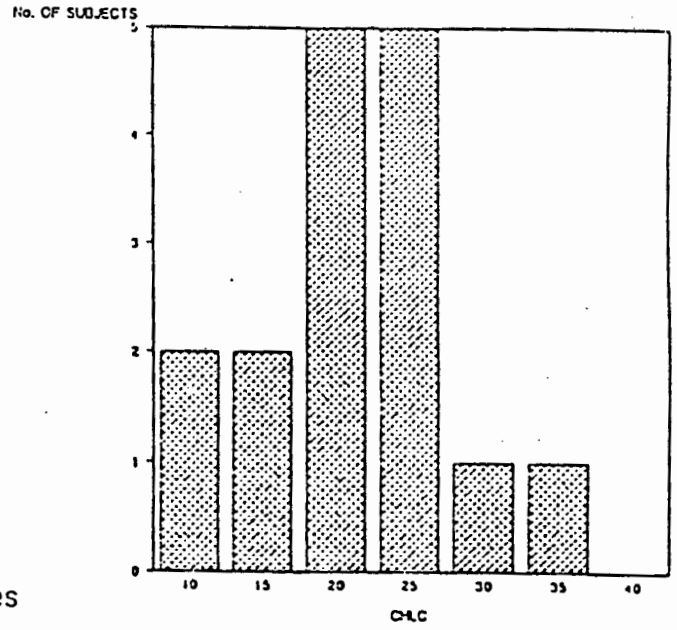
The large variation of scores on the PHLC scale (see Figure 1.5) reflect the extent to which this group differ in their perceptions of the role of professional health care workers in controlling their health. Ideal scores for chronic patients on this measure would be high scores on the PHLC and IHLC scales. Managing RA requires a combination of strict compliance to treatment and medication regimens as well as reliance on one-self to manage one's health (such as preventing damage to joints through inappropriate activity). In this sense, the latter two scales could be interpreted as a single scale depicting "internality" for a chronic illness sample.

Figure 1.5: Distribution of Health Locus of Control scores Sample at Pre-intervention

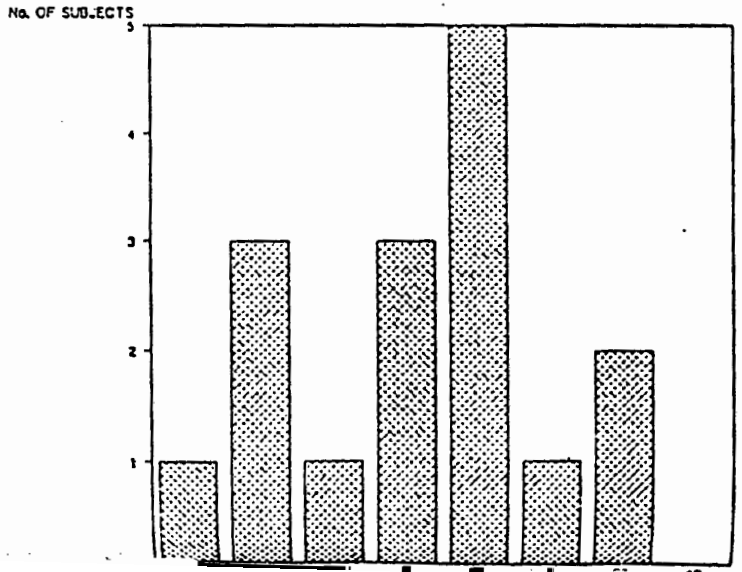
Distribution of Internal Health Locus of Control scores
Sample at Pre-intervention



Distribution of Chance Health Locus of Control s
Sample at Pre-intervention



Distribution of Powerful Others Health Locus of Control scores
Sample at Pre-intervention



2.2.2 Pain

The results of the measures assessing the qualitative aspects (McGill Pain Questionnaire - Pain Rating Index), Intensity (Present Pain Intensity), and impact of pain (Pain subscale of the AIMS) were consistent with predicted levels for arthritis sufferers. These results illustrate the importance of assessing multiple aspects of the subject's pain experience. Table 3 provides a breakdown of the baseline pain scores as rated by the MPQ, Pain subscale of the AIMS, and Ritchie Articular Index.

In order to account for difficulties in making comparisons between the pain dimension sub-scales yielded by the MPQ, an additional scoring procedure (as suggested by Kremer, Atkinson & Ignelzi, 1981) was utilized. A score for each dimension (sensory, affective, evaluative and miscellaneous) was calculated by summing the rank order intensity value for that dimension and dividing the summated score by the total possible score on that dimension. The calculation was as follows:

$$\frac{\text{sum of rank values}}{\text{total possible score}} = \text{pain rating index score}$$

This procedure yields values ranging from 0 to 1.0, "with 0 indicating that the patient selected no words from a particular dimension to describe [her] pain, and 1.0 indicating that the patient selected all of the highest ranked descriptors in a particular dimension to describe [her] pain" (Kremer et al., 1981). This procedure has an advantage in that it makes sub-scale scores directly comparable without altering the amount of information provided by the sub-scales scores (Karoly & Jenson, 1987).

The evaluative ($\underline{M}$ = 0.43) and sensory ($\underline{M}$ = 0.38) dimensions of the sample's pain scores seem to be the most significant qualitative aspects of their pain experience. The former score indicating the extent to which the subject's subjective experience of her pain is strongly influenced by her perception of the meaning of such pain. An example of this would be a patient interpreting pain in the joint as further damage to that joint (i.e., "I feel pain, therefore there must be something wrong"). These values are well above the means reported in an earlier study (Melzack, 1975) on RA patients. The mean $\pm\underline{SD}$ total pain rating index (25.31 $\pm$13.87) demonstrated the extent of the heterogeneity of the subjective pain experience of this group (see Figure 1.6).

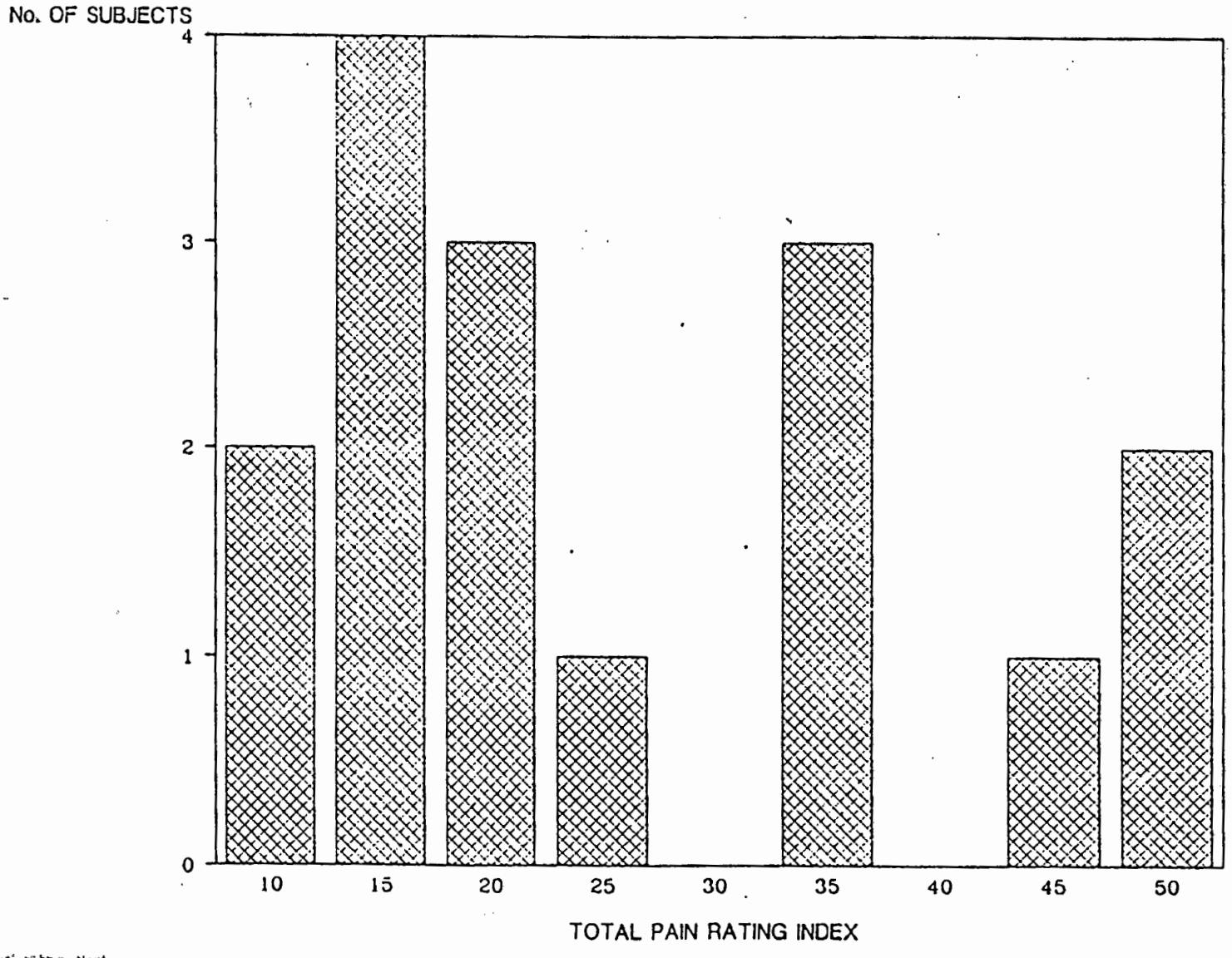
The mean $\pm\underline{SD}$ Present Pain Intensity (PPI) score indicated that the group were uniform in their assessment of the intensity of their pain. A score of 2 on this measure indicates that one rates one's present pain as "discomforting". The AIMS-pain subscale mean (6.5 $\pm$2.02) was moderately high reflecting the extent to which pain has had an major impact on the patients' lives during the month preceding the assessment.

The AIMS also produces total psychological (the sum of depression and anxiety subscales) and physical (the sum of a number of functional status related subscales) health status scores. The mean $\pm\underline{SD}$ psychological (4.16 $\pm$1.84) impact of RA is slightly greater than that of the physical (3.80 $\pm$1.16) impact, both scores indicating moderate health status. In the following section, the results of the sample's mean $\pm\underline{SD}$ scores on the 9 subscales of the AIMS are reported, to provide an indication of the health status of the subjects in this study.

Table 3: Baseline pain scores along the three pain measures.

Measure	Mean score	±SD
McGill Pain Questionnaire (0= low, 1.0= high index)		
PRIS (Sensory Dimension)	0.38	0.21
PRIA (Affective Dimension)	0.21	0.22
PRIE (Evaluative Dimension)	0.43	0.36
PRIMT (Miscellaneous Dimension)	0.26	0.22
PRIT (Total Pain Rating Index)	0.32	0.18
Verbal Analogue Scale (0= no pain, 5= unbearable)		
PPI (Present Pain Intensity)	2.25	0.77
Ritchie Articular Index (0 to 78)		
RITCHIE	13.26	6.17
Arthritis Impact Measurement Scale (0= good health, 10= poor)		
PAIN	6.50	2.02

Figure 1.6: Distribution of Total Pain Rating Index scores Sample at Pre-intervention



2.2.3 Disease Activity

The AIMS has measures of functional status which provide an index of physical well-being (see Table 4). These scores indicate the extent to which arthritis has had a relatively major impact on the sample's physical (6.37 ±2.33) and social (4.13 ±1.9) activities, and dexterity (5.5 ±2.97). These findings support the high pain scores reported above, indicating the extent to which pain is an important index of the health status of the RA patient.

The moderately high scores on the Ritchie Articular Index (13.26 ±6.17) substantiated the above assessment of physical well-being. This index indicates that the degree of pain and inflammation in the actual joints is widespread but mild, providing a more clinical estimate of disease activity for this sample. Figure 1.7 plots the distribution of scores on this variable.

Mean ±SD pre-intervention serum measures of ethrocyte sedimentation rate (37.27 ±28.72) provide a further index of the level of disease activity, with the sample's average ESR result being well above the range for healthy women (range = 0 to 7). Figure 1.8 plots the distribution of scores on this variable.

2.2.4 Lymphocyte Proliferation Rate

Lymphocyte proliferation rates (see Table 1) to the three mitogens: Phytohaemmaglutinin (stimulating mainly T-cells), Pokeweed (B-cell stimulation), and Conconavilin-A (mainly T- but also B-cell stimulation); for the sample were well within the normal range of response (i.e., greater than ±75% of the control serum). Only one subject (see Table 14.2) displayed abnormally low response rates for PHA (29.6%), PWM (27.8%), and CON-A (10%).

These results provide a measure of the immune status of the subjects, indicating healthy levels of T- and B-lymphocyte function. The percentage score for each subject was obtained by dividing the subject's cell count by that of the control factor. Because of the clinical (i.e., qualitative) nature of the assay, it is not possible to report on any other characteristics of these haematological results. Furthermore, as noted in Tables 14.1 and 14.2, it is important to recognize that different control serum (the serum decays over time, gradually loosing its ability to respond to mitogenic stimulation) was used in the assays for the two groups at all assessment points. Logistics prevented all subjects (i.e., both groups) to be assessed on the variable on the same day. The percentage score is thus relative to the control serum for that group on a particular day. The results in Table 14.2 confirm this change in the control serum over time for the two baseline assessment points, where a drop in all subjects' immune response is more a function of the deteriorating control than a decline in their immune systems ability to respond to these mitogens.

2.3 Baseline Correlational Analysis: Testing Hypothesis 1

To test the hypothesis that there is a significant relationship between the psychological and pain, disease activity and immunological variables, a correlation matrix (Pearsons correlation, SAS) was constructed to assess the strength of these relationships. Key variables (n=18) were chosen in this analysis, and the large matrix was broken

Table 4: Breakdown of samples scores on the 9 scales of the AIMS at pre-intervention.[1]

AIMS	Mean Score	±SD
Mobility (MOB)	2.50	3.16
Physical Activity (PACT)	6.38	2.33
Dexterity (DEX)	5.50	2.97
Household Activity (HACT)	2.60	1.34
Social Activity (SACT)	4.13	1.90
Activities of Daily Living (ACDL)	1.72	1.88
Pain	6.50	2.02
Depression (DEPR)	3.88	1.97
Anxiety (ANXI)	4.43	2.06

[1]Each scale has a range of 0-10, where 0=good health status, and 10=poor health status.

Figure 1.7: Distribution of Ethrocyte Sedimentation Rate Sample at Pre-intervention

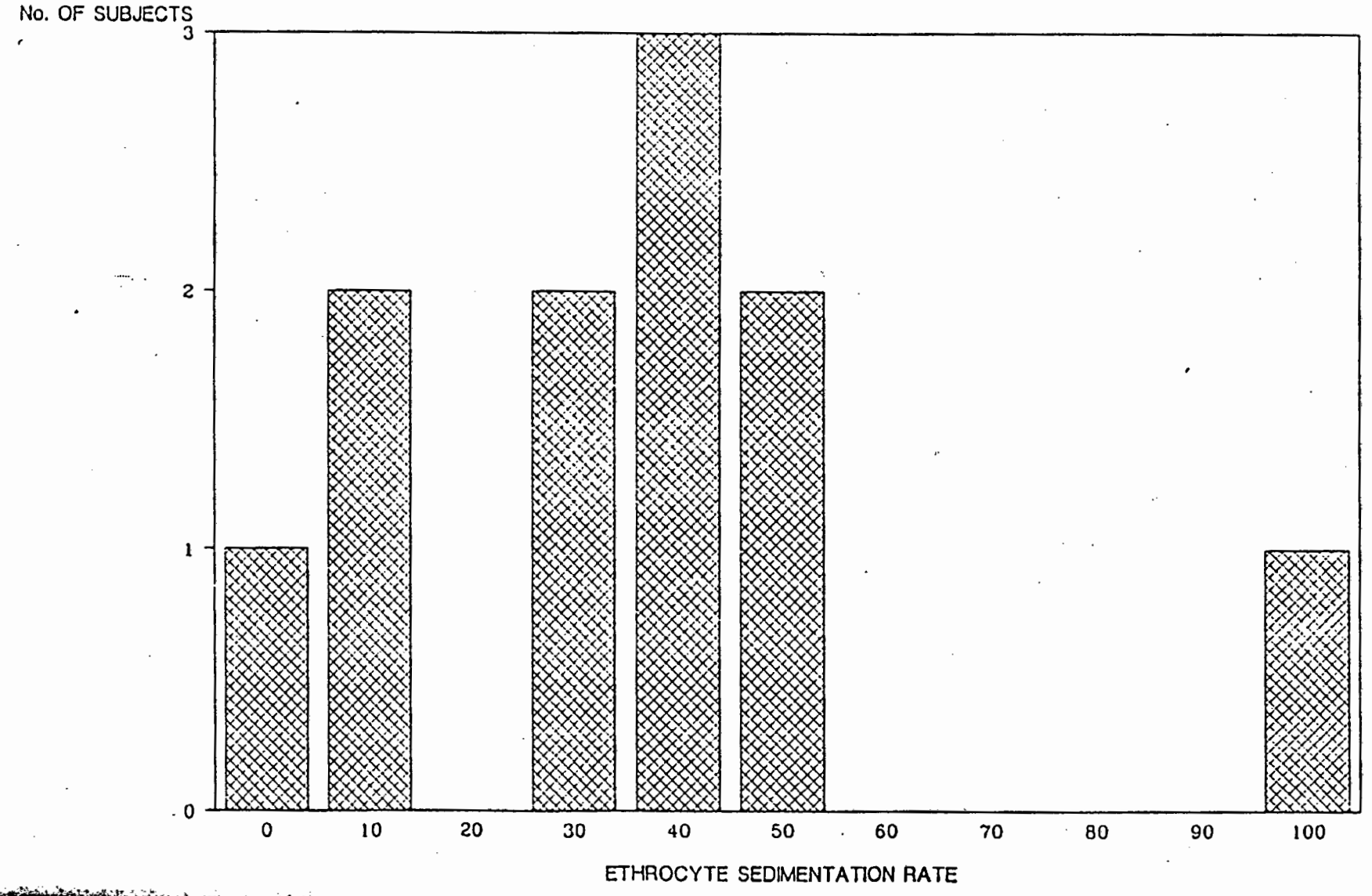


Figure 1.8: Distribution of Ritchie Articular Index scores Sample at Pre-intervention

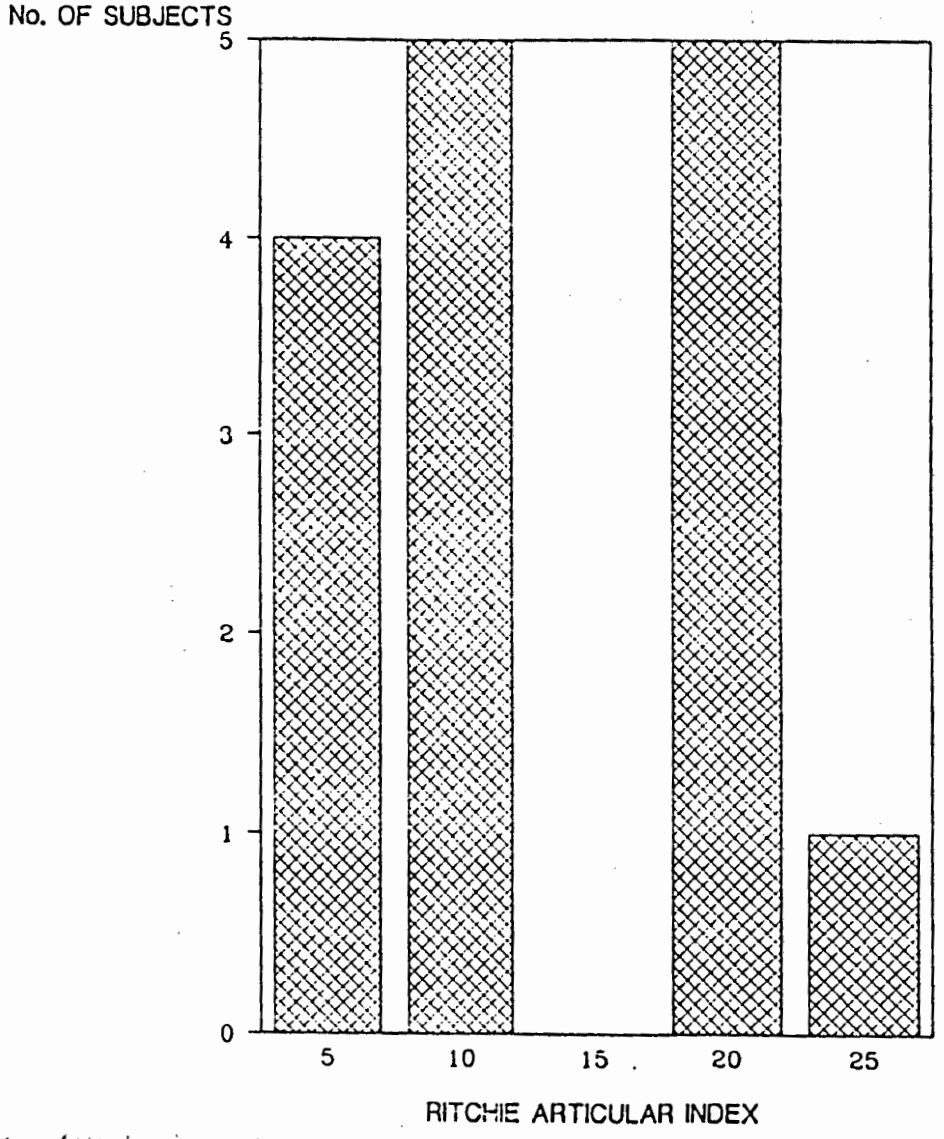


Table 14.1: Mean Baseline and Post-intervention lymphocyte proliferation response rates for treatment and control groups.[1]

	Proliferation Response Rate			
	Baseline		Post-Intervention	
Variable	Mean (%)	±SD	Mean (%)	±SD
Treatment:				
BPHA	204.7%	75.06	714.53	998.87
BPWM	210.6%	65.09	1384.31	1965.70
BCON	403.8%	197.54	229.71	203.60
Control:				
BPHA	126.8	23.79	1407.97	1152.40
BPWM	210.2	30.67	3295.51	3084.64
BCON	585.7	936.18	107.40	60.67

[1]Expressed as a percentage of the control serum used for that assay

BPHA= Baseline Phytohaemaglutinin percentage response rate, BPWM= Baseline Pokeweed Mitogen percentage response rate, BCON= Baseline Conconavilin A percentage response rate.

Table 14.2: Baseline and Post-Intervention treatment effects: Individual Lymphocyte Proliferation Rates

	BASELINE								
	FIRST			SECOND			POST		
Subject	PHA (%)	PWM (%)	CON-A (%)	PHA (%)	PWM (%)	CON-A (%)	PHA (%)	PWM (%)	CON-A (%)
Treatment group									
101	316.0	203.5	202.5	100.6	416.0	1109.4	1372.0	5401.8	63.5*
102	29.6*	27.8*	10.0*	60.4*	447.1	64.9*	293.9	345.5	123.3
103	368.6	320.1	807.9	150.2	117.0	151.8	200.0	372.8	253.0
106	390.4	341.4	982.6	121.0	187.7	140.9	3149.8	4073.1	96.1
107	272.1	145.6	378.5	172.3	158.7	390.4	131.7	153.4	120.7
110	310.8	224.3	636.8	71.0*	54.0*	88.5	169.7	169.5	264.4
111	606.3	331.7	1123.1	-----	-----	-----	272.3	223.5	178.5
112	269.9	148.0	221.3	233.2	157.0	467.4	126.8	334.9	738.2
Control group									
201	116.8	198.5	109.0	70.7	150.1	187.0	2610.2	5285.0	208.8
202	-----	-----	-----	-----	-----	-----	144.3	173.8	38.0*
206	165.8	235.4	84.5	100.8	280.8	322.8	3207.4	9040.0	100.6
207	119.1	121.9	35.6	106.8	299.2	196.7	287.4	344.6	86.3
209	177.0	178.4	138.9	102.2	215.2	264.0	1795.3	4667.2	87.8
210	-----	-----	-----	43.3*	97.5	90.4	162.1	243.8	45.6*
211	174.4	187.9	407.6	134.8	234.3	4110.5	1649.1	3314.2	184.7

* Value ≤75% (where 75% = cut-off to distinguish between normal and abnormal proliferative response rate

1 It is important to note that these results cannot be compared with each over time, as each percentage is relative only to the control it was compared to on a particular day. As such, the test is purely qualitative, and is used for clinical purposes.

2 Rate is expressed as a percentage of the particular control on that day.

PHA= Phytohaemaglutinin percentage response rate, PWM= Pokeweed Mitogen percentage response rate, CON=

down into smaller parts to facilitate analysis (see Tables 5 to 10, wherein correlation coefficients and significance levels are reported).

Two important limitations of such an analysis should be mentioned. Firstly, strong correlations (at the $p \leq 0.05$, $p \leq 0.01$ and $p \leq 0.001$ significance levels) between these variables cannot be taken as proof of a causal (viz., linear) relationship. Secondly, the significance of any one relationship should be viewed with caution, considering the small sample size, and the large number of variables included in the matrix (i.e., the number of correlations involved). It is important that the researcher does not "shop for significance".

2.3.1 Mood Disturbance and Pain

As was predicted, significant interrelationships were obtained between the subjects' mood disturbance (TMD) and their total pain rating index (PRIT) ($r(16) = 0.63$, $p \leq 0.05$), and present pain intensity (PPI) ($r(16) = 0.59$, $p \leq 0.05$). Mood disturbance was not shown to be significantly correlated to actual pain (i.e., Ritchie Articular Index) felt in individual joints, nor to scores on the AIMS Pain subscale. The former indicating a possible dysjuncture between pain experience on a purely organic/physical level and the pain experienced on a personal/psychological level. Table 5 provides a breakdown of the correlation between these pain and mood variables.

Total mood disturbance (TMD) correlated most strongly with the affective (PRIA) ($r(16) = 0.58$, $p \leq 0.05$) and miscellaneous (PRIMT) ($r(16) = 0.82$, $p \leq 0.001$) dimensions of the McGill measure. The strongest correlations in this mood--pain relationship being with the Depression, Anger and Fatigue mood factors. An important finding is the particularly strong correlations between the mood factors and the Miscellaneous dimension of the McGill measure. The descriptors in this pain dimension (e.g., "spreading", "squeezing", "agonizing") seem more able to tap the patient's feelings of tension, depression, anger and fatigue in response to the pain experienced, than the descriptors in the Affective pain dimension (.e.g., "exhausting", "sickening", "cruel").

The Anger mood factor correlated strongly with scores on all but one (i.e., Ritchie Articular Index) of the pain measures, demonstrating the extent to which high levels of reported pain are closely linked to feelings of anger, frustration and hostility. The significant correlation with the Sensory pain dimension ($r(16) = 0.52$, $p \leq 0.05$) demonstrates the extent to which heightened feelings of frustration and anger may possibly aggravate or influence the individual's sensory experience of the painful joints. It is also possible that heightened sensory experience of pain contributes to such moods.

Unexpected nonsignificant correlations were found between mood disturbance and the Sensory pain dimension. According to the tension--pain cycle of experience, it was predicted that mood disturbance was related to aspects of this pain dimension. In this case, the Anger-PRIS relationship seems to be based on the frustration experienced by these subjects as a result of their pain and the concomitant increase in muscle tension and heightened awareness of the sensory input from the inflamed joints.

Table.5: Correlation matrix of all Mood and Pain variables

PAIN VARIABLES	MOOD VARIABLES						
	TEN1	DEP1	ANG1	FAT1	VIG1	CON1	TMD_1
PRIS1	0.36	0.47	0.52	0.12	0.15	0.14	0.37
p=	0.18	0.07	0.04*	0.65	0.57	0.59	0.16
PRIE1	0.34	0.30	0.18	0.44	0.02	0.31	0.41
p=	0.20	0.25	0.49	0.09	0.94	0.25	0.12
PRIA1	0.38	0.66	0.61	0.52	-0.10	0.06	0.58
p=	0.15	0.01**	0.01**	0.04*	0.72	0.82	0.02*
PRIMT1	0.75	0.79	0.64	0.61	-0.07	0.39	0.82
p=	0.001**	0.0003***	0.008***	0.01**	0.80	0.14	0.0001***
PRIT1	0.55	0.68	0.65	0.41	0.06	0.26	0.63
p=	0.03*	0.004**	0.01**	0.12	0.82	0.34	0.01**
PPI1	0.53	0.46	0.65	0.40	-0.16	0.18	0.59
p=	0.03*	0.07	0.007**	0.12	0.56	0.51	0.02*
PAIN_1	0.31	0.35	0.62	0.33	-0.17	-0.09	0.41
p=	0.24	0.19	0.01**	0.22	0.53	0.74	0.11
RITCHIE1	0.13	0.10	0.06	0.04	-0.01	0.10	0.12
p=	0.65	0.74	0.83	0.90	0.97	0.72	0.68

*p≤0.05 **p≤0.01 ***p≤0.001
PRIS=Sensory Dimension; PRIE=Evaluative Dimension; PRIA=Afective Dimension;
PRIMT=Miscellaneous; PRIT=Total Pain Rating Index; PPI=Present Pain Intensity;
PAIN_1=Subscale of AIMS; RITCHIE1=Articular Index.
TEN1=Tension; DEP1=Depression; ANG1=Anger; FAT1=Fatigue; VIG1=Vigor; CON1=Confusion;
TMD_1=Total Mood Disturbance.

Confusion is notably not related to these pain dimensions, which was expected. This provides further confirmation and validation of the significance of the above mentioned correlations. The feelings associated with this mood factor should not necessarily affect the subjects pain ratings nor be affected by elevated pain levels (although extreme pain could influence concentration levels). The Vigor mood factor was not significantly correlated to any of the pain dimensions or measures.

2.3.2 Mood Disturbance and Other Variables

Total mood disturbance (TMD) was not found to be significantly related to the other physiologic variables (see Table 10 below), where low correlations with ethrocyte sedimentation rate (ESR) ($r(10) = -.31$) and physical health status (HSCPHY) ($r(16) \doteq 0.43$) were found. The nonsignificant inverse correlation with ESR is contrary to expectations. It was expected that elevated mood disturbance would be related to increased ESR values.

Consistent with expectations, Mood disturbance (TMD) was significantly related to the other measures of psychological adjustment. Strong correlations were found with stress totals (SEITOT) ($r(16) = 0.62$, $p \leq 0.05$), psychological health status (HSCPSY) ($r(16) = 0.71$, $p \leq 0.01$), and perceived coping efficacy (PCE) ($r(16) = 0.49$, $p \leq 0.05$). No significant correlations were found between mood factors and health locus of control scores.

2.3.3 Perceived Coping Efficacy and Key Variables

Perceived coping efficacy was not found to relate significantly (with the exception of mood disturbance) with any of the other key psychological, pain and immunological variables. Table 6 provides a summary of these correlations. These results did not concur with predictions which anticipated significant inverse correlations with mood disturbance, stress, "powerful others externality", "chance externality", pain variables, ethrocyte sedimentation rate and lymphocyte proliferation response. Being a mediator in the coping process between stressful events and the outcome of coping efforts, it was thought that this variable would be significantly related to these other key variables. It was predicted that the greater the subject's appraisal of their ability to cope effectively with stressful events (including those related to particularly painful episodes), the more improved their psychological adjustment and lower levels of reported pain.

The non-significant results presented in Table 6 are contrary to the above argument, despite the moderate number of inverse correlations recorded. This analysis shows that the level of pain that the subject experienced, did not influence her perception of her efficacy in coping with stressful events (see Table 2), despite the fact that some of these events included painful episodes or "arthritis flare-up". The significant correlation between TMD and SATSLF ($r(16) = 0.52$, $p \leq 0.05$) was particularly unexpected, where the subject's greater satisfaction with her coping efforts was related to higher levels of total mood disturbance.

Table.6: Correlation matrix of Coping Efficacy and key variables.

KEY VARIABLES	PERCEIVED COPING EFFICACY			
	SATSLF_1	SATOTH_1	BLFFTR_1	PCETOT_1
TMD_1	0.52	0.37	0.38	0.49
p=	0.04*	0.15	0.14	0.06
SEITOT_1	0.30	0.16	0.28	0.28
p=	0.26	0.56	0.29	0.29
IHLC_1	0.10	-0.35	-0.10	-0.12
p=	0.72	0.19	0.70	0.65
PHLC_1	0.15	-0.23	-0.04	-0.03
p=	0.58	0.39	0.89	0.90
CHLC_1	0.32	0.04	0.16	0.21
p=	0.22	0.88	0.56	0.45
PRIT1	0.39	0.30	0.07	0.28
p=	0.14	0.26	0.81	0.29
PPI1	-0.04	0.12	-0.12	-0.02
p=	0.89	0.67	0.65	0.93
PAIN_1	0.13	0.17	-0.06	0.09
p=	0.63	0.52	0.82	0.75
RITCHIE1	-0.09	0.02	-0.19	-0.10
p=	0.75	0.94	0.52	0.73
ESR1	-0.26	0.41	0.13	0.12
p=	0.47	0.24	0.71	0.75

*$p \leq 0.05$
SATSLF_1=Satisfaction with self; SATOTH_1=Satisfaction with others; BLFFTR_1=Belief in future coping efficacy; PCETOT_1=Total perceived coping efficacy; SEITOT_1=Total stress experience; IHLC_1=Internal locus of control; PHLC_1=Powerful others locus of control; CHLC_1=Chance locus of control.
(See Table.5 for explanation of other variables)

2.3.4 Stress Levels and Key Variables

Contrary to predictions, stress levels as measured by the Stress Evaluation Inventory (the frequency and intensity of stressful events) were only found to be significantly related to mood disturbance and total pain rating index scores. Table 7 provides a breakdown of the correlations between the three stress factors and the other key variables. Family and personal stress levels were strongly related to Total Mood Disturbance ($r(16) = 0.64$, $p \leq 0.01$; and $r(16) = 0.78$, $p \leq 0.001$ respectively) and Total Pain Rating Index scores ($r(16) = 0.66$, $p \leq 0.001$; and $r(16) = 0.71$, $p \leq 0.001$ respectively). As the majority of the sample did not work, the results for the Job stress factor should not be considered meaningful.

The particularly low correlations between stress factors and the Ritchie Articular Index (RAI) indicate that there is no relationship between the degree of joint inflammation and the frequency of stressful experience. The inverse correlations between these stress factors and ethrocyte sedimentation rate (ESR), although non-significant, are difficult to interpret, as they are contrary to the prediction that elevated stress experience would be result in greater joint inflammation. These results could possibly be due to the relatively low levels of stress reported by this sample, and the inadequacies of the instrument, rather than the absence of possible relationships between these variables.

2.3.5 Health Locus of Control and Key Variables

The sample's Health Locus of Control scores on the three dimensions were not found to be significantly related to any of the key variables (see Table 8). These findings were contrary to the prediction that locus of control was an important psychological variable in RA and the management of stress and pain. It was expected that greater "internality" would be inversely related to mood disturbance and pain variables. Only the near significant correlation with ethrocyte sedimentation rate ($r(16) = -.57$, $p \leq 0.1$) was consistent with this prediction that an internal locus of control would mediate the effect of stress and pain on the inflammatory response. A high degree of internality would indicate greater compliance with treatment regimens, rest, safe-activity as well as a personal involvement in arthritis care. These behaviours play a role in determining physical health status and concomitant disease activity.

The low correlations with the rest of the variables indicates that the subjects' health locus of control was not strongly related to psychological adjustment, physical health, or pain experience. The fact that this sample did not differ greatly from expected norms, perhaps contributed to these nonsignificant correlations.

2.3.6 Health Status and Key Variables

Health status as measured by the AIMS was found to be significantly related to mood disturbance and scores on the pain measures. Table 9 provides a summary of the correlations of psychological and physical (i.e., functional) health status with key variables. Psychological health status was particularly significantly correlated with Total Mood Disturbance ($r(16) = 0.71$, $p \leq 0.01$), with the Tension, Depression, and Fatigue mood factors contributing to

Table.7: Correlation matrix of Stress factors and Key variables

KEY VARIABLES	STRESS EVALUATION INVENTORY			
	JOBSTR_1	FAMSTR_1	PERSTR_1	SEITOT_1
TMD_1	0.11	0.64	0.78	0.62
p=	0.68	0.01**	0.0004***	0.01**
PCETOT_1	0.07	0.27	0.36	0.28
p=	0.80	0.30	0.17	0.29
IHLC_1	0.43	0.30	0.15	0.38
p=	0.09	0.26	0.59	0.14
PHLC_1	-0.13	0.13	0.21	0.08
p=	0.62	0.62	0.43	0.78
CHLC_1	-0.56	-0.15	0.24	0.24
p=	0.02*	0.58	0.37	0.37
PRIT1	0.11	0.66	0.71	0.60
p=	0.68	0.006**	0.002**	0.01**
PPI1	0.21	0.51	0.42	0.48
p=	0.44	0.04*	0.11	0.06
PAIN_1	-0.04	0.17	0.37	0.19
p=	0.89	0.53	0.15	0.48
RITCHIE1	-0.05	0.09	0.06	0.04
p=	0.86	0.75	0.83	0.88
ESR1	-0.24	-0.29	-0.31	-0.33
p=	0.50	0.42	0.39	0.35

*p≤0.05 **p≤0.01 ***p≤0.001
JOBSTR_1=Job stress; FAMSTR_1=Family stress; PERSTR_1=Personal stres
(See Table.5 for explanation of other variables)

Table. 8: Correlation matrix of Health locus of control factors and key variables.

KEY VARIABLES	MULTIDIMENSIONAL HEALTH LOCUS OF CONTROL		
	IHLC_1	PHLC_1	CHLC_1
TMD_1	-0.06	0.20	0.09
p=	0.82	0.45	0.73
PCETOT_1	-0.12	-0.03	0.21
p=	0.65	0.90	0.45
SEITOT_1	0.38	0.08	-0.24
p=	0.14	0.78	0.37
PRIT1	0.11	0.16	0.09
p=	0.68	0.52	0.73
PPI1	-0.01	0.15	-0.16
p=	0.98	0.57	0.56
PAIN_1	-0.01	0.09	0.03
p=	0.96	0.73	0.90
RITCHIE1	0.05	-0.30	-0.30
p=	0.85	0.30	0.30
ESR1	-0.57	-0.02	0.05
p=	0.09	0.95	0.89

(See Table.5 for explanation of variables)

Table. 9: Correlation matrix of Health status, Inflammation measures and key variables

KEY VARIABLES	HEALTH STATUS		INFLAMMATION	
	HSCPSY_1	HSCPHY_1	ESR1	RITCHIE1
TEN	0.69	0.09	-0.33	0.13
p=	0.003**	0.74	0.35	0.65
DEP1	0.63	0.58	0.10	0.10
p=	0.01**	0.02*	0.79	0.74
ANG1	0.48	0.42	-0.03	0.06
p=	0.06	0.10	0.95	0.83
FAT1	0.49	0.50	-0.23	0.04
p=	0.05*	0.05*	0.53	0.90
VIG1	-0.13	0.03	0.49	-0.01
p=	0.64	0.90	0.15	0.97
FAT1	0.49	0.50	-0.23	0.04
p=	0.05*	0.05*	0.53	0.90
CON1	0.38	0.17	-0.42	0.10
p=	0.15	0.53	0.22	0.72
TMD_1	0.71	0.43	-0.31	0.124
p=	0.002**	0.10	0.39	0.68
PCETOT_1	0.47	-0.10	0.12	-0.10
p=	0.06	0.70	0.75	0.73
SEITOT_1	0.52	0.13	-0.33	0.04
p=	0.04*	0.62	0.35	0.88
IHLC_1	0.11	-0.17	-0.57	0.05
p=	0.67	0.52	0.09	0.85
PHLC_1	0.22	0.02	-0.02	-0.30
p=	0.40	0.95	0.95	0.30
CHLC_1	0.17	-0.14	0.05	-0.30
p=	0.52	0.62	0.89	0.30

Continued...

*p≤0.05 **p≤0.01 ***p≤0.001
HSCPHY_1=Physical health status; HSCPSY_1=Psychological health status; ESR1=Ethrocyte sedimentation rate.
(See Table.5 for explanation of other variables)

Table. 9: Continued...

KEY VARIABLES	HEALTH STATUS		INFLAMMATION	
	HSCPSY_1	HSCPHY_1	ESR1	RITCHIE1
PRIT1	0.88	0.34	-0.23	-0.001
p=	0.0001***	0.20	0.52	0.10
PPI1	0.60	0.23	-0.07	0.01
p=	0.01**	0.39	0.84	0.98
PAIN_1	0.61	0.13	-0.37	-0.45
p=	0.01**	0.62	0.29	0.10
HSCPHY_1	0.23	-----	0.03	0.41
p=	0.39		0.94	0.14
HSCPSY_1	-----	0.23	-0.45	-0.08
p=		0.39	-0.19	-0.78
RITCHIE1	-0.08	0.41	0.52	-----
p=	0.78	0.14	0.12	
ESR1	-0.45	0.03	-----	0.52
p=	0.19	0.94		0.12

*p≤0.05 **p≤0.01 ***p≤0.001
HSCPHY_1=Physical health status; HSCPSY_1=Psychological health status; ESR1=Ethrocyte sedimentation rate.
(See Table.5 for explanation of other variables)

this high correlation. These results providing an independent measure of the validity of these two instruments (POMS and AIMS) in RA populations.

The non-significant correlation between Physical health status and Total Mood Disturbance indicates the extent to which the subject's functional status (in terms of ability to perform various activities) was not related to their overall mood state. Depression ($r(16) = 0.58$, $p \leq 0.05$) and Fatigue ($r(16) = 0.50$, $p \leq 0.05$) were the only mood factors significantly related to functional status (HSCPHY).

The significant correlation between Psychological health status (HSCPSY) with Total Pain Rating Index (PRIT) ($r(16) = 0.88$, $p \leq 0.001$), confirmed the extent to which pain levels are the most powerful predictors of the psychological impact of RA. This finding was supported by the non-significant correlations (see Table 10) between Physical health status and the pain variables. This nonsignificant finding is extremely important, indicating the extent to which pain, in this sample, was more associated with the psychological status of RA patients than with their functional status (i.e., mobility, dexterity, and activity). It is thus possible that pain is more a function of psychological distress than physical impairment. Neither Psychological nor Physical health status correlated significantly with any of the other key variables.

2.3.7 Disease Activity and Key Variables

No significant correlations were found to exist between ethrocyte sedimentation rate (ESR) and the other key variables. This was also true for the Ritchie Articular Index. Table 9 provides a summary of the correlations between these variables and other key variables.

2.3.8 Lymphocyte Proliferation Rate and Key Variables

Each subject's lymphocyte proliferation rate was expressed as a percentage of the control used for that particular assay. A baseline proliferation rate was obtained by calculating the mean proliferation rate for each subject over the first two assessment points (one week difference). These values were entered into the correlation matrix. As the sample was well within the normal or healthy range of response (with the exception of 3 subjects) to all three mitogens (see Table 1), these results should be viewed with caution. The central problem is that one cannot describe such values as better or worse, as there is a cut-off point (70% of the control factor) whereby one makes a decision regarding the immunocompetence of the individual. Furthermore, the small number of observations in terms of these lymphocyte values ($n=12$) may have obscured possible relationships between these and the other key variables.

Table 10 provides a summary of the correlations between the baseline lymphocyte response rates and other key variables. Very few significant correlations were found, with most of the correlations being particularly low or meaningless. It was predicted that good psychological adjustment would be significantly related to healthy proliferative response rates to mitogenic stimulation.

Table. 10: Correlation matrix of Lymphocyte proliferation data and key variables

KEY VARIABLES	BASELINE LYMPHOCYTE PROLIFERATION RATE[1]		
	BPHA	BPWM	BCON
TEN1	-0.25	0.18	0.21
p=	0.46	0.59	0.54
DEP1	-0.42	-0.24	-0.04
p=	0.20	0.47	0.90
ANG1	-0.34	0.04	-0.26
p=	0.31	0.91	0.43
FAT1	0.08	-0.34	-0.23
p=	0.81	0.31	0.50
VIG1	-0.90	0.18	-0.18
p=	0.0002**	0.59	0.59
CON1	0.04	-0.57	0.03
p=	0.90	0.07	0.93
TMD_1	-0.06	-0.28	-0.05
p=	0.87	0.40	0.88
PCETOT_1	0.04	0.30	-0.11
	0.90	0.37	0.75
SEITOT_1	-0.45	0.01	-0.32
p=	0.17	0.98	0.34
IHLC_1	-0.19	-0.01	-0.39
p=	0.57	0.97	0.24
PHLC_1	-0.44	-0.53	-0.07
p=	0.20	0.09	0.83
CHLC_1	-0.20	-0.18	-0.10
p=	0.56	0.59	0.76
HSCPSY_1	-0.16	-0.21	0.06
p=	0.64	0.55	0.87
HSCPHY_1	-0.21	-0.28	-0.18
p=	0.54	0.41	0.59

Continued...

***p≤0.01

[1]Expressed as a percentage of the control serum used in the assay.

Table. 10: Continued...

KEY VARIABLES	BASELINE LYMPHOCYTE PROLIFERATION RATE[1]		
	BPHA	BPWM	BCON
PAIN_1	0.30	-0.43	-0.11
p=	0.37	0.18	0.75
ESR1	0.04	0.60	0.19
p=	0.93	0.12	0.66
RITCHIE1	-0.12	0.67	0.22
p=	0.71	0.02*	0.49
PRIT1	-0.43	-0.22	-0.19
p=	0.19	0.53	0.57
PPI1	-0.04	-0.30	0.23
p=	0.90	0.37	0.50

**$p \leq 0.05$
[1]expressed as a percentage of the control serum used in the assay.

Lymphocyte proliferation to Phytohaemmaglutinin (PHA) did not correlate well with other key variables. The significant inverse correlation between PHA and the Vigor mood factor ($r(11) = -.90$, $p \leq 0.001$), was not consistent with such a prediction. As the Vigor mood factor is a measure of the level of energy or ebullience that the individual has, it is not clear why the negative correlation is so significant. Although non-significant, the correlations between PHA and the: Tension, Depression, and Anger mood factors; Stress totals; Powerful others locus of control; and Total Pain Rating Index are more consistent with expected findings (see Table 10). Negative affect, stressful experience and an over reliance on significant others (i.e., helplessness) have all been shown to dysregulate immune function, particularly T-Lymphocyte activity.

Lymphocyte proliferation to Pokeweed Mitogen (PWM) was only marginally related to mood disturbance, health locus of control, Articular Index and pain. The near-significant correlations between PWM and the Confusion mood factor ($r(11) = -.57$, $p=0.07$); and Powerful others locus of control ($r(11) = -.53$, $p=0.09$), were consistent with expected findings. The significant correlation between the Articular Index and PWM ($r(12) = 0.67$, $p \leq 0.05$), indicates the extent to which the degree of joint inflammation is related to B lymphocyte activity.

No meaningful correlations were found between Conconavilin A (CON) and other key variables. The extremely high mean $\pm SD$ percentage scores for the two groups on this variable, may have obscured possible significant relationships.

2.4 Summary of Results Testing First Hypothesis

The results reported above provide little evidence that there is a significant relationship between psychological adjustment and disease activity, health status, and immune function variables. There is evidence the central role of psychological adjustment in the pain experience, given the significant correlations between the variables operationalized as constituting psychological adjustment (particularly mood disturbance), and health status (psychological and functional disability) and pain.

It is not possible to determine the direction of causality in the significant correlations observed. By examining patterns of change in the variables over time in the context of the above results (see section 3), the role of psychological adjustment in RA may possibly be clarified.

3. Group Comparisons: Pre-, Mid-, and Post-Intervention

3.1 Differences at Pre-Intervention

To determine whether or not significant differences existed between the experimental and control groups prior to the intervention, a series of t-tests (parametric) and Wilcoxon Rank Sum tests (non-parametric) for independent groups were conducted for the key variables. Table 11.1 and Table 11.2 summarize the results of these analyses.

-able 11.1: Group comparisons on key variables at Pre-intervention[1]

	Treatment Group		Control Group			
'ariable	Mean	±SD	Mean	±SD	t	p
Personal						
Age	48.14	8.34	51.38	10.64	-0.65	0.53
Onset	1984.36	3.50	1982.33	3.91	1.13	0.28
POMS						
TMD	205.86	30.12	196.22	46.09	0.46	0.65
MHLC						
IHLC	24.14	6.07	24.56	5.41	-0.14	0.89
PHLC	20.29	9.62	20.89	8.54	-0.13	0.90
CHLC	21.57	3.21	21.22	8.03	0.12	0.91
AIMS						
PAIN	7.71	1.60	5.56	1.84	2.45	0.03*
HSCPSY	4.48	1.86	3.91	1.90	0.61	0.55
HSCPHY	4.17	1.66	3.52	0.49	1.01	0.35

*$p \leq 0.05$

[1]The 2-sample t-test (parametric).

TMD=Total Mood Disturbance, MHLC=Multidimensional Health Locus of Control, IHLC=Internality, PHLC=Powerful others externality, CHLC=Chan externality, AIMS=Arthritis Impact Measurement Scales, HSCPSY=Psychological Health Status, HSCPHY=Physical Health Status.

ble 11.2: Group comparisons on key variables at Pre-intervention[1]

	Treatment Group		Control Group			
riable	Mean	±SD	Mean	±SD	z	p
SEI						
Total	10.29	4.50	14.33	10.16	-0.69	0.49
PCE						
Self	10.86	3.72	11.44	4.25	-0.16	0.87
Others	10.29	4.57	10.89	2.32	-0.80	0.42
Future	9.71	4.27	10.89	3.66	-0.59	0.56
Total	30.86	11.85	32.22	8.41	-0.90	0.37
MPQ						
PRIT	0.34	0.20	0.31	0.17	0.00	1.0
PRIS	0.40	0.25	0.37	0.17	-0.27	0.79
PRIE	0.40	0.37	0.44	0.38	-0.16	0.87
PRIA	0.27	0.27	0.16	0.18	0.81	0.42
PRIMT	0.27	0.17	0.25	0.26	0.37	0.71
PPI	2.57	0.53	2.00	0.87	1.31	0.19
RAI	13.63	7.84	12.86	4.10	0.17	0.86
ESR	51.00	51.39	32.13	17.68	0.31	0.76
LYMPHOCYTES						
BPHA	204.72%	75.06	126.84%	23.79	-1.95	0.05*
BPWM	210.59%	65.09	210.17%	30.67	0.00	1.00
BCON	403.78%	197.54	585.66%	936.18	-0.97	0.33

*p≤0.05
[1]The Wilcoxon Rank Sum test (non-parametric).
SEI=Stress Evaluation Inventory; PCE=Perceived Coping Efficacy;
MPQ=McGill Pain Questionnaire; PRIT=Total Pain Rating Index;
PRIS=Sensory; PRIE=Evaluative; PRIA= Affective; PRIMT= Miscellaneous;
PPI=Present Pain Intensity, RAI=Ritchie Articular Index;
ESR=ethrocyte sedimenation rate; BPHA=Baseline Phytohaemaglutinin;
PWM=Baseline Pokeweed Mitogen; CON=Baseline Conconavilin A)

Of the 25 variables selected, significant group differences were found on only the Pain sub-scale of the AIMS (t = 2.45, $p \leq 0.05$), and on the Phytohaemmaglutinin (z = -1.95, $p \leq 0.05$) lymphocyte proliferation measure. In both cases, the treatment group had higher mean $\pm$SD scores than the control group (7.71 $\pm$1.60 and 204.72 $\pm$75.06, for Pain and PHA respectively). As the results on the more comprehensive McGill Pain Questionnaire did not concur with the findings on the Pain subscale, this difference in pain scores was not considered a serious confounding factor. The difference in PHA stimulation rate was also not considered significant, as both groups remained well within the normal or healthy range of lymphocyte response (+70% of the control's response) to the mitogen.

The particularly high probability values (i.e., $p \geq 0.05$) for the remainder of the key variables provided sufficient evidence to conclude that the two groups were similar on all the important variables prior to the initiation of the intervention programme. This was further supported by the two groups having similar perceptions of how they were coping with their arthritis as rated on a 10-point rating scale (where 0 = extremely poor coping and 10 = extremely well). Mean $\pm$SD group ratings for the treatment and control groups were 5.5 $\pm$2.65 and 5.13 $\pm$2.67 respectively, indicating moderate coping abilities.

The matched-pairs (matched on onset date and coping rating) and random allocation to condition procedures, successfully controlled for random error variation.

3.2 Treatment effects: Testing Hypothesis 2

3.2.1 Change over Time

To test the second hypothesis that:

(i) the SIPMT intervention will improve psychological adjustment, and pain management; and

(ii) such improvement would result in changes in, disease activity, and immune function;

change scores were calculated for the key variables. The following calculations were used to obtain these scores:

MID - PRE = Change 1

POST - PRE = Change 2

These change scores were expressed as a positive or negative value (the direction) of change as a function of time. Only mood disturbance, perceived coping efficacy, pain rating index, ethrocyte sedimentation rate and Ritchie Articular Index had two change scores (as these variables were assessed mid-intervention).

A series of univariate tests (paired-difference t-test and Wilcoxon signed rank test for paired groups) were conducted on these change scores to determine inter- and intra-group differences over time. Tables 12.1 and 12.2 (pre- to mid-assessment), and Tables 13.1 and 13.2 (pre- to post-assessment) provide summaries of the results of these analyses.

Table 12.1: Pre- to Mid-intervention treatment effects[1]

	Intra Group changes[2]								
	Treatment Group			Control Group			Inter Group changes		
Variable	Mean±SD	t	p	Mean±SD	t	p	Mean±SD	t	p
Mood Disturbance									
TEN	0.00±16.7	0.00	1.0	0.43± 2.4	0.46	0.66	0.21±11.5	0.07	0.95
ANG	1.29±13.8	0.25	0.81	1.57± 7.5	0.56	0.60	1.43±10.6	0.5	0.62
DEP	0.43±14.1	0.08	0.94	2.86± 9.2	0.83	0.44	1.64±11.5	0.53	0.60
VIG	0.57± 6.7	0.22	0.83	-3.14± 7.8	-1.07	0.33	-1.29± 7.3	-0.66	0.52
FAT	-2.43±11.2	-0.57	0.59	0.43± 3.5	0.33	0.75	-1.00± 8.1	-0.46	0.65
CON	0.71±10.7	0.18	0.87	0.00± 6.9	0	1.0	0.36± 8.6	0.15	0.88
TMD	-0.57±61.3	-0.02	0.98	8.42±22.4	1.00	0.36	3.93±44.6	0.33	0.75

[1]The paired t-test (parametric) for paired groups.
[2]Change expressed as Mean±SD difference (mid minus pre) with the sign (+ or -) indicating direction.
(See Table.5 for explanation of variables)

Table 12.2: Pre- to Mid-intervention treatment effects[1]

	Intra Group changes[2]								
	Treatment Group			Control Group			Inter Group changes		
Variable	Mean±SD	Sgn Rank	p	Mean±SD	Sgn Rank	p	Mean±SD	Sgn Rank	p
Coping Efficacy									
Satslf	0.14± 4.7	0.0	1.0	-0.29± 3.3	-1.0	0.92	-0.07± 3.9	-2.5	0.89
Satoth	-0.29± 5.6	3.0	0.67	0.14± 3.4	0.5	1.0	-0.07± 4.5	6.0	0.70
Blfftr	-1.29± 4.1	-5.0	0.45	1.00± 3.7	4.0	0.55	-0.14± 3.9	-2.5	0.90
Pcetot	-1.43±10.7	-1.5	0.83	0.86± 8.8	2.5	0.62	-0.29± 9.5	0.0	1.0
Pain Index									
Prit	0.43±15.6	-4.5	0.50	-1.57± 7.2	-2.0	0.75	-0.57±11.7	-12.0	0.42
Pris	-0.71± 8.6	-1.5	0.83	0.71± 5.5	2.0	0.80	0.00± 7.0	-2.0	0.92
Pria	0.86± 3.3	0.5	1.0	-0.71± 2.3	-2.5	0.59	0.07± 2.9	-3.5	0.76
Prie	0.00± 0.6	0.0	1.0	0.14± 0.7	1.0	0.77	0.07± 0.6	1.5	0.77
Primt	0.14± 4.5	-4.5	0.40	-1.57± 4.1	-5.0	0.34	-0.71± 4.2	-12.0	0.37
PPI	-0.86± 0.7	-7.5	0.05*	0.00± 0.8	0.0	1.0	-0.43± 0.9	-13.5	0.10
Inflammation									
ESR	-3.00± 8.5	-1.0	0.79	1.63±13.8	1.5	0.83	0.36±12.3	0.5	1.0
Ritchie	0.25± 6.6	0.5	1.0	-0.57± 2.4	-3.5	0.52	-0.13± 4.9	-5.0	0.72

*$p \leq 0.05$
[1]The Wilcoxon Signed Rank test (non-parametric) for paired groups.
[2]Change expressed as Mean±SD difference (mid minus post) with the sign (+ or -) indicating direction and tested at the $p \leq 0.05$ significance level for change from zero.
(See Table.11.2 for explanation of variables)

Figure 2.1: Change by Mid-Intervention: TMD
Comparison between Groups

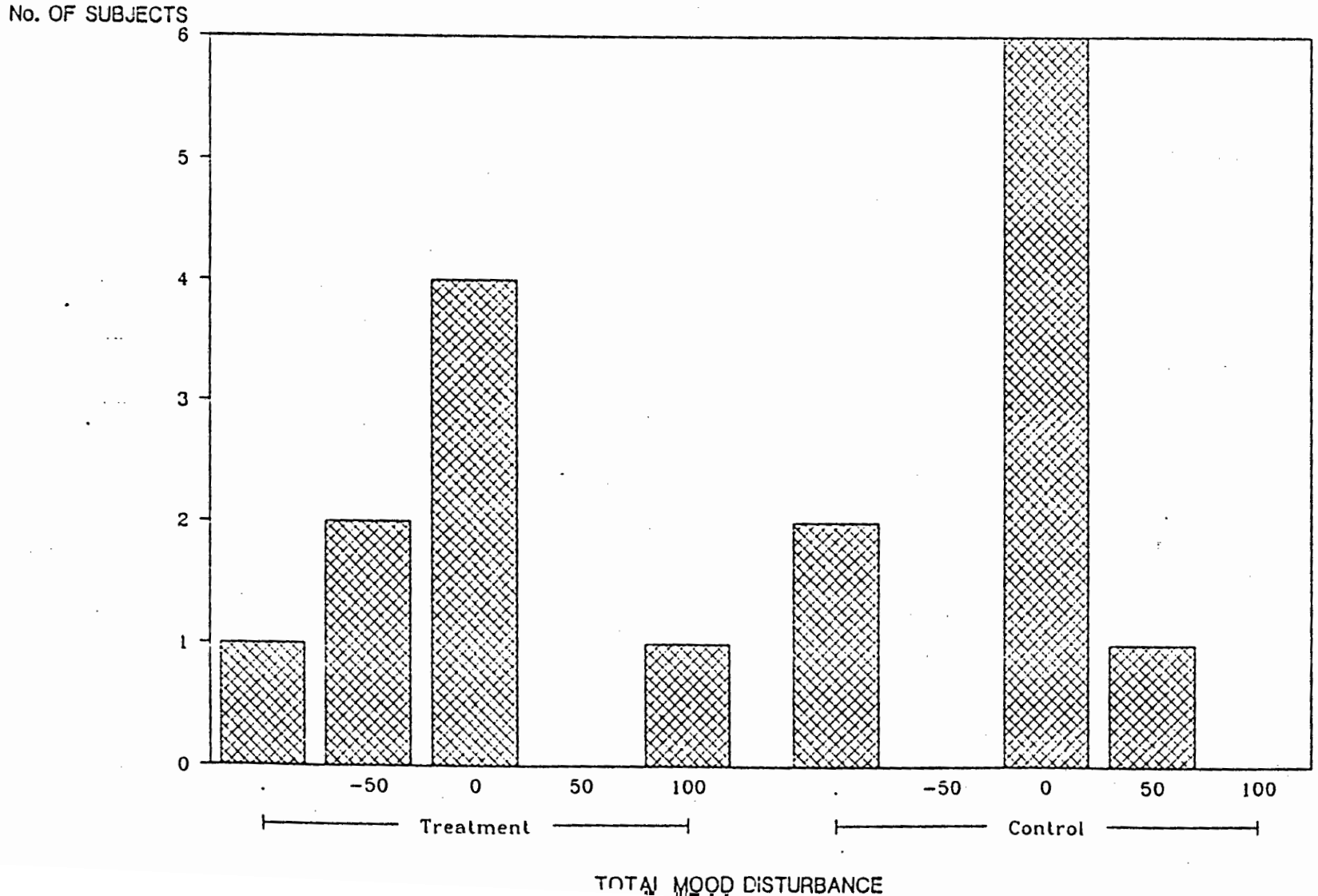


Figure 2.2: Change by Mid-Intervention: Tension Comparison between Groups

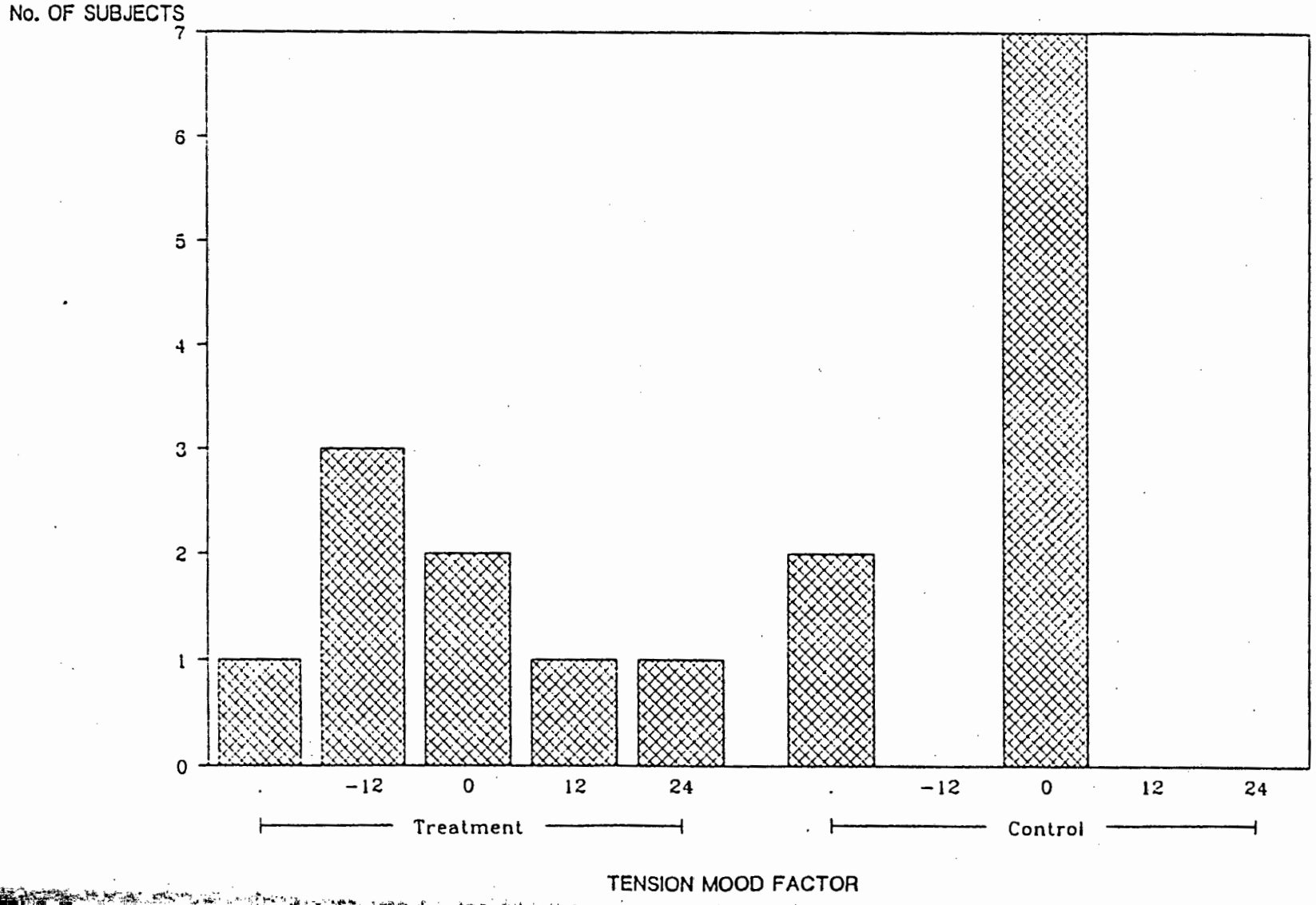


Figure 2.3: Change by Mid-Intervention: Fatigue Comparison between Groups

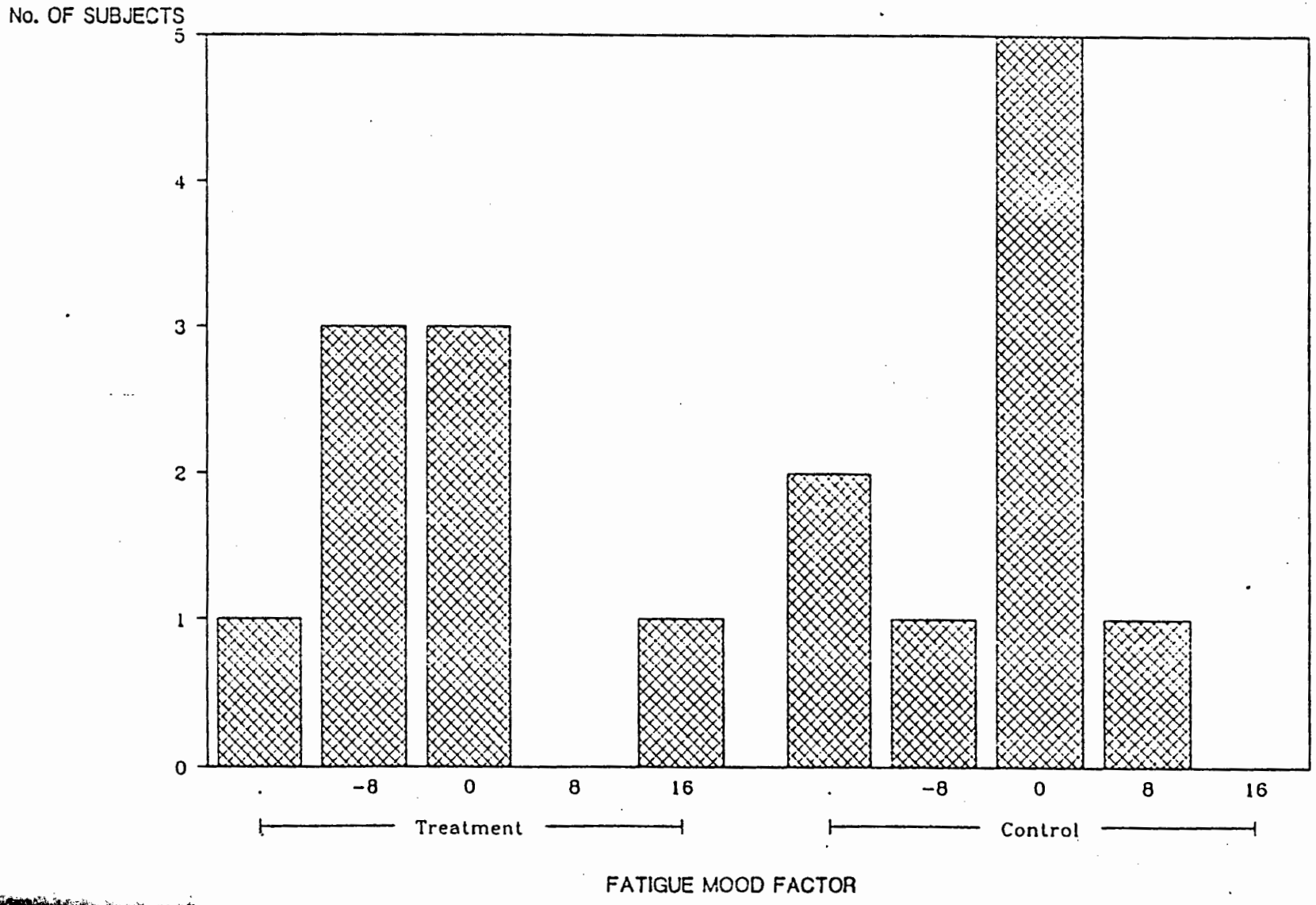
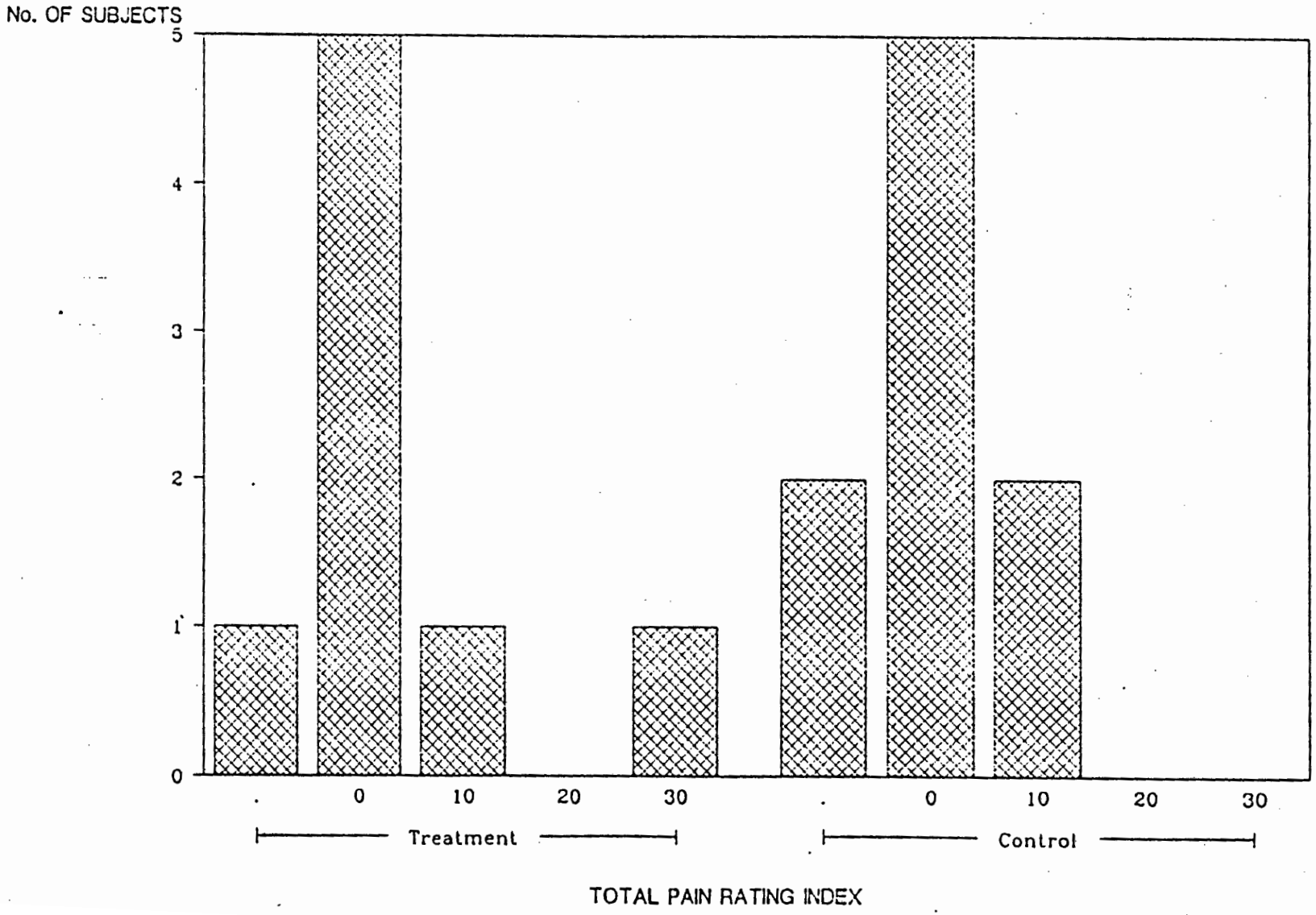


Figure 2.4: Change by Mid-Intervention: Total Pain Rating Index Comparison between Groups



3.2.2 Change over Time: Pre- to Mid-Intervention

No significant inter-group changes had occurred by mid intervention along the mood disturbance (TMD), coping efficacy (PCE), Pain Rating index (PRIT), Ritchie Articular Index, or ethrocyte sedimentation rate variables (see Tables 12.1 and 12.2). The SIMPT intervention produced significant reductions only in the treatment group's Present Pain Intensity ratings (Sgn Rank = -7.5, $p \leq 0.05$), yet this change did not reach significance (Sgn Rank = -13.5, $p \leq 0.1$) at an inter-group level of analysis. No significant changes occurred in the control group along these variables.

Figure 2.1 shows the distribution of TMD change scores for the two groups on a single axis, where the marked deterioration (an increase of 100 in TMD score) of one subject in the treatment group, and the lack of data for three subjects, may have biased the mean intra group changes in mood disturbance. Considering the small sample size, it is possible that the problem of missing data for three subjects (two in the control group) may have affected the distribution of change scores in the sample considerably.

Similar patterns can be observed in the 6 mood factors, where the treatment group showed a moderately larger intra group variance in their mean $\pm$SD changes along these variables (see Table 12.1) than the controls. This was particularly so for the Tension (0.00 $\pm$16.7 compared to 0.43 $\pm$2.4) and Fatigue (-2.43 $\pm$11.2 compared to 0.43 $\pm$3.5) mood factors. Figures 2.2 and 2.3 provide an indication of the differences between the two groups in the distribution of change scores along these two variables.

The intra group changes in scores on the Total Pain Rating Index (PRIT) also demonstrates the extent to which the existence of an extreme score in the treatment group, and the lack of data for two subjects in the control group possibly affected the significance of possible changes. Figure 2.4 shows the distribution of these scores.

The results of intra and inter group changes in ethrocyte sedimentation rate (ESR) must be viewed with caution, considering the lack of data for five subjects in the treatment group.

The non-significant intra and inter group changes observed by mid-intervention, do not concur with the prediction that the SIMPT programme would improve psychological adjustment (in terms of mood disturbance and perceived coping efficacy), and pain management (i.e., lowered pain rating index). It is thus not possible to draw any conclusions at this point regarding the possible effects any improvement in psychological adjustment would have had on the disease activity (Ritchie Index and ESR) variables.

3.2.3 Change over Time: Pre- to Post-Intervention

No significant inter group differences were found in the change scores for all key variables over the full duration of the treatment programme (see Tables 13.1 and 13.2). Based on these findings, it would be necessary to reject the alternative hypothesis and accept of the null hypothesis that the group mean change scores are equivalent. These

Table 13.1: Pre- to post-intervention treatment effects[1]

	Intra Group changes[2]								
	Treatment Group			Control Group			Inter Group changes		
Variable	Mean±SD	t	p	Mean±SD	t	p	Mean±SD	t	p
Mood Disturbance									
TEN	2.29±13.6	-0.44	0.67	1.50± 7.4	0.57	0.59	1.87±10.3	0.70	0.50
ANG	1.14±13.6	0.44	0.83	6.12± 5.7	3.04	0.02*	3.80±10.1	1.46	0.17
DEP	2.29±11.8	0.51	0.63	0.88± 6.9	0.36	0.73	1.53± 9.2	0.65	0.58
VIG	-3.29± 7.7	-1.13	0.30	-4.63±11.2	-1.16	0.28	-4.00± 9.4	-1.64	0.12
FAT	1.43±11.4	0.33	0.75	0.38± 9.2	0.11	0.91	0.87± 9.9	0.34	0.74
CON	2.29±14.2	0.43	0.68	-2.63± 4.2	-1.76	0.12	-0.33±10.1	-0.13	0.90
TMD	12.72±55.9	0.61	0.57	10.88±30.9	1.00	0.35	11.73±42.6	1.07	0.30
Health locus of control									
IHLC	3.00±6.2	1.28	0.20	-1.25±2.3	-1.53	0.17	0.73±4.9	0.58	0.57
PHLC	-0.71±5.3	-0.36	0.73	1.00±4.0	0.71	0.50	0.20±4.6	0.17	0.87
CHLC	1.86±6.8	0.72	0.50	0.25±3.8	0.18	0.86	1.00±5.3	0.73	0.48
Health Status									
Psych	-0.00±2.5	-0.00	1.0	-0.00±1.6	-1.21	1.0	-0.00±2.0	-2.85	1.0
Physic	0.00±1.5	0.01	0.99	-0.01±0.5	-0.03	0.98	-0.00±1.0	-0.00	1.0
Pain Exp.									
PAIN	0.14±1.6	0.24	0.82	0.13±1.0	0.34	0.74	0.13±1.3	0.41	0.69

*$p \leq 0.05$

[1]The paired t-test (parametric) for paired groups.

[2]Change expressed as Mean±SD difference (post minus pre) with the sign (+ or -) indicating direction

Table 13.2: Pre- to post-intervention treatment effects[1]

	Intra Group changes[2]								
	Treatment Group			Control Group			Inter Group changes		
Variable	Mean±SD	Sgn Rank	p	Mean±SD	Sgn Rank	p	Mean±SD	Sgn Rank	p
Stress Experience									
Seitot	0.00± 3.9	-0.5	1.0	1.38± 4.0	6.0	0.35	0.73±3.9	11.0	0.46
Coping Efficacy									
Satslf	-1.14± 5.1	-3.0	0.67	-0.71± 2.9	-4.0	0.55	-0.93± 4.0	-12.5	0.45
Satoth	0.14± 3.1	1.0	0.93	1.00± 2.6	6.0	0.35	0.60± 2.8	14.5	0.38
Blfftr	-1.14± 3.8	-3.5	0.53	0.25± 5.0	1.5	0.89	-0.40± 4.4	-4.5	0.80
Pcetot	-2.14±10.9	-4.5	0.50	2.00± 7.5	1.5	0.83	-0.07± 9.2	-1.5	0.94
Pain Index									
Prit	8.86±17.6	6.5	0.31	-1.00±10.7	-3.0	0.72	3.60±14.7	9.0	0.63
Pris	0.29± 8.9	-2.5	0.73	0.50± 8.0	2.0	0.83	0.40± 8.1	-1.5	0.95
Pria	2.57± 4.7	4.5	0.28	-0.38± 2.3	-1.5	0.78	1.00± 3.8	7.0	0.50
Prie	1.29± 2.5	3.5	0.27	-0.88± 2.1	-6.5	0.30	0.13± 2.5	0.5	1.0
Primt	4.57± 6.6	8.5	0.09**	-0.13± 2.8	0.5	1.0	2.07± 5.4	0.5	0.15
PPI	0.57± 1.1	3.5	0.27	0.38± 0.5	3.0	0.15	0.47± 0.8	11.0	0.06
Inflammation									
ESR	-1.33± 3.8	-1.5	0.59	-5.75±15.4	-7.0	0.27	-4.55±13.2	-13.5	0.18
Ritchie	-1.25± 5.8	-3.5	0.67	-1.43± 6.2	-2.0	0.80	-1.33± 5.8	-10.0	0.59

*p≤0.1
[1]The Wilcoxon Signed Rank test (non-parametric) for paired groups.
[2]Change expressed as Mean±SD difference (post minus pre) with the sign (+ or -) indicating direction.
(See Table.11.2 for explanation of variables)

Figure 3.1: Change by Post-Intervention: Anger Comparison between Groups

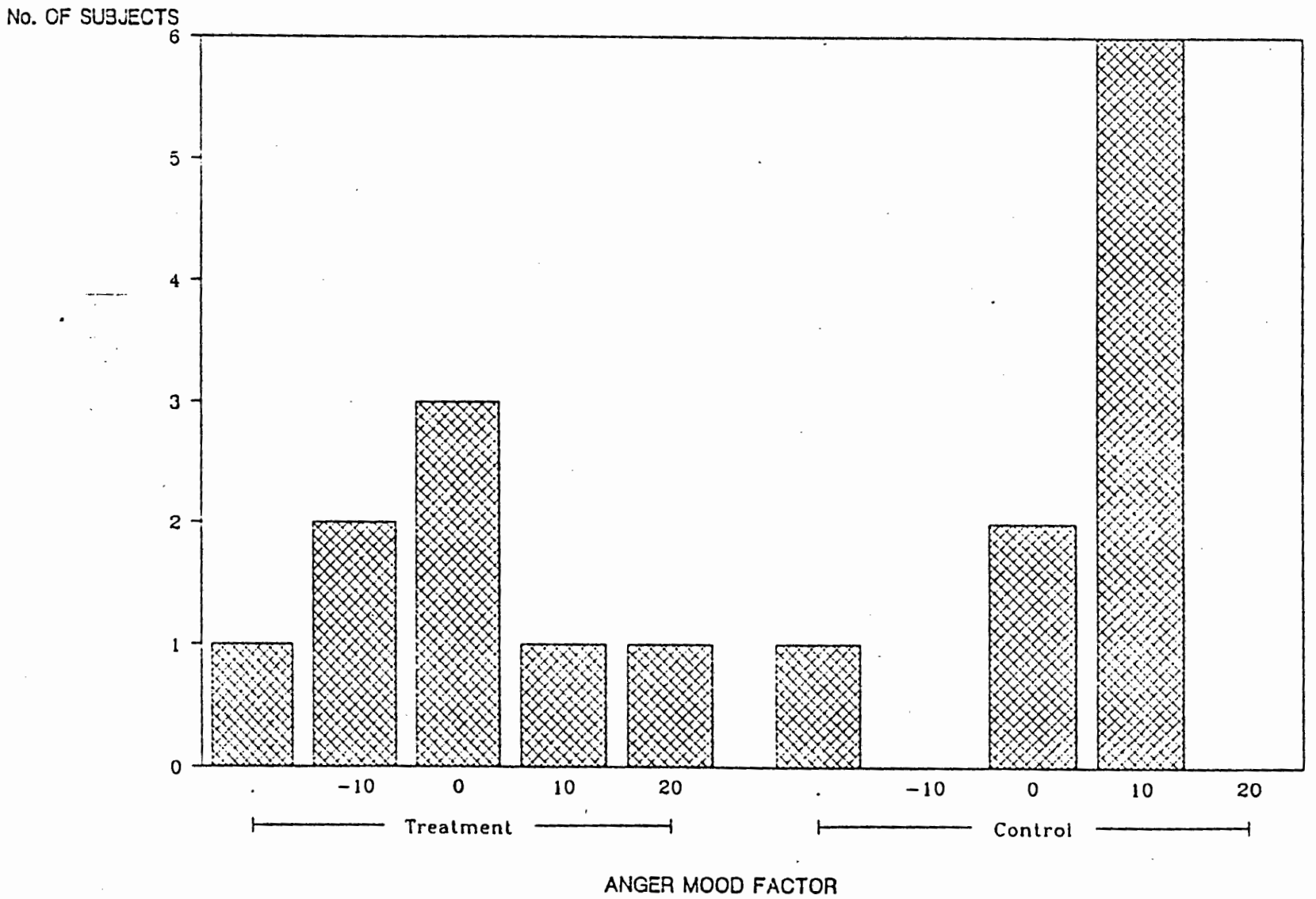


Figure 3.2: Change by Post-Intervention: TMD
Comparison between Groups

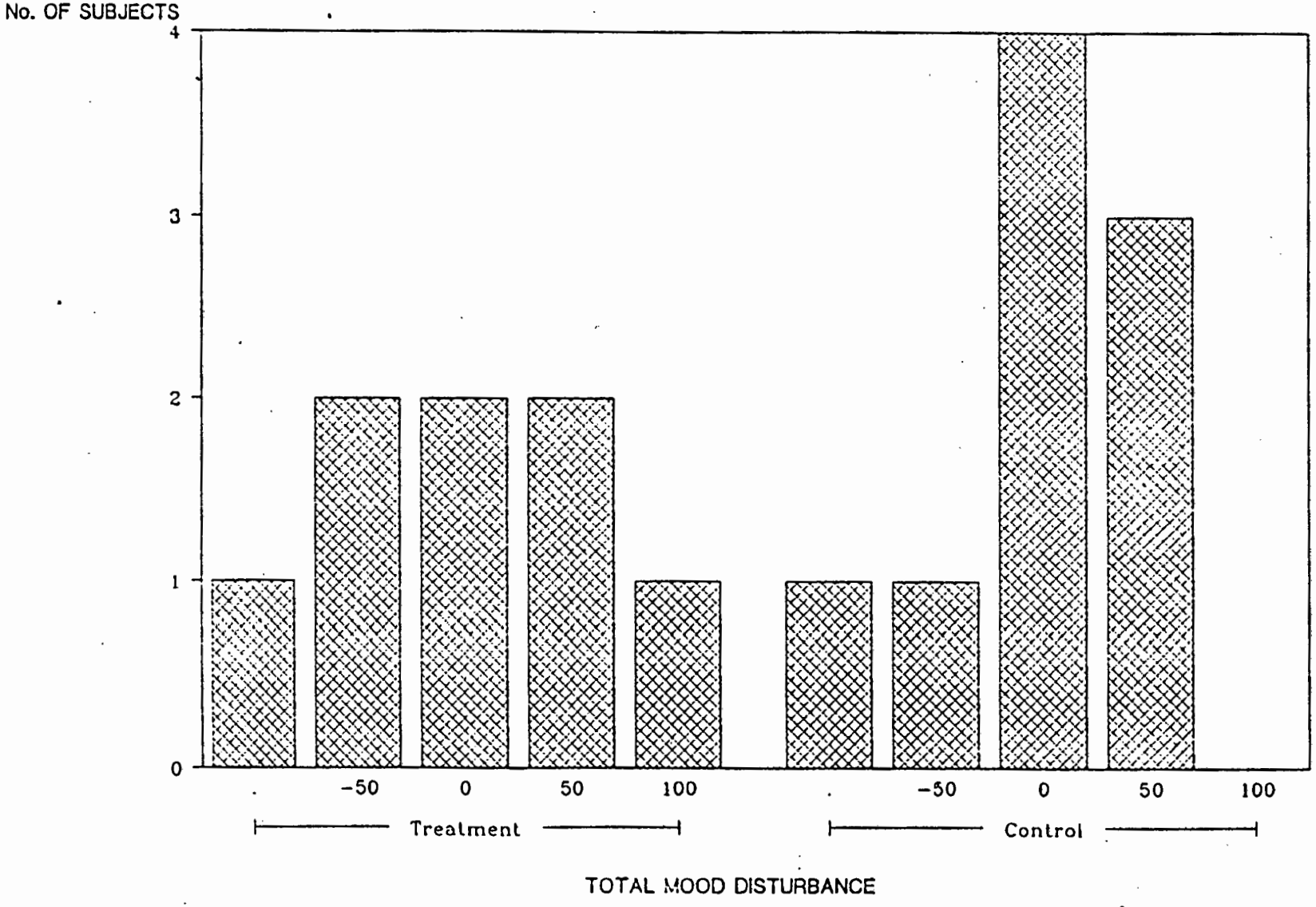


Figure 3.3: Change by Post-Intervention: IHLC
Comparison between Groups

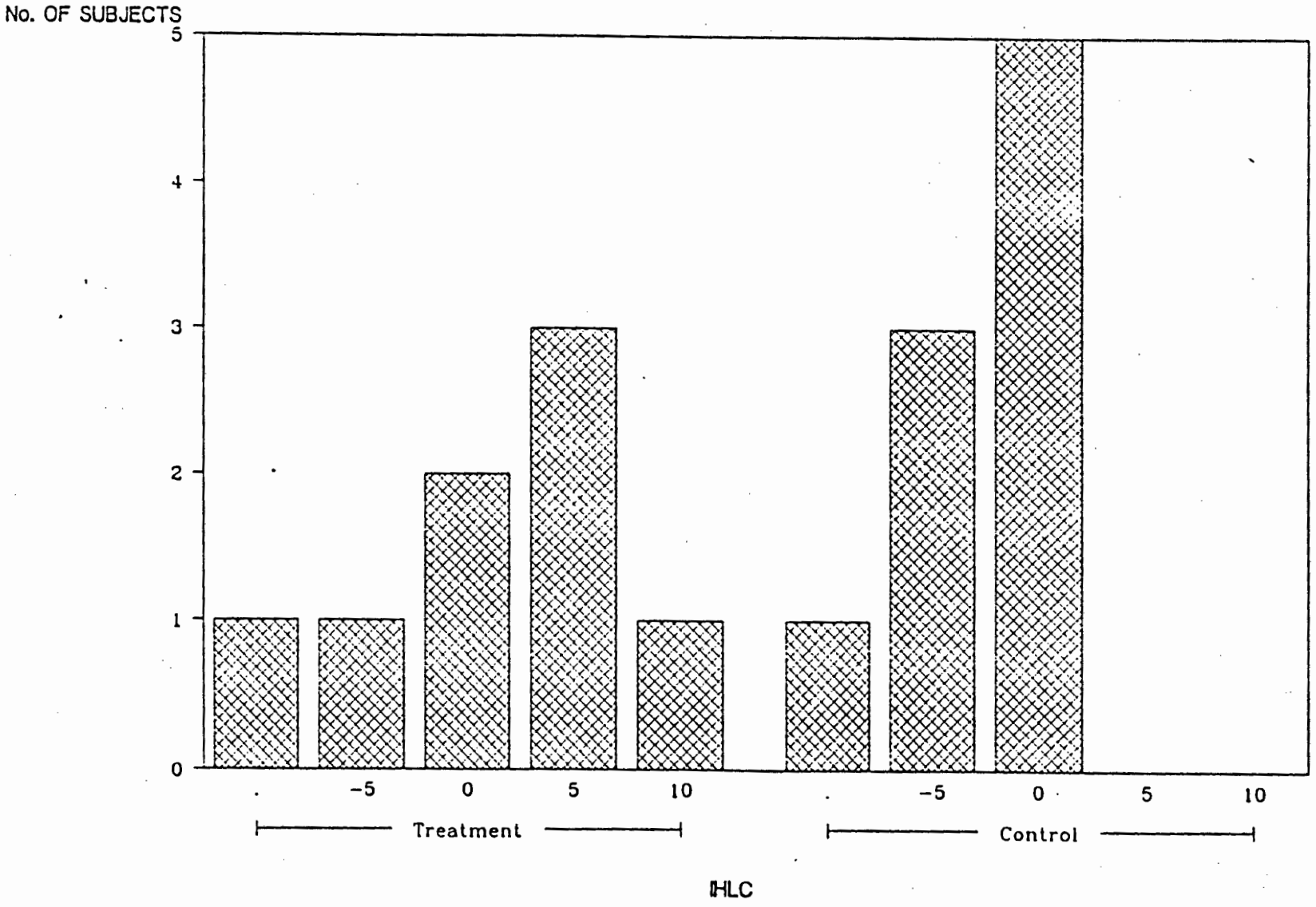


Figure 3.4: Change by Post-Intervention: Perceived Coping Efficacy Comparison between Groups

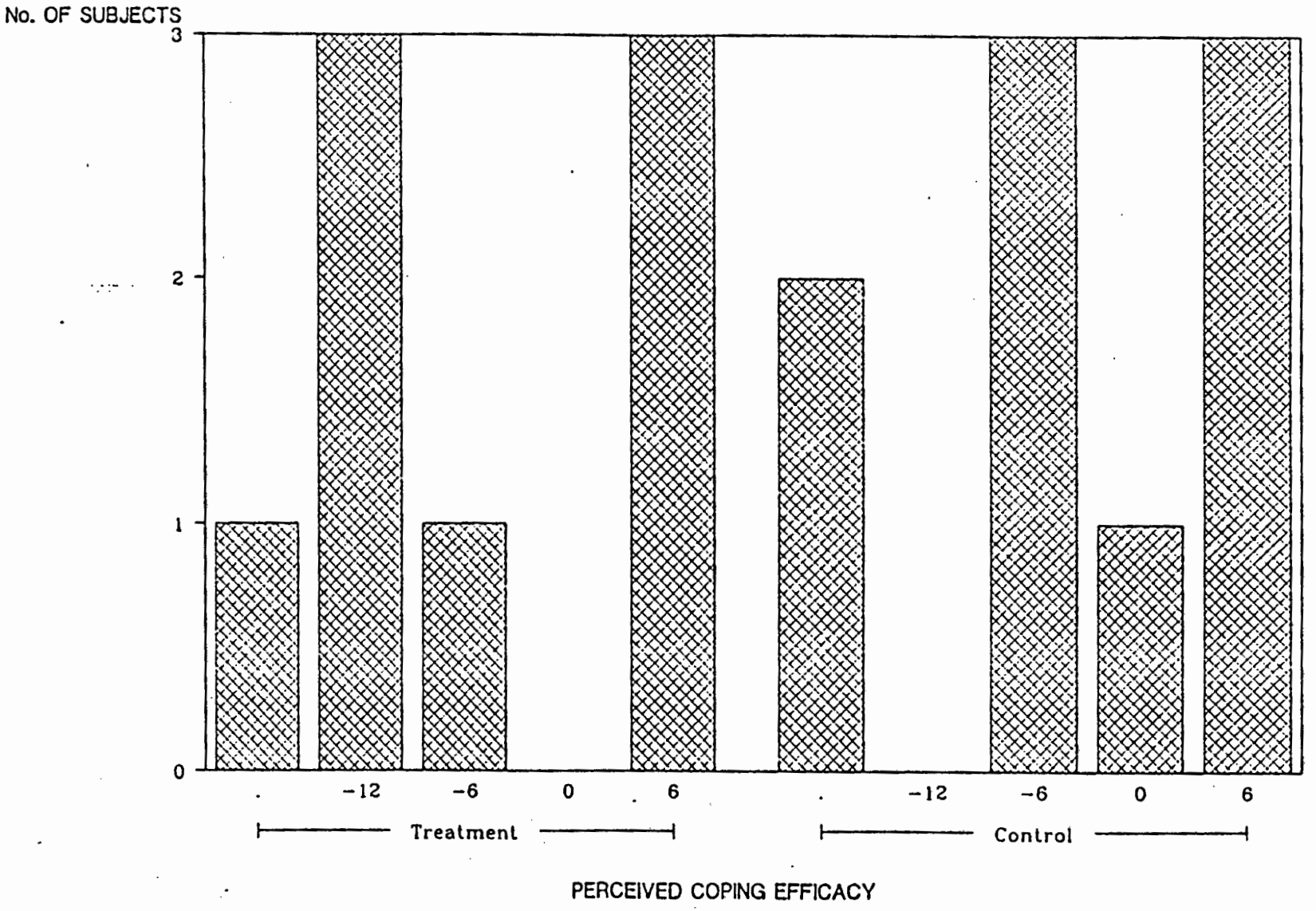


Figure 3.5: Change by Post-Intervention: Total Pain Rating Index Comparison between Groups

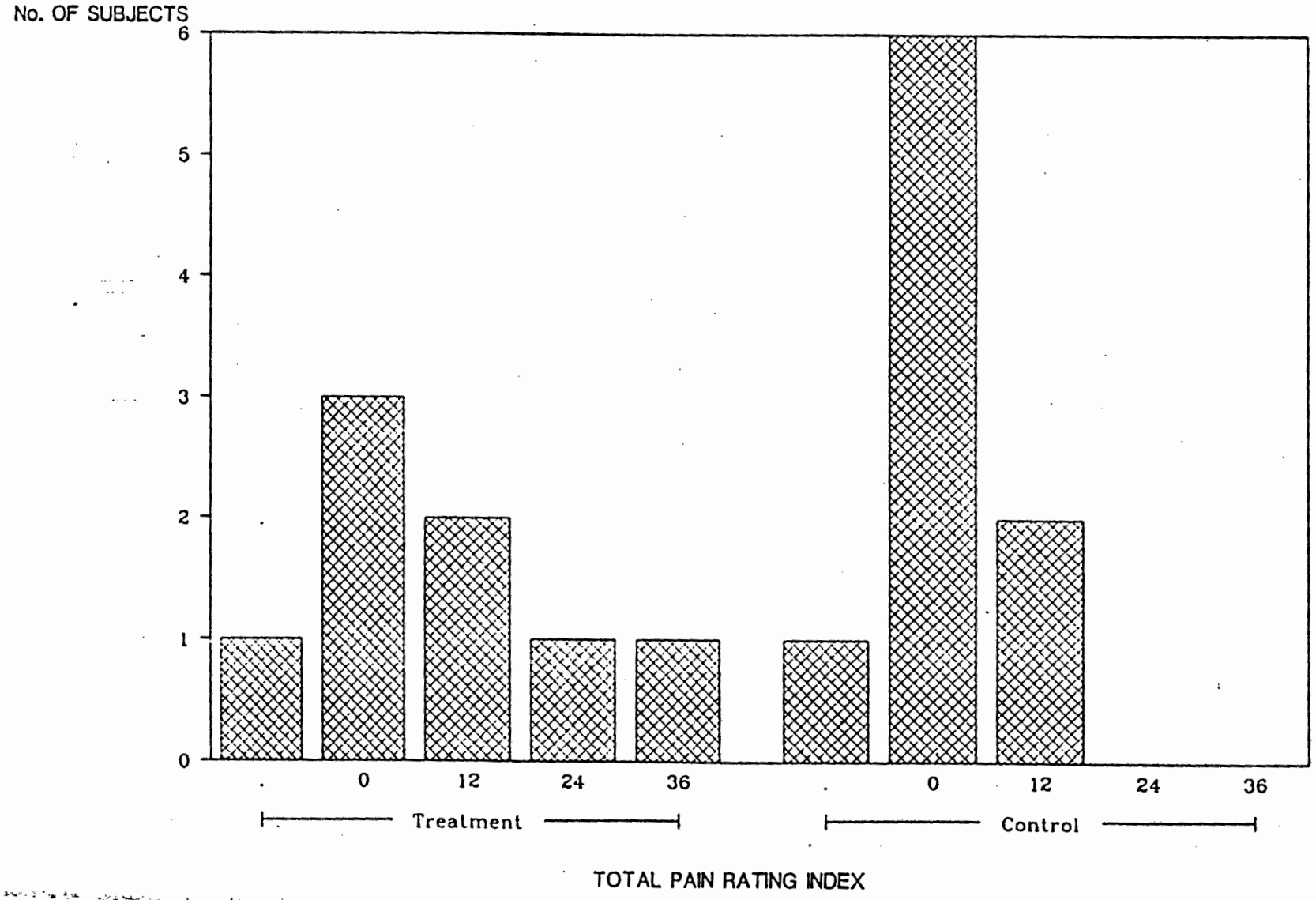
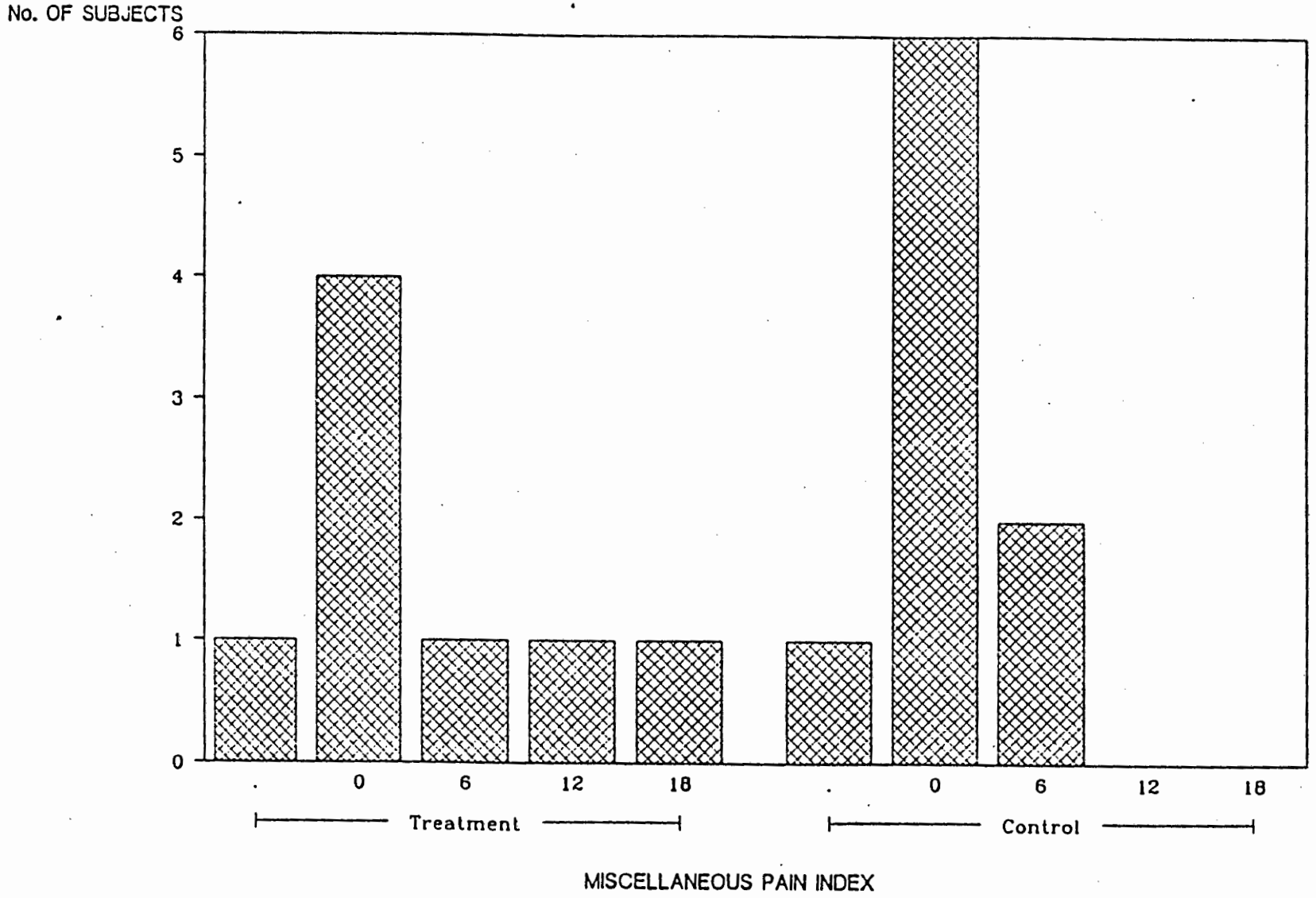


Figure 3.6: Change by Post-Intervention: Miscellaneous Pain Rating Index Comparison between Groups



Note: Period (.) equals missing values

results indicate that the SIMPT intervention did not provide significant improvement the treatment group's scores on the dependent measures utilized as compared to the control group.

Present Pain Intensity was the only variable indicating near significant (Sgn Rank = 11.0, $p=0.06$) inter group differences over the duration of the programme. The overall increase in the treatment group's mean $\pm$SD scores on this variable indicated that the initial improvement by mid-intervention (see section 3.2.2) was only temporary.

Only one intra group changes proved significant: the control group showed a significant increase in scores on the Anger mood factor ($t = 3.0$, $p \leq 0.05$). Figure 3.1 shows the distribution of Anger change scores for the two groups, where six of the subjects in the control group had a slight increase in feelings related to this mood factor.

Closer examination of some of the mean $\pm$SD inter- and intra-group change scores revealed interesting trends. Near significant inter group changes (see Table 13.1) existed for the Anger ($t = 1.46$, $p=0.17$) and Vigor ($t = -1.64$, $p=0.12$) mood factors. In the former, the treatment group improving over the duration of the intervention (i.e., a decrease in Anger compared to the control group), and in the latter, both groups showing a reduction in Vigor levels. Both groups showed an increase in their mean $\pm$SD TMD change scores over the duration of the study (see Figure 3.2). This deterioration in mood state, however, was non-significant at the intra and inter group ($t = 1.07$, $p=0.30$) levels of analyses. Intra group mean $\pm$SD change scores for the treatment group showed a relatively larger variance in scores on the six mood factors than did the control group. These results (i.e., large variance in scores) are similar to those of the Mid-intervention analyses, and could have played an important role in affecting the statistical significance of some of the expected changes in mood disturbance.

The changes in internal locus of control, although non-significant, indicate to some extent a possible difference in the two groups' perceptions of their role in maintaining their health. Figure 3.3 shows the differences in IHLC change scores between the two groups. These slight changes in locus of control were not evident in the powerful others, and chance locus of control dimensions.

Inter- and intra-group changes in Health status (psychological and functional disability) as measured by the AIMS, were virtually non-existent (see Table 13.1). Considering the strength of the correlations (see section 2.3.6) between Psychological Health Status and Total Mood Disturbance and Total Pain Rating Index at pre-intervention analysis, it is not clear why the slight changes along the latter two variables did not influence change in the former.

The slight reduction in the treatment group's mean $\pm$SD perception of coping efficacy (-2.14 ± 10.29), compared to the control group's (2.00 ± 7.5), was not significant at the intra and inter group levels of analyses. These results indicate that the SIMPT intervention was not successful in altering the subjects' perception of their ability to cope with stressful events. As Figure 3.4 demonstrates, three subjects in the treatment group actually showed a relatively large (-12) reduction in their perceived coping efficacy. The missing data for two subjects in the control group confound interpretation of the changes in that group.

The distribution of PRIT change scores (see Figure 3.5) for the two groups indicate the extent to which the treatment group provided higher ratings their pain post-intervention. The increases in mean $\pm$SD pain index

performance on the other key variables over the duration of the study. The clinical nature of the study as well as the clinical nature of the lymphocyte data, facilitates such an approach to understanding these results. This is particularly so for the five subjects who showed subnormal response rates to the mitogens at baseline and/or at post-intervention.

3.3 Summary of Results Testing Second Hypothesis

The results presented in section 3.2 do not provide evidence that the SIPMT programme produced significant change in the treatment group's scores on the dependent measures. Furthermore, there is no evidence that even the slight changes observed in some of these variables resulted in changes in disease activity and immune function.

4. Understanding Individual Treatment Effects: A Case Study Approach.

The clinical nature of the study indicates that a closer analysis of the possible benefit of the SIMPT for individual subjects, would yield interesting information. To determine the interplay of the psychological, pain, and disease activity variables over the duration of the study, it was necessary to reduce all these variables to the same scale of measurement. The following procedure was used to allow for such analysis:

$$\frac{\text{obtained score}}{\text{maximum possible score}} \times \frac{100}{1}$$

The subject's score on each variable was thus expressed as a percentage (0 to 100%) of the maximum possible score for that variable, making it possible to plot these scores on a single axis (see Figures 4.1 to 4.14). This procedure has the advantage of making the variables directly comparable without altering the amount of information given by each respective measure. Special attention was given to those subjects having subnormal lymphocyte proliferation response results, to determine whether or not any unusual psychological and/or disease activity factors could be identified.

The cases identified as T or C are subjects from the treatment and control groups respectively. This is followed by their identification number and a pseudonym to facilitate the discussion. Each case is discussed in terms of her medical history and disease condition, medication, personal characteristics, attitude and reaction to the treatment, and changes along key variables over time.

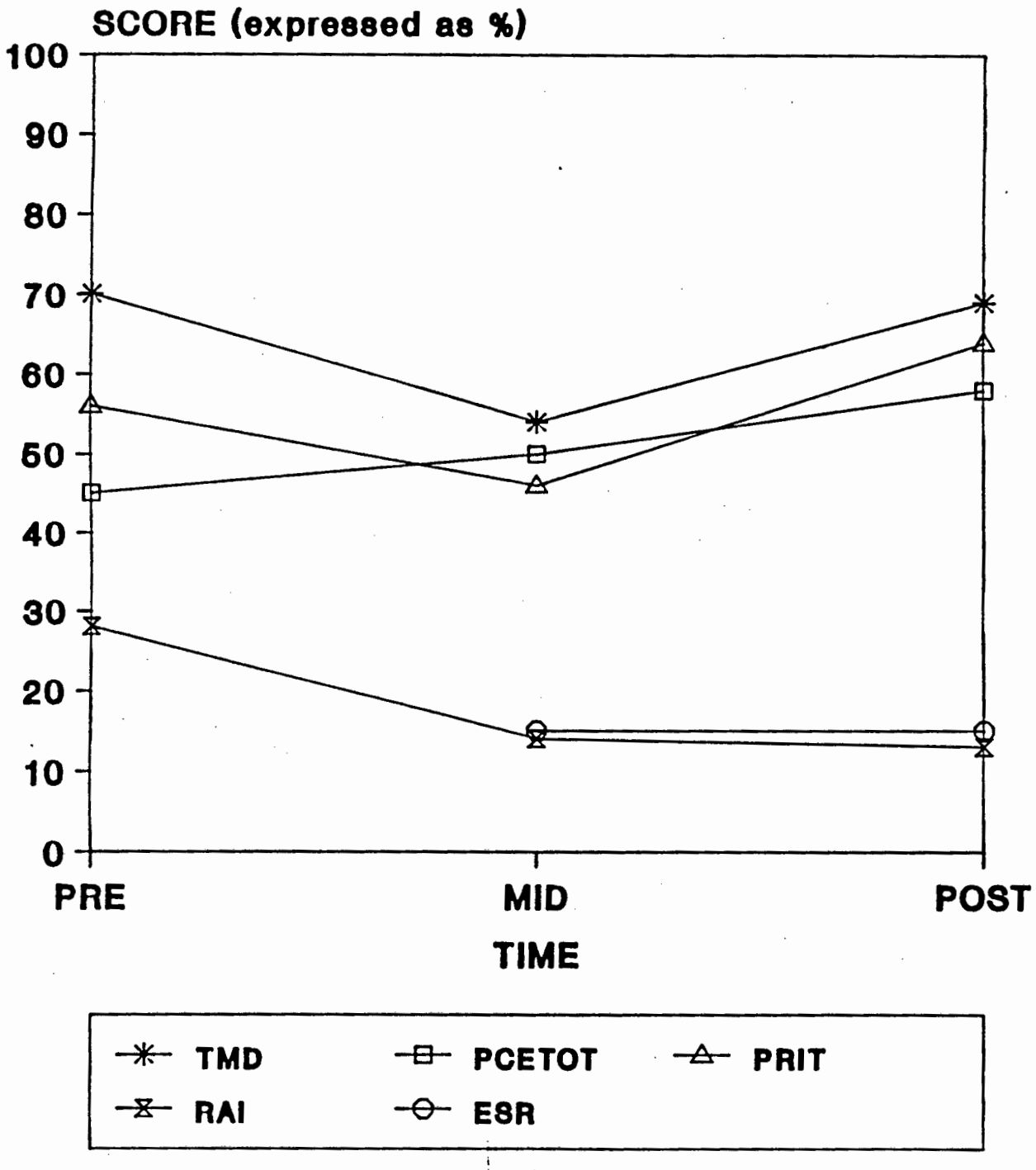
4.1 Case T(102): "Gafsa".

Medical History:

Diagnosed with classic RA in 1988. Moderate disease activity and joint impairment in hands, knees and feet. Extreme, "distressing" (Present Pain Intensity) pain in elbows, wrists, hands, knees, and feet. Treatment included

FIGURE 4.1: Case T(102) Gafsa

Treatment Group



monthly visits to the clinic, and daily anti-inflammatory medication (i.e., the Non-steroidal Anti-inflammatory Drugs--hereafter NSAID--: Feldene and Naproxen).

Subject:

"Gafsa" is a 42 year old, married, Moslem woman, living in Mitchells Plein and supporting more than four children. She is no longer able to work because of extreme pain. Her pre-intervention scores showed moderate mood disturbance (high scores on Tension, Depression and Anger mood factors) and perceptions of her ability to cope with stressful events effectively. Upon entry into the study, her pain was worst in the mornings, and she relied on her anti-inflammatory medication and heat applications for pain relief. She felt that not much could be done for her pain, but tried to control it "in the best and most simple possible way".

SIMPT:

She was "most positive" (i.e., 7/7 ratings on the Treatment Perception Questionnaire - TPQ) that the intervention would be successful in helping her to cope with her RA and decreasing its impact on her life. Her participation in the sessions and enthusiasm was excellent. She set herself a long term goal of becoming more independent by earning money through the sale of bed linen that she could make at home. By the end of the intervention she had secured a number of sales which considerably alleviated the financial strain they were experiencing. Short term goals included increasing activity, and improving her relationship with her spouse.

As Figure 4.1 shows, her initial progress was good, and by mid-intervention had an improved mood state (TMD) and pain rating (PRIT). The close relationship between the mood and pain variables are clearly demonstrated in this subject's progress over time. Her Ritchie Articular Index (RAI) score followed a similar pattern. Despite the gradual increase in her perceived coping efficacy (PCETOT) over the duration of the intervention, the reversal in mood and pain at the end of the treatment was difficult to explain. It is possible that she received more benefit from the first (information) and second (skills training) phases of the treatment, than the third (application). Her extremely high expectations of the value of the treatment (i.e., idealized) may not have concurred with the actual or more realistic outcomes (i.e., that pain would not be "cured") of the intervention.

The reduction in disease activity in terms of localized joint inflammation (RAI), and stable abnormal ESR were not affected by the mid-to-post deterioration in mood and pain. As she had been on the NSAIDs for some time prior to the intervention, it is possible that the intervention played a role in reducing inflammation in her joints. She had particularly subnormal T- and B-lymphocyte proliferative response rates to PHA (29.6%), PWM (27.8%), and CON-A (10.0%) at baseline assessment. However, by the end of the intervention, there was a dramatic improvement in her lymphocytes' ability to respond to in vitro stimulation (PHA = 293.9%, PWM = 345.5%, and CON-A 123.3%). As there was little change in her medication over this period and she did not report any changes in her health, it is difficult to interpret such change in immune status. It is possible that the reduction in joint inflammation played a contributary role. The changes in mood disturbance are also not consistent with such improvement. Only perceived coping efficacy to stressful events followed a similar pattern of progress (i.e., improvement), and it is possible that it mediated the negative effects of such adverse psychological experience.

4.2 Case T(103): "Miriam"

Medical History:

First diagnosed with RA in 1980. Relatively mild disease activity and joint impairment in wrists and hands only. Extreme, "distressing" (Present Pain Intensity) pain in shoulder, wrists, hands, and ankle. Her treatment regimen consisted of monthly visits to the clinic and daily medication. This medication included non-steroidal anti-inflammatory drugs (i.e. NSAID) (Arthrexin and Panamor) and gastro-intestinal ulcer treatment (Zantac).

Subject:

"Miriam" is a 46 year old married "coloured" woman from Mitchells Plein. She no longer has children to support and considers herself to be a housewife. Her elevated mood disturbance (high scores on Tension, Depression, Anger and Fatigue factors) was not consistent with a good perception of coping efficacy in stressful events. Her pain was worse in the early mornings, in cold weather, and after too much activity. Her pain seemed to decrease "when relaxed or having been out visiting for a few hours". Miriam relied on medications , relaxation, massage, and ignoring her pain, for pain relief. She was not clear what could be done for her pain. Miriam was an anxious person, and indicated that she frequently felt tense and uptight at home. This possibly could have accounted for the need for ulcer treatment.

SIMPT:

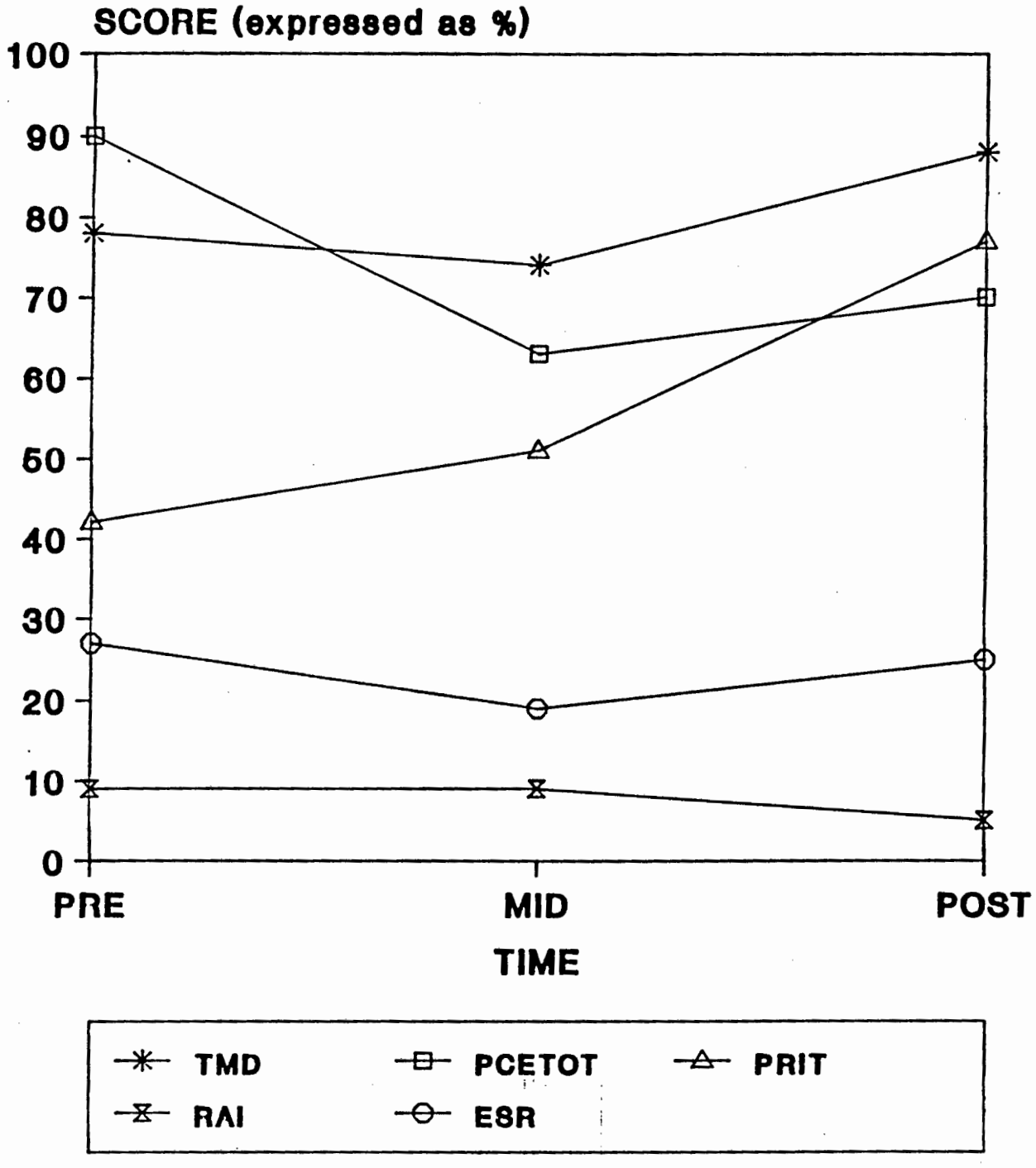
Miriam was "moderately positive" (4/7 on the TPQ) that the intervention would be of value in helping her to cope with RA. She seemed to enjoy the sessions, and participated lively in the group discussions. She did not enjoy the relaxation training as she found it "scary to be out of touch with one's body". Her long term goal was to be more independent and active. Short term goals included increased activity (walking around the block) and to start knitting again.

Miriam made very little progress over the duration of the intervention, despite an anecdotal account of the benefit of the programme. Figure 4.2 shows the extent to which there was an definite increase in her overall rating of her pain (PRIT), and partial increases in mood disturbance. Although there was a drop in her perceived coping efficacy (PCETOT), these ratings remained relatively high. There was a slight reduction in her Ritchie Articular Index (RAI) at post-intervention. Her anecdotal account of improved medication usuage, following education on the nature of her medication and the increased incidence of ulcers through inadequate medication usuage (e.g., between meals), probably contributed to the reduction in joint inflammation. Despite her poor psychological adjustment and increase in pain ratings, lymphocyte proliferation results were well above the 75% cut-off point at baseline and post-intervention (see Table 14.2).

Considering the stability of the disease activity indexes and the marginal changes in mood disturbance and coping efficacy, the sharp increase in Miriam's pain ratings are difficult to explain. Closer analysis of the subscales of this

FIGURE 4.2: Case T(103) Miriam

Treatment Group



measure revealed a considerable increase in her pain ratings on the sensory dimension. It is possible that instruction in learning to communicate the nature of her pain more effectively (in terms of "what it feels like", to reduce significant others perception of her as "always complaining about her pain and suffering"), may have biased these results. No changes occurred on the other dimensions. These results, furthermore, indicate the extent to which changes in the sensory dimension of the pain experience may not necessarily influence changes on the other dimensions.

4.3 Case T(106): "Dorothy".

Medical History:

RA was first diagnosed in 1989, following a long history of unexplained pain and malaise. Moderate disease activity present, though little joint impairment present. Moderate, "discomforting" (Present Pain Intensity), generalized pain experienced in shoulders, neck, abdomen, pelvis, fore-arms, wrists, and ankles. Treatment included monthly visits to the clinic, and medication. The latter included a remission-inducing drug (Metalcaptase), and a steroidal anti-inflammatory drug (Meticorten).

Subject:

"Dorothy" is a 49 year old, married, "white" woman from a middle-class suburb of Cape Town. She is a housewife, with two mid-adolescent children to care for. Her mild mood disturbance (elevated scores on Tension, and Fatigue factors) and moderate perception of coping efficacy concurred well with her condition. Her pain was worse following "over-activity", and only medication and bed-rest brought pain relief. Trying to ignore the pain and massaging the painful area also seemed to control her pain. Upon entry into the programme, Dorothy was an anxious person who still found it difficult to accept that she had RA. She felt that: "they still have not uncovered the true cause for my pain."

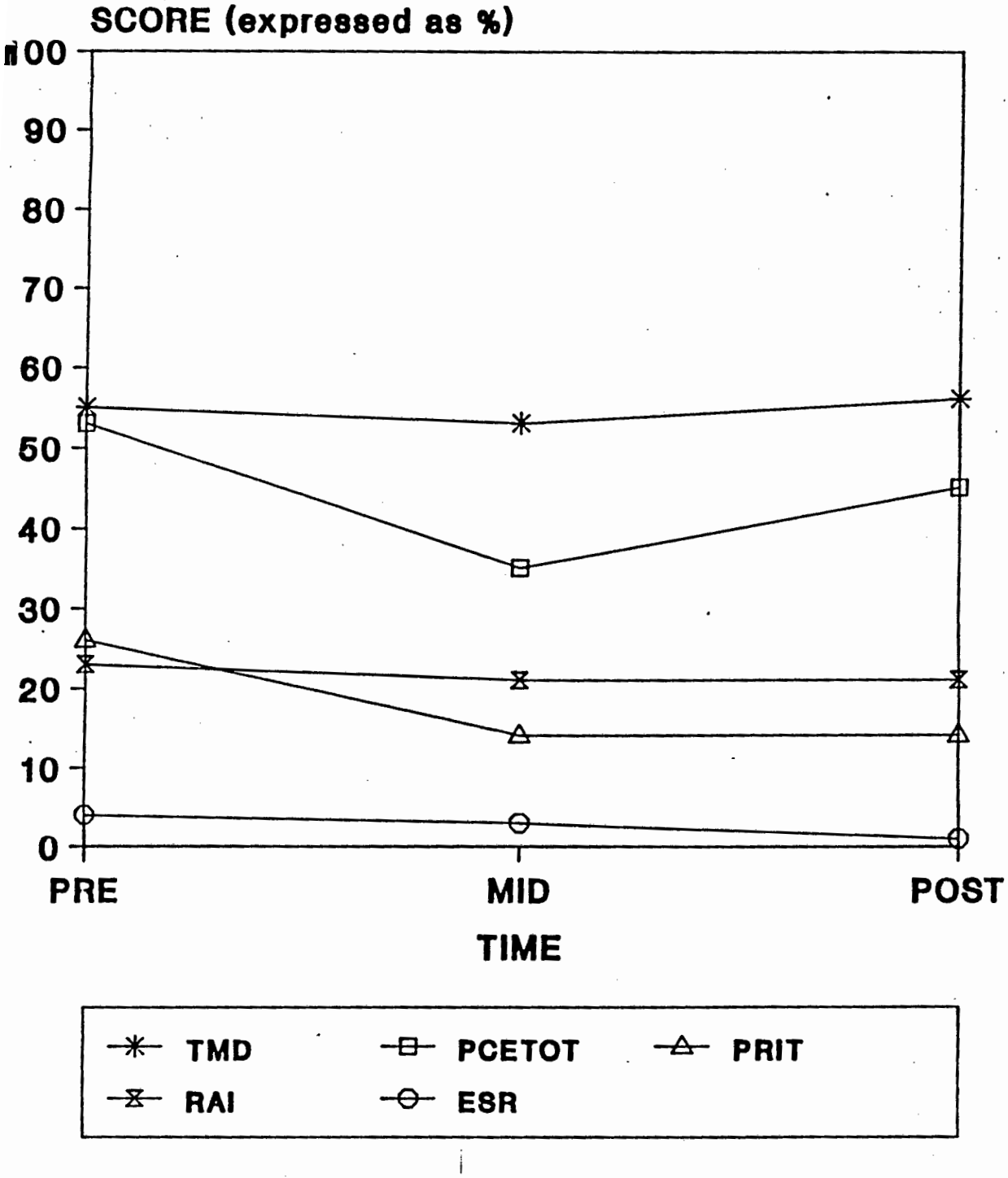
SIMPT:

Dorothy was reluctant at first to participate in the group discussions, and expressed the wish to "just listen to what was being said". Her moderate ratings of the success of the programme (4/7 on the TPQ) in helping her to cope with her RA indicated this uncertainty regarding the value of the group discussions. She frequently complained of not feeling well (nauseous, dizzy) as a result of her medication. Her understanding of what RA entailed was minimal, and found great benefit from hearing about the other women's' experiences.

Despite her anecdotal account of benefitting from the programme, Dorothy made very little progress along the key variables. Figure 4.3 shows the stability in her scores on the key measures. This was particularly so for the disease activity (RAI and ESR) and mood (TMD) variables. A slight drop in her pain rating index (PRIT) indicated a mild reduction in pain by mid-intervention which remained so at post-intervention. Generally, the lack of any improvement in scores, indicates that Dorothy received little benefit from the SIMPT programme. Dorothy found it difficult to accept the long-term nature of her remission-inducing medication, and this was reflected in poor

FIGURE 4.3: Case T(106) Dorothy

Treatment Group



compliance (anecdotal) to the drug. It proved difficult to convince her of the extent to which she was furthering her disease through poor medication usuage account of poor compliance with daily dosage. By th end of the programme, however, Dorothy had achieved a stability in medication intake, and more appropriate behaviours at home (e.g., more careful use of joints and increased rest). The greatest benefit she recieved from the programme (based on anecdotal accounts, and interaction in the group) was the recognition of the role she played in determing the nature and extent of her pain. She claimed that the realization that she could actually cause more pain through inapropriate actions (e.g., excessive gardening when she felt better), was of immense help to her.

Her lymphocyte proliferation results (see Table 14.2) were well above the 75% cut-off point, indicating healthy T- and B-lymphocyte response to mitogenic stimulation. A poor response to CON-A (96.1%) relative to PHA (3149.8%) and PWM (4073.1%) at post-intervention is difficult to explain.

4.4 Case T(107): "Dorian".

Medical History:

RA was diagnosed in 1985. This followed a history of chronic illness (including a stroke and subsequent paralysis, and removal of a metastatic tumor) and extreme personal loss, where "Dorian" showed a remarkable recovery despite poor prognoses. High disease activity, and moderate joint inflammation and impairment (hands, hips, knees, and feet) existed on entry into the programme. Pain was generalized, with local sites (e.g., fingers) of extreme, "distressing" (Present Pain Intensity) pain and discomfort. Her treatment included monthly visits to the clinic, and medication to control inflammation.

Subject:

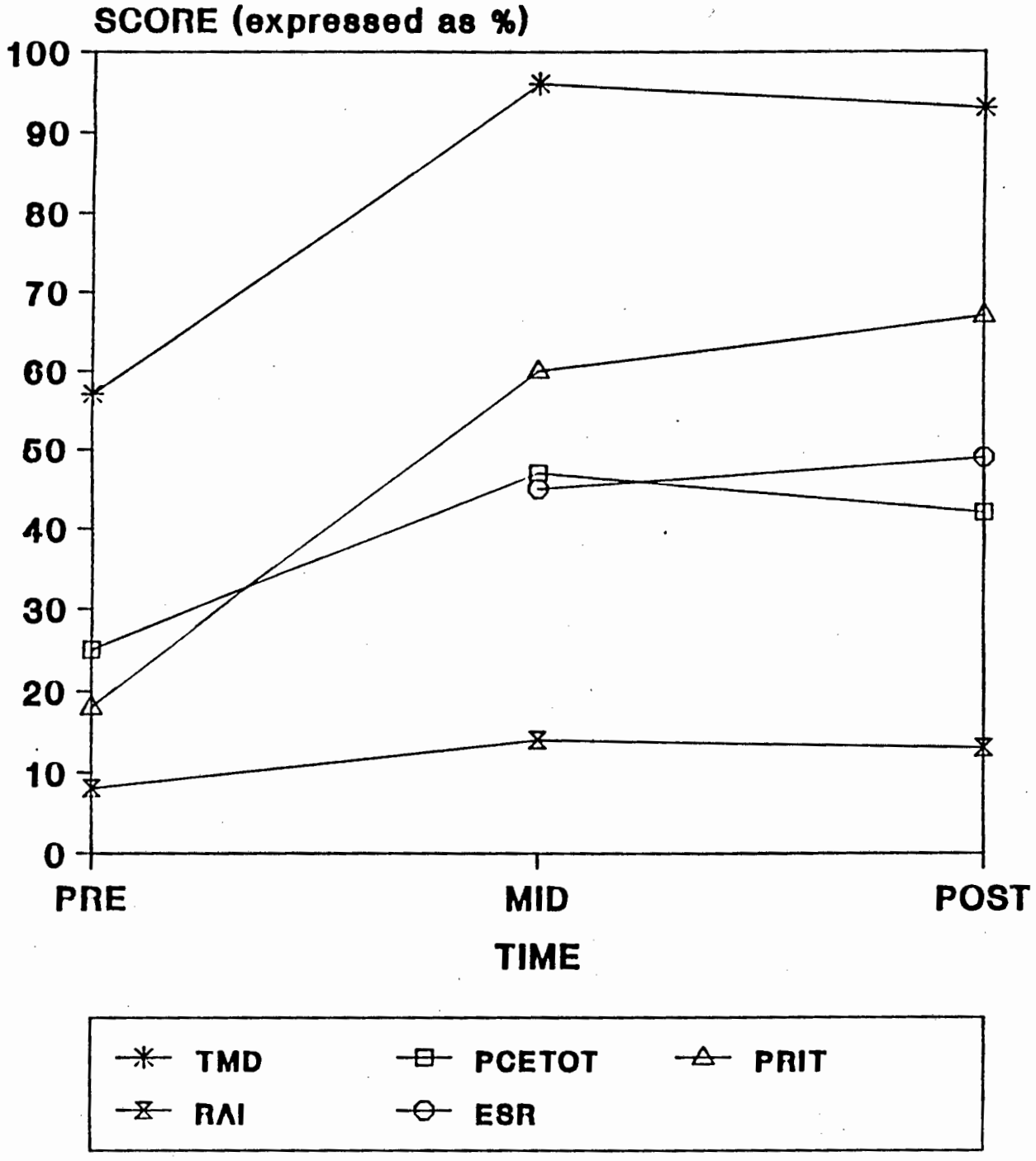
"Dorian" is 52 year old, widowed, "coloured" woman living in Grassy Park. Being on a disability pension, she is able to support her daughter's child in a reasonably furnished first floor flat. Despite poor perceptions of coping efficacy, she showed minimal mood disturbance (elevated scores on Anger and Confusion factors only). Increases in her pain, according to her, were associated with "upsetness", too much activity (e.g., house work) and the weather. She found that medication, moderate activity ("I must keep moving."), and having "less worries" seemed to alleviate her pain. She had also developed unusual pain management techniques which included talking to the painful joint/limb and treating it "as if it does not belong to me" (i.e., dissociation); immersing the joint into hot water; or wrapping the joint up tightly in a bandage. She also wore sweat bands on her wrists and ankles, and thermal underwear throughout the year. She had her own set of "exercises" which she followed daily to manage her pain and maintain strength in her arms and legs. She felt that the best thing that could be done about her pain was the discovery of a cure for RA.

SIMPT:

Dorian was an dedicated participant in the programme who contributed greatly to the content of the sessions. She felt comfortable discussing her experiences, and sharing her pain management techniques. This was despite her

FIGURE 4.4: Case T(107) Dorian

Treatment Group



moderate rating (4/7 on the TPQ) of the treatment's ability to help her cope more successfully with RA. She was most positive (7/7 on TPQ) that the treatment would reduce the effect that RA has on her social life, and enjoyed the support she received from the other subjects.

Her deterioration in mood (TMD) and pain ratings (PRIT) by mid-intervention were unexpected. Figure 4.4 shows these changes clearly, demonstrating the close relationship between mood disturbance and pain experience. Such changes occurred despite an increase in perceived coping efficacy to stressful events (PCETOT), and little change in joint inflammation (RAI). As no pre-intervention ethrocyte sedimentation rate data was available, it is difficult to determine what caused her deterioration in mood and pain. Anecdotal accounts of the "upsetting" disruption of her home environment during the intervention by builders renovating the building, possibly contributed to these changes. Furthermore, Dorian's health did deteriorate over the duration of the intervention, and it is possible that an increase in disease activity caused the increase in mood disturbance.

No changes occurred in her lymphocyte proliferation results (see Table 14.2) over the duration of the intervention, values remaining well within the healthy range of response.

4.5 Case T(110): "Mary".

Medical History:

RA was first diagnosed in 1983, following a few years of pain in the hips and hands. Disease activity was mild and extreme, "distressing" (Present Pain Intensity) pain was present in the hips, upper thighs and hands. Great discomfort yet mild joint impairment was experienced in the hips and hands. Treatment entailed monthly visits to the clinic, and a daily NSAID (Feldene) for pain relief.

Subject:

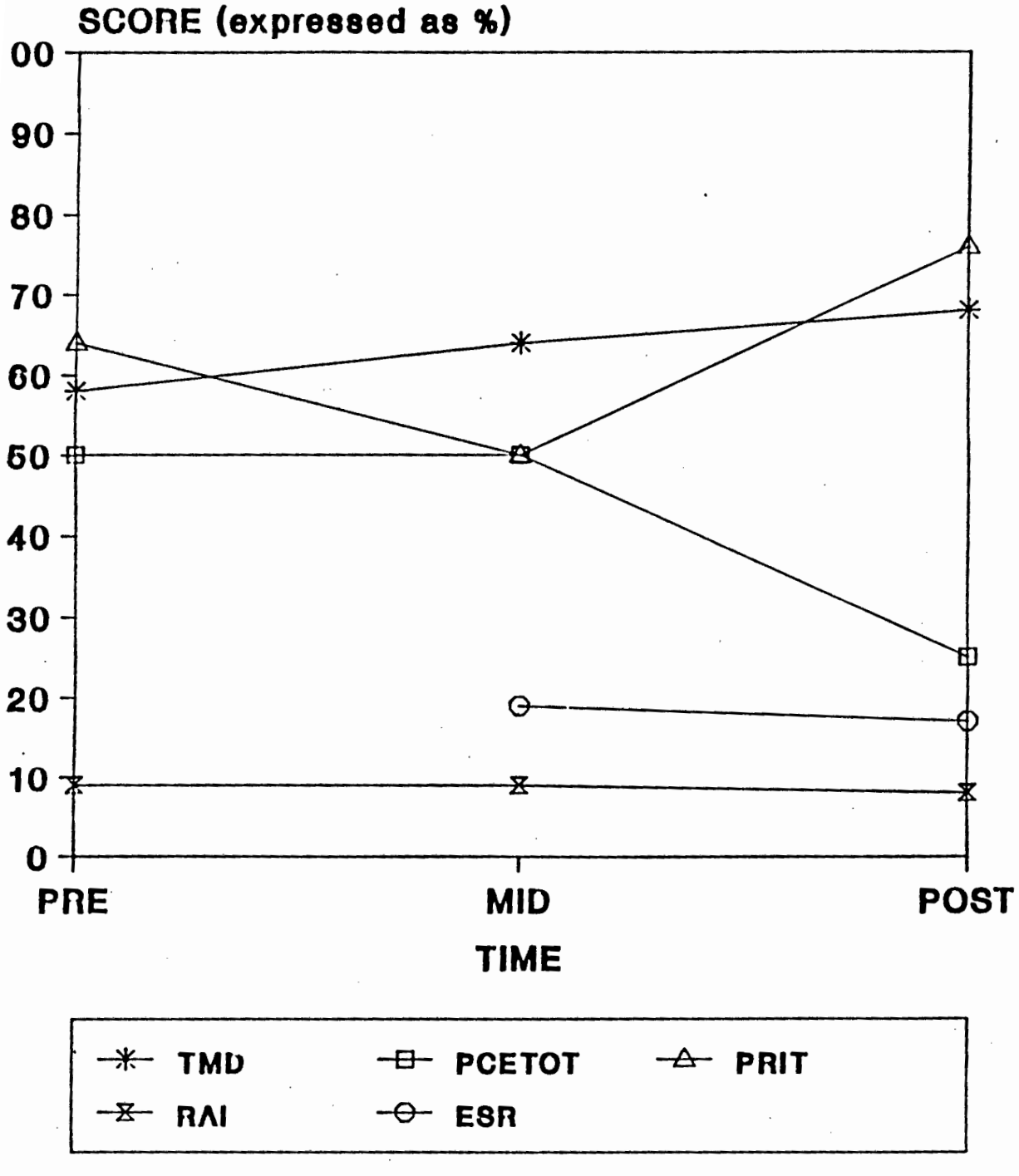
"Mary" is a 63 year old, widowed, "coloured" woman from Lavander Hill (a lower class "coloured" suburb). Her state pension enabled her to share a house with her sister and her daughter's two pre-school children. Her mild mood disturbance (elevated scores on Anger and Confusion factors) was accompanied with moderate perceptions of coping efficacy to stressful events. Her pain was worst in the early morning and evening. She found that doing her housework seemed to decrease her pain as it helped her "take her mind off the pain". Mary also relied on her daily medication, bedrest and heat application to control her pain. Being able to relax was also associated with reductions in her pain. She felt that advice would help her pain, and felt moderately able to decrease her pain when it was present.

SIMPT:

Mary was a cheerful and talkative participant over the duration of the intervention. Her above moderate (6/7 on the TPQ) perceptions of the value of the programme in terms of her ability to cope with RA and to reduce its impact, concurred with her enthusiasm in the sessions. Her long term goal was "to learn to stop worrying so much

FIGURE 4.5: Case T(110) Mary

Treatment Group



about other people". Short term goals included increasing her activity, and reducing the amount of time spent satisfying her sister's demands.

Figure 4.5 shows the progress that Mary made over the duration of the treatment. Only pain ratings (PRIT) had improved by mid-intervention, though this reduction was not significant. The gradual deterioration in mood (TMD), poor perceived coping efficacy (PCE) and elevated pain rating scores at post-intervention indicate that the treatment did not succeed in improving Mary's stress and pain management. It is clear that the third phase of the treatment programme was not as successful as the first two phases. This result is consistent with that of Gafsa (subject 102 above). Mary's greatest daily hassle was having to look after her daughters children and ignore her sister's (who also suffers from RA) complaints. Over the duration of the intervention, she found it increasing difficult to cope with the demands at home, and still manage to "live with her pain in her hip". This is possibly reflected in the deterioration in PCE by post-intervention

The stability of the joint inflammation (RAI) and ethrocyte sedimentation rate (ESR) scores over the duration of the treatment indicated that disease variables were not responsible for the above results. Furthermore, no change occurred in lymphocyte proliferation results (see Table 14.2), with values remaining well within the healthy range of response.

4.6 Case T(111): "Alfreda".

Medical History:

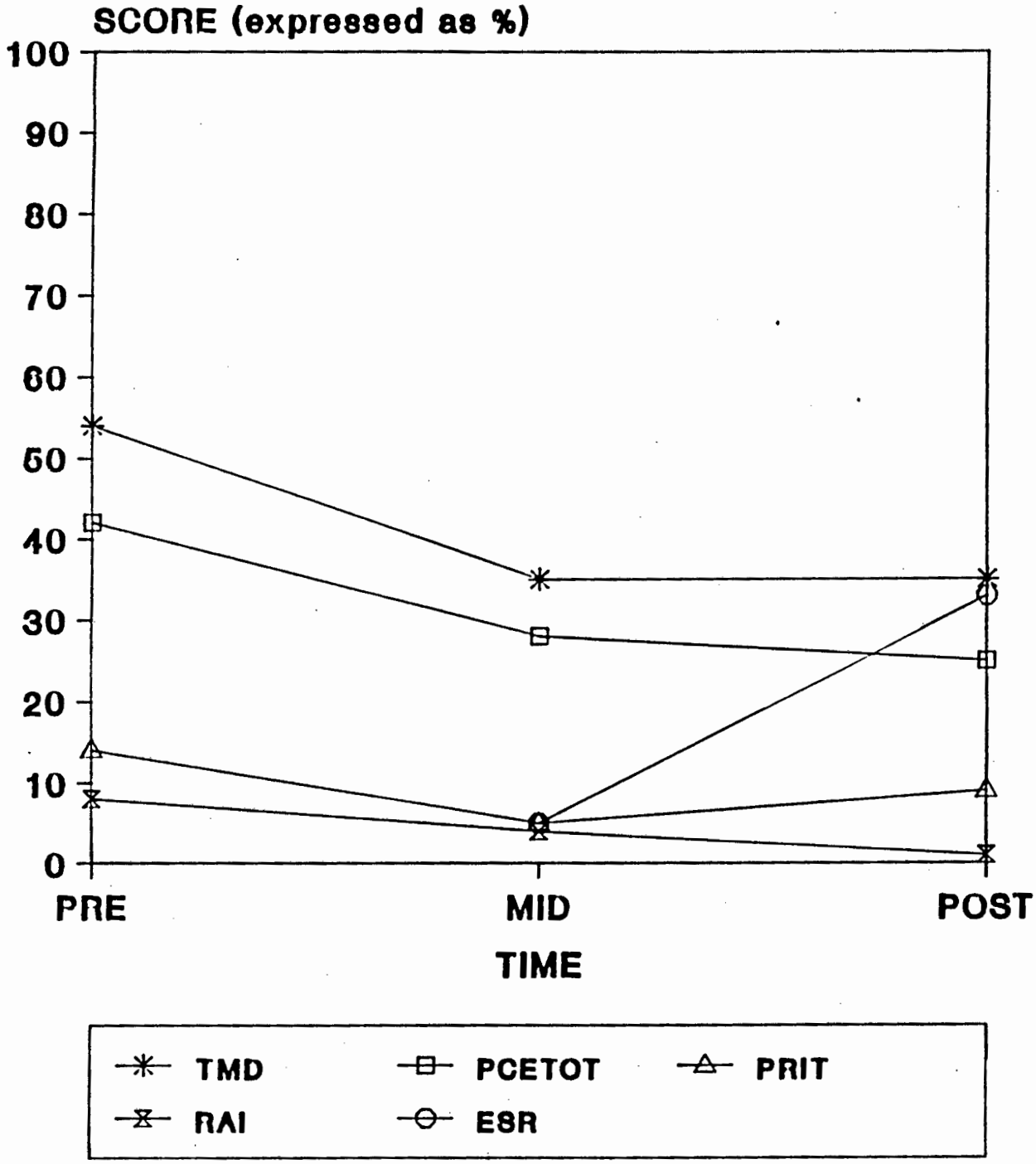
RA was first diagnosed in 1985. Disease activity was mild, with little joint impairment or deformation. Moderate, "discomforting" (Present Pain Intensity) pain was experienced, localized in the elbows and right-hand wrist. She claimed that her RA had improved considerably over the last few years. Her treatment regimen included monthly visits to the clinic and daily medication. The latter included NSAIDs (Naprosyn, Indocid) and a remission-inducing drug (Sulphasalozyn).

Subject:

"Alfreda" is a 38 year old, recently widowed, "coloured" woman from a middle-upper class area. She is temporarily unemployed and supports two children. Although she had a generally poor perception of her ability to cope with stressful events effectively, she showed minimal mood disturbance. Her pain was worse following "too much activity" and towards the late afternoon. Being "too upset" also exacerbated her pain. When she felt cheerful and full of energy, her pain decreased. She relied on her medication, exercise, relaxation, and occasional physiotherapy to control her pain. She also tried to structure her work load (e.g., laundry) to avoid over activity. She felt that her pain "should just be controlled". Alfreda saw herself as being a very capable person, who was able to manage her pain and adapt to her RA as well as possible.

FIGURE 4.6: Case T(111) Alfreda

Treatment Group



SIMPT:

Alfreda rated the value of the treatment very highly (7/7 on the TPQ), and was an animated participant in the programme. She set herself the long term goal of being able to enjoy a hobby that helped her to relax and distract her from her pain. She chose knitting as a short term goal and was proud to wear her first jersey on the final session. She responded well to the assignments set during the first two phases of treatment.

Figure 4.6 shows Alfreda's progress over the duration of the treatment, where there was a substantial drop in her mood disturbance (TMD) by mid-intervention. This occurred despite a slight deterioration in perceived coping efficacy (PCETOT). A significant feature of Alfreda's reaction to the intervention, was the unexpected return of feelings of loss related to her husband's death the year before. She found the programme helped her to think about it more clearly, and be able to express her grief over the loss.

There was a dramatic increase in disease activity as measured by ethrocyte sedimentation rate (ESR) from mid- to post-intervention, with no corresponding increase in the other variables. This change was possible associated with her coming to terms with her deceased husband and the strong emotions which the group sessions elicited. Little change in overall pain rating (PRIT) and joint inflammation (RAI) occurred over the duration of the programme. Finally, lymphocyte proliferation response rates to in vitro mitogenic stimulation were well within a healthy range of response at baseline and post-intervention assessment (see Table 14.2).

The lack of clear relationships between the variables are not consistent with predicted patterns of change. The reduction in mood disturbance score and stable low pain rating index, however, indicate the extent to which the treatment was particularly beneficial for Alfreda during the first two phases. This concurs with the findings for Gafsa and Mary above.

4.7 Case T(112): "Francis".

Medical History:

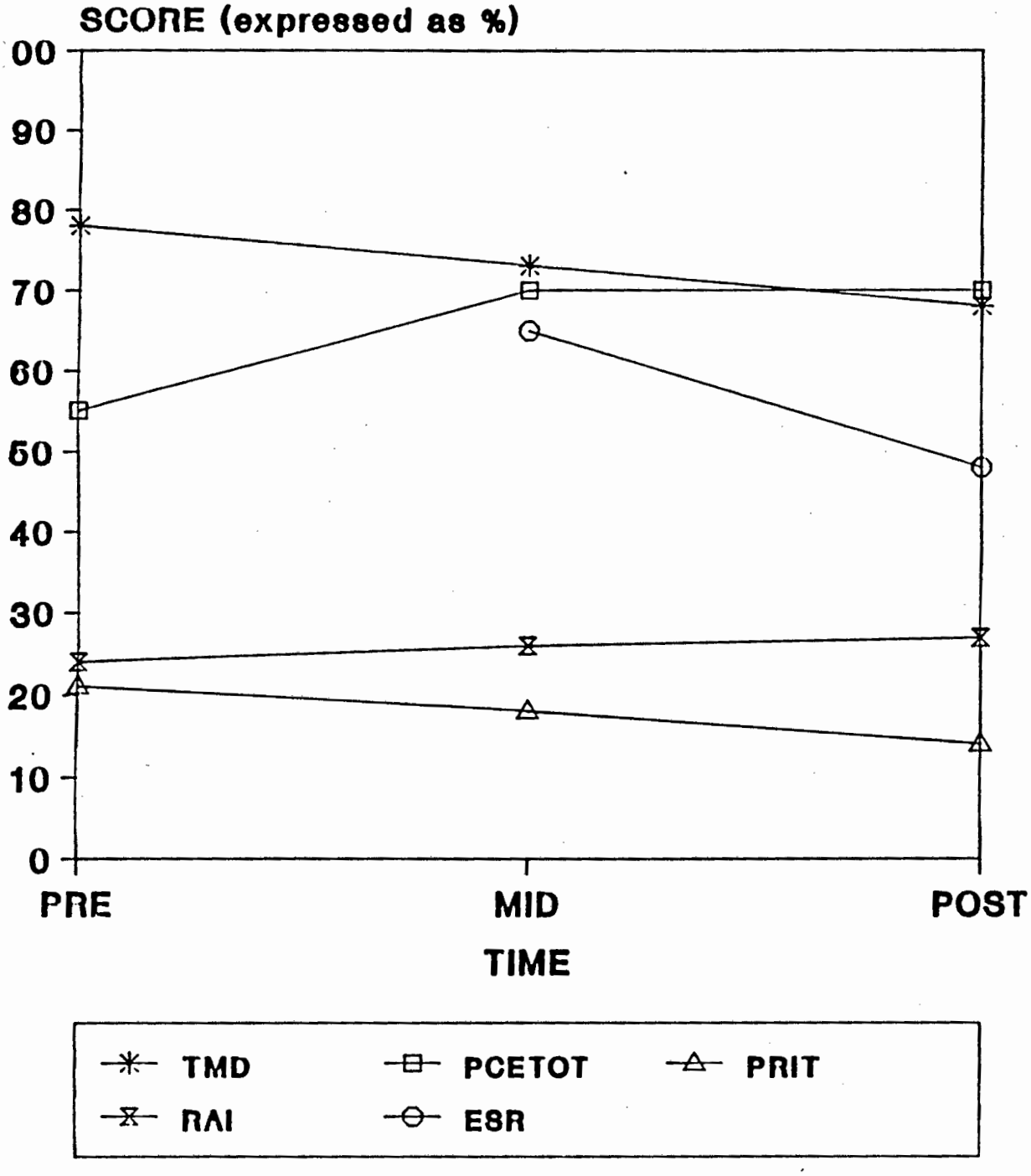
RA was first diagnosed in 1980, and showed rapid deterioration in recent years. Disease activity was relatively high with marked joint deformity and impairment in the knees, ankles, shoulders and hands. Mobility was restricted, and a wheelchair was considered necessary for locomotion. Moderate, pain existed, experienced as "discomforting" (Present Pain Intensity) in the affected joints. Treatment included monthly visits to the clinic, and medication, including: a remission-inducing drug (Penicillamine), anti-inflammatories (Prednizone--steroidal--and Voltarin), an analgesic (Panadine), and an anti-depressant (Surmontil).

Subject:

"Francis" is a 45 year old, married, "white" woman from a middle class area. She was declared medically unfit to work in 1986. She showed marked mood disturbance (extremely high scores on Depression, Fatigue and Confusion factors), yet moderate perceptions of coping efficacy. Her pain seemed to increase following too much

FIGURE 4.7: Case T(112) Francis

Treatment Group



activity, during cold weather, and in the early morning and late afternoon. She found that rest, warmth, and resting her joints using splints were associated with decreases in her pain. Francis relied on medication, bedrest, relaxation, heat applications and "ignoring the pain", to control her pain. She felt that the best thing for her pain would be "to find a cure for RA".

Francis was an anxious, depressed person who seemed to have resigned herself to her condition. She felt that she was "being punished", and was bitter that her condition was so much worse than the other women in the group. The number of different medications she was on is indicative of her condition. She felt disillusioned with the fact that neither medication, surgery, nor strict compliance with medical and occupational advice had been able to halt the progressive course of her disease.

SIMPT:

Francis was very confident that the treatment would be of benefit to her in helping her to cope more effectively with her RA (7/7 on the TPQ). She was only moderately convinced that the treatment would reduce RA's impact on her social and personal life. Despite the strong emotions the sessions seemed to evoke in her, Francis was an active participant in the discussions, talking about her experiences and feelings related to RA and stressful events. She found the treatment to be particularly effective in helping her to come to terms with her condition; that group support made it easier for her to accept her RA. During the individual interveiws, she found it a relif to begin to come to terms with the effect her disease had on her marraige. She was able to aknowledge the true reasons (i.e., withdrawal) for her husband's behaviour (staying away all day, and becomming over-involved in his church group).

Figure 4.7 shows the progress that she made over the duration of the treatment. The reduction in mood disturbance (TMD) concurred with a reduction in pain rating index (PRIT) and ethrocyte sedimentation rate (ESR). This improvement occurred in the context of an increase perception of coping efficacy to stressful events (PCETOT), and a stable Ritchie Articular Index (RAI). Lymphocyte proliferation rates remained well within the healthy range of response at baseline and post-intervention assessment (see Table 14.2), indicating that her steroidal medication had little effect on lymphocyte activity.

The changes demonstrated in Figure 4.7 indicate the extent to which Francis benefited from the treatment seesions, as well as the extent to which changes in psychological adjustment are related to changes in pain experience. It is possible that the programme is more applicable to more extreme cases of RA.

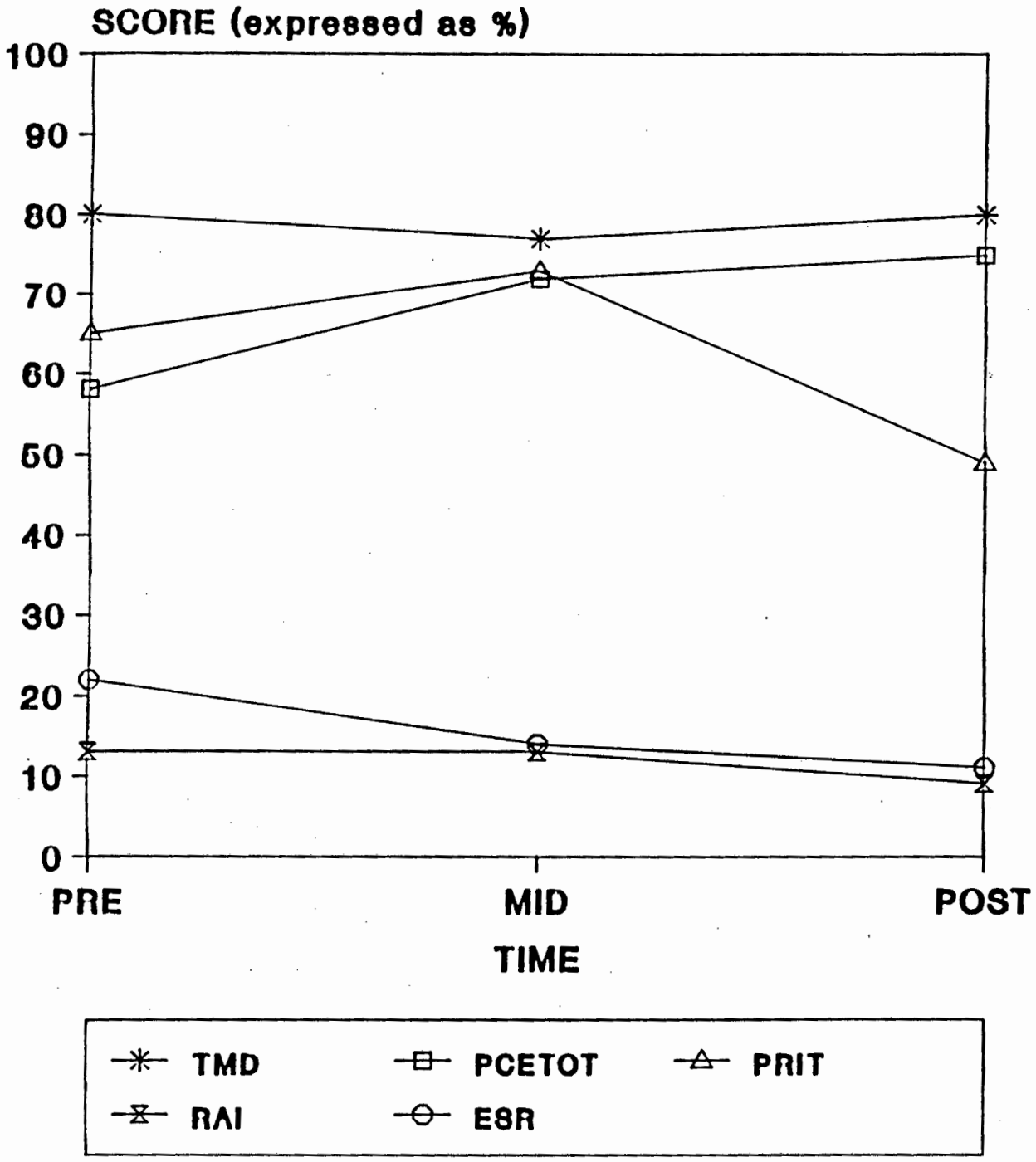
4.8 Case C(201): "Isabel".

Medical History:

First diagnosed with RA in 1984. Moderate disease activity, little joint deformity or impairment, and extreme, 'distressing' (Present Pain Intensity) pain in arms, neck, and knees. Constant complaints of abdominal distress, and medication side-effects. Her treatment regimen included monthly visits to the clinic, and daily medication.

FIGURE 4.8: Case C(201) Isabel

Control Group



The latter included anti-inflammatory (Voltaren), a remission-inducing agent (Chloroquine), and analgesic (Paracetamol) drugs.

Subject:

"Isabel" is a 52 year old, "coloured" woman from Mitchells Plein who was separated from her husband (drinking problems). She is a semi-trained nurse, but is no longer able to work. Her extremely poor mood disturbance (elevated scores on all six mood factors) was accompanied by relatively good perceptions of coping efficacy. Pain seemed to increase following periods of inactivity and "when upset or tired". She relied on medication and bedrest to control her pain. When pain was severe, taking a hot bath provided relief. Isabel did not know what else could be done for her pain. She seemed an anxious person who was struggling to cope with her condition and personal circumstances.

Evaluation:

Extremely high mood disturbance (TMD) and pain rating index (PRIT) scores. Figure 4.8 shows the extent to which these variables changed over time. Despite the reduction in her overall rating of pain (PRIT) by post-intervention, and the gradual increase in perceived coping efficacy (PCETOT) scores, little changes occurred in mood disturbance. Ritchie Articular Index (RAI) and ethrocyte sedimentation rate (ESR) scores indicating little change in moderate disease activity. Her medication clearly contained her RA.

These changes observed, indicate the extent to which any meaningful changes in the treatment group would have to entail significant (i.e., dramatic) improvements to confirm the second hypothesis. Lymphocyte proliferation results were well within the healthy range of response over the duration of the study (see Table 14.2).

4.9 Case C(202): "Helen".

Medical History:

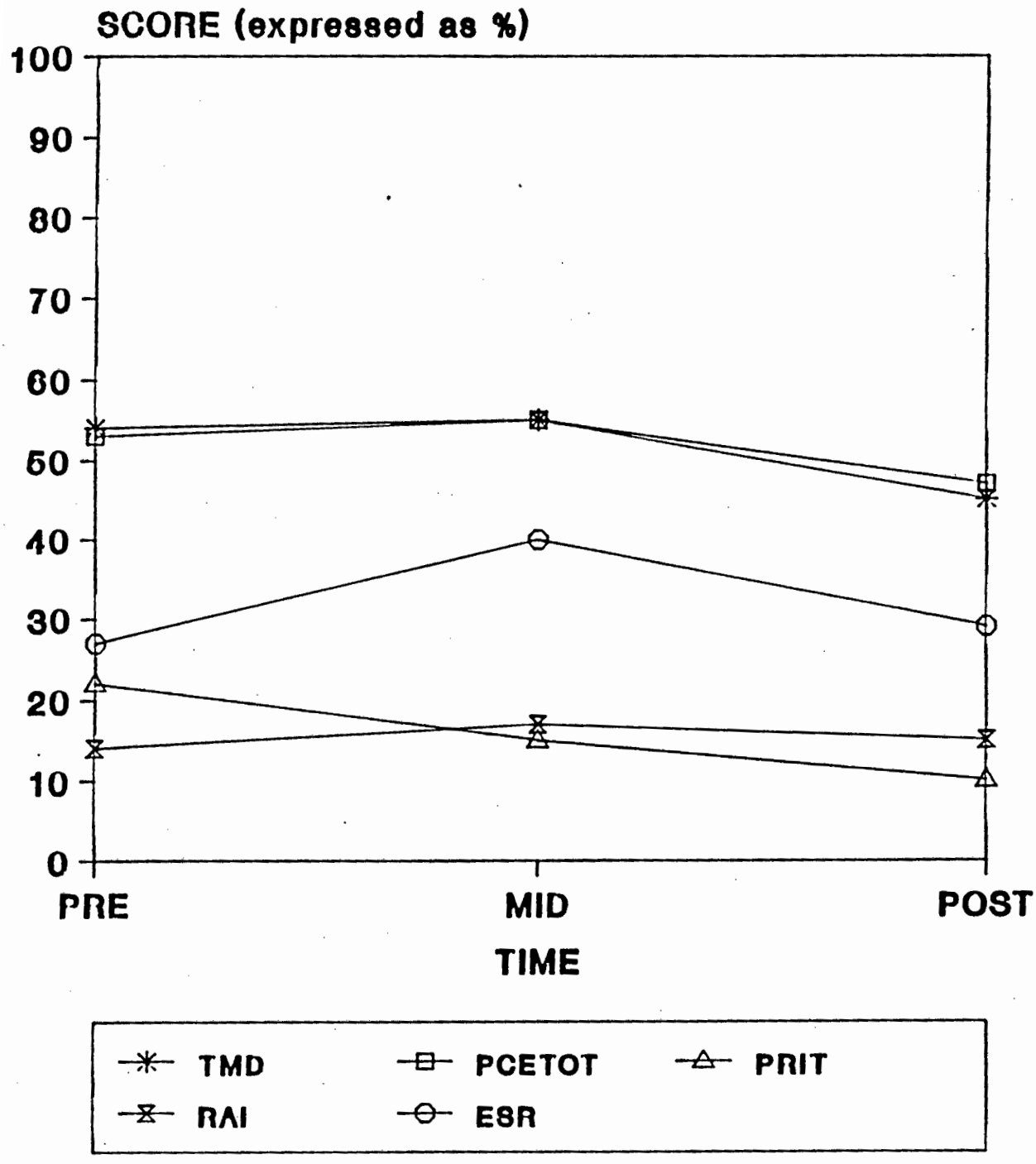
RA was first diagnosed in 1985, following a period of painful swelling in the legs and arms. Widespread but mild disease activity was present with "mild" (Present Pain Intensity) pain in the hands, calves, and ankles. Little joint deformity or impairment was present. Her treatment regimen included monthly visits to the clinic, and daily medication. The latter included: a remission-inducing agent (Sulphasalazine), NSAIDs (Voltarin and Arthrexin), and analgesics (Paracetamol and Codeine).

Subject:

"Helen" is a 57 year old, divorced, "white" woman from Observatory. A trained nurse, she went on disability pension a year prior to the study. Moderate perceptions of coping efficacy accompanied mild mood disturbance (elevated scores on Depression and Confusion factors). Increases in pain followed "too much activity", and she found that lying in a relaxed position on her bed reading with soft relaxing music helped decrease her pain. Helen

FIGURE 4.9: Case C(202) Helen

Control Group



also relied on medication, exercise, and ignoring the pain to control her pain. She felt that the best thing that could be done about her pain was "taking and coping with each day in its own way as it comes and not getting agitated with myself". Helen seemed to be determined to cope with her RA, and had adopted an attitude of acceptance and patience (particularly with the long-term benefits of Sulphasalazine) with her condition.

Evaluation:

Little changes occurred in her scores on the key measures. Figure 4.9 shows the extent to which there was minimal change in mood disturbance (TMD), perceived coping efficacy (PCETOT), and inflammation (RAI). A slight reduction occurred in her pain rating index (PRIT), which was unrelated to the changes in ethrocyte sedimentation rate (ESR). Helen maintained her status as someone with a moderate perception of her ability to cope with stressful events, and with a somewhat unstable mood disturbance profile.

Incomplete lymphocyte proliferation data prevents a meaningful analysis of the possible status and changes over time in immune function (see Table 14.2). At post-intervention, however, Helen showed a subnormal lymphocyte proliferation response to CON-A (38.0%). This result is difficult to interpret considering her average health status, moderate disease activity, and slightly unstable mood profile. It is possible that the medication contributed to this result.

4.10 Case C(206): "Asha".

Medical History:

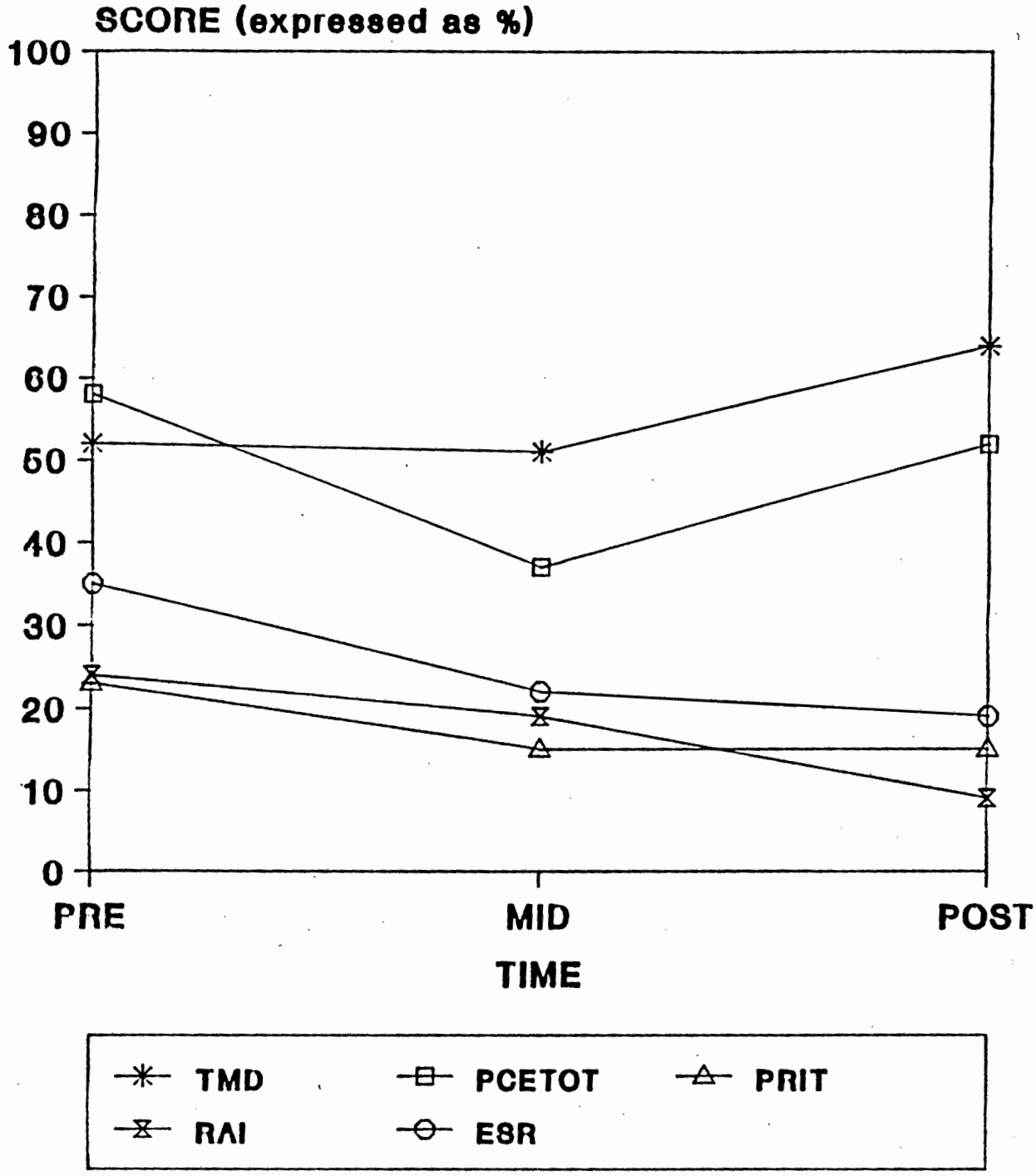
RA was first diagnosed in 1986. RAI scores indicated widespread but mild disease activity. Moderate joint deformity had occurred in the hands and fingers and splints were necessary to protect the joints from further destruction. "Discomforting" (Present Pain Intensity) pain was experienced in the neck, shoulders, wrists, fingers, lower back, hips, knees and ankles. Her treatment regimen included monthly visits to the clinic, and daily medication. The latter included: NSAIDs (Feldene and Arthrexin), and an analgesic (Dolorol).

Subject:

"Asha" is a 34 year old, married, "coloured" woman from Mitchells Plein. Although "medically unfit for work", she earned a living as a seamstress from home, supporting three children. Her minimal mood disturbance (elevated scores on Anger factor only) was accompanied by a relatively good perception of coping efficacy in stressful events. Increases in her pain followed periods of "too much activity" (e.g., cutting fabric with scissors), and Asha found that rest decreased her pain. She also relied on medication, massage, and monthly physiotherapy to control her pain. Asha had developed her own daily exercise programme lying in a hot bath. She found that "taking life as it comes and avoiding stress" were essential to managing her pain.

FIGURE 4.10: Case C(206) Asha

Control Group



Evaluation:

Figure 4.10 shows the extent to which changes occurred in the key variables over the duration of the intervention. A general reduction occurred in ethrocyte sedimentation rate (ESR), Ritchie Articular Index (RAI), and pain rating index (PRIT), indicating an improvement in disease activity. These improvements occurred independent of the increase in mood disturbance (TMD), and drop in perceived coping efficacy (PCETOT). Lymphocyte proliferation results were well within the healthy range of response at all assessment points (see Table 14.2).

4.11 Case C(209): "Anna".

Medical History:

RA first diagnosed in 1978. Current disease activity mild, but widespread. "Distressing" (Present Pain Intensity) pain experienced in upper back, forearms, hands, and feet. Other complaints include gastric ulcers and endometrical pain. Joint deformity minimal (i.e., no overt signs of RA), with mild joint impairment. Her treatment regimen included monthly visits to the clinic, and daily medication. The latter included: a NSAID (Indocid), an analgesic (Syndol), and a sedative (Serepax).

Subject:

"Anna" is a 43 year old, divorced, "white", woman working as a estate agent. Her extremely poor mood disturbance (extremely high scores on Tension, Fatigue and Confusion factors), was accompanied by a relatively good perception of coping efficacy in stressful events. Her pain increased during periods of fatigue, at midday, "sitting in one position for too long", and overuse of limbs (e.g., typing). Events which decreased her pain included walking, swimming, resting and relaxation (e.g., painting, reading). She relied on daily medication, bedrest, and heat applications to control her pain. She felt that her pain should be stopped before it got too severe.

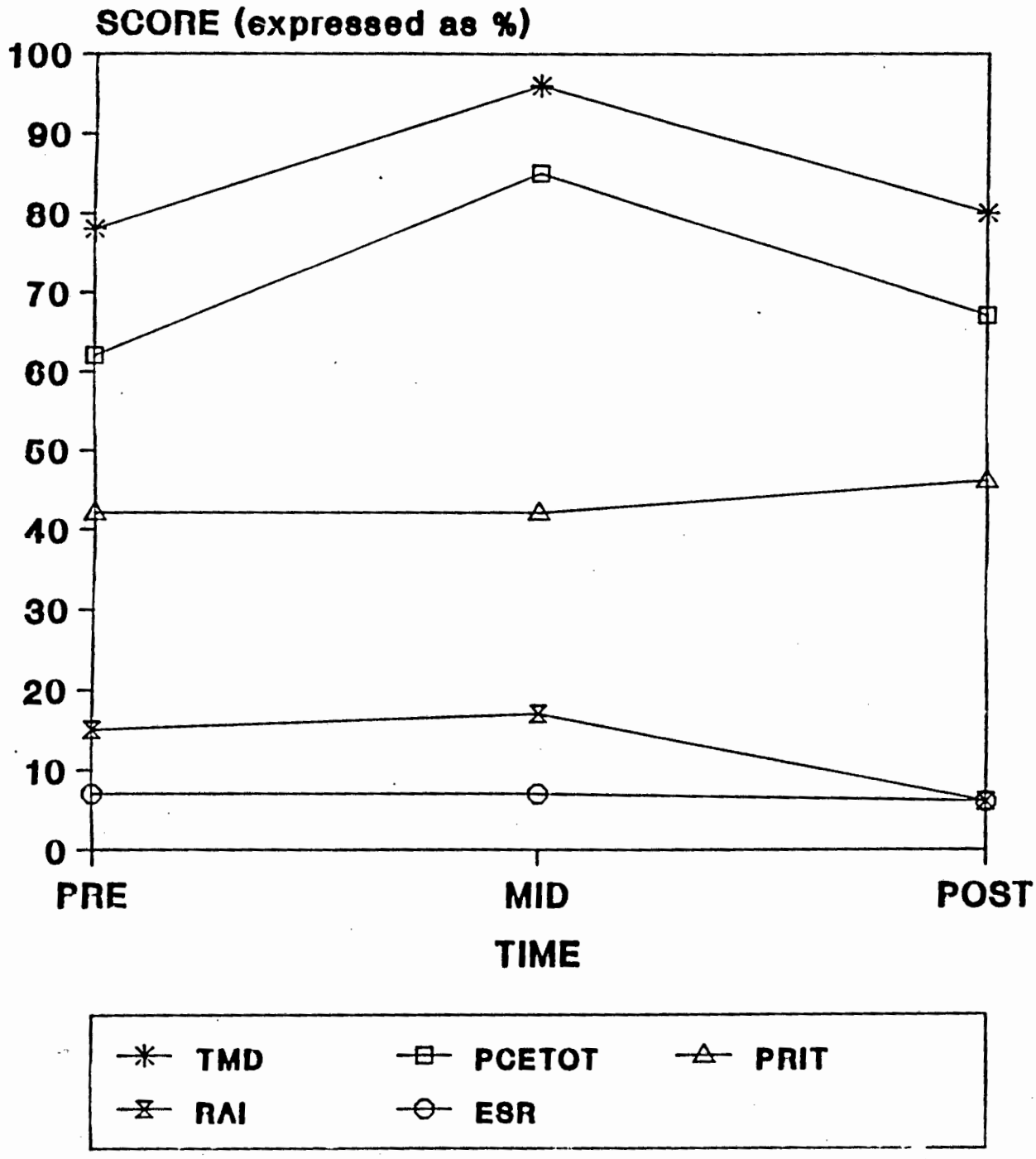
Evaluation:

No changes occurred in her high pain rating index (PRIT) scores, and low ethrocyte sedimentation rate (ESR) results over the duration of the intervention (see Figure 4.11). Her Ritchie Articular Index (RAI) scores showed an improvement by post-intervention. The dramatic increase in mood disturbance (TMD) by mid-intervention presented an extremely unstable mood profile. Ironically, this was associated with an increase in perceived coping efficacy (PCETOT). Both these latter two changes returned to baseline levels at post-intervention. Lymphocyte proliferation rates were within the normal range of response over the duration of the intervention (see Table 14.2).

The pattern of changes shown in Figure 4.11 do not present any meaningful relationships between the variables over time. Changes in mood disturbance did not affect pain levels, disease activity or immune response. It is possible that the use of a sedative influenced her mood state through elevated fatigue scores.

FIGURE 4.11: Case C(209) Anna

Control Group



4.12 Case C(210): "Shamilla".

Medical History:

RA was first diagnosed in 1980. Mild disease activity indicated, with joint impairment in the hips and legs. Mobility restricted, requiring walking stick during recovery from hip replacement operation. "Discomforting" (Present Pain Intensity) pain experienced in legs, hip, and arms. Her treatment regimen included monthly visits to the clinic, and daily medication. The latter included: NSAIDs (Arthrexin and Voltarin) and an analgesic (Syndol).

Subject:

"Shamilla" is a 56 year old, married, Moslem woman from a middle class area. She showed moderately high mood disturbance (high scores on the Tension and Anger mood factors), yet relatively good perceptions of coping efficacy in stressful events that she had experienced. Upon entry in the study, pain was worse in the early morning, following over activity, and "when having to worry about my daughter". Decreases in pain were associated with bedrest, medication, and "by taking my mind off the pain".

Evaluation:

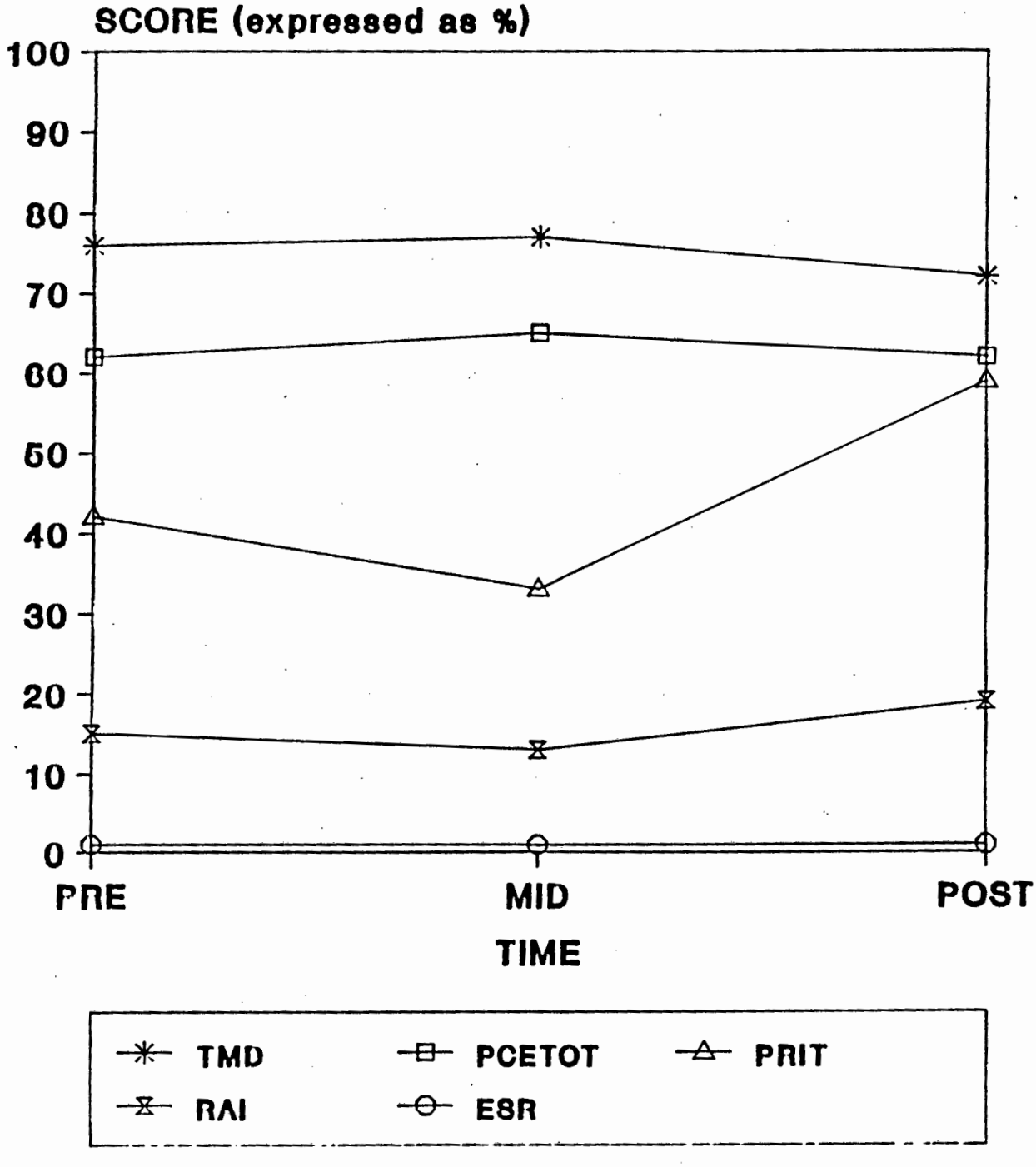
Little change occurred in mood disturbance (TMD) and perceived coping efficacy (PCETOT) scores. Figure 4.12 shows the extent to which changes in pain rating index (PRIT) did not influence the former two variables. Ethrocyte sedimentation rate (ESR) and Ritchie Articular Index (RAI) results indicated mild disease activity not related to the sharp increase in pain from mid- to post-intervention.

Missing data prevented a conclusive account of baseline lymphocyte proliferation results (see Table 14.2). However, subnormal response to PHA (43.3%) and low responses to PWM (97.5%) and CON-A (90.4%) indicated poor lymphocyte responsivity to in vitro stimulation. A similar subnormal response to CON-A (45.6%) was evident at post-intervention, despite response rates to PHA and PWM being well within the healthy range. These changes in lymphocyte proliferation results are difficult to interpret, considering the stability of medication, disease activity indices, and mood disturbance. It is possible that changes were occurring which the measures were unable to identify (e.g., the lack of extensive haematological data).

The pattern of changes that occurred in Shamilla's results are not consistent with expected inter variable relationships.

FIGURE 4.12: Case C(210) Shamilla

Control Group



4.13 Case C(211): "Rose"

Medical History:

Following an acute onset of symptoms, RA was diagnosed in 1976. Current disease activity is moderate and widespread, with "distressing" (Present Pain Intensity), "rhythmic" pain experienced in jaw, shoulders, back, arms, wrists, hands, knees, ankles, and feet. Joint deformity and impairment in hands and feet. Other complaints include fatigue, abdominal discomfort and extreme headaches. Her treatment regimen included monthly visits to the clinic, and medication. The latter included: a remission-inducing agent (Chloroquin), a NSAID (Voltaren), a relaxant (Ativan), and an analgesic (Para-Codeine).

Subject:

"Rose" is a 62 year old, widowed, "white", pensioner from a middle-class area. Her fairly high mood disturbance (elevated scores on Tension, Depression, and Confusion factors) was accompanied with a moderate perception of coping efficacy to stressful events. Pain was worsened following too much activity, in the early morning, and late at night. She relied on medications, bedrest, relaxation (e.g., reading), hot baths, and immersion of hands into cold water to control her pain.

Evaluation:

No meaningful patterns emerged in the small changes in Rose's scores on the key measures. Figure 4.13 shows the extent to which an increase in mood disturbance (TMD) was unrelated to the reduction in pain rating index (PRIT), ethrocyte sedimentation rate (ESR), and perceived coping efficacy (PCETOT) variables. Her anecdotal account of a deteriorating relationship with her daughter possibly contributed to her overall mood disturbance. The increase in Ritchie Articular Index (RAI) indicating increased disease activity and joint inflammation was the only variable to follow a similar pattern to that of TMD. This increase in disease activity, however, could be accounted for by her cessation of NSAID medication during the intervention. Lymphocyte proliferation results were well within the healthy range of response at baseline and post-intervention assessment (see Table 14.2).

FIGURE 4.13: Case C(211) Rose
Control Group

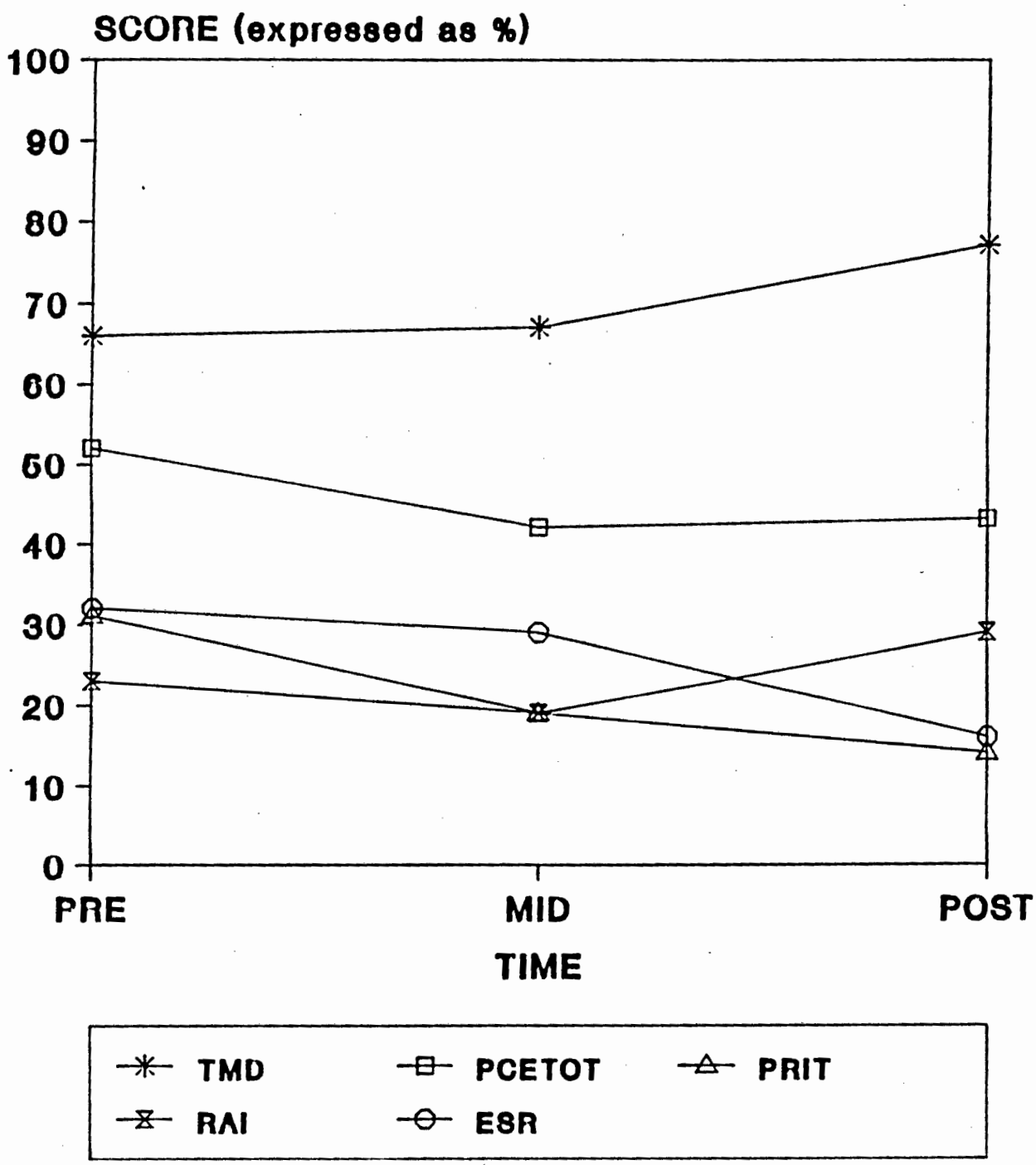
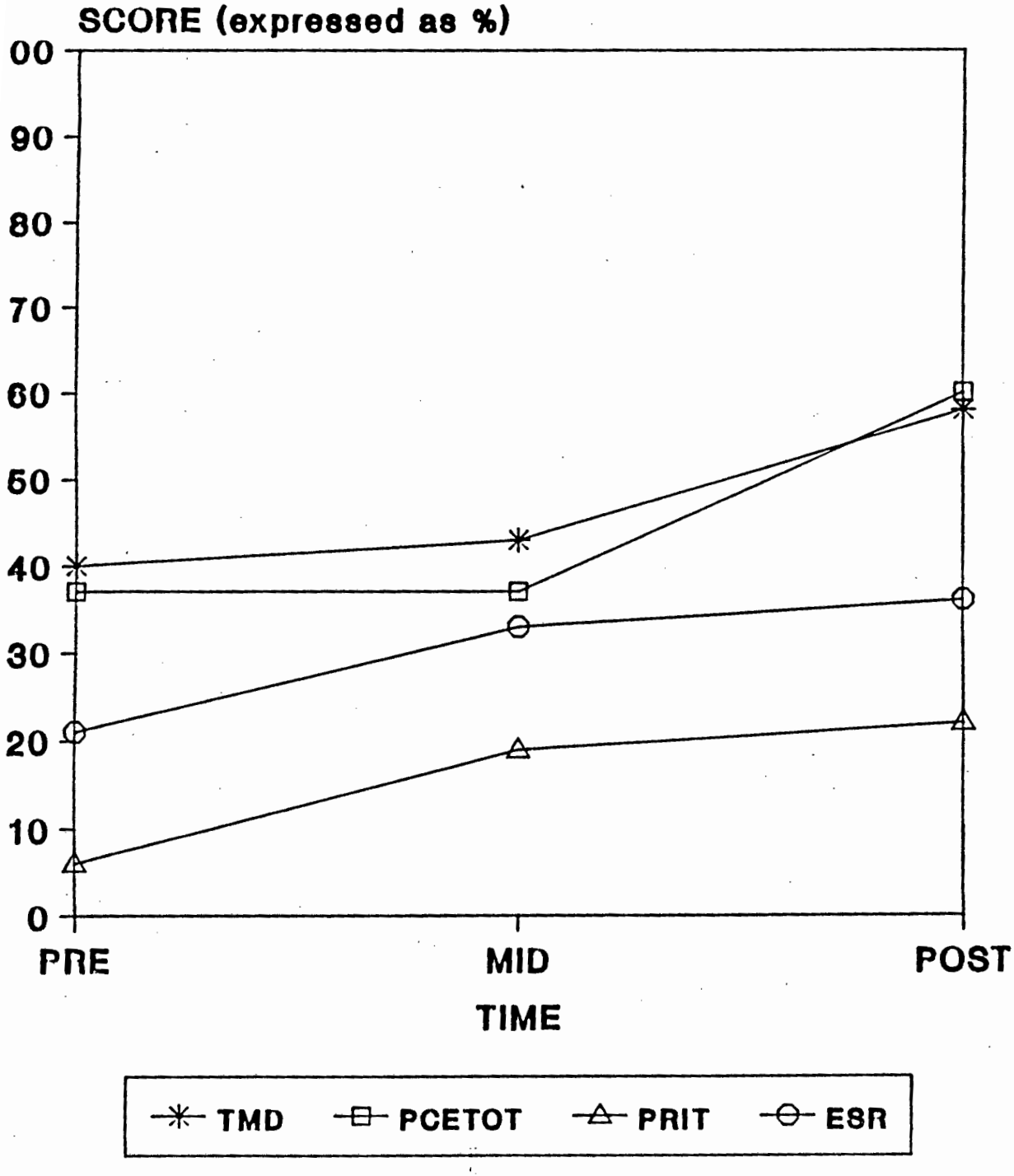


FIGURE 4.14: Case C(212) Jane

Control Group



4.14 Case C(212): "Jane".

Medical History:

RA diagnosed in 1981. Mild disease activity, with little joint impairment or discomfort. "Mild" (Present Pain Intensity) pain was confined to shoulders, knees, and ankles. Her treatment regimen included monthly visits to the clinic, and daily analgesic (Panadeine) medication.

Subject:

"Jane" is a 50 year old, single, "coloured" nurse from a middle class area. She showed minimal mood disturbance and had moderate perceptions of coping efficacy to stressful events. Pain increased during the night, early morning (i.e., stiffness.), and following "too much or too little activity". Daily medication, daily massage, heat application (e.g., hot baths), and ignoring the pain, provided pain relief. She felt that physiotherapy would be of benefit to her shoulders.

Evaluation:

Figure 4.14 shows the gradual deterioration in Jane's health over the duration of the intervention. The close relationship between the psychological, pain and disease activity variables concur with the predicted nature of such relationships. Despite an increase in perceived coping efficacy (PCETOT); mood disturbance (TMD), ethrocyte sedimentation rate (ESR), and pain rating index (PRIT) scores followed a similar deteriorating pattern. Missing data for Ritchie Articular Index (RAI) and lymphocyte proliferation variables (see Table 14.2) prevent a comprehensive analysis of the predicted psychological, pain, and immunological relationships involved. Despite the deterioration, Jane's results concur with the predicted nature of the relationships between these variables where changes in mood disturbance were accompanied by changes in pain and disease activity.

CHAPTER SEVEN

DISCUSSION

1. The Impact of RA

The results of this study demonstrate that RA has a significant impact on the psychological adjustment of RA patients, thereby confirming one of the predictions of this thesis. Poor psychological adjustment was characterized by marked mood disturbance (i.e., elevated feelings relating to depression, anger, tension, and fatigue), and poor perceptions of coping efficacy in stressful events. The finding that RA patients in this study generally selected interpersonal conflict/problems as being important stressful events is unusual. It was expected that more patients would have identified acute disease/pain episodes as being particularly stressful. The poor perceptions of coping efficacy thus seem to be more a function of factors in the patient's interpersonal environment (i.e., "external") than perceptions of an inability to cope with factors relating to their illness experience (i.e., "internal"). It seems that these patients feel that they are less able to respond effectively in stressful events involving significant others than in coping with pain and discomfort associated with their disease (see section 2.1.4 below). This process of negative self-appraisal may have adverse effects of their general sense of well-being, influencing their ability to adapt to their disease. As Liang et al. (1984) wrote in their discussion of their findings regarding the psychosocial impact of RA: "they appear to be reacting both to the symptoms of disease and to the life changes created by those symptoms." (p. 17) In the case of these RA patients, it is the latter which constitutes the key stressor (see section 2.1.2 below). The fact that the patients' internal health locus of control was relatively normal for a chronic patient population, is perhaps a further indication that perceived helplessness in controlling health is not necessarily an underlying factor affecting these patients' negative self-appraisal of coping efficacy.

The high levels of pain reported were consistent with expected findings, with elevated scores on the cognitive (i.e., evaluative and sensory pain dimensions) and behavioural (i.e., impact in terms of psychosocial functioning) aspects of the pain experience. Such high levels of reported pain occurred in the context of mild yet widespread disease activity in the joints. These findings demonstrate that pain experienced by the RA patient, is not merely a function of the degree/amount of tissue or joint inflammation. Whilst the intensity of pain experienced by these patients was relatively moderate, the meaning that they attached to their pain was particularly salient. The RA patient's subjective experience of her pain was thus primarily determined by her appraisal of the pain sensation. Given the tendency for patients and medical practitioners to generally interpret pain sensations as indicative of damage to the joints, it is not unusual for these patients to interpret their pain as being a further threat to their well-being. A common response by these patients to their pain being: "I feel pain, therefore there must be something wrong". Liang et al.'s (1984) finding that RA patients had elevated scores on the Hyponchondriasis and Hysteria scales of the MMPI, possibly confirms the extent to which they show a negative appraisal of their pain sensations. This

interpretation would also account for the finding that these RA patients were preoccupied with the nature of the actual pain sensation.

To demonstrate this point more clearly, it is necessary to relate the researcher's personal experience whilst working with the patients. During the group discussions, the patients had to describe the effects of their disease on their lives and their perceptions of themselves as able women. There was a strong tendency for these RA patients to "communicate through" their pain experience. This was particularly salient when addressing the researcher (who was initially addressed as "doctor"; i.e., perceived as an important member of the health care profession). Their response to questions about their well-being typically entailed a detailed description of their pain. Such a description included: (a) the site and nature of the pain sensation, (b) their suffering as a result of the pain, and (c) the effect the pain was having on their lifestyle (i.e., appetite, sleep, energy levels, house work, etc.). As one subject said: "I am so sore, the pain in my hips is like a hot knife sticking through me, I can't seem to do anything at all at home when the pain gets so bad, and my children do not understand how this arthritis takes over your body". Such a description was generally accompanied by facial and bodily signals of despair and suffering. An interesting observation was the initially angry response of the patient when the researcher purposefully ignored this "meta-communication" (i.e., the researcher interpreted such communication as an expression of need: e.g., "help me, I am suffering"). The patient would then offer a description of her well-being wherein less emphasis was placed on symptom complaints and her suffering, than on practical issues and difficulties. Previous research's findings that pain is the best indicator of health status for RA patients in terms of perceptions of well-being and medication usage supports this observation (see Kazis et al., 1983).

The findings from the initial analyses of the sample's baseline characteristics, thus indicate that RA was characterized more by poor psychological health status (i.e., the patients' illness experience) than physical disability (i.e., functional status and disease activity). This finding is significant in the sense that RA is generally more known for its impact on the physical well-being of such patients, than the psychosocial ramifications. An interesting phenomenon is the extent to which the subjects complained how family members and friends only expressed concern and support when there were observable manifestations of the disease (e.g., swelling in the joints, nodules, or joint disfigurement). This point highlights the emphasis usually placed on the physical aspects of the disease. An "error of judgement" is thus common in the illness experience of the patient, as well as the attitudes of health care workers or significant others. This process entails limiting one's attention and concern to the physical dimension of RA (i.e., the disease itself) at the expense of the psychological and social dimensions. Given the extent to which the prognosis of the RA patient is determined by the adherence to complex and long term treatment goals, there is clearly the risk that such an "error of judgement" would adversely affect the patient's disease status through maladaptive responses to the disease. Such treatment would include compliance with medication usage, adaptations in life-style such as limiting injury to joints through safe exercise and appropriate joint use, and maintaining a positive and constructive attitude towards their disease.

2. Support for the Proposed Model

The results of the correlational analyses only partially support the model proposed in Chapter Four. The predicted nature of the interrelationships among the psychological, pain, disease activity, functional status and immune function variables, could only be partly confirmed. The correlational analyses sought to establish both the general interrelationship among the key variables, as well as the extent to which psychological adjustment could be seen as playing a mediating role as indicated by the model. The results indicate that pain was the only variable significantly associated with psychological adjustment. Few significant correlations were found among the other key variables.

2.1 Important Findings

The non-significant results from the pre-intervention correlational analyses have value in that they highlight both the complexity of the relationships under investigation, as well as some of the problems inherent to research in this area.

2.1.1 Health Locus of Control: A poor Predictor of Psychological Adjustment in RA

These RA patients' health locus of control (particularly their internal health locus of control) was unrelated to general psychological adjustment. This finding does not concur with the predicted role of patients' beliefs regarding their ability control or determine their health, in influencing their response to their disease. It is possible that the particular focus of this variable (i.e., it is directly related to factors or events contributing to general health) resulted in the exclusion of an analysis of the beliefs the patients' held regarding their ability to manage the particular characteristics of RA. An earlier study had similar inconclusive findings regarding the role of RA patients' health locus of control in terms of the impact of RA, and in relation to other psychological factors (see Liang et al., 1984).

Locus of control has generally been considered an important psychological factor in determining an individual's perception of his/her ability to control or cope with stressful events or circumstances. It is possible that the emphasis on health biased an evaluation of learned helplessness in this study. The recent construction of an Arthritis Health Locus of Control measure (Wallston, personal communication, June, 1990) might perhaps clarify the role of RA patients' cognitions regarding factors determining their disease progression, and their responses to their disease and/or treatment. Wallston et al. (1983), for example, demonstrated the extent to which individual's expectancies about their control over their health (i.e., their health locus of control) were related to their preferences to get actively involved in their own health care. It is possible that certain sub-groups of RA patients would derive greater benefit from cognitive-behavioural therapy (which promotes and encourages self-efficacy in the management of the disease effects) than others. An important finding from Wallston et al.'s (1983) study was the extent to which persons who believe their health is controlled by powerful others, are less likely to comply with treatment encouraging self-treatment or active behavioural involvement in medical care. Cognitive-behavioural interventions in RA should thus consider and address the beliefs patients hold regarding their control over their

health, to ensure maximum co-operation in treatment programs. Later on, this point will be illustrated in the case of two of the participants in the treatment group (see section 3.1).

2.1.2 Mood Disturbance in RA

Mood disturbance was not only found to be a particularly important index of psychological adjustment in RA patients, but also the only psychological variable to be associated with any of the other non-psychological variables (i.e., pain). The inverse relations between mood disturbance and both perceptions of coping efficacy and psychological health status, is self evident. The greater the mood disturbance in RA patients, the less adequate their self-perceptions of coping efficacy in stressful events. Mood disturbance can thus be considered an important index of psychological well-being in RA. The latter finding highlights the extent to which successful pain management should include a consideration of the RA patient's mood state.

The exact nature of the relationship between mood disturbance and coping efficacy, in terms of the direction of causality, remains a moot point. On the basis of Parker et al.'s (1988b) findings that coping processes mediate the relationship between disease effects and psychological status, it was predicted that mood disturbance would be more a function of self-perceptions of coping efficacy than vice versa. Given the surprisingly poor relationship found between coping efficacy and stressful life experience (considering the type of events identified as stressful), the findings of this study possibly clarify this difficult question. It seems that it is the degree of mood disturbance which determines the RA patient's self-perceptions of their ability to cope with stressful events in their lives. In other words, the greater the mood disturbance, the poorer the RA patient's perception of her ability to respond effectively to stressful events. The psychological impact of the disease effects thus seem to adversely affect the RA patient's self-perception of her ability to cope with daily hassles. As one subject said, "when my pain is very bad, and I feel I cannot go on anymore, I just can't face the world." In this sense, affective factors are shown to influence the cognitive appraisal processes in response to stressors. This interpretation probably indicates that an intervention which concentrates solely on cognitive factors, would have difficulty in improving their perceived coping efficacy. The affective state of the RA patients must be addressed. This will be discussed in greater detail below (section 3.2).

Such findings can be corroborated by the researcher's experiences during the intervention. The individual and group discussions consistently indicated the extent to which the mood of the participant influenced either her account of how she was coping with having RA, or her response to instruction in the various coping strategies during the second phase of the intervention. Furthermore, whilst the subjects identified (a) difficulties in their family life, or (b) a preoccupation with their private thoughts/emotions, as factors influencing their mood, they all consistently highlighted pain and discomfort as the major factors influencing how they were feeling. Problems with family members who are not being considerate or trustworthy, financial strain, feeling old and unattractive or lonely, being rejected by spouse or family, or self-appraisal of one's ability to continue coping with disease effects were all secondary factors influencing mood.

This anecdotal observation is supported by the significant association found between measures of pain experience and mood disturbance (see 2.1.3 below). It is interesting to note that these findings do not support Hawley and

Wolfe's (1988) observations. Hawley and Wolfe (1988) found that patient characteristics and life experience, rather than possibly the disease itself, contributed to RA patient's psychological adjustment. In a sense, Hawley and Wolfe (1988) possibly underestimated the psychological impact of RA disease effects.

2.1.3 Mood Disturbance and Pain Experience

The significant relationship which was found to exist between psychological adjustment (particularly mood disturbance) and pain experience corroborates the findings discussed in the section above. Elevated feelings associated with depression, anger, and tension were common disturbances in mood in this sample. This finding is supported by evidence from the research literature (see Chapter Three, section 3.1.2).

The exact nature of the relationship (in terms of causality) between mood and pain in RA has yet to be clarified. . Again, the findings from this study may provide some pointers to this problem. It is possible that disturbed mood states play an important role in determining the overall pain experience. That is, the level of mood disturbance influences the RA patient's appraisal of her pain sensations (i.e., the meaning of the pain). Given the findings that pain in these RA patients was more a function of cognitive/affective and behavioural factors than a function of tissue damage/disease activity (i.e., the meaning of the pain was the most salient factor of the pain experience: see section 1 above, and section 2.1.4 below), it is possible that psychological factors contributed to exacerbating the pain experienced by these patients.

The central role of affective factors (i.e., in this instance, mood states) in the appraisal process (in terms of the subjects' perceptions of coping efficacy as well as their pain experience) are thus is being highlighted in the findings of this study. Reisenzein's (1983) critical appraisal of the Schachter theory of emotion demonstrates the extent to which affective factors (i.e., emotional beliefs) "colour" the cognitive appraisal of an event, ultimately determining the experienced emotion (in this instance the pain experience). Everly's (1988) model does account for the central role that affective factors play in the appraisal of stressors. In this instance, the stressor is the pain sensation(s). Elevated mood disturbance possibly influence the patients' appraisal of their pain, thereby heightening the subjective experience thereof.

Such an observation can be supported by the results regarding the non-significant correlation between disease activity variables and pain measures. This important finding indicates that the pain experience of the sample is more a function of psychological distress than actual physical impairment or joint destruction. The subjective or phenomenological experience seem to outweigh the physical level of the pain experience. This is discussed in greater detail in the next section.

2.1.4 Pain: Not merely a Function of Disease Activity

A poor relationship was found to exist between pain experience and disease activity. It is argued that this finding provides substantial support for the above delineation of the role of mood states in determining pain as experienced in RA. The finding also confirms the prediction that pain is more a function of psychological

adjustment than actual disease status, with psychological adjustment mediating the effects of the disease symptoms and the patient's experience thereof.

Given these above findings, it is possible to postulate the causal process linking disease symptoms (including pain sensations) and the ultimate subjective (personal) experience and the behavioural impact thereof. A process of appraisal mediates the pain sensations, and thus determines the ultimate pain experience of the RA patient. This appraisal process, however, is influenced by affective (i.e., mood state) factors. These factors influence and determine the meaning of the pain sensation to the patient in terms of the threat to his/her well-being.

2.1.5 Disease Activity and Immune Function: Unrelated to Psychological Adjustment

A final important finding of the correlational analyses is the extent to which variables indicating disease activity and lymphocyte function were not related to the psychological, pain, or functional status of these patients. This finding was unexpected, as it was predicted that patients' perceptions of their ability to cope with stressful events (including acute disease episodes), and their mood state, would mediate the relationship between disease activity and functional status. The patient's appraisal of the extent and nature of her disease should determine, to some extent, the influence that the disease has on physical functioning; particularly when such functioning was rated by the patient. As functional status was assessed through a self-report measure (i.e., the AIMS), it was expected that the response of the patient to items concerning the impact that RA has on mobility, activities of daily living, etc., may be influenced by underlying cognitive and affective factors. This was not demonstrated in this study.

The non-significant relationship demonstrated between lymphocyte function and psychological adjustment did not support the findings in the psychoimmunological literature. Given the mood disturbance of these patients, and the associations between depression, distress and anxiety, and variables of immune function demonstrated in earlier research (see Chapter Two, section 5), it was predicted that these patients would show some immune function "dysregulation" (term borrowed from Jasnoski & Schwarz, 1985). The finding that all of the patients had lymphocyte proliferation response rates which were well within the healthy range of response, can be interpreted in two ways. Firstly, this demonstrates that this index of immune function is not the most appropriate index to assess in psychoimmunological interventions with RA patients. Secondly, lymphocyte function in RA is unrelated to the disease process. O'Leary (personal communication, 1990) argues that given the heterogeneity of RA immunology, psychoimmunological research with such patient populations would require extensive measures of immune status and function. The present finding, according to Dowdall (personal communication, 1990), demonstrates that lymphocyte proliferation rate is only an appropriate measure of immune function in patient populations characterized by gross immune deficiency (e.g., HIV infected individuals, patients receiving immunosuppressive therapy, etc.).

2.2 Conclusions

The above findings confirm the value of determining the psychosocial impact of RA on such patients. That psychological factors are directly related to pain experience, independent of the level of disease activity, clearly

demonstrates the need for consideration thereof. The findings also indicate the complex nature of the relationships between anomalous variables. These relationships are partly determined by the specific operationalizations of the variables, and their relevance to RA. Health locus of control, and lymphocyte proliferation rate are two measures which seem to have little relevance in RA.

The next section discusses the results of this study in terms of the value of a second important area of psychological intervention in RA: the benefits of running cognitive-behavioural pain and stress management programs for RA patients.

3. The Value of Cognitive-Behavioural Intervention

The results of the analyses of intra- and inter-group changes over time along the key variables, did not corroborate other findings (see Chapter Four, section 4.2) of the benefit of cognitive-behavioural treatment of RA.

The Stress Inoculation and Pain Management Training Programme (SIPMT) was not shown to produce significant change along any of the measures of the key variables over time. The prediction that the intervention, would lessen the impact of the disease (i.e., improve pain experience and psychological adjustment) could thus not be confirmed. This finding is contrary to those of similar interventions with RA patients reviewed in Chapter Three (e.g., Randich, 1982; Bradley et al., 1984; Bradley et al., 1987; Parker et al., 1988b; and O'Leary et al., 1988).

A common finding of cognitive-behavioural treatment of RA, is the change in perceptions of self-efficacy and an improvement in coping strategies to reduce the impact of the disease, rather than any significant improvement in the psychological well being (i.e., in terms of depression etc.). This was not demonstrated in the above analyses of change in perceived coping efficacy. It is possible, however, that the bias the subjects showed towards selecting interpersonal events as stressors prevented any consideration of change in their self-perception of coping efficacy with their pain. In this study then, the poor results are probably also the result of a poor operationalization of coping efficacy. A measure of the pain coping strategies employed by the subjects would have shed more insight into the effects of the intervention. The non-significant change in mood disturbance and psychological health status (i.e., depression and anxiety), concurs with Parker et al.'s (1988b) findings. Whilst Parker et al. (1988b) were unable to demonstrate significant change in psychological well-being, they were able to show improved coping skills and enhanced self-efficacy in their sample. Other studies though (e.g., Bradley et al., 1984, 1987; O'Leary et al., 1988), have demonstrated significant improvements along measures of psychological status. These contradictory findings indicate both (1) the difficulty in achieving some consensus in the operationalization of treatment outcome in clinical interventions, and (2) the differential effect of cognitive-behavioural programs.

The inability of the intervention to produce change along disease activity (i.e., joint inflammation and ethrocyte sedimentation rate) and immune function variables concurs with the findings of Parker et al. (1988b) and O'Leary et al. (1988). An important finding of this study is the difficulty of determining change in lymphocyte proliferation rate. In this study, almost all the patients had response rates which were well within the normal range of response. According to the researcher running the proliferation assays, if there is a healthy response rate at onset, and there

is no subsequent drop to an abnormal level of response, it is meaningless to talk about an "improvement" in this index of immune function (L. Weight, personal communication, September, 1990). The failure of previous studies to report the extent to which their sample fell within a "normal" range of lymphocyte response, highlights a significant methodological inadequacy of a number of studies in psychoimmunology. There is a tendency to report significance, when in fact it is meaningless to report "improvement" over time. If one's results are within the normal range, one cannot "get better".

The fact that all of the patients' lymphocyte proliferation rate measures were normal, also indicates that it is not an appropriate index of immunocompetence in RA. It is possible that analyses of T- and B-cell ratios, as well as circulating immunoglobulins, would have been better indices of immune function in RA. This corroborates the findings of O'Leary et al. (1988).

3.1 Factors Affecting Poor Outcome

There are a number of possible factors contributing to the inconsistency between this study's findings in terms of the benefit of cognitive-behavioural intervention with RA, and those cited in the literature. Such factors include methodological inadequacies in terms of the sampple size and assessment of treatment outcome, as well as the relevance of the statistical procedures employed. In the former, one factor is the use of dependent measures which did not: (1) assess changes in the subjects' perception of their ability to cope with pain in particular, nor (2) establish the types of coping strategies being utilized. In the latter, the limitations in the actual data undermined the value of the statistical procedures utilized to test change over time. Such limitations included: (a) the small sample size; (b) the large intra-group variance (particularly in the treatment group) in scores along the key variables; (c) the existence of extreme scores; (d) the heterogeneity of the sample; and (e) the incidence of missing data. The analyses relied on mean change scores, and it is possible that the clinical benefit for individual patients may have been obscured in the process. This can best be demonstrated in the changes in Total Mood Disturbance in the two groups. The treatment group showed a slight drop in their mean TMD change score (-0.57 $\pm$61.3), which differed from the increase in the control group's TMD (8.42 $\pm$22.4). The greater variance in the former (i.e., existence of possible extreme scores) possibly masking any significant change that may have occurred.

It is possible to argue that an important finding in this study is the value of combining both quantitative and qualitative procedures to evaluate the benefit of cognitive-behavioural intervention with RA patients (or any patient population for that matter). It is argued that to solely focus on results of statistical analyses, and draw conclusions based on the significance or non-significance thereof is a self-defeating exercise.

A central problem inherent to this research, thus involves the difficulty assessing the clinical benefit (i.e. treatment outcome) of psychological intervention for a small number of patients.

3.2 Individual Change: The Findings of the Case Studies

The findings from the case studies, adopted to examine the individual patients' reactions to the treatment and/or disease effects, provided further insight into clarifying the nature of the interrelationship among the key variables.

Individual assessment allowed for closer analysis of possible patterns of changes occurring which an analysis of group means masked. The heterogeneity of the treatment group became apparent, where the psychological adjustment, pain experience, and disease status of each subject was considered in the context of that subject's particular characteristics.

3.2.1 Patients in the Treatment Group

Three out the seven patients in the treatment group were found to benefit from the intervention. All three (Gafsa, Alfreda, and Francis) showed reductions in mood disturbance and pain experience, and improved perceptions of coping efficacy. Such change was in accordance with the predicted nature of the relationship among these variables. An important finding, moreover, was that such changes were independent of any observable change in disease activity or joint involvement (despite the fact that both Alfreda and francis were on remission-inducing therapy). In the case of one patient, the improvement in mood and stability of pain experience and joint inflammation occurred despite a significant (30%) increase in ethrocyte sedimentation rate. A further important finding from the case study approach was that two of these three patients who benefitted from the intervention had relatively high scores on the internal health locus of control measure. Such scores, furthermore, reflected a transparent belief that "it is up to me to do something about my pain and my arthritis". These two patients demonstrated an array of idiosyncratic coping strategies to deal with their pain, and found great comfort from the realization that what they were doing was considered "proven" (i.e., sanctioned by the health profession) ways of coping with the disease. In this sense, the programme concurred with their expectations about their ability to control their disease. This finding corroborates Wallston et al.'s (1983) research. In a clinical sense then, the intervention was of benefit for these three patient's psychological health status.

Of the four other subjects in the treatment group, only one (Dorian) showed a marked deterioration on most of the variables (despite an increase in perceived coping efficacy). No clear pattern emerged in the changes in the other three subjects' (Miriam, Dorothy, and Mary) progress over the duration of the intervention. These patients showed varying, and at times contradictory, beliefs about their ability to control their disease. For example, saying that "you are the doctor, can't you do something about my pain", yet at the same time complaining that their doctors did not give them any help in finding ways for they themselves, to do something about their pain. The responses of these other treatment group patients thus demonstrated the complex and individual nature of each patient's reaction both to their disease, and to psychological intervention. Whilst the researcher observed a remarkable change in the treatment group patients' attitude towards, and understanding of: (a) their disease and pain; (b) the treatment goals in terms of medication usage; (c) their own idiosyncratic responses to cope with pain; as well as (d) their interaction with significant others; these patients demonstrated no measurable benefit from the intervention. It is possible, therefore, that the measures utilized could not identify changes in knowledge and understanding of the disease, changes in appraisal of self-efficacy in managing disease effects and pain, and changes in appraisal of well-being despite the impact of RA.

The general increase in these patients' ratings of the quality of their pain is difficult to interpret. It was predicted that improving their understanding of their pain, teaching them to verbalize their pain more effectively, and instructing them in techniques to manage their pain, would result in lower pain rating index scores. This, however,

was not the case. A possible explanation for this result could be that the MPQ is a reactive measure in that it may be reflecting an improvement in the latter groups' ability to express the qualities of their pain experience, rather than an increase in actual pain. Such an explanation could possibly be an important finding from this study, as the McGill Pain Questionnaire, to the researchers knowledge, has not been evaluated in terms of this feature.

The description of the subjects' response to the intervention in section 3.3, has value in that it contributes to an evaluation of the clinical relevance of the study. Furthermore, it would highlight some of the problems inherent in cognitive-behavioural interventions of this nature.

3.2.2 Patients in the Control Group

With the exception of one subject (Jane), no consistent relationship between mood disturbance (TMD) and pain rating index (PRIT) was found in the control group. The same held true for disease activity. Unlike the treatment group, change in mood was not a good predictor of change in pain experience in this group. The relatively stable mood disturbance (TMD), perceived coping efficacy (PCETOT), and pain rating index (PRIT) scores relative to the treatment group are difficult to interpret. It is possible that for the treatment group, the intervention succeeded in exposing deep emotions regarding the impact that RA has had on their lives, as well as teaching them to describe their pain more effectively.

An interesting finding in the control group was the extent to which disease activity was shown to be dependent on medication usage. One of the subjects, Rose, showed an increase in RAI and ESR scores. Closer analysis revealed that she was no longer taking anti-inflammatory medication. It is essential for any research in this area to be able to account for changes in mediation when evaluating the success, or lack thereof, of psychological intervention. No psychoimmunological relationships could be confirmed in these individual case studies. The lymphocyte proliferation results are difficult to interpret as, where subnormal responses occurred, little evidence was present to explain such results. Such subnormal responses did not seem to be related to type of medication (e.g., steroidal treatment).

3.3 Clinical Experiences During Intervention

3.3.1 The Needs of the Patients

Upon entry into the programme, the patients' presented themselves as victims of both their disease, as well as the health care system. There was thus an fundamental expression of their needs. A frequent complaint was that they felt that not enough was being done to find a cure for the disease nor to alleviate their symptoms. They also felt that they were not given enough time with the consultants at the two out-patient clinics, and felt powerless to enquire about the meaning of their symptoms and the goals of the treatment/medication. They also felt that no one seemed to care about how they felt about the disease, and how it was affecting their lives. All patients felt that RA prevented them from leading normal lives, and that they all have had to come to terms with radical changes in their life-styles.

They indicated that they felt great loss in terms of their independence, their youth, and their vitality. They no longer felt attractive, and were fearful that the disease would further estrange them from their husbands or significant people in their lives. Given the negative social stereotype of RA, as being a disease affecting the elderly or weak, they felt frustrated with family members and friends who did not seem to understand the nature of the disease, particularly when there were no overt symptoms. On this point, the treatment group were very keen to praise the researcher as someone "who knew what they were going through". Such a response was even more extraordinary, as it was expressed during the first session. The researcher, however, recognized the extent to which the patients were both idealizing his capabilities (i.e., seeing the researcher as somehow providing that "cure", or answer to their problems), as well as attempting to set the standard of health care and support they expected.

3.3.2 Group Processes

Whilst the cognitive-behavioural intervention was directed at the cognitions and actions of the patients in response to their disease, it was apparent that it was imperative that their needs were addressed. The researcher responded by focussing the group discussions more on developing an emotional support group wherein these needs could be discussed and shared, than merely a discussion of coping strategies. In this sense, it is possible that the lack of improvement in mood state for most of the treatment group, can be explained by the effect that such group processes had on allowing these women to ventilate their feelings of loss in their lives as a result of RA. The researcher had a difficult task of adhering to the highly structured treatment manual, yet at the same time, addressing the needs of the group.

Both the treatment and control groups thus saw the researcher as someone who was going to help them with their disease and pain: that something was "going to be done to them". Despite the researcher's attempt to provide some education for the control group during their four sessions, it was apparent that they expected far more input (despite the initial explanation that they were merely going to be monitored to determine the impact of RA on their lives). A great deal of anger and hostility was thus expressed by the control group when they realized that nothing was "going to be done to them".

Group discussions in the treatment group revealed a sense of desperation or frustration as a result of not being able to communicate their needs to medical personal and significant others. Most felt that all they could do was "to suffer in silence". They also felt that they were an encumbrance to their family and husband, as their disease prevented them from leading "normal lives". Two patients reported a breakdown in marital relations, hinting that they were no longer able to satisfy the needs (i.e., sexual) of their husbands. A common fear in the married patients, was the husband having affairs "with younger, more attractive and able women".

Given the characteristics of the subjects in the treatment group (i.e., middle-aged women, coming from a low income group, having little education, having poor marital or familial support networks, and suffering from chronic pain), the researcher was faced with a particularly difficult group to "move" psychologically. Apart from their astonishing ignorance of the nature of their disease, it was necessary to translate somewhat difficult concepts into expressions which they could understand and relate to. Such a process included translating the concept into its Afrikaans equivalent, relying on colloquial expressions to confer meaning. The researcher thus relied on the

subjects' collaboration and assistance in helping each other integrate the information at a personal level. "Stress" for example, was understood as "senuwee", "nerves" with no apparent cause. They had great difficulty separating the event or stressor, from the phenomenological experience.

As the treatment progressed, a startling change began to occur in the patients' reaction to the attention, interest and concern of the researcher. Each session showed the patients undergoing a metamorphosis, beginning to dress more elegantly, be more preoccupied with their appearance, changing their hairstyle, and almost competing for attention. The powerful placebo effect of the male researcher's presence and concern was an important component of the intervention. As it was difficult to ignore this covert expression of sexuality and improved body image, the researcher decided to focus on these changes, and interpret the patients behaviour towards the researcher as a manifestation of their needs. In one particular group discussion following input from the researcher regarding effective communication techniques, the researcher hinted at the effect that RA had on the patients' sexuality, and the extent to which they experienced great loss of self-esteem and self-appraisal of sexual worth. In the intense and emotional response to this suggestion, four of the patients voiced their relief in finally recognizing "the extent to which RA had changed them", and "being given the opportunity to share their feelings without fearing further rejection".

The issue of dependency seemed to be of central importance in the patients' response to RA disease effects. At the onset of the treatment, the patients assumed a passive role in the programme. It was apparent to the researcher that there was an underlying expectation that they would receive treatment for their pain. When it was made clear to them that the effectiveness of the pain and stress management strategies to be taught to them relied on their personal and active involvement in the treatment, the response was unexpected. Most of the patients remarked that they were glad to realize that there was something they could do to cope with their pain.

Towards the end of the programme, however, a marked change occurred where the patients began to express their disappointment that "our relationship is coming to an end". It was clear that the group was entering into the termination phase. The task of the researcher to ensure that the degree of independence achieved by the individual patients was not adversely affected by this phase, was particularly important. If the cognitive-behavioural treatment is to succeed in enhancing self-efficacy and coping ability in RA patients, it was imperative that these patients did not develop a dependence on the person conducting the programme. The researcher's response was to prepare them for the final session by: (a) getting them to discuss their personal experiences during the programme in terms of self-development and interaction with other group members; (b) encouraging them to start thinking about what they would do when the programme was over in terms of support, utilization of skills, and making constructive changes in their lives; (c) allowing them to come to terms with the "loss" of the group, and ensuring that they did not perceive the ending as a further loss in their lives, but an important step in the right direction (i.e., cognitive restructuring). The researcher also ensured that they could all contact each other, and gave them a list of support services available to RA patients.

3.4 Conclusions

From the above findings, it is clear that a central factor to determining the efficacy of the cognitive-behavioural treatment of RA, entails the examination of the individual and phenomenological response of the patients to both the researcher and the treatment programme. The clinical benefit of cognitive-behavioural interventions may possibly only be confirmed through the juxtaposition of empirical data and clinical observation.

The value of cognitive-behavioural interventions also becomes a debatable issue. When and is how is it appropriate for psychological intervention of this nature? Considering the extent to which affective processes have been shown to influence the RA patient's appraisal of her ability to cope with both her pain as well as daily hassles, any intervention in RA should first address the emotional needs of the participants. Cognitive-behavioural interventions such as the SIPMT programme used in this study may possibly only have benefit following a support group wherein the RA patients are able to explore the extent to which RA has changed their lives. Such a support group should give them the opportunity to ventilate or come to terms with feelings associated with the psychological impact of the disease.

Only once the affective component of RA has been dealt with, would such patients be able to derive maximum benefit from cognitive-behavioural intervention. Such intervention helping them to develop a more adaptive cognitive appraisal of their disease and pain, and use of pain coping strategies to reduce the adverse effect of chronic pain on their psychological well being. Success in such instruction would possibly (given the findings of this study), reduce the pain experience as a result of RA, improve compliance to treatment regimens, and enhance the RA patient's psychosocial functioning. Ultimately, the rehabilitation of the RA patient would possibly be optimized.

4. Limitations of the Study

This study sought to test the validity of the conclusions reached by similar previous studies, by including the recommendations of earlier researchers (see O'Leary et al., 1988). Considering the findings of this study, a number of limitations are apparent (those which have already been discussed, will be summarized here).

4.1 Dependent Measures

It is possible that the dependent measures utilized may have been either too crude or inappropriate to assess the variables identified in the proposed model (i.e., in Chapter Four). Given the known validity and reliability features of the measures used, the latter option is more plausible. In retrospect, the variables in the model, particularly, psychological adjustment, were poorly operationalized. In RA, it is clear that psychological adjustment is a complex process involving mood state, personality characteristics, and coping behaviours. Considering that this study did not account for the latter two factors in terms of <u>how</u> the patient was responding to their disease (i.e., what coping strategies were they employing?), it is not unusual that "psychological adjustment" was not shown to be influenced by the intervention.

This operationalization of psychological adjustment also defines treatment outcome. To determine the value of cognitive-behavioural intervention, one should have clearly defined parameters of what would constitute meaningful psychological change. Given the general findings in the literature, at best one would expect cognitive-behavioural intervention to improve the coping strategies employed by the participants. The nature of the intervention defines the parameters of expected gain: cognitive and behavioural (see section 4.5 below). As no provision was made to determine change in coping strategies, a limitation of this study would be its inability to provide an account of, or measure, possible changes occurring in the subjects' pain coping strategies.

The findings of this study, in terms of the psychoimmunological relations in RA, indicate that lymphocyte proliferation rate is not a good measure of immunocompetence. There is limited value in including only one index of immune function. Financial (i.e., the high cost of laboratory materials), practical (i.e., lack of laboratory space and expertise), and "political" (i.e., achieving support and interest from professionals in the medical fraternity in terms of the relevance and value of the research) constraints are significant factors which determine the choice of immunological assay. This study was unable to rely on measures of immunoglobulin levels, percentage circulating T-cells, and ratios between helper- and suppressor T-cells.

4.2 Statistical Procedures

The value of the statistical procedures one employs are based primarily on whether or not one's data satisfies the assumptions upon which that statistical procedure is based. Furthermore, such statistical procedures rely on mean and standard deviation scores. Given the small sample size in this study, the heterogeneity in subject characteristics, the large variance in intra-group scores along the key variables (e.g., the existence of extreme scores), and the incidence of missing data, the value of the statistical procedures employed in this study are questionable. This is particularly so for the correlational matrix, t-tests and Wilcoxon Rank Sum tests employed to determine intra- and inter-group change over time.

There is much value in examining the effects of cognitive-behavioural intervention for RA patients, by examining changes in the key variables for individual subjects. It is argued that the procedure employed in the case studies (wherein all variables are reduced to the same unit of measure with change then expressed as a percentage of the total possible score on each particular measure) is a valuable procedure to determine change in small sample interventions.

4.3 Subject Characteristics

Cognitive-behavioural programs rely primarily on the participants' ability to integrate the information provided during the conceptual phase, in order for successful instruction and training in the application of cognitive and behavioural skills. Such programs, furthermore, rely on the ability of the participant to apply the newly learned skills in their daily environment. Given the low socioeconomic status of the sample, their poor educational level, it is possible that this group of women with RA are not the most ideal group to investigate the potential value of such psychological intervention. The subjects possibly lacked the basic resources (i.e., both material and psychosocial

factors such as financial constraints, education, familial support, etc.) necessary for cognitive and behavioural change.

If one is constantly faced with insurmountable financial (e.g., concern whether one has the money to pay for basic bills) and familial (e.g., one's spouse has a drinking problem or has left you; no assistance to do daily household chores) hardships, it is unlikely that cognitive-behavioural would be the panacea for all your difficulties.

All the subjects in this study were considered chronic RA patients in the sense that most of them had experienced chronic pain for over 7 years, or were in remission. This in itself, makes them a particularly difficult group to introduce change.

4.4 The Nature of the Intervention

A possible limitation of the SIPMT programme, was the lack of a central focus on the emotional difficulties the subjects were struggling to cope with. The needs of the subjects were relegated to secondary concern, as the study sought to evaluate the benefit of a programme where cognitive processes and resultant actions were of primary concern.

4.5 Multidisciplinary Involvement

Behavioural medicine relies on multidisciplinary involvement in both research programs and treatment regimens. The difficulties of co-ordinating a study which relies on the co-operation of various skilled professionals can be insurmountable at times. In this study, the high incidence of missing data in terms of disease activity and immunological variables is a case point. It proved difficult to ensure that bloods were drawn from all patients on each particular day. Furthermore, the planning of the intervention had to accommodate for the daily work load of the other professionals involved. This was particularly so for the immunological assays, each of which took three days to complete.

The patients, furthermore, had a number of questions and queries which ideally required the participation of other allied health professionals, such as a nursing sister, physiotherapist, or occupational therapist. The emotional needs of this group, discussed earlier, also included the desire for more information regarding their medication regimens and symptoms. The researcher's experience of patient education in this context is worthy of comment. It seems that the information these RA patients received concerning the nature of their disease, and what was happening to each of them in particular, was not wholly constructive. The patients became more preoccupied and concerned with what is happening, and what could possibly happen in the future (e.g., one patient gave a very detailed and clinical account of the destruction of the joints in her hands. Her particular condition, however, was not nearly as severe as what she made it out to be). The extent to which an RA patient, particularly those of lower educational status, should receive a detailed and graphic description of joint damage or anticipated joint destruction, is questionable. There is perhaps more value in patient education which focuses on treatment goals, instruction in self-help skills, and group support.

5. Recommendations for Future Research/Intervention

In the above discussion (i.e., section 4) of the limitations of this particular study, a number of implicit recommendations for future research and/or intervention with RA patients are apparent.

Firstly, the planning and co-ordination of the cognitive-behavioural programme should include the efforts and involvement of other allied health professionals. Such assistance would focus particularly on patient education and instruction in safe exercise/activities. If the aim of cognitive-behavioural intervention is to improve the self-efficacy of its participants, then they need to know: (a) what activities they can or should not do (e.g., physiotherapist); (b) how they should make adjustments in their home environment to ensure minimum stress on inflamed joints (e.g., occupational therapist); (c) how and when they should take their medication (e.g., nursing sister); and what community services they could utilize (e.g., social worker). Whilst cognitive-behavioural intervention is considered a psychological treatment for RA, it should include the professional services and expertise of other health care workers within the clinics.

Secondly, subject selection should include more specific inclusion/exclusion criteria. The reason being that the patients may only benefit from cognitive-behavioural intervention if they have the capacity and resources to respond to treatment goals. Ideally, such criteria should therefore include:

(a) Verbal proficiency in language of intervention;
(b) Secondary educational level;
(c) A restricted age range, with preference for the 30-40 age group;
(d) Acute disease onset;
(e) Moderate (i.e., II classification by ARA standards) functional status;
(f) Great deal of reported pain;
(g) Active phase of the disease (i.e., the onset of RA should be relatively recent);
(h) Seroposivity to the rheumatoid factor;
(i) Similar treatment regimens, including medication usage (e.g., analgesic, anti-inflammatory, or remission-inducing agents); and,
(j) A generally healthy physical status (i.e., in terms of disease other than RA).

Thirdly, the variables to be examined should be carefully operationalized, to coincide with definitions of treatment outcome. "Psychological adjustment", therefore, should therefore include measures of personality characteristics, coping strategies/behaviour, perceptions of self-efficacy/helplessness, mood states, and social support networks. "Pain" should also include an objective measure of pain behaviours, to determine the extent to which the subjective (i.e., phenomenological) level of experience, in terms of the meaning of the pain, is translated to the physical/functional level of experience. "Immune status" should include measures of percentage circulating T- and B-cells, and ratios between helper- and suppressor T-cells for a more accurate estimation of psychoimmunological relations in RA. Lymphocyte proliferation rate is not an appropriate index of immunocompetence to rely on.

It is also important that other dependent measures are considered in terms of determining treatment outcome. These include measures of compliance to medication regimens (e.g., pill counts or correct usage), as well as questionnaires to determine which components of the cognitive-behavioural intervention were most effective for the individual patients. Follow-up data would also provide some indication of the long-term benefits of such intervention.

Finally, future research in determining the value of cognitive-behavioural intervention with RA patients should pay closer attention to the response of the individual patients throughout the duration of the study. The data analyses should account for the heterogeneity in RA in terms of patient characteristics and response to their disease and the intervention. The daily response of the RA patient to his/her disease is of particular importance.

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APPENDIX A

UNIVERSITY OF CAPE TOWN

FACULTY OF MEDICINE

APPLICATION TO CONDUCT CLINICAL RESEARCH OR A CLINICAL DRUG TRIAL

Note: (i) It is in the interests of the patients, the investigator, the hospital and the pharmaceutical company that drug trials are approved by the Pharmaceutical Advisory Committee of the hospital and the Faculty Ethics & Research Committee. Final responsibility for the trial rests with the Investigator.

(ii) In the case of a drug trial involving an unregistered medicine, approval must be obtained from the Medicines Control Council. The pharmaceutical firm concerned will assist in this regard.

(iii) Informed consent must be obtained from subjects.

(iv) Confidentiality must be respected.

(v) Subjects must be informed that they are free at any time to withdraw from participation in the study, without prejudice to their clinical care.

Directions:

(i) Please complete the questionnaire overleaf and return to the Chairman, E & R Committee, Dean's Office, Medical School.

(ii) Include a detailed protocol of the investigation or trial where appropriate.

(iii) A six-monthly progress report is required.

CHAIRMAN
ETHICS & RESEARCH COMMITTEE

APPENDIX: B

UNIVERSITY OF CAPE TOWN

Department of Psychology

University of Cape Town · Rondebosch 7700 · South Africa
Telephone: (021) 650-9111
Fax No: (021) 650-3726

TO WHOM IT MAY CONCERN

I am a final year Masters Psychology student doing research on **pain** and it's effects on one's well-being. I am particularly interested in **Arthritic pain** and discomfort. Various **non-medical** treatments for such pain exist, and I am interested in examining their ability to alleviate your pain experience.

The University's Rheumatology department, who run your arthritis clinic at Grootte Schuur/Princes Alice, is assisting me in this investigation.

I require your assistance and/or participation in an **8-week program** to investigate the extent of your pain experience, the impact this pain has on your daily life, and your ability to cope/manage this pain. We hope that such information will increase our understanding of arthritis, and improve subsequent treatment of the pain and discomfort it causes.

I hope to initiate the full investigation in the 2nd/3rd week of June 1990, and completing the program late July 1990.

If you have any queries or questions, please feel free to contact me or leave your name, telephone number and address at the clinic. I can be contacted at the Telephone Number listed below.

Kindest Regards,

SEAN GERMOND TEL: 650 3434 / 650 3430

DEPARTMENTS OF PSYCHOLOGY AND RHEUMATOLOGY

CONSENT TO PARTICIPATE IN RESEARCH PROGRAM

I,_____, THE UNDERSIGNED,

AGREE TO:

* PARTICIPATE FREELY IN THIS RESEARCH PROGRAM (DESCRIBE BRIEFLY).
* UNDERSTAND THAT ANY INFORMATION OBTAINED OR GATHERED WILL BE HELD STRITLY CONFIDENTIAL.

I,_____, AS THE RESEARCHER,

WILL ENSURE THAT:

* THE INTERESTS OF THE PARTICIPANTS BE OF PRIORITY
* ALL DATA GATHERED WILL REMAIN STRICTLY CONFIDENTIAL AND BE MADE AVAILABLE (ON REQUEST) TO THE PARTICIPANTS ON TERMINATION OF THE STUDY.
* THE PROGRAM WILL NOT INTERFERE WITH THE PARTICIPANTS' MEDICAL TREATMENT

DATED THIS DAY _____ AND MONTH _____, 19____

PARTICIPANT _____ WITNESS (1) _____

RESEARCHER _____ WITNESS (2) _____

oooooooooooo

STRESS INOCULATION AND PAIN MANAGEMENT TRAINING

PRE-INTERVIEW QUESTIONNAIRE

A: PERSONAL AND BIOGRAPHICAL DETAILS

NAME: _____ **DATE:** ___/___/____

ADDRESS: _____

_____

DATE OF BIRTH: ___/___/____ **TEL:** _____

For Office use only

[ ][ ][ ] 1-3

AGE: _____ [ ][ ] 4-5

SEX: Male [1] Female [2] [ ] 6

MARITAL STATUS

Single	1
Married	2
Divorced	3
Separated	4
Widowed	5

[ ] 7

HIGHEST EDUCATIONAL LEVEL ATTAINED:

Std 9 or lower	1
Std 10	2
Std 10 plus 1 or 2 years further training	3
University degree	4
Other (please specify)	5

[ ] 8

WHAT IS YOUR POSITION ON THE LABOUR MARKET?

For Office use only

Employer (other people work for you)	1
Self-employed (you work for yourself)	2
Employee (you work for other people)	3
Temporarily unemployed (looking for a job)	4
Housewife	5
Pensioner	6
Medically unfit for work (please specify)	7

☐ 9

WHAT IS YOUR NET INCOME PER MONTH?

R0,00 - R1 000,00	1
R1 000,00 - R1 999,00	2
R2 000,00 - R2 999,00	3
R3 000,00 - R3 999,00	4
R4 000,00 - R4 999,00	5
R5 000,00 +...	6
Do not wish to say	7

☐ 10

HOW MANY CHILDREN DO YOU STILL SUPPORT FINANCIALLY?

None	1	2	3	4/MORE

☐ 11

B: DESCRIPTION OF YOUR PAIN

For Office use only

1. On a scale of 0 to 100, with 0 meaning "no pain" and 100 meaning "pain as bad as it could be", **how much** pain do you have **on the average?**

☐ ☐ 12

2. On the same scale of 0 to 100, how much pain do you have when it is **at it's worst?**

☐ ☐ 13

3. How much pain do you have when it is **at it's least?**

☐ ☐ 14

4. How much pain do you have **right now?**

☐ ☐ 15

5. What seems to make your pain **worse** (such as time of day, too much or too little activity, boredom, etc.)?

_____

_____

6. What events seem to be associated with **decreases** in your pain?

_____

_____

7. How many hours per day (i.e. in a 24-hour period) do you usually spend out of bed?

☐ 16

C: PAIN-COPING STRATEGIES

8. Using the scale below, please indicate how often you use the pain-coping strategy listed to control your pain (if you have never heard of or used a particular skill, then circle "0" for "never").

Activity	Never	Once a month/ less	Once a week	2-4 times week	About once a day	More than once
Medications	0	1	2	3	4	5
Bedrest	0	1	2	3	4	5
Exercise	0	1	2	3	4	5
Relaxation	0	1	2	3	4	5
Massage/rubbing	0	1	2	3	4	5
Ignore the pain	0	1	2	3	4	5
Heat	0	1	2	3	4	5
Physiotherapy	0	1	2	3	4	5

9. Using the scale below, please indicate how effective each pain-coping strategy is for you. If you do not use the pain-coping strategy now, indicate how effective you think the strategy would be for your pain if you were to use it.

Activity	Never	Once a month/ less	Once a week	2-4 times week	About once a day	More than once
Medications	0	1	2	3	4	5
Bedrest	0	1	2	3	4	5
Exercise	0	1	2	3	4	5
Relaxation	0	1	2	3	4	5
Massage/rubbing	0	1	2	3	4	5
Ignore the pain	0	1	2	3	4	5
Heat	0	1	2	3	4	5
Physiotherapy	0	1	2	3	4	5

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10. If you take medications to control your pain, please indicate below which ones you use.

Name of medication	Dose	Average number taken per day.

11. What else do you do to control your pain?

_____

_____

12. What do you think should be done about your pain?

_____

_____

13. On a scale of 0 to 10, with 0 meaning "not at all able" and 10 meaning "very able", rate your ability to **decrease** pain when it is present.

☐ ☐ 33

oooooooooooo

THANK-YOU FOR COMPLETING THIS QUESTIONNAIRE, YOUR PARTICIPATION IN THIS PROGRAM IS MUCH APPRECIATED.

Department of Psychology
University of Cape Town · Rondebosch 7700 · South Africa
Telephone: (021) 650-9111
Fax No: (021) 650-3726
SEAN GERMOND
ROOM 2.28
1st JUNE 1990

DEAR _____

Thank-you for your interest in this research! I think that you will benefit considerably from participating in this investigation.

I am conducting this program for your benefit, and the results will be used to improve treatment strategies for your, and others', arthritis.

I have arranged with Lentegeur Public Library (in Merrydale road, next to the civic center) to use their "activities" room. This is a suitable venue, and I hope that it is more accessible to you (near bus/taxi routes and on the station). I have also organised a venue at Groote Schuur (UCT Medical School), and will confirm this arrangement with you (if you would prefer to meet here) as soon as possible.

If you have any transport difficulties, please let me know, and we can arrange financial assistance for you.

I hope to start the program on 18th/19th June, at 8am -> 9am. We will meet twice a week at this venue as a group (with 10 other ladies who also have arthritis). Only 4 of the sessions will begin at 8am, the rest will start at 9am to 10am. The program will be over 8 weeks, and then I will remain in contact with you for 6 months after that.

In order for me to ensure that you go into the correct group, I require you to to complete the attatched questionnaire and return it to me as soon as possible. I have included an addressed envelope to facilitate this.

Remember:

1. I need your collaboration! Just as you may learn from me, so I need to learn from your personal experience of arthritis. You are the best judge of that.
2. You will each receive a full report on your conditon and progress during the program.
3. You are encouraged to participate as actively as is possible. What you put into the program is important for you and your fellow arthritis sufferers.
4. All information you supply is held strictly confidential. I am bound by the regulations layed down by Grootte Schuur/Princes Alice, and by my Professional Board.
5. I can assist you with any problems you may have in participating in this investigation (such as transport etc...).

6. Your Doctor has recommended your involvement in this program, and will be kept informed of your progress/condition.

I hope to receive your questionnaire soon, and will contact you very shortly!

Kindest regards

SEAN GERMOND TEL: 650 3434/3430.

Department of Psychology

University of Cape Town · Rondebosch 7700 · South Africa
Telephone: (021) 650-9111
Fax No: (021) 650-3726

SEAN GERMOND
ROOM 2.28
6th JUNE 1990

DEAR _____

Thank-you for, your interest in this research! I think that you will benefit from the feedback and information that I can offer you if you complete the questionnaires sent to you.

The whole purpose of such an investigation is to determine the extent to which Arthritis, particularly the **pain** and discomfort, has an impact on **your** life. The information you provide would be of great benefit to us to design and implement **effective** treatment programs to suit your needs.

I am being assissted by the Dept. of Rhuematology at Groote Schuur/Princes Alice and the University of Cape Town Medical School. All information you provide will be held strictly confidential, and I am bound by the regulations of these institutions as well as by my Professional Board.

I will be sending you questionnaires over a period of 8 weeks (3 times within this period) which I need you to complete as honestly as possible. Furthermore, I will also require some blood samples from you over this period, and will contact you shortly to determine the most appropriate days/times (perhaps on your monthly visit to the clinic). We need this information to monitor your condition/progress.

Your Doctor has recommended your involvement in this investigation, and will be kept informed of all results from these questionnaires and blood tests.

Finally, as this is the first of hopefully a number of programs for Arthritis Sufferers, I'm sure that you will be able to participate in a pain management program at a later date.

Please complete the attatched questionnaire, and return it to me. I have provided an addressed envelope for this purpose. If you have any queries or further questions that you might like to ask, please feel free to contact me at one of the telephone numbers listed below. Your interest and participation in this study is greatly appreciated!

Yours sincerely

SEAN GERMOND TEL No: 650 3434 (W) 650 3430 (W)

STRESS INOCULATION AND PAIN MANAGEMENT TRAINING FOR RHEUMATOID ARTHRITIS PATIENTS

PROGRAM OUTLINE (8 WEEKS)

SION	DATE	GROUP (*)	ASSESSMENT (**)	VENUE	TIME	PHASE
	MON 25/6 TUE 26/6	EXP CONT	BLOOD**; QUEST. BLOOD**; QUEST.	G12 SEMINAR ROOM	9-10 AM	I (2 WKS)
	THUR 28/6 FRI 29/6	EXP CONT	BLOOD** BLOOD**	SAME	9-10 AM	
	MON 2/7 TUE 3/7	EXP CONT	BLOOD** BLOOD**	SAME	9-10 AM	
	THUR 5/7	EXP		SAME	9-10 AM	
	MON 9/7	EXP		SAME	9-10 AM	II (4 WKS)
	THUR 12/7	EXP		SAME	9-10 AM	
	MON 16/7	EXP		SAME	9-10 AM	
	THUR 19/7 FRI 20/7	EXP CONT	BLOOD**; QUEST. BLOOD**; QUEST.	SAME	9-10 AM	
	MON 23/7	EXP		SAME	9-10 AM	
	THUR 26/7	EXP		SAME	9-10 AM	
	MON 30/7	EXP		SAME	9-10 AM	
	THUR 2/8	EXP		SAME	9-10 AM	
	MON 6/8	EXP		SAME	9-10 AM	III (2 WKS)
	THUR 9/8	EXP		SAME	9-10 AM	
	MON 13/8	EXP		SAME	9-10 AM	
	THUR 16/8 FRI 17/8	EXP CONT	BLOOD**; QUEST. BLOOD**; QUEST.	SAME	9-10 AM	

(* = 12 per group; ** = Sister to draw blood + Richie Index)

Germond Dept. of Psychology Tel No. 650 3434/3430

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INTERVIEW FORMAT

NAME: _____ DATE: ___/___/_____

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1-3

A. SELECT THE 4 MOST STRESSFUL EVENTS IN THE MONTH PRIOR TO NOW.

(1) _____

_____

(2) _____

_____

(3) _____

_____

(4) _____

_____

B. **EVENT ONE:**

"How did you respond to the event...?"

_____

_____

_____

_____

For Office use

B1. "How satisfied were you with how you responded?"

very satisfied	..1..
somewhat satisfied	..2..
neither satisfied nor dissatisfied	..3..
somewhat dissatisfied	..4..
very dissatisfied	..5..

☐ 4

B2. "How satisfied were you with how others responded? Were you..."

very satisfied	..1..
somewhat satisfied	..2..
neither satisfied nor dissatisfied	..3..
somewhat dissatisfied	..4..
very dissatisfied	..5..

☐ 5

B3. "If this event were to happen in the future, how certain are you that you would be able to adjust well to it's negative aspects...?"

very certain	..1..
fairly certain	..2..
in some ways certain/ in some ways uncertain	..3..
fairly uncertain	..4..
very uncertain	..5..

☐ 6

B. **EVENT TWO:**

"How did you respond to the event...?"

_____

_____

_____

_____

For Office use

B1. "How satisfied were you with how you responded?"

very satisfied	1
somewhat satisfied	2
neither satisfied nor dissatisfied	3
somewhat dissatisfied	4
very dissatisfied	5

☐ 7

B2. "How satisfied were you with how others responded? Were you..."

very satisfied	1
somewhat satisfied	2
neither satisfied nor dissatisfied	3
somewhat dissatisfied	4
very dissatisfied	5

☐ 8

B3. "If this event were to happen in the future, how certain are you that you would be able to adjust well to it's negative aspects...?"

very certain	1
fairly certain	2
in some ways certain/ in some ways uncertain	3
fairly uncertain	4
very uncertain	5

☐ 9

B. **EVENT THREE:**

"How did you respond to the event...?"

_____

_____

_____

_____

For Office use

B1. "How satisfied were you with how you responded?"

very satisfied	1
somewhat satisfied	2
neither satisfied nor dissatisfied	3
somewhat dissatisfied	4
very dissatisfied	5

☐ 10

B2. "How satisfied were you with how others responded? Were you..."

very satisfied	1
somewhat satisfied	2
neither satisfied nor dissatisfied	3
somewhat dissatisfied	4
very dissatisfied	5

☐ 11

B3. "If this event were to happen in the future, how certain are you that you would be able to adjust well to it's negative aspects...?"

very certain	1
fairly certain	2
in some ways certain/ in some ways uncertain	3
fairly uncertain	4
very uncertain	5

☐ 12

B. **EVENT FOUR:**

"How did you respond to the event...?"

_____

_____

_____

_____

For Office use

B1. "How satisfied were you with how you responded?"

very satisfied	..1..
somewhat satisfied	..2..
neither satisfied nor dissatisfied	..3..
somewhat dissatisfied	..4..
very dissatisfied	..5..

☐ 13

B2. "How satisfied were you with how others responded? Were you..."

very satisfied	..1..
somewhat satisfied	..2..
neither satisfied nor dissatisfied	..3..
somewhat dissatisfied	..4..
very dissatisfied	..5..

☐ 14

B3. "If this event were to happen in the future, how certain are you that you would be able to adjust well to it's negative aspects...?"

very certain	..1..
fairly certain	..2..
in some ways certain/ in some ways uncertain	..3..
fairly uncertain	..4..
very uncertain	..5..

☐ 15

oooooooooooo

NAME _____ DATE _____

IDENTIFICATION

Below is a list of words that describe feelings people have. Please read each one carefully. Then fill in ONE space under the answer to the right which best describes HOW YOU HAVE BEEN FEELING DURING THE PAST WEEK INCLUDING TODAY.

The numbers refer to these phrases.
0 = Not at all
1 = A little
2 = Moderately
3 = Quite a bit
4 = Extremely

	0 NOT AT ALL	1 A LITTLE	2 MODERATELY	3 QUITE A BIT	4 EXTREMELY
Friendly					
Tense					
Angry					
Worn out					
Unhappy					
Clear-headed					
Lively					
Confused					
Sorry for things done					
Shaky					
Listless					
Peeved					
Considerate					
Sad					
Active					
On edge					
Grouchy					
Blue					
Energetic					
Panicky					
21. Hopeless					
22. Relaxed					
23. Unworthy					
24. Spiteful					
25. Sympathetic					
26. Uneasy					
27. Restless					
28. Unable to concentrate					
29. Fatigued					
30. Helpful					
31. Annoyed					
32. Discouraged					
33. Resentful					
34. Nervous					
35. Lonely					
36. Miserable					
37. Muddled					
38. Cheerful					
39. Bitter					
40. Exhausted					
41. Anxious					
42. Ready to fight					
43. Good natured					
44. Gloomy					
45. Desperate					
46. Sluggish					
47. Rebellious					
48. Helpless					
49. Weary					
50. Bewildered					
51. Alert					
52. Deceived					
53. Furious					
54. Efficient					
55. Trusting					
56. Full of pep					
57. Bad-tempered					
58. Worthless					
59. Forgetful					
60. Carefree					
61. Terrified					
62. Guilty					
63. Vigorous					
64. Uncertain about things					
65. Bushed					

MAKE SURE YOU HAVE ANSWERED EVERY ITEM.

NCS Data Reflex W 1268-543

M 021

1

McGILL PAIN QUESTIONNAIRE

NAME: _____ DATE: ___/___/_____

PRI: S____ A____ E____ M(S)____ M(AE)____ M(T)____ PRI(T)____ PPI____
(1-10) (11-15) (16) (17-19) (20) (17-20) (1-20)

PART 1. What Does Your Pain Feel Like?

Some of the words below describe your **PRESENT** pain. Tick **ONLY** those words that best describe it. **LEAVE OUT** any category that is **not** suitable. Use only a **SINGLE WORD** in each appropriate category (the one that applies best).

1 FLICKERING	8 TINGLING	16 ANNOYING
QUIVERING	ITCHY	TROUBLESOME
PULSING	SMARTING	MISERABLE
THROBBING	STINGING	INTENSE
BEATING		UNBEARABLE
POUNDING	9 DULL	
	SORE	17 SPREADING
2 JUMPING	HURTING	RADIATING
FLASHING	ACHING	PENETRATING
SHOOTING	HEAVY	PIERCING
3 PRICKING	10 TENDER	18 TIGHT
BORING	TAUT	NUMB
DRILLING	RASPING	DRAWING
STABBING	SPLITTING	SQUEEZING
LANCINATING		TEARING
	11 TIRING	
4 SHARP	EXHAUSTING	19 COOL
CUTTING		COLD
LACERATING	12 SICKENING	FREEZING
	SUFFOCATING	
5 PINCHING		20 NAGGING
PRESSING	13 FEARFUL	NAUSEATING
GNAWING	FRIGHTFUL	AGONIZING
CRAMPING	TERRIFYING	DREADFUL
CRUSHING		TORTURING
	14 PUNISHING	
6 TUGGING	CRUEL	PPI
PULLING	VICIOUS	0 NO PAIN
WRENCHING	KILLING	1 MILD
		2 DISCOMFORTING
7 HOT	15 WRETCHING	3 DISTRESSING
BURNING	BLINDING	4 HORRIBLE
SCALDING		5 EXCRUCIATING
SEARING		

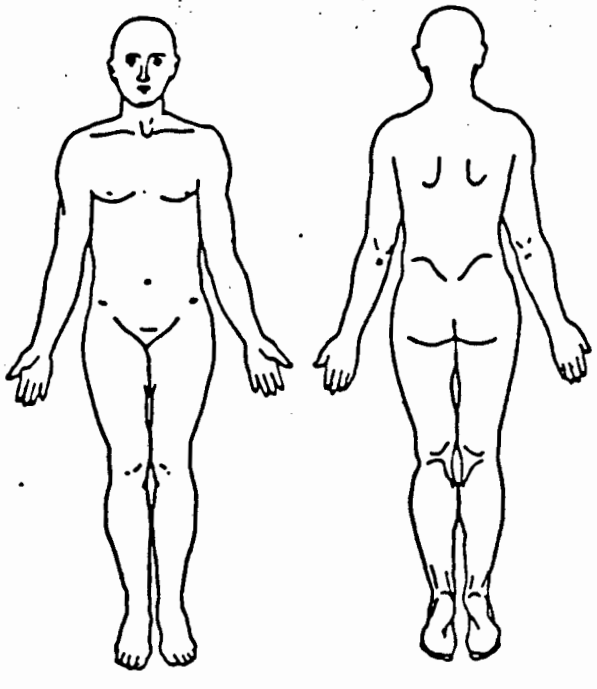
PART 2. How Does Your Pain Change With Time?

Which word or words would you use to describe the PATTERN of your pain?

BRIEF	RHYTHMIC	CONTINUOUS
MOMENTARY	PERIODIC	STEADY
TRANSIENT	INTERMITTENT	CONSTANT

PART 3. Where Is Your Pain?

Please mark on the drawings below, the areas where you feel pain. Put **E** if external, or **I** if internal, near the area which you mark. Put **EI** if both external and internal.



COMMENTS:

MHLC - FORM B

NAME: _____ DATE: ___/___/1990

This is a questionnaire designed to determine the way in which different people view certain important health-related issues. Please answer these items carefully, but do not spend too much time on any one item. As much as you can, try to respond to each item independantly. When making your choice, do not be influenced by your previous choices. It is important that you respond according to your actual beliefs and not according to how you feel you should believe or how you think we want you to believe. Each item is a belief statement with which you may agree or disagree. Below each statement is a scale which ranges from "strongly disagree (1) ....to.... strongly agree (6)". For each item we would like you to circle the number that represents the extent to which you disagree or agree with the statement. The more strongly you agree with a statement, then the higher will be the number you circle. The more strongly you disagree with a statement, then the lower will be the number you circle.

For example:...

Exercise is only good for healthy people.

Strongly Disagree 1	Moderately Disagree (2)	Slightly Disagree 3	Slightly Agree 4	Moderately Agree 5	Strongly Agree 6

Please make sure that you answer every item and that you circle only one number per item. This is a measure of your personal beliefs; obviously there are no right or wrong answers.

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1 - 3

-1-

For Office use

1. If I become sick, I have the power to make myself well again.

Strongly Disagree 1	Moderately Disagree 2	Slightly Disagree 3	Slightly Agree 4	Moderately Agree 5	Strongly Agree 6

☐ 4

2. Often I feel that no matter what I do, If I am going to get sick, I will get sick.

Strongly Disagree 1	Moderately Disagree 2	Slightly Disagree 3	Slightly Agree 4	Moderately Agree 5	Strongly Agree 6

☐ 5

3. If I see an excellent doctor regularly, I am less likely to have health problems.

Strongly Disagree 1	Moderately Disagree 2	Slightly Disagree 3	Slightly Agree 4	Moderately Agree 5	Strongly Agree 6

☐ 6

4. It seems that my health is greatly influenced by events beyond my control.

Strongly Disagree 1	Moderately Disagree 2	Slightly Disagree 3	Slightly Agree 4	Moderately Agree 5	Strongly Agree 6

☐ 7

5. Whenever I don't feel well, I should consult a medically trained professional...

Strongly Disagree 1	Moderately Disagree 2	Slightly Disagree 3	Slightly Agree 4	Moderately Agree 5	Strongly Agree 6

☐ 8

For Office use

6. I am directly responsible for my own health.

Strongly Disagree 1	Moderately Disagree 2	Slightly Disagree 3	Slightly Agree 4	Moderately Agree 5	Strongly Agree 6

☐ 9

7. Other people play a big part in whether I stay healthy or become sick.

Strongly Disagree 1	Moderately Disagree 2	Slightly Disagree 3	Slightly Agree 4	Moderately Agree 5	Strongly Agree 6

☐ 10

8. Whatever goes wrong with my health is my own fault.

Strongly Disagree 1	Moderately Disagree 2	Slightly Disagree 3	Slightly Agree 4	Moderately Agree 5	Strongly Agree 6

☐ 11

9. When I am sick, I just have to let nature run it's course.

Strongly Disagree 1	Moderately Disagree 2	Slightly Disagree 3	Slightly Agree 4	Moderately Agree 5	Strongly Agree 6

☐ 12

10. Health professionals control my health.

Strongly Disagree 1	Moderately Disagree 2	Slightly Disagree 3	Slightly Agree 4	Moderately Agree 5	Strongly Agree 6

☐ 13

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11. When I stay healthy, I am just very lucky.

Strongly Disagree 1	Moderately Disagree 2	Slightly Disagree 3	Slightly Agree 4	Moderately Agree 5	Strongly Agree 6

☐ 14

12. My physical well-being depends on how well I take care of myself.

Strongly Disagree 1	Moderately Disagree 2	Slightly Disagree 3	Slightly Agree 4	Moderately Agree 5	Strongly Agree 6

☐ 15

13. When I feel ill, I know it is because I have not been taking care of myself properly.

Strongly Disagree 1	Moderately Disagree 2	Slightly Disagree 3	Slightly Agree 4	Moderately Agree 5	Strongly Agree 6

☐ 16

14. When I recover from an illness, it's usually because other people (for example, doctors, nurses, family, friends) have been taking good care of me..

Strongly Disagree 1	Moderately Disagree 2	Slightly Disagree 3	Slightly Agree 4	Moderately Agree 5	Strongly Agree 6

☐ 17

15. Even when I take care of myself, it's easy to get sick.

Strongly Disagree 1	Moderately Disagree 2	Slightly Disagree 3	Slightly Agree 4	Moderately Agree 5	Strongly Agree 6

☐ 18

NAME: _____ DATE: ___/___/_____

AIMS

Arthritis Impact Measurement Scales

ID ___ 1-3/
4/

Arthritis Impact Measurement Scales

Instructions: Please answer the following questions about the way your arthritis affects your health. Circle the appropriate number to indicate your answer. Try to answer every question.

1. When you travel around your community, does someone have to assist you because of your health? (circle one number)
   - Yes ____ 1 5/
   - No ____ 2

2. Are you able to use public transportation?
   - No, because of my health ____ 1 6/
   - No, for some other reason ____ 2
   - Yes, able to use public transportation ____ 3

3. Do you have to stay indoors most or all of the day because of your health?
   - Yes ____ 1 7/
   - No ____ 2

4. Are you in bed or in a chair for most or all of the day because of your health?
   - Yes ____ 1 8/
   - No ____ 2

5. Does your health limit the kind of vigorous activities you can do such as running, lifting heavy objects or participating in strenuous sports?
   - Yes ____ 1 9/
   - No ____ 2

6. Do you have any trouble either walking several blocks or climbing a few flights of stairs because of your health?
   - Yes ____ 1 10/
   - No ____ 2

7. Do you have trouble bending, lifting or stooping because of your health?
   - Yes ____ 1 11/
   - No ____ 2

(circle one number for each question)

8. Do you have any trouble either walking one block or climbing one flight of stairs because of your health?

Yes	1	12/
No	2	

9. Are you unable to walk unless you are assisted by another person or by a cane, crutches, artificial limbs, or braces?

Yes	1	13/
No	2	

10. Can you easily write with a pen or pencil?

Yes	1	14/
No	2	

11. Can you easily button articles of clothing?

Yes	1	15/
No	2	

12. Can you easily turn a key in a lock?

Yes	1	16/
No	2	

13. Can you easily tie a pair of shoes?

Yes	1	17/
No	2	

14. Can you easily open a new jar of food?

Yes	1	18/
No	2	

15. If you had the necessary transportation:
Could you go shopping for groceries or clothes...

Without help (taking care of all shopping needs yourself)	1	19/
With some help (need someone to go with you to help on all shopping trips)	2	
Or are you completely unable to do any shopping	3	

(circle one number for each question)

22. During the past month, about how often did you get together with friends or relatives?

Every day	1	26/
Several days a week	2	
About once a week	3	
Two or three times in the past month	4	
Once in the past month	5	
Not at all in the past month	6	

23. During the past month, about how often have you had friends or relatives over to your home?

Every day	1	27/
Several days a week	2	
About once a week	3	
Two or three times in the past month	4	
Once in the past month	5	
Not at all in the past month	6	

24. During the past month, how often have you visited with friends, or relatives at their homes?

Every day	1	28/
Several days a week	2	
About once a week	3	
Two or three times in the past month	4	
Once in the past month	5	
Not at all in the past month	6	

25. About how often were you on the telephone with close friends or relatives during the past month?

Every day	1	29/
Several days a week	2	
About once a week	3	
Two or three times in the past month	4	
Once in the past month	5	
Not at all in the past month	6	

26. When you bathe, either a sponge bath, tub or shower, how much help do you need?

No help at all	1	30/
Help with bathing one part of your body, like back or leg	2	
Help in bathing more than one part of your body	3	

(circle one number for each question)

27. How much help do you need in getting dressed?

No help at all	1	31/
Only need help in tying shoes	2	
Need help in getting dressed	3	

28. How much help do you need to use the toilet?

No help at all	1	32/
Only need help in getting to or using the toilet	2	
Not able to get to the bathroom at all	3	

29. How well are you able to move around?

Able to get in and out of bed or chairs without the help of another person	1	33/
Need the help of another person to get in and out of bed or chair	2	
Not able to get out of bed	3	

30. During the **past month**, how would you describe the arthritis pain you usually have?

Very severe	1	34/
Severe	2	
Moderate	3	
Mild	4	
Very mild	5	
None	6	

31. During the past month how often have you had severe pain from your arthritis?

Always	1	35/
Very often	2	
Fairly often	3	
Sometimes	4	
Almost never	5	
Never	6	

32. During the past month, how long has your morning stiffness usually lasted from the time you wake up?

Over four hours	1	36/
Two to four hours	2	
One to two hours	3	
Thirty minutes to an hour	4	
Less than thirty minutes	5	
Do not have morning stiffness	6	

(circle one number for each question)

33. During the past month, how often have you had pain in two or more joints at the same time?

Always	1	37/
Very often	2	
Fairly often	3	
Sometimes	4	
Almost never	5	
Never	6	

34. During the past month, how much of the time have you enjoyed the things you do?

All of the time	1	38/
Most of the time	2	
A good bit of the time	3	
Some of the time	4	
A little of the time	5	
None of the time	6	

35. During the past month, how much of the time have you felt tense or "high strung"?

All of the time	1	39/
Most of the time	2	
A good bit of the time	3	
Some of the time	4	
A little of the time	5	
None of the time	6	

36. How much have you been bothered by nervousness, or your "nerves" during the past month?

Extremely so, to the point where I could not take care of things	1	40/
Very much bothered	2	
Bothered quite a bit by nerves	3	
Bothered some, enough to take notice	4	
Bothered just a little bit by nerves	5	
Not bothered at all by this	6	

37. How often during the past month did you find yourself having difficulty trying to calm down?

Always	1	41/
Very often	2	
Fairly often	3	
Sometimes	4	
Almost never	5	
Never	6	

(circle one number for each question)

38. During the **past month**, how much of the time have you been in low or very low spirits?

All of the time	1	42/
Most of the time	2	
A good bit of the time	3	
Some of the time	4	
A little of the time	5	
None of the time	6	

39. How much of the time during the past month did you feel relaxed and free of tension?

All of the time	1	43/
Most of the time	2	
A good bit of the time	3	
Some of the time	4	
A little of the time	5	
None of the time	6	

40. How much of the time during the past month have you felt downhearted and blue?

All of the time	1	44/
Most of the time	2	
A good bit of the time	3	
Some of the time	4	
A little of the time	5	
None of the time	6	

41. How often during the past month did you feel that nothing turned out the way you wanted it to?

Always	1	45/
Very often	2	
Fairly often	3	
Sometimes	4	
Almost never	5	
Never	6	

42. How much of the time during the past month have you felt calm and peaceful?

All of the time	1	46/
Most of the time	2	
A good bit of the time	3	
Some of the time	4	
A little of the time	5	
None of the time	6	

(circle one number for each question)

. During the **past month**, how often did you feel that others would be better off if you were dead?

Always	1	47/
Very often	2	
Fairly often	3	
Sometimes	4	
Almost never	5	
Never	6	

. How much of the time during the past month were you able to relax without difficulty?

All of the time	1	48/
Most of the time	2	
A good bit of the time	3	
Some of the time	4	
A little of the time	5	
None of the time	6	

. How often during the past month have you felt so down in the dumps that nothing could cheer you up?

Always	1	49/
Very often	2	
Fairly often	3	
Sometimes	4	
Almost never	5	
Never	6	

. In general would you say your health is excellent, good, fair or poor?

Excellent	1	50/
Good	2	
Fair	3	
Poor	4	

. Thinking about the past month, how much of the time has your health kept you from doing the kinds of things that you should be able to do?

All of the time	1	51/
Most of the time	2	
Some of the time	3	
None of the time	4	

(circle one number for each question)

48. During the **past month** how active has your arthritis been? 52/

Very active	1
Moderately active	2
Mildly active	3
Not at all active	4

Note: In answering the next four questions, please circle the number that best describes how you feel about each statement.

49. I seem to get sick a little easier than other people. 53/

Definitely true	1
Mostly true	2
Don't know	3
Mostly false	4
Definitely false	5

50. I never worry about my health. 54/

Definitely true	1
Mostly true	2
Don't know	3
Mostly false	4
Definitely false	5

51. My body seems to resist illness very well. 55/

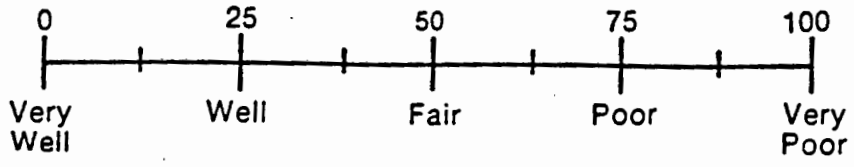
Definitely true	1
Mostly true	2
Don't know	3
Mostly false	4
Definitely false	5

52. When there is something going around, I usually catch it. 56/

Definitely true	1
Mostly true	2
Don't know	3
Mostly false	4
Definitely false	5

53. Considering all the ways your arthritis affects you, mark (X) on the scale for how well you are doing.

57-58/



(circle one number for each question)

54. During the **past month** how often have you had to take medication for your arthritis?

Always	1	59/
Very often	2	
Fairly often	3	
Sometimes	4	
Almost never	5	
Never	6	

55. Is your health currently affected by any of the following medical problems? (please circle yes or no for **each one**)

	1	2	
High blood pressure	Yes	No	60/
Heart disease	Yes	No	61/
Mental illness	Yes	No	62/
Diabetes	Yes	No	63/
Cancer	Yes	No	64/
Alcohol or drug abuse	Yes	No	65/
Lung disease	Yes	No	66/
Kidney disease	Yes	No	67/
Liver disease	Yes	No	68/
Stomach or blood disease	Yes	No	69/

56. Do you take medicine every day for any problem other than your arthritis?

Yes	1	70/
No	2	

57. Did you see a doctor more than three times last year for any problem other than arthritis?

Yes	1	71/
No	2	

1

LIFE EXPERIENCE EVALUATION

NAME: _____ DATE: ___/___/____

ADDRESS: _____

_____

_____

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1-3

A. PERSONAL DETAILS

A.1 Sex

Male [1] Female [2] 4

A.2 Age

under 30	1
30 - 40	2
40 - 50	3
50 - 60	4
60 - 70	5

5

B. INSTRUCTIONS

The following 30 items ask that you evaluate each statement in terms of your own recent life experience. For each statement, please check one, and only one, of the three choices provided: "Frequently Applies to Me", "Sometimes Applies to Me", or "Does Not Apply to ME". Your response to each item is important in helping us structure a tailored program to suite your needs. Please do not skip any questions.

	Frequently Applies to Me	Sometimes Applies to Me	Does Not Apply to Me	For Office use
1. My current job makes great demands in terms of my performance.	1	2	3	6
2. I constantly seem to have problems getting along with members of my family.	1	2	3	7
3. I prefer to spend less time with my friends now than I did before.	1	2	3	8
4. The job I hold has very little career potential for me.	1	2	3	9
5. The demands made on me by my family are very stressful.	1	2	3	10
6. Many of the people I meet during the week seem to make me anxious and upset.	1	2	3	11
	Frequently Applies to Me	**Sometimes Applies to Me**	**Does Not Apply to Me**	**For Office use**
7. I have difficulty performing my present job to my satisfaction.	1	2	3	12
8. I have heated arguments with at least one member of my family.	1	2	3	13
9. Most non-job relation= ships with other people are very demanding of my time and effort.	1	2	3	14

	Frequently Applies to Me	Sometimes Applies to Me	Does Not Apply to Me	For Office use
10. Being able to satisfy supervisors is hard to do in my present job.	1	2	3	15
11. My living situation is strained and tense.	1	2	3	16
12. I have difficulty getting on with those whom I regard as my superiors.	1	2	3	17
13. The policies followed where I work tend to decrease my on-the-job performance.	1	2	3	18
14. I wish that I could change my personal relationship with a member of my family.	1	2	3	19
15. Because I am so busy, I prefer to keep my social contacts to a minimum.	1	2	3	20

	Frequently Applies to Me	Sometimes Applies to Me	Does Not Apply to Me	For Office use
16. My work requires that I put in a lot of extra time outside of regular hours.	1	2	3	21
17. The type of life I presently lead inter= feres with my family activities.	1	2	3	22
18. Lately, I have been loosing my temper more often than usual.	1	2	3	23

	Frequently Applies to Me	Sometimes Applies to Me	Does Not Apply to Me	For Office use
19. I have recently taken on a new job that demands much more of me than my last job.	1	2	3	24
20. I have recently taken on a new family responsibilty such as marriage, a new child, or a live-in.	1	2	3	25
21. At present, it would require a lot of effort on my part to make new friends.	1	2	3	26
22. I should be paid more for the amount and type of work I perform on the job.	1	2	3	27
23. I wish I could spend more time at home with my family.	1	2	3	28

	Frequently Applies to Me	Sometimes Applies to Me	Does Not Apply to Me	For Office use
24. Overall, I have trouble meeting the demands placed on me by each day's activities.	1	2	3	29
25. I am seriously thinking about leaving my job for one with better working conditions.	1	2	3	30
26. Recently, I have, or am planning to, change my place of residence.	1	2	3	31
27. Just being pleasant to friends is getting very hard for me.	1	2	3	32

5

	Frequently Applies to Me	Sometimes Applies to Me	Does Not Apply to Me	For Office use
28. My job really leaves me worn out at the end of the day.	1	2	3	33
29. At present, my personal finances are somewhat strained.	1	2	3	34
30. I get bored because I have too much leisure time on my hands.	1	2	3	35

Thank you for completing this questionnaire. Your participation in this program is much appreciated.

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E: _____ DATE: ___/___/1990

Please circle the number that is most applicable to your belief about this program. Answer honestly, and there are no right or wrong responses. I need to know your feelings about the value of this treatment.

FOR OFFICE USE ONLY

		1-3
How confident are you that this treatment will successfully help you to cope more effectively with your rheumatoid arthritis?	Most Positive 7 6 5 4 3 2 1 Most Negative (Moderate)	4
How successful do you feel this treatment will be in decreasing the extent to which rheumatoid arthritis interferes with your work/social life?	Most Positive 7 6 5 4 3 2 1 Most Negative (Moderate)	5
How confident would you be in recommending this treatment to a friend/associate who suffered with rheumatoid arthritis?	Most Positive 7 6 5 4 3 2 1 Most Negative (Moderate)	6

APPENDIX: O

SCORING FOR INDICES OF INFLAMMATORY ACTIVITY.

NAME:

JOINT SCORES

DATE:		Visit 1	Visit 2	Visit 3	Visit 4	Visit 5	Visit 6	Visit 7
Temp.mand								
Cx. spine								
St. Clavic								
Acromio clavic								
Shoulder	R							
Shoulder	L							
Elbow	R							
Elbow	L							
Wrist	R							
Wrist	L							
MCP	R	R/L 1/1 2/2	R/L 1/1 2/2	R/L 1/1 2/2	R/L 1/1 2/2	R/L 1/1 2/2	R/L 1/1 2/2	R/L 1/1 2/2
	L	3/3 4/4 5/5	3/3 4/4 5/5	3/3 4/4 5/5	3/3 4/4 5/5	3/3 4/4 5/5	3/3 4/4 5/5	3/3 4/4 5/5
PIP	R	R/L 1/1 2/2	R/L 1/1 2/2	R/L 1/1 2/2	R/L 1/1 2/2	R/L 1/1 2/2	R/L 1/1 2/2	R/L 1/1 2/2
	L	3/3 4/4 5/5	3/3 4/4 5/5	3/3 4/4 5/5	3/3 4/4 5/5	3/3 4/4 5/5	3/3 4/4 5/5	3/3 4/4 5/5
HIP	R							
	L							
KNEE	R							
	L							
ANKLE	R							
	L							
TALO-CALC	R							
	L							
MIDTARSAL	R							
	L							
MTP	R	R/L 1/1 2/2	R/L 1/1 2/2	R/L 1/1 2/2	R/L 1/1 2/2	R/L 1/1 2/2	R/L 1/1 2/2	R/L 1/1 2/2
	L	3/3 4/4 5/5	3/3 4/4 5/5	3/3 4/4 5/5	3/3 4/4 5/5	3/3 4/4 5/5	3/3 4/4 5/5	3/3 4,4 5/5
NO.SWOL.JTS								
ARTICULAR INDEX								
GRIP STRENGTH	R							
	L							
ESR								
CRP								
VISCOSITY								

APPENDIX: P

STRESS INOCULATION AND PAIN MANAGEMENT TRAINING (SIPMT)

OVERVEIW:

(1) 16 sessions, structured over 3 phases:

A. Conceptual (sessions 1-4)
B. Skills Acquisition and Training (sessions 5-12)
C. Application and Follow-through (sessions 13-16)

(2) These sessions are of 1 hour duration.

(3) Venue: UCT psychology department (the most likely setting).

(4) Time: Late afternoon (approximately 5.30-6.30; but to be arranged).

(5) Assisstance: Qualified nurse to draw blood.

(6) Equipment:

* 10 floor mats and blankets
* tape recorder
* hand-outs and questionnaires
* overhead projector
* test-tubes, needles and box to transport blood (including stick labels etc...)

A. CONCEPTUAL

General: Introductory phase to the program. In these sessions, the subjects are educated in terms of: the rationale for the psychological (or non-medical) techniques for managing pain; the effects of chronic pain; the nature of stress and it's effect on the body; and the two pain cycles that have been identified (the depression/inactivity-pain, and the anxiety/tension-pain cycles). A model of pain is introduced, explaining the role of psychological and behavioural processes contributing to the pain experience. A model of stress is introduced, explaining the role that stressful experience and coping techniques play in adapting to the pain and disability as a result of RA. The treatment program is outlined, and emphasis is placed on goal setting and achieving those goals.

Session 1: "Introduction to course"

(1) Introduction (rationale, objectives, practical issues).
(2) Getting to know each other (group contract, confidentiality, personal objectives and expectations).
(3) Lecturette: The effects of chronic pain (the two pain cycles).
(4) Questions and discussion.
(5) Wrap up, blood drawn, questionnaires and pain and tension diaries handed out and explained.

Session 2: "Goal setting"

(1) Lecturette: Biopsychosocial model of pain.
(2) Discussion and questions.
(3) Goal setting: Rationale, types of goals, how to set and achieve them.
(4) Blood drawn.

Session 3: "Stress and pain"

(1) Lecturette: Stress, tension and pain.
(2) Discussion: Identify and list types of stressors associated with RA.
(3) Wrap up: Facilitator identifies central concerns.
(4) Blood drawn.

Session 4: "Coping with stress and pain"

(1) Goals feedback.
(2) Lecturette: Coping strategies.
(3) Discussion: Personal coping strategies and techniques, sharing personal experience.
(4) Goal setting.

B. SKILLS ACQUSITION AND TRAINING

General: Training and introduction of coping skills to manage stress and pain. Identify two major forms of coping: palliative and cognitive. The former utilizing relaxation and the latter utilizing cognitive pain coping skills such as attention refocussing, dissociation, relabelling, etc. Subjects receive the rationale for each technique, are given instruction in the technique, and demonstrated when and how the technique can be used. It is emphasised that it is necessary to practice the technique for optimal efficacy. A "buddy system" is set up whereby subjects keep in contact with each other telephonically to promote goal setting and the reinforcement thereof.

Session 5: "Pain diary and relaxation"

(1) Pain and tension diary feedback.
(2) Lecturette: Relaxation training and the relaxation response.
(3) Training: Progressive muscle relaxation.
(4) Discussion: Personal experience of relaxation.

Session 6: "Relaxation and guided imagery"

(1) Goals feedback.
(2) Lecturette: Use of engaging and relaxing past-times.
(3) Training: Progressive muscle relaxation and guided imagery.
(4) Goal setting.

Session 7: "Cognitive skill: Attention refocussing"

(1) Introduction: Demonstration of skills effectiveness -Part 1.
(2) Lecturette: Attention refocussing.
(3) Training: Demonstration of skills effectiveness - Part 2.
(4) Discussion.
(5) Relaxation.

Session 8: "Cognitive skill: Vivid imagery"

(1) Goals feedback.
(2) Lecturette: Vivid imagery and attention refocussing.
(3) Training: Demonstration of skills effectiveness.
(4) Goal setting..
(5) Blood drawn and tests distributed.

Session 9: "Cognitive skill: Dissociation"

(1) Lecturette: Dissociation and pain awareness.
(2) Training: Demonstration of skills effectiveness.
(3) Relaxation and Neurolinguistic programming.
(4) Discussion.

Session 10: "Cognitive skill: Relabelling"

(1) Goals feedback.
(2) Lecturette: Relabelling and pain experience.
(3) Training: Demonstration of skills effectiveness.
(4) Relaxation.
(5) Goal setting.

Session 11: "Self-Appraisal and Self Encouragement"

(1) Lecturette: Appraisal and pain coping -what we can do to aleviate pain.
(2) Discussion: Personal experience so far.
(3) Training: Using self-encouragement.
(4) Relaxation.

Session 12: "Social Factors and Communication"

(1) Goals feedback.
(2) Discussion: Support of spouse/family and other factors influencing pain coping.
(3) Lecturette: Pain and communication.
(4) Relaxation.
(5) Goal setting.

C. APPLICATION AND FOLLOW-THROUGH

General: This final phase focusses on consolidating the skills and knowledge acquired in the first two phases. Emphasis is now placed on communication and support within the group, on reinforcement, and encouragement in utilizing the coping skills learned. The syndrome of "learned helplessness" or passive coping is addressed, and appraisal of coping efficacy by self, group and facilitator occurs. Problem solving in terms of difficulties encountered in utilizing the coping skills is a central concern in this phase. Relaxation continues, with emphasis more on "hypnotic relaxation" or neurolinguistic programming.

Session 13: "Pain/tension diary and personal experiences"

(1) Feedback: Pain/tension diary progress.
(2) Discussion: Personal experiences using the techniques.
(3) Relaxation.

Session14: "Learned helplessness and Self-Appraisal"

(1) Goals feedback.
(2) Discussion: Learned helplessness and self-appraisal.
(3) Relaxation.
(4) Goal setting.

Session 15: "Feedback and Follow-through"

(1) Feedback from facilitator.
(2) Discussion: Where to from here?
(3) Relaxation.

Session 16: "Closure"

(1) Goals feedback and long-term goal setting.
(2) Discussion: Personal and groups' experience of the program.
(3) Blood drawn and tests distributed, and future follow-up discussed.
(4) Closure: Debriefing, saying goodbye.

FOLLOW-UP

(1) Welcome and feedback of data.
(2) Assessment: Completion of test instruments.
(3) Discussion: Use of the techniques.
(4) Blood drawn.
(5) Closure: Encouragement to continue, referral...

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STRESS INOCULATION AND PAIN MANAGEMENT TRAINING

TREATMENT MANUAL

BY
SEAN GERMOND

(BASED ON THE STANFORD ARTHRITIS CENTER PAIN MANAGEMENT PROGRAM)

TABLE OF CONTENTS

SESSION 1: INTRODUCTION TO COURSE

1) Introduction [10 MIN]

Thanks to participants for deciding to participate in the research, for giving blood and completing the questionnaires.

Trainer gives name and background and why the course is being run.

The objectives of the program are stated:

(1) To teach each of you ways of dealing with your pain; ways that are non-medical and have no side-effects.

(2) To teach you about pain and stress/tension, and how stress can affect your pain.

(3) To demonstrate to you how one plays a role in increasing one's pain by reducing activity, and being preoccupied with the pain.

(4) To enable you to share your experiences and help each other to live with RA.

(5) To give you a glimpse of life beyond pain; of life where pain is not the most central issue of your existence; to gain a different perspective of, and attitude towards pain.

The rationale is stated:

Considerable research has demonstrated the extent to which psychological (i.e. emotional, experiential and ways of thinking about one's experiences) factors play a role in contributing to RA and pain. Anecdotal reports of factors such as personality types, attitudes and behaviours influencing RA have been extensively researched and science is still trying to unravel this mind-body relationship.

Numerous techniques have evolved for coping with stress and pain, and have been utilized in arthritis centers world-wide. The program you are now on is based on one being run at the influential "Stanford Arthritis Center" in New Jersey in the United States. It has been modified to suit your requirements and certain additions have been made by the researcher based on contemporary research findings.

Your collaboration is required in this program. Although I will be demonstrating and teaching you various techniques, I need your

participation, your assistance and your knowledge/understanding of what you're going through in order for the program to be of maximal benefit. As much as I will give you feedback, so should you give feedback to me.

I want us all to enjoy this program and to learn as much as possible about your experiences and how we can improve your present condition. Remember, I'm not offering a cure for RA. I'm demonstrating more constructive ways of dealing with its symptoms, particularly the pain and discomfort.

You must continue your medication and visits to the clinic; and must feel free to approach me with any problems you may be experiencing. If you have any queries or require assistance in finding out as much as you can about your disease, please feel free to consult with me about what books to read or what questions to ask your Doctor.

2. Getting to know each other [15 MIN]

(Each member gets a name tag so that they are easily identifiable)

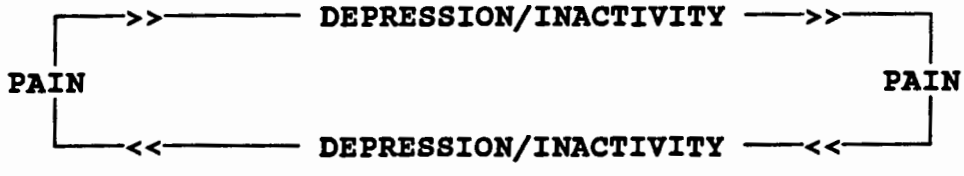
Participants introduce selves by stating name, background, what they would like to get out of the course. Participants also share their personal reasons/explanations of <u>why</u> they're suffering from RA/pain and what such experience means to them.

(If they mention things that are not directly related to pain/stress/RA, either draw connections between what they say/reveal and pain/RA or explain that while the course is geared to help people to cope with pain/RA, it will be possible to use goal-setting to achieve other objectives.)

Group draws up a "contract" wherein they specify their personal wishes/demands/"boundaries" so as to clarify what governs their group and the nature of their relationships within the group. (It is noted that this "contract" can always be re-worked as time progresses.)

(3) Lecturette: The effects of chronic pain (the pain-depression-inactivity-pain cycle) [15 MIN]

(Use a poster depicting each cycle)



Pain in RA is often made worse by moving. Because of this, many people with the disease decrease the number of things they do and the places they go. The combination of chronic pain extending into the future with unpredictable fluctuations and an uneventful life can lead to severe depression. Inactivity can also, by itself, make pain worse in RA since it is necessary to have the right balance of activity and rest for healthy functioning of the joints.

When people with RA were asked what their greatest concerns were, almost all of them said "pain!" Pain is the reason that people make changes in their lives, and gaining control over pain can enable you to reverse the changes brought about by arthritis.

(4) Discussion [10 MIN]

How has chronic pain affected your life? What changes have you had to make in your life because of RA? Everyone should be encouraged to participate and share their experiences.

(5) Wrap-Up [10 MIN]

Whilst blood is drawn, questionnaire packages are handed out and explained. The tension/pain diary record cards are explained and participants are encouraged to complete them daily for the next two weeks. The researcher will then discuss them with each of you privately to see **where** changes can be made.

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SESSION 2: GOAL SETTING

(1) Lecturette: A biopsychosocial model of pain [15 MIN]

Pain is a complex phenomenon that is psychological as well as physical. As an introduction, a few concepts about different aspects of the pain experience.

Factors **besides** the physical that contribute to pain.

Appraisal: How we think about pain affects our experience. If we think of pain as indicating extreme damage, our pain will be worse. For example: a woman had pain in her chest that was quite severe. She was worried that something serious was wrong, that perhaps she was suffering from angina or the pain possibly indicated cancer. When she went to the doctor and had tests performed she learned that there was nothing wrong. The pain felt much better right away, and she was able to ignore it and continue her daily activities. Part of the **appraisal** of pain has to do with how prepared we think we are to cope with it. Someone with arthritis who knows a number of exercises and pain control techniques will not be so upset when she feels pain beginning, and this will actually lessen her pain.

If we think of pain as bringing us benefit, it doesn't seem so bad (consider the "no pain no gain" maxim in most exercise clubs). In RA, activity which can cause pain is not harmful unless overdone, and is actually helpful in improving your condition.

Emotions: When we are anxious and under stress, pain can become worse. For example, dental pain is worse for people who are very nervous about dental procedures. In RA, there is evidence that stress makes the physical disease process worse and that tension accentuates the pain in the limbs or joints. Depression can also increase pain in RA because it can cause inactivity and foster poor perceptions of self-esteem and self-worth.

Attention: The reason that athletes often continue playing even after a serious injury is that they are so engrossed in the game that they simply don't notice their pain. Our mind is like a searchlight: it can only shine on a limited number of things at one time. Or think of a television. It can only be tuned into one channel at a time. If you're concentrating hard on something else, the pain can't enter your experience.

The fact that what you are thinking determines your pain does not mean that pain is "all in your head", or imagined. We are not trying to say that you are responsible for causing your pain. The pain is real!, you are suffering from arthritis which does

inflame the joints and muscles. But, psychological, emotional and physical factors all work together to ultimately determine your experience of pain.

Treatment can thus focus on components besides the physical one. Just taking aspirin affects your pain through physical means, learning effective mental strategies can help you to control your pain through psychological and emotional means.

Questions and discussion.

(2) Goal setting: Rationale, types and setting and achieving goals. [30 MIN]

The key to success in any undertaking is first learning a set of skills and then practicing them until they have been mastered. Children cannot read before first learning to recognize the letters of the alphabet. They then learn the sounds of combinations of letters. Later, they learn the meanings of simple words and phrases. It is only after years of practice that one is able to read a novel. Think about it. The same is true with almost anything we do from baking a cake to driving a car. These tasks are all based on learning skills and mastering them.

Success in arthritis management is the same. One needs to learn a set of skills that can help relieve some of the pain caused by arthritis. However, we have learned that knowing skills is not enough. Most of us need a way of incorporating these skills into our daily lives. Unfortunately, whenever we try a new skill, the first attempts are clumsy, slow, and show few results. It is easier to return to our old ways than to continue to try to master new and sometimes difficult skills. One of the best ways to master new skills is through gaol-setting. In the next few minutes, we will try to outline some of the principles of goal setting. If you **use** these principles, the success of an arthritis self-management program is almost assured.

TYPES OF GOALS

There are two types of goals, long term and short term. An example of a long term goal is wanting to climb stairs, lose 20 kilograms, or walk 4 kilometers every day. Most of us are pretty good at naming our long term goals, and do so very easily. The question to ask yourself is, "What would I like to do that I am unable to do now because of my arthritis?" Take a few moments to jot down one or two long-term goals. ... Long-term goals are important as they set an objective. However, they are of little help in achieving successful skill mastery. To do this, one needs to work on short term goals.

A short-term goal is just what it says it is, a goal to be accomplished today or, at the most, this week. Short-term goals are the steps to achieving a long-term goal. Examples of short-term goals might be, "Today, I will do two strengthening exercises for my knees," or "Today, I will not eat anything after 7 PM," or "Today, I will walk around the block", or "This week I will spend 2 hours each afternoon in the garden. As you can see, these short-term goals relate to the long-term goals mentioned before, but they demand a specific action NOW!

Since short-term goals are so important, let's look at them more carefully.

HOW TO FORM A SHORT-TERM GOAL

First, a short-term goal must call for a specific action that you can realistically expect to accomplish now. Most of us can do things which could make us more healthier but often fail to do them. For example, most people with arthritis can walk. Some of them can only just walk across a room, others half a block, most can walk several blocks, and some can walk for a kilometer or more. However, we seldom do this even when we know that it is good for us. Therefore, a short-term goal might be to walk around the block four times this week.

By being specific, we mean that a goal must have the following parts:

1. **An action** - something you do, such as walk, do exercise, call a friend, take medicine, or lose weight.

2. **Description** - This gives the detail so that anyone listening to or reading your goal should be able to know exactly what you mean. The description always includes one of the following:

    a. What kind - for example, instead of "exercise", you might be more specific by saying, "stretching exercise for my shoulder."

    b. How far, how much, how many - for example, instead of "walk" you might say "walk around the block", or instead of "stretching exercise of the shoulder", you might say "three stretching exercises of the shoulder".

3. **How often** - This is the final part of the goal, but again, be so specific that anyone reading or listening to your goal would be able to know exactly what you expect to do. For example, instead of "walk around the block", say, "walk around the block once a day", or "walk around the block four times this week".

Thus a goal must:

1. Have an **action.**

2. Give **details** about the action.

3. Describe **how often** the action will be done.

Earlier we said that at short-term goal must be both specific and realistic. The above discussion should help you to be specific. Being realistic is just as important. Goals should be set in such a way that they can be accomplished fairly easily. On the other hand, they should require some change in your behaviour. For example, if you never eat after supper, setting a goal of not eating after supper may be specific but will accomplish nothing as it requires no change in your behaviour.

Where most people have problems is that they make the goals too difficult. It is either too big or it is done too often. It is best to give yourself some room to slip up. The best goals are those that you accomplish. This in turn helps you to set more goals. One of the best examples of this aspect is Alcoholics Anonymous (the AA). The members do not set goals of never drinking. Rather, their goal is not to drink today.

In setting goals for arthritis, there are a couple of rules that may help towards success.

1. **Start where you are or start slowly.**

   If you can now walk around the block, start your walking program with walking around the block, not walking a kilometer.

2. **Give yourself some time off.**

   All people have days when they don't feel like doing something. Therefore, it is best that you will do something 3 to 4 times a week, but not every day. That way, you don't feel like walking today, you can still meet your goal tomorrow. In review, there are five guides for goal setting:

1. Choose an action.

2. Give specific details about the action such as what kind, how far, how much, how many, and when and where.

3. Decide how often you will do whatever you have decided to do.

4. Start where you are.

5. Give yourself time off.

Now that you know about short-term goals, write one or two short-term goals which will help you to reach your long-term goals.

As we continue to set goals during the next few weeks, keep these things in mind:

(i) **Remember** the 2-hour pain rule: If your joint still hurts two hours after performing an activity, cut back!

(ii) **Self-reward** for achieving gaols, on a daily basis, is very important. Choose something that is enjoyable but not so time consuming or expensive that it won't be done or causes undue stress or tension.

You may set as many goals as you wish, but at least **one** should involve increasing painful activity.

(Pause for three minutes)

(Trainer hands out and explains goal charts)

Goal sheets should be put on the fridge door and filled out every day. Participants should bring them back next week.

Sharing: Go around the circle and tell what goals you have set yourself for this week. Each person should set a long-term goal. Daily sub-goals are set for the next week (i.e. your short-term goals), and recorded on the goal sheets. In addition to the activity goal(s), personal goals such as weight loss or stress management may be set. Daily rewards for achieving goals should be set and listed on the goal sheet. For example, someone may specify walking one block as a goal to be followed by reading the evening newspaper. You can specify longer-term rewards as well; for example, at the end of the week you may wish to treat yourself to dinner or a movie.

:ach person should telephone at least one other from the group during
:he next week, so that each person both makes and receives a call.
'hone numbers can be obtained right after this session.

3) Blood drawn: [5 MIN]

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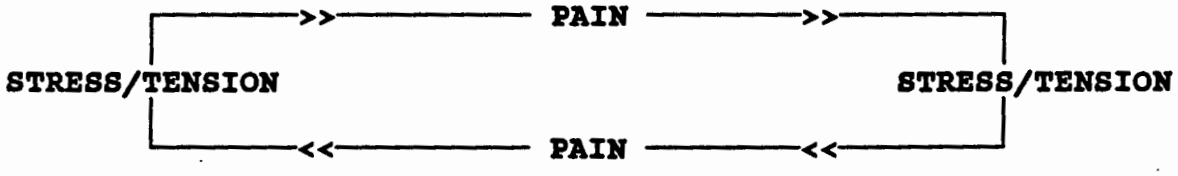
ESSION 3: STRESS AND PAIN

) Lecturette: Stress and the tension-pain cycle [30 Min]]

(Use poster to depict cycle and overheads)



During the last two sessions, we have seen that psychological experience plays an important role in managing RA and its related pain and discomfort. We have also discussed the importance of increasing activity and setting goals for ourselves. Today, we're going to discuss the role of stress in affecting pain and general well-being.

When we say stress/tension aggravates pain, we are not implying that your pain is "all in the mind". Rather, we are saying that your "state of mind" greatly colours your pain experience. Stress is one of the most important psychological factors that aggravates RA and pain. Extensive research has documented the extent to which greater life stress is associated with greater frequency and intensity of RA relapse. Put differently, stress can accelerate the progression of RA. Stress also increases tension in our muscles and limbs, this tension promotes stiffness and pain in these areas.

Stress affects us both psychologically and physically. Perhaps an example might clarify this point more aptly.

> Imagine that you have had a very busy and tiring morning. You have got a deadline to meet by 1 p.m. You finally manage to finish what you had to do and jump into your car at 12.45. Now you know that the drive takes at least 15 mins, but you're sure you can make it. On the way, unfortunately, you seem to catch every red light, and each short cut you take runs into difficulties. You eventually get stuck in a traffic jam and its almost 1 p.m. and you're nowhere near where you need to be... [How are you feeling..?]

My answer is that...

> I would be feeling extremely "up-tight", frustrated, angry and "tense". So, psychologically you are experiencing negative feelings, you can't think straight, you are angry; you feel on edge and irritable and may even be overcome with despair with what is happening to you. Physically, a whole

host of things are happening in your body. Your muscles become tense, tight. You might be trembling, feeling shaky. You may notice that your breathing is faster and shallow, feeling smothering sensation, you feel light-headed. Your hands may feel clammy, and you may be sweating. Your mouth feels dry, you may feel exhausted and even a bit nauseous.

This is obviously a highly dramatized reaction or response to being late for an appointment but it serves as a picture of stress. Each of us experience these symptoms to lesser or greater degree in our everyday life, and at times we know that our response to something can be highly inappropriate. (e.g. When we "flare-up" for no reason at all). Inappropriate because we may appraise the situation, event or experience as stressful simply because we are feeling taxed, not coping at all.

Stress then, is a highly complex experience, but can be broken down into its component parts and made sense of. (Keep in mind the example that we have used). Stress is made up of three stages:

Neutral event or stimulus	How you make sense of it. How you feel about it, or right now.	Your psychological and physical response

If we superimpose our example on this diagram, we may understand what is actually happening in such a situation.

Long tiring morning, Late for an appointment, heavy traffic, short cuts are not effective.	Your short-cuts are not a good idea. Why is there so much traffic? What is going to happen if I am late?	Feeling frustrated angry, on edge, up-tight, light-headed tense, shaky, trembling, sweaty hands, fatigued

Stress is effectively, **how you feel.** The event, situation or experience is not in itself stressful. Just as I may enjoy jumping out of aeroplanes, this may be decidedly stressful to you. What is happening is that you and I "appraised" the event

differently. I might look forward to jumping out, anticipating the sensation of free-falling and floating down to earth. You may see this as completely crazy and would die of fright. We differ in our appraisal of what is happening. So, I will enjoy free-falling, feel completed exhilarated, and look forward to stepping out on to the wing. You, on the other hand, will feel sick with fright, tremble and want to faint the thought of stepping out onto the wing of the plane.

What makes the event stressful is your appraisal there-of. This appraisal is based on your:

(1) beliefs, (e.g. the parachute will not open)

(2) past experiences (e.g. the last time you went sky-diving one of the members had a tragic accident).

(3) life-style/habits (e.g. you don't enjoy doing things out-of-the-ordinary, and prefer more low-risk sports)

(4) state of mind/mood at the time (e.g. feeling anxious or nervous)

(5) personality (e.g. you worry a great deal about dying or don't like not being in control of what is happening to you).

HOW DOES THIS RELATE TO PAIN?

Stress effects pain in two major ways:

(i) When under stress, we feel overwhelmed, we feel we cannot cope with what is happening. If you happen to be in pain at the time, your experience thereof is heightened. The pain seems unmanageable and just the "last straw". The pain becomes stressful in itself.

(ii) Stress causes tension in our body's muscles and tendons. This tension, causing tightness or stiffness and aching in the muscles contributes to the pain caused by RA, and the total amount of pain is increased (it feels worse, more than you can bear).

This is the vicious circle of tension - pain - tension where tension causes pain and pain in itself becomes tension.

Pain causes physical tension and stress in two ways:

(i) When we are in pain, we tend to hold our bodies very rigid and in unusual postures to try to protect the painful areas. When "trying to get comfortable" we are actually causing physical tension. Limping, sitting side-ways or awkwardly, or holding the spine/body in a lopsided way are

some of the ways in which pain sufferers try to "ease the pain".

This **"muscle guarding"**, the uneven body postures we may adapt, strains different muscle joints which, in turn, begin to hurt. We thus increase our total experience of pain by causing pain in otherwise healthy muscles, tissue. Finally, this guarding may leave muscles unused, weak and wasted, and it hurts when trying to use them again.

(ii) The pain that we are experiencing can make us feel angry and "fed-up", it can also make us feel very anxious about our health and thus we may worry about it continuously. The pain in itself becomes stressful.

Our ability to cope with and manage stress has fundamental implications in terms of our ability to manage pain and our experience of pain itself. We need to identify types of stress in our daily life, particularly those associated with RA, and discuss what effects they have on us, in light of this model we now have.

(2) Discussion: Identify and list types of stressors. [15 MIN]

(3) Wrap-up: Identify central concerns [10 MIN]

(4) Blood drawn: [5 MIN]

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SESSION 4: COPING WITH STRESS AND PAIN

(1) Goals feedback [15 MIN]

Each participant tells:

(a) Previous weeks goals and rewards

(b) Whether achieved - why or why not?

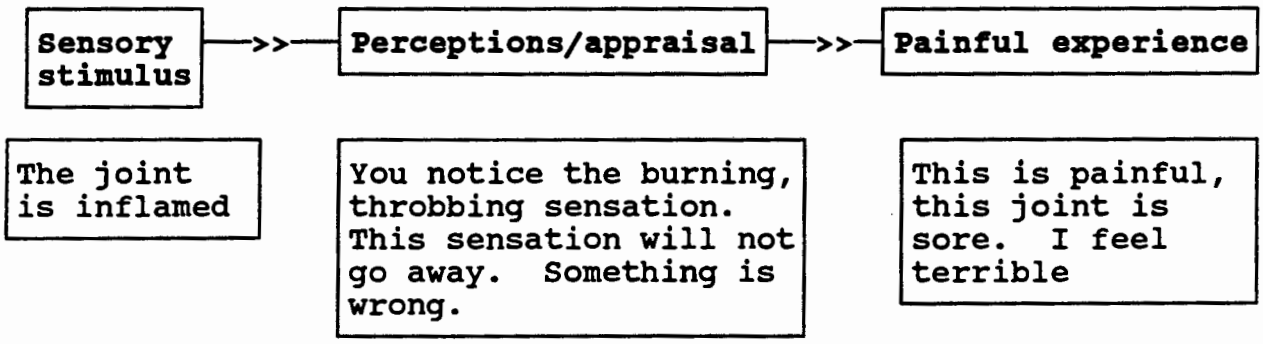
(c) Problem-solve by asking questions, not directing. If goal was too ambitious, reduce it next time.

(2) Lecturette: Coping strategies [15 MIN]

In session 3 we discussed the stress experience and its relation to our experience of pain. Today we are going to examine ways of intervening in such experiences, ways of coping with stress and with the tension - pain cycle.

Consider again the model we used in the last session.

We can also superimpose a similar (though slightly simplified) model of pain



Generally we are unable to avoid the stressors, these events, experiences or sensations occur and are usually beyond our control. Your pain exists because of RA; the traffic jam occurs because an accident on the road is blocking the traffic. It is difficult to intervene effectively with the source. We cannot remove the smashed vehicles. Sometimes our medication does not relieve the inflammation at the joint.

But, we can intervene in terms of:

(1) Our appraisal of what is happening/has happened.

We are in pain, how do we make sense of it? We can alter our beliefs, attitudes and perceptions of what is happening. This can be a very satisfying revelation for those who feel that they are unable to cope.

We can cope with what is happening to us, by re-examining our attitudes and beliefs about what it **means** to us.

For example: "It doesn't matter if we're late for the appointment", or "This pain is not a punishment, I'm not the only one suffering".

We can re-define the way we perceive or think about it (re-interpret what is happening or, "this pain could be worse").

For example: "Traffic jams are a fact of modern life, it is not my fault".

Finally, in terms of appraisal, we can change our life-style or behaviour which may be contributing to what is happening to us.

For example: Structure day to anticipate possible traffic jams, or avoid situations, activities that increase pain unnecessarily).

(2) We can also intervene with the "stress response", or pain experience.

When we feel the stress, we can learn to relax, psychologically and physically. We can turn on the music, take a deep breath and settle back in our seat. We can take our mind off the pain by thinking about something else, by engaging in some activity that prevents the pain from taking up our whole experience.

Coping strategies can be adaptive or maladaptive; constructive or destructive. The above strategies are adaptive, they seek to actively deal with the experience; to reduce or re-interpret what is happening to you. Maladaptive strategies do not effectively deal with stress or your pain. Yelling and driving recklessly will not get you to your appointment on time. Thinking about your pain, being preoccupied with your pain experience and guarding your muscles to "try and be comfortable", will not effectively reduce the pain you are experiencing.

This program is specifically geared towards helping you develop more adaptive, constructive and effective strategies to cope with pain and stress. But, in order to do so, it is necessary that you examine your own coping strategies and make decisions or choices about what is effective and what is not.

(3) Discussion: List and discuss personal coping strategies [15 MIN]

(4) Goal setting: [15 MIN]

(hand out goal sheets)

Break up into two smaller groups and discuss goals for next week. New goals are set based on success during this last week. Activity goals should be set on a daily basis again. Each of you must share these goals with the rest of the group.
Participants are reminded to bring their pain/tension diaries to the next session and make another phone call.

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B. SKILLS ACQUISITION AND TRAINING

SESSION 5: PAIN DIARY AND RELAXATION

(1) Pain and tension diary feedback. [20 min]

(The facilitator provides each participant with two sheets of paper to record their daily tension and pain levels over the previous two weeks. They utilize their pain/tension diaries to extract this information. The group is sub-divided into two smaller groups).

In your groups, discuss your pain/tension charts, looking particularly at how these levels fluctuate. Try and give reasons why there is such variation and explore what this information means to you in terms of understanding your pain and it's relationship with the level of tension in your life.

(2) Lecturette: Relaxation training and the relaxation response [10 MIN]

We have discussed the psychological and physical effects of stress. Nature has a complementary or opposite set of psychological and physical processes which counter those negative feelings, sensations caused by stress. It is called the "relaxation response", and has been extensively researched in terms of its beneficial effects for the individual.

It is characterized by a feeling of warmth, heaviness and looseness in the body, and associated feelings of peace, tranquility and self-awareness - of "being in touch" with one's inner-self.

Researchers have developed some highly effective relaxation techniques for inducing this relaxation response. They are simple activities that make you feel relaxed and at peace. There are, however, many other activities that can serve this relaxation function. For example, knitting, watching a sunset on the beach, or lying back and listening to music, can all help us to relax and reduce tension.

Relaxation is an effective way to manage pain, and it can be utilized:

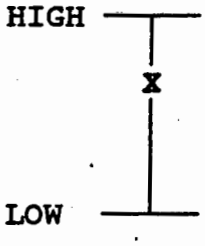
(a) when pain makes it difficult to get to sleep;

(b) when experiencing stress (and associated tension, worry and depression) which increases pain;

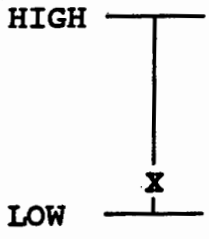
(c) to relieve symptoms of RA such as stiffness;

(d) to reduce fatigue, normalize blood pressure, and reduce all these symptoms of anxiety.

Relaxation, however, is a skill that must be developed. Few people can do it successfully the first time. Our ambient stress level, i.e. our own particular individual level of tension, is generally quite high (see diagram). Repeated practice is necessary to successfully overcome this tension we normally carry around with ourselves.



NORMAL LEVEL OF TENSION



OPTIMAL LEVEL OF TENSION

(3) Training: Progressive muscle relaxation [20 MIN]

(Participants to lie down on the mats and make themselves as comfortable as possible. Facilitator utilizes the Jacobson technique of first tensing and then relaxing the main muscle groups)

(4) Discussion: Describe your personal experiences [10 MIN]

Each person describes his/her experiences. Was she able to relax? What problems, if any, were encountered? How might they be solved? Some suggestions: Don't be concerned if thoughts of your own keep creeping in. Relaxation is a skill that doesn't develop overnight.

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SESSION 6: RELAXATION AND GUIDED IMAGERY

(1) Goals feedback [15 MIN]

Each participant tells:

(a) Previous week's goals and rewards

(b) Whether achieved - why, why not?

(c) Problem-solve by asking questions, not directing.Give out several copies of the goal sheet to each person and encourage them to continue the goal setting process on their own.

(2) Lecturette: Use of engaging and relaxing pastimes [10 MIN]

Part of the relaxation technique's efficacy lies in its ability to take your mind off the pain, and onto something else that is pleasant and comfortable. By taking your attention off your pain, you find that it actually decreases. Over the next few sessions, we will learn some very effective, systematic methods to manage pain in specific situations/activities. But, today, we are going to discuss one of the most important ways to use attention in the control of chronic pain. This is simply acquiring very engaging pastimes and hobbies. If you have something that you are really interested in doing, that is very involving of your attention, then you will have something to turn to on a "bad day". You may already have such activities (e.g. reading, drawing, etc.).

Think about which activities are available to you now, and which you could think about adopting, or re-adopting. For next week's goal, try to use some engaging activity at times when you are in pain. Remember, your leisure time should include such an activity which helps you to relax and take your mind off your daily hassles. Try it for yourself, and note your improvement. You need to list any such activities and hobbies, and make a realistic choice of which would be the most successful in capturing your attention. Remember, managing pain is learning to make choices, choices which are constructive in helping you adapt to your pain.

(3) Training: Progressive muscle relaxation and guided imagery [20 MIN]

(Facilitator leads the group through Jacobsonian relaxation training into an autogenic technique to enhance perception of the state of relaxation)

(4) Goal setting: [15 MIN]

The group will again break up into two smaller groups to discuss goals for next week. Please try to incorporate the use of some of the techniques we've just learned as part of your goal.
Continue practicing relaxation before you go to sleep. Share your goals with the rest of the group, and arrange to call one other person during the week.

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SESSION 7: COGNITIVE SKILL - ATTENTION REFOCUSSING

(1) Introduction: Demonstration of skills effectiveness -Part 1 [15 MIN]

(Facilitator should have two calibrated visual analogue pain scales and one strategy work-sheet for each participant. A stopwatch must also be on hand).

As a demonstration of the effectiveness of some specific pain control techniques that we will learn today, we're going to conduct an exercise. Each of you will actually experience some pain right now, but this is necessary so that we can see how great a difference it makes to use the techniques that you'll learn today. Each person should select an exercise that they can perform for thirty seconds, twice during the session. Lifting the arms or legs, getting up and down from a chair, walking rapidly around the room are all examples. First the activities will be performed without the use of strategies.

After each participant has selected an activity, the trainer says, "begin". The trainer times the thirty seconds of activity and says "stop". Then each participant indicates the greatest level of pain experienced during the thirty seconds and marks it on the analogue scale.

(2) Cognitive skills training: Attention refocussing [20 MIN]

(Hand out "Attention refocussing" work-sheet)

As we discussed in the last session, one important factor in addition to physical stimulation that influences how much pain we feel, is the amount of attention we focus on our pain sensations. When we concentrate only on our pain sensations, it takes up almost all of our awareness; we can hardly think about anything else. But if we pay less attention to the pain, we become less aware of it and, as a result, it bothers us less.

There are many examples of people who have been hurt but who concentrate so much on something else that they don even realize that they have been injured until sometime later. Many athletes are able to compete in spite of injuries because they focus their attention so completely on their performance and what is happening around them, that they cannot think about anything else. If they don't think about how much pain they are in, then they don't feel it as much.

Discussion: Can you think of examples of attention affecting pain in your own life?

The first technique I will describe is called "attention refocussing". It involves shifting your attention away from thinking about your pain to some other activity. By thinking about or doing something else, the pain will not intrude upon you as much and you will be less bothered by it. These strategies are often used by people who have not learned them specifically. You yourself may use techniques like these. Even children find them useful when, for example, dealing with pain when at the dentist (some find it easier to get through the pain by counting the holes in the tiles of the ceiling, or by counting backwards or staring out the window).

These activities can only be practiced when your mind is relatively free of other concerns, and only for relatively short periods of time. You may find them most useful when you are doing something physical that you find particularly painful, such as walking or climbing stairs, carrying parcels, etc.

Here are some examples of things you could do to divert your attention. If you have your own, continue to use them. Be creative in your selection of activities. For example

(1) While climbing stairs, you could plan exactly what you are going to do and say at the top of the stairs. Imagine every single thing that you will be doing for the next few minutes.

Or,

You could name a different kind of animal/motor vehicle/food for each stair that you climb.

(2) While you are buttoning your shirt, you could give people's names for each letter of the alphabet in order.

(3) While you are getting up from bed, or a chair, or out of a car, you could imagine that you are suspended on a rope where you are light and almost weightless, floating effortlessly upward.

Or,

You could count backwards from 100 by two's each time as quickly as possible. Each time, try to beat your last record.

(4) While you are carrying something heavy, you could imagine that you are transporting miniature versions of all the people you know.

Or,

You could try to remember exactly what is in the bag/parcel, and how much it cost you.

In addition to these structured techniques, just finding an activity that is very engaging for you can achieve the same effect. Engage in a hobby that is fascinating to you and notice how you can 'escape' the pain. Gardening, playing a musical instrument, making things, reading/writing, watching T.V. are all

examples. You can use any kind of mental or any combination of activities, so long as it occupies your mind and makes you focus your attention on it. By concentrating on these kinds of activities you will keep your attention occupied and you will find yourself thinking less about your pain.

Please use the work-sheet to describe at least five things that you think would work well to divert your attention from your pain at times when you want to alleviate it. At least one should be some sort of hobby that you currently enjoy or would like to adopt. It should be something that you could work on at home whenever you are in pain, not something that you need to go elsewhere to do. List the hobby under (e) on the work-sheet.

In order to see how easily you can focus your attention on these things, we will try them out. Although we can't engage in the activities here, we can at least try out the mental techniques. Let us start with the things described in,

(a)...(20-sec. pause).

Now let us try the things listed under,

(b)...(20-sec. pause).

Now what about the things described in,

(c)...(20-sec. pause).

Now the things in,

(d)...(20-sec. pause).

Do you have any questions?... Now that you have tried using these examples, do you think this technique will work well? Is there anything you wish to change?

Discussion: Techniques one can use to refocus one's attention.

(3) Demonstration of skills effectiveness: Part 2 [15 MIN]

Now, time to practice these skills! This time we will practice the same activity as at the beginning of the session, but this time using the techniques. Each person should take a few minutes to think of some strategies that might work while performing her activity. Write them down on the "Strategies Work-sheet".

The trainer should read all of the strategies and check them to make sure that they are engaging enough, and take up enough time during which they are used. Everyone should be encouraged to skip around between strategies at will. Again, the trainer times the thirty seconds of activity and says "stop". Then each participant indicates the greatest level of pain experienced during the thirty seconds and marks it on the analogue scale.

Discussion: Each person reports how much pain they experienced before and after learning the techniques. Also report any difficulties encountered in use of the techniques; which ones seemed to work best.

(4) Brief relaxation [10 MIN]

(A shortened version of Jacobson is run with the participants).

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SESSION 8: COGNITIVE SKILL - VIVID IMAGERY

(1) Goals' feedback: [15 MIN]

Each participant tells:

(a) Previous week's goals and rewards

(b) Whether achieved - why, why not?

(c) Problem-solve by asking questions, not directing. Give out copies of goal chart to each person, and encourage them to continue the goal setting process on their own.

(d) Discussion: Use of pain strategies thus far. Who used them? Which used? Did they work? If not, how could they be improved upon.

(2) Cognitive skills training: Vivid imagery [20 MIN]

(Facilitator hands out work-sheet: "Vivid imagery")

Let's move on to the second technique. People can remove themselves from their present situation by using their imagination to pace themselves in some more pleasant situation. Sometimes when we fantasize or daydream about things that we wish would happen, it is because we want to make our current situation bother us less. This technique is called "vivid imagery". It is related to the guided imagery that we practiced in conjunction with relaxation, but is more involving and can be used even when you are physically active. It is better for longer-term painful activities, such as walking some distance or having to sit and stand for a long period of time, as when you are waiting at the doctor's office.

(A). One way to use imagery to cope with pain is to use your memory to recall pleasant scenes from your past. For example, you could try to remember every detail of a recent event that made you feel happy. Who was there, what happened, what did you have to eat and drink, what did you talk about. Or you could think about a pleasant holiday that you had recently: where you went, what you saw, what you did, what the weather was like, everything exactly as it happened. Fantasies that might be especially useful for people with arthritis, are imagining very warm experiences - such as lying on a beach and absorbing the sun. Or, if you find that cold is more effective than heat as a treatment for your arthritis, to imagine cold experiences. For example, swimming in the cold sea.

During the minute, try to remember two good things that happened to you in the past and that you could use effectively to direct your attention away from any pain that you might experience during your daily activities. To help you to remember these

things, please write four or five key-words for each of them in the space provided on your handout.

To find out how easily you can focus your attention on these scenes, you will try to remember each scene as vividly and completely as possible, as if you were actually reliving them. Try to remember every detail: where you were, who else was there, what you did said and felt, and so on.

Let us start with scene 1...(20-sec pause).

Now let us try scene 2...(20-sec. pause).

Do you have any questions or comments?

(B). Another way to use imagery is to dream up fantasy scenes, situations filled with positive feelings and pleasant events. For example, you could fantasize about a romantic encounter you would like to have, how you would spend a million Rand, what your dream clothes would look like if you were not limited to by money or how you would furnish your present house or flat, what kind of car you would by if you had unlimited funds, or what an ideal trip might be like. Imagine any scene that is pleasant and positive for you. Use the following minute to choose two scenes that you think would help you to direct you attention away from any pain that you might experience, and write down four or five key words as a reminder for each scene in the space provided on your handout.

In order to see how easy it is for you to focus your attention on these scenes, you will try to immerse yourself in each scene. You will use your imagination to fill in all of the details of each situation.

Let us start with scene 1...(20-sec pause).

Now let us try scene 2...(20-sec. pause).

Do you have any questions or comments? Have you used this technique yourself at times?

Discussion: Ask participants to describe their experiences. Are any problems foreseen in using this technique?

(3) Goal setting [15 MIN]

The group breaks up into two smaller groups to discuss goals for next week. Please try to incorporate use of some of the techniques we've learned as part of your goal. For example, if your activity goal is climbing stairs, you might set the goal of trying two different cognitive strategies while you are climbing them. Daily practice of relaxation is still recommended.

Each participant shares goals with the rest of the group.

(5) Blood is drawn and tests distributed. [10 MIN]

Participants are reminded of the nature of the questionnaires and to complete them truthfully and honestly. It is for their own benefit that the program is being run.
Participants are thanked for donating blood samples for analysis.

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SESSION 9: COGNITIVE SKILL - DISSOCIATION

(1) Lecturette: Dissociation of pain awareness [15 MIN]

(Facilitator hands out work-sheet: "Dissociation")

Dissociation involves trying mentally to separate yourself from the part of your body that is in pain. It is especially effective when your pain is acute and very severe, so severe that it seems impossible to distract yourself from it. You may have already experienced a sense of dissociation from your body, particularly your hands, arms and legs, during the relaxation exercises we have been through. It is a sense of your limb not belonging to you. This can be a mildly disturbing sensation; after all, we all like to be in touch with our bodily parts.

A good example of what dissociation should feel like if practiced effectively, is to compare it to the sensation of your leg or arms falling asleep. As when you wake up during the night to find that you've stopped the blood circulation in your arms by pinching them underneath your head/torso. That sense of these arms not "belonging" to you, is the sensation we want to achieve with the dissociation technique. Think of the relief you can achieve if you are able to successfully dissociate yourself from severe pain in your limbs. Consider the fire-walkers who do not experience any pain in their feet!

Discussion: Personal experience of dissociation

(2) Training: Demonstration of skills effectiveness [10 MIN]

(Participants make themselves comfortable)

To use this technique, we will use the next few minutes to familiarize ourselves with it. Let us start now. Please sit comfortably and quietly, without moving any part of your body.

Imagine that your joint is in pain. Any joint. Picture that joint (or a larger part of the body, such as your hand) is separate from the rest of your body. Imagine that your joint is completely insensitive and therefore does not feel any pain. For example, tell yourself that your joint does not belong to you; it is completely separate from you, and just happens to be there. Think of your joint as rubbery or waxen. Think of your joint as numb. Because it is not a part of your body, you cannot feel anything that happens to it; whatever happens to your joint does not affect you at all.

Just do this for the next little while ... feel the sense of dissociation between you and the joint.

Now take a minute to write down two or three key words to help you remember the ideas that you might want to use some time you are in pain.

Do you have any questions or comments. Have you used this technique before?

Discussion.

(3) Relaxation: [20 MIN]

(Facilitator takes participants through Jacobsonian and autogenic techniques, emphasizing sense of dissociation, warmth, heaviness)

(4) Discussion: [10 MIN]

How did you feel? Did you notice a sense of being cut-off from your body? Did this exercise help you?

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SESSION 10: COGNITIVE SKILL - RE-LABELLING

(1) Goals feedback: [15 MIN]

Each participant tells:

(a) Previous week's goals and rewards

(b) Whether achieved - why, why not?

(c) Problem-solve by asking questions, not directing. Give out copies of goal chart to each person, and encourage them to continue the goal setting process on their own.

(d) Discussion: Use of pain strategies thus far. Who used them? Which used? Did they work? If not, how could they be improved upon.

(2) Lecturette: Re-labelling and pain experience [15 MIN]

(Facilitator hands out work-sheet: "Re-labelling")

In the last three sessions, we have examined ways in which we can refocus our attention away from the pain. Ways in which we can "push", as it were, the pain beyond our conscious experience. Sometimes this may not be possible, or only partly successful, especially if the pain is very severe or very intense.

Another way to manage intense pain, to make it more bearable, is to pick out all the sensations that are there, but do not think of them as painful or bothersome. One's attention is drawn to the sensation of the pain experience. Instead of merely feeling the pain, try to be aware of the quality and nature of the pain. A common saying is to "ride the pain". This is precisely what this technique aims to do. Here, you can go with the sensations, experience them, but do not let yourself be bothered by them. Perhaps you know of people who must endure a lot of pain while they work - athletes or dancers for example who work with the maximum "no pain, no gain" - but who carry on because they think of the pain as a nuisance, as nothing serious. Maybe this person is you yourself.

This is an example of a technique called "re-labelling". In the case of arthritis, it is important not to think of the minor pain that comes with moderately increased activity as the result of damage to your joint, but rather as part of a beneficial process that is actually making your body healthier - the "no pain, no gain" maxim. Of course, this is not true if pain is made more severe.

It is important to remember that you can alter your experience of pain by re-interpreting, re-labelling it. This is changing your appraisal of your pain, making sense of it in a different way,

seeing it in a different light. Especially when it accompanies increased activity to reduce stiffness and tension in the joints and muscles which is beneficial in the long-term, but discomforting whilst being undertaken.

3) Demonstration of skill's effectiveness [15 MIN]

'Participants to make themselves comfortable and either focus on pain present, or imagine existence of pain in one of their joints, muscles)

Imagine that one of your joints is hurting. Focus on that part of your body that gives you the most pain and discomfort. If you are in pain right now, focus your attention on that pain. Feel the intensity of the sensations, ... and think about them - analyze them.... Try to find all kinds of sensations: tingling, burning, pressure, numbness, piercing, throbbing, stabbing... Concentrate.... feel your pulse or muscle tension in and around your joint.... but don't think of the sensations of pain..... Instead of thinking of the sensations as painful, think of them as unusual..... the sensations may seem to increase, decrease or level off.... Just go along with them... See if you can begin to think of the sensations as mere dullness as if the joint has become completely numb.. Remember, you have the ability to re-interpret, re-lable the pain you are experiencing.

Now take a minute to write down a few words on you work-sheet to help you remember the ideas that you might want to use at times when you are in pain.

Discussion: How did it feel Could you grasp what was being required of you. In what ways could it be improved.

4) Relaxation: [10 MIN]

(A brief relaxation session: shortened version of Jacobson technique).

5) Goal setting: [15 MIN]

(Facilitator hands out new goal sheets)

The group will again break up into two small groups to discuss goals for next week. Please try to incorporate use of some of the techniques we have just learned as part of your goal. Daily practice of relaxation is still recommended.

Each participant shares goal(s) with the rest of the group.

How did phone calls go last week? Each person agrees to call one other person by next session.

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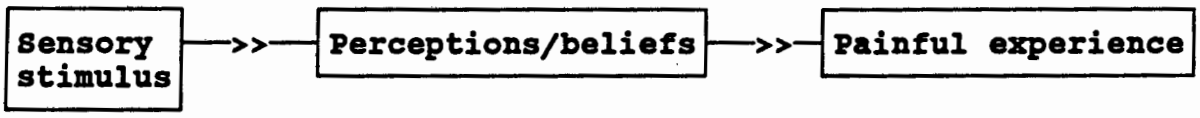
SESSION 11: SELF-ENCOURAGEMENT AND SELF-APPRAISAL

1) Lecturette: Appraisal and pain coping - the role of self-encouragement [15 MIN]

Recall session 3 when we discussed the model of stress and related it to pain.

and



In this programme thus far we have tried to demonstrate the extent to which our appraisal of the painful stimulus influences our experience thereof. We have practiced ways in refocusing our attention using vivid imagery, dissociating ourselves from the pain. In last session we practiced re-labelling the pain experience, focussing on the sensations of the pain rather than the pain itself.

We have attempted to change or alter our appraisal of the painful stimulus, the pain in the joint or ligament. To the extent that we are successful in achieving these goals, our perceptions or appraisal of our ability to manage our pain is enhanced. We begin to feel that we are coping better with our pain. Appraisal thus works on two levels in pain management:

(1) the level of the pain experience

(2) the level of our perceptions of being able to manage our pain

Today we are going to address this second level. By attaining at least some control over our pain, we are able to alter our appraisal of ourselves, as persons who can cope with our pain. Consider your experiences over the last 10 sessions. Think of yourself now, and how you were at the onset of this programme. Have you learned anything about how to manage pain, could you utilize any of these techniques. If so, how does it make you feel about yourself. Have you been rewarding yourself for achieving your goals that you have set yourself?

(2) Discussion: [15 MIN]

What we have learned and experienced thus far. What changes, if any, have occurred in our appraisal of our pain and ability to manage it.

Our perception of ourselves, and our ability to manage pain play a fundamental and integral part in the success of any of these non-medical techniques we have demonstrated.

(3) Training: Using self-encouragement [15 MIN]

While you are practicing your increased activity, and you are using all of the techniques you have learned, you may sometimes have difficulties and may find yourself becoming discouraged and no longer trying to cope with it. What you do and what you say to yourself can affect your ability to cope with the pain. The most important thing to focus on is how much progress you have already made. You deserve to be proud of yourself. You need to be your own cheerleader. setting goals and achieving them, trying to actively deal with stress and pain is hard work and requires a great deal of time and practice. You have required some simple, but great skills now. You can deal with pain. When you are in pain, take time to think creatively about what you can do to deal with it. Think about a plan. Make active attempts to manage or re-appraisal the pain. Thinking about the pain will not help anything.

What are some of the things you can do instead?

Imagine that your pain is getting to you. The first thing you must do is rest. Then concentrate fully on one coping technique - the one that works best for you. Try a relaxation technique, or turn to a distracting past-time to ease the build up of tension and the sense of your pain taking over your world/experience. If your pain demands your attention, use dissociation or re-labelling. Remember that you do not need to eliminate pain entirely, you just need to keep it manageable. That in itself, "eliminates" pain.

You **can** keep pain under control. Whenever you have achieved success in coping with your pain, remember to always tell yourself, "I deserve a pat on the back for having tried. Even if I brought the pain under control just a little bit, I deserve to feel proud". This is thinking positively and constructively about yourself, and your ability to control your pain.

Discussion: Do you have any comments? When and how has each of you used this technique in your own life. What things can each of you think of, that another person in the group should feel proud about?

4) Relaxation: [15 MIN]

(Participants to lie down and make themselves comfortable. Facilitator leads them through Jacobson technique, into guided visualization exercise.)

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SESSION 12: SOCIAL FACTORS AND COMMUNICATION

(1) Goals feedback [10 MIN]

Each participant tells:

(a) Previous week's goals and rewards

(b) Whether achieved - why, why not?

(c) Problem-solve by asking questions, not directing. Give out copies of goal chart to each person, and encourage them to continue the goal setting process on their own.

(2) Discussion: Support of spouse/family and significant others [15 MIN]

You are not the only one who lives with your pain, your family does too. How does your pain affect those around you? What are their reactions to it? Do you think these reactions are helpful? If not, what can be done to make them react in more helpful ways? Share your experience with the groups.

(3) Lecturette: Pain and communication [10 MIN]

How do you communicate with your spouse on issues related to pain/discomfort as a result of RA? When do you communicate your experience of pain and for what reasons? We are alone in our pain experience and sometimes we desperately need to communicate our experience thereof to significant persons in our lives. Unfortunately, we tend to communicate with what can be called "you-messages". Consider this scenario in which a woman with arthritis is talking with her husband.

Scenario 1: "You-message" example

(The husband is complaining because the wife does not ever want to go out and the wife is unhappy because her husband is not more considerate of her arthritis.)

Husband: Hurry up. I don't want to be late.

Wife: You're always in a hurry. Don't you understand yet that I hurt?

Husband: You always complain about your arthritis. How do you think I feel, never getting to go anywhere?

Wife: All you ever think about is yourself, its no wonder I have pain.

etc...

When we are angry or upset, most of us tend to blame someone else. This is human nature. The result is that the other person also becomes angry and defensive, and often nothing is accomplished except bad feelings. One way of avoiding this rather unpleasant happening is to utilize "I-messages". The concept is simple. Whenever you want to express a feeling, express it in terms of how you feel.

This scenario has a different "climate" to it when "I-messages" are being used.

Scenario 2: "I-message" example

Husband: Hurry up. I don't want to be late.

Wife: I have a lot of pain today.

Husband: When I hear you complain about your arthritis, it makes me angry. Sometimes I feel your pain won't let me go anywhere.

Wife: I see this pain is a problem for both of us.

Although there is conflict, it is recognized and honestly expressed. Using "I-messages", in a sense withdraw us from being caught up in a vicious cycle of blame and anger. They can be used in all contexts of communication. For example:

Instead of:

"Why can't you children be more helpful? You are just selfish and inconsiderate".

Try:

"I am really upset that you children have not made your beds and tidied your rooms".

Instead of:

"Why are you always late? You must love burned suppers".

Try:

"I really get angry when you are late and the supper gets spoilt".

In the second examples, the person is reporting his/her own feelings without blaming the other person. "I-messages" tend to open up communication rather than close it down. They make our personal relationships less stressful and enable us to deal with tension in a constructive and adaptive manner.

(4) Demonstration of skill's effectiveness [15 MIN]

(Overhead of examples of using "I-messages")

One or more participants should respond to each situation using "I-messages". If someone is having a problem, get another (successful) participant to act as a coach. Be sure everyone in the programme actually says an "I-message".

a. You want to tell your doctor he really does not understand the pain.

b. Your children are pressurizing you to prepare a favourite family supper that you don't feel up to.

c Your friends are pushing you to walk faster.

d. You need help carrying the groceries, even-though it is a small bag.

e. The receptionist at the doctor's office tells you that you can't have an appointment for 3 weeks and you hurt now.

f. Your boss wants you to work extra hours.

g. Your neighbour's dog keeps making a mess on your lawn or keeps barking all night.

Like any other new skill, these "I-messages" take practice. Start by just listening to yourself and others. How often do you hear blaming? Then try to use an "I-message" at least once a day. You will be surprised how quickly they can become a habit.

(5) Goal setting [10 MIN]

The group separates into two smaller groups to discuss personal goals for the next week. Try to improve your communication skills and use "I-messages" as one of your goals. Continue to practice relaxation daily.

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C. APPLICATION AND FOLLOW-THROUGH

SESSION 13: PAIN - TENSION DIARIES AND PERSONAL EXPERIENCES THUS FAR.

(1) Feedback: Pain-Tension diary progress [15 MIN]

(2) Discussion: [20 MIN]

Personal experiences using the techniques and self-management of pain. What has worked for you and what have you found particularly valuable about these techniques. How has your life changed since you have been using these pain management techniques?

(3) Relaxation training: [25 MIN]

(Participants asked to make themselves comfortable; the trainer takes them through a deep autogenic training session)

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SESSION 14: LEARNED HELPLESSNESS AND SELF-APPRAISAL

(1) Goals feedback: [10 MIN]

Each participant tells:

(a) Previous week's goals and rewards

(b) Whether achieved - why, why not?

(c) Problem-solve by asking questions, not directing. Give out copies of goal chart to each person, and encourage them to continue the goal setting process on their own.

(2) Discussion: [20 MIN]

Learned helplessness and self-appraisal. How well am I coping, how able am I to alleviate my pain? Is there anything I can do about my pain. I know that my pain is never going to go away, can I live with this realization? If the doctors cannot relieve my pain, what can I do?

(3) Relaxation training: [15 MIN]

(Participants are taken through an autogenic training session)

(4) Goal setting: [10 MIN]

The group separates into two smaller groups to discuss personal goals for the next week. Try to improve your perception of yourself as someone who can cope with their pain. Use this as one of your goals and reward yourself if you can achieve this goal. Continue to practice relaxation daily.

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SESSION 15: FEEDBACK AND FOLLOW-THROUGH

(1) Feedback from trainer: [20 MIN]

Feedback is given to the group as a whole as well as to individual persons. Each participant receives a report of their progress over the previous few weeks by the trainer, and the members give each other feedback on their progress on the program.

(2) Discussion: Where to from here? [20 MIN]

The trainer facilitates the discussion on future plans and use of the techniques learned and practiced on the program, and how they can continue to manage their pain effectively when the program ends. Participants are encouraged to continue setting goals for themselves and to utilize these techniques whenever they want to alleviate their pain. Discussion should focus on practical issues, such as who to call if pain becomes worse, what advice to seek. The emphasis is on problem-focussed coping, and the participants are encouraged to share their ideas of how they would resolve such problems as they may encounter in their daily lives.

Participants are asked to bring a snack for next session's refreshments. The program will end with a little party.

(3) Relaxation training: [20 MIN]

(Participants are taken through an autogenic training session)

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SESSION 16: CLOSURE

(1) Goals feedback and long-term goal setting: [15 MIN]

How to set and achieve long-term goals. Each participant should take time to think about what changes they would like to continue to make in their lives to help them live with arthritis. Goals should be set for the end of the year and even in the next five years. Short term goals should then be set in order to achieve these long-term goals. Participants should discuss these goals with the group.

(2) Discussion: Personal and groups' experience of the program [20 MIN]

How has this program influenced your ability to manage pain. What did you gain from this program? Discuss your experiences over the last eight weeks and what achievements, if any, you have made. Participants are encouraged to give feedback to each other of their progress in the program. Each person states what she has accomplished, and what she plans to continue.

(3) Blood drawn and tests distributed and future follow-up discussed: [15 MIN]

(4) Closure: Debriefing and saying good-bye [15 MIN]

Participants are thanked for their participation in the program, and their enthusiasm they have shown to learn and practice these techniques. The trainer promises to contact each one in the next month or so to check on their progress. Telephone numbers and contact addresses are shared. The group may wish to continue meeting. This is fine, as long as they organize the meetings themselves.

Refreshments.

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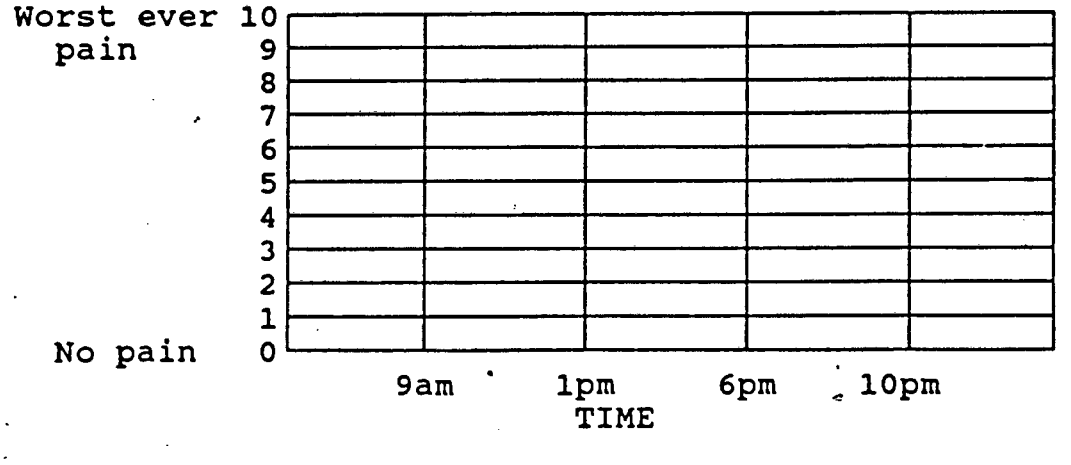
PAIN AND TENSION RECORD

DATE: ___/___/1990 MONDAY

TIME	TENSION (0-10)	PAIN (0-10)	WHAT AM I DOING?	WHAT AM I THINKING?
9am				
1pm				
5pm				
10pm				

PAIN DIARY



GOAL SHEET

STEP 1. LONG-TERM ACTIVITY GOAL(S): _____

_____

_____

STEP 2. SHORT-TERM ACTIVITY GOAL(S): _____

_____

_____

_____

STEP 3.

GOAL/ACTIVITY		DETAILS	
Action:	_____	When:	_____
		Where:	_____
		How far/much how many:	_____
		How often:	_____
Action:	_____	When:	_____
		Where:	_____
		How far/much how many:	_____
		How often:	_____

STEP 4. REWARD FOR ACHIEVING GOAL(S):

(Specify) _____

_____

ATTENTION REFOCUSSING WORKSHEET

	STRATEGY	FOR WHAT ACTIVITIES?
(A)	_____	_____
	_____	_____
		_____
(B)	_____	_____
	_____	_____
		_____
(C)	_____	_____
	_____	_____
		_____
(D)	_____	_____
	_____	_____
		_____

(E) (HOBBY): _____

_____

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DISSOCIATION WORKSHEET

STRATEGY/IDEA: 1. _____

_____

Key words: _____

STRATEGY/IDEA: 2. _____

_____

Key words: _____

STRATEGY/IDEA: 3. _____

_____

Key words: _____

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VIVID IMAGERY WORKSHEET

(A) PAST PLEASANT SCENE: 1. _____

_____

_____

Key words: _____

2. _____

_____

_____

Key words: _____

(B) FANTASY SITUATION/SCENE: 1. _____

_____

_____

Key words: _____

2. _____

_____

_____

Key words: _____

RELABELLING WORKSHEET

STRATEGY/IDEA: **1.** Sensation as..._____

_____

Key words: _____

STRATEGY/IDEA: **2.** Sensation as..._____

_____

Key words: _____

STRATEGY/IDEA: **3.** Sensation as..._____

_____

Key words: _____

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