ALEXITHYMIA CHRONIC PAIN AND DEPRESSION

by

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ABSTRACT

Questionnaires from 107 subjects were analysed to test the following hypotheses: (i) there is a significant difference in alexithymic scores, between groups on certain biographical variables, such as age, sex, race, marital status, education and income; (ii) there is a significant difference in alexithymic scores between subjects with chronic non-organic pain (CNOP), chronic organic pain (COP) and subjects without any pain (NO PAIN); (iii) there is a significant difference in levels of alexithymia in patients who are experiencing clinical levels of depression as opposed to non-depressed subjects. (iv) the levels of an individual's alexithymic scores will be jointly determined by their levels of depression and the organicity of their pain. Instruments for assessment included the Zung Self-Rating Depression Scale (SDS) and the Toronto Alexithymia Scale-Revised (TAS-R). Results show that there are significant differences in alexithymia levels within the education and income groups; between the depressed and "normal" group and the chronic pain (CP) group (COP and CNOP) and NO PAIN group. No differences are noted between the COP and

CNOP groups. No interaction between CNOP and depression is noted. Research in alexithymia is in its infancy as the first two reliable and valid instruments (Toronto Alexithymia Scale (TAS); Toronto Alexithymia Scale-Revised; (TAS-R)) have only recently been formulated. The beginning of the identification of alexithymic groups is necessary for ongoing research.

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This thesis is the result of my own work except where otherwise indicated.

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1. INTRODUCTION

The term, "alexithymia" or "no words for mood" is relatively new. Little empirically supported information and a considerable amount of speculation exists in this area. The time lag between the formulation of the concept in 1973, and the formulation of a reliable and valid measurement instrument in 1988 accounts for the dearth of information in this area. Research in this area is really just beginning.

Alexithymia has been linked to chronic pain (CP) and depression, where similar symptoms are shared, such as disinterest, dullness and anhedonia. Alexithymia has also been proposed as an etiological factor, a maintaining factor and an adaptive response to both chronic pain (CP) and depression. Many hypotheses have been generated to account for this.

On the basis of these existing hypotheses it is necessary to establish empirically whether patients with chronic pain (CP), particularly chronic non-organic pain (CNOP), and/or depression have elevated alexithymic levels. Once alexithymic groups are identified, it will be feasible for ongoing research to explore the nature of alexithymia, how these clinical entities are etiologically related and therapeutic interventions designed.

The aim of this thesis is to establish: (i) whether there are any differences in alexithymic scores between groups on certain biographical variables; (ii) whether patients with chronic non-organic pain (CNOP) are more alexithymic than those with chronic organic pain (COP) and those without any pain (NO PAIN). The distinction in chronic pain (CP) is controversial and will be discussed in detail; (iii) whether patients with clinical depression are more alexithymic than normals; (iv) whether alexithymic scores will be jointly determined by elevated levels of depression and chronic pain (CP), i.e. an interaction.

The literature review will provide a discussion of alexithymia, chronic pain (CP) and depression. An account of the methodology, results, discussion and concluding remarks will follow.

2. LITERATURE REVIEW

2.1. ALEXITHYMIA

2.1.1. Introduction

The term "alexithymia", meaning literally "no words for mood", was coined by Sifneos in 1973, and as the name implies, is a disturbance between cognition and affect. It was originally associated with psychosomatic illnesses, but has since been recognised as a more widely occurring phenomenon. The background, clinical features, mechanisms, research and therapeutic aspects will be discussed.

2.1.2 Background

Although alexithymia was only formally described in 1973, cognitive-affective type disturbances were reported prior to that. Ruesch, in 1948, noted a disturbance of verbal and symbolic expression in psychosomatic patients, whom he referred to as "infantile personalities". Maclean (cited in Taylor, 1984) was developing his concept of the "triune brain" when he observed that many psychosomatic patients showed an intellectual inability to verbalize emotional feelings; he suggested that these emotions found immediate expression through the body, instead of being relayed to the neocortex and finding expression in the symbolic use of words.

"Psychosomatics" originally referred to the physical manifestation of psychological conflicts in specific illnesses such as asthma, ulcerative colitis, rheumatoid arthritis, migraine and peptic ulcers. Gannon and Haynes (1981, p.1) defines these psychosomatic disorders as, "those disorders which are accompanied by observable or self-reported physical change, are exacerbated by psychological states or environmental stress, and are commonly although not exclusively treated with psychotherapies." It was hypothesised that alexithymia played an etiological role in psychosomatics, where the individual's inability to express their emotions or conflicts was expressed in illness.

However, this notion has changed. Firstly, the term "psychosomatics" is outdated (this shift is illustrated by the changes that have occurred in DSM-II and DSM-III), as all illness is now seen to have a psychological component. In spite of this, references to the term, as a concept applying to a particular class of disease, still occur which contributes to confusion in this field. Current references to psychosomatics refer to any physical condition that is precipitated, exacerbated or prolonged by psychological factors, rather than to any distinct group of disorders.

Secondly, research on alexithymia (Sifneos, 1973) indicates that although it exists amongst many patients who would originally have been referred to as psychosomatic, there are many of these disorders which are not associated with alexithymic characteristics. Furthermore, alexithymic characteristics have been found among patients with a wide range of medical and psychiatric disorders, such as somatization disorders (Shipko, 1982); psychogenic pain disorders (Mendelson, 1982); hypochondriasis (Barsky & Klerman, 1983); sexual perversions (McDougall, 1982); substance abuse disorders (Kauhanen et al., 1992) and posttraumatic stress disorders (Krystal, 1968; 1979). Alexithymia has also been described as one of the risk factors that seem to increase the susceptibility to disease, and has been indicated in the research of Todarello et al., (1989), which found a relationship between alexithymia and cancer. Alexithymia has been referred to as a personality trait that exists in the normal population, with a more pronounced presence of alexithymic characteristics among lower levels of intelligence, education and socio-economic status (Pierloot & Vinck, 1977).

2.1.3 Clinical features

Nemiah and Sifneos (cited in Taylor, 1984, p.726) describe alexithymia as a,

> "specific disturbance in an individual's psychic functioning that is manifest primarily in his or her communicative style. This is characterized by markedly reduced or absent symbolic thinking so that inner attitudes, feelings, wishes and drives are not revealed".

These patients have a reality-based, concrete cognitive style, minimal ability to fantasize, stereotypic interpersonal relationships and resistance to traditional psychotherapeutic interventions.

Sifneos (1988, p. 288) describes alexithymics as not being able to link their thoughts with a specific emotional state,

> "One hears a suspicious noise during the night but starts thinking of food; one attacks someone but has no thoughts about what made him angry .... In sum, one can think, can act, can talk and can recognise emotions but cannot connect the thoughts with one's emotions".

Nemiah (cited in Taylor, 1984) adds that alexithymics occasionally have outbursts of rage or tearfulnesss, but few can elaborate on what they were feeling. Alexithymics report few dreams and when they do, they lack colour, bizzareness and symbolism. They follow a robot-like existence, as if they were following an

instruction book. Their constricted emotional functioning and inner psychic life is sometimes revealed by a stiffness of posture and lack of expressive facial movements. Krystal (1979) adds that they have an impaired capacity for empathy in interpersonal relationships.

2.1.4. Mechanisms of alexithymia

The mechanisms, causes or processes of alexithymia have been commented on by many authors, with many variable opinions. Warnes (1986, p.96) reflects this,

> "Is alexithymia a pathology of affect or character neurosis; is it primary or secondary; genetic or developmental? Is it an adaptational deformation related to social class and low psychological sophistication, a life style or a cerebral deficit? Is it global and consistent (trait) or partial and temporary (state)...."

The various explanations for alexithymia will be subsumed under Freyberger (1977) and Sifneos' (1988) distinction between "primary" and "secondary" alexithymia, to facilitate reading of this section. It is important to note that this distinction is only proposed by a few authors, and different writers have different definitions of primary and secondary alexithymia (for example, section 2.1.4.3.)

2.1.4.1. Primary alexithymia

Primary alexithymia has been described as a hereditary interruption of the communication between the limbic system and neocortex due to neuroanatomical defects or a neurobiological deficiency in the form of biochemical or physiological abnormalities (Sifneos, 1988).

Heiberg and Heiberg (cited in Sifneos, 1983) found in a Norwegian study of twins that there was a strong hereditary predisposition to alexithymia; monozygotic twins had a higher concordance for these characteristics, which was absent in the dizygotic pairs. However, these results were inconclusive as the methodology was unsatisfactory; there were a small number of twins - only 33 pairs and the twins were raised in their own families and so were exposed to similar environmental influences.

Sifneos (1988) reviews Hoppe's studies on epileptics who had surgical commisurotomies, and noted the similarities in their, and alexithymics' cognitive style and affective expression. Sifneos (ibid) suggests that hemispheric specialization plays an important role in alexithymia where an interruption of input from the left cerebral hemisphere, in a right handed person may cause an inability to connect

images, fantasies and thoughts with visceral emotions and the use of appropriate language to express feelings. Similarly, an interruption of input from the right hemisphere may be equally important, as it plays a major role in modulating an affective component and in giving to language a special intonation and colouring. Patients with right-hemispheric damage cannot assess emotional priorities and have difficulties in emotional communication in the form of "aprosody".

2.1.4.2 Secondary alexithymia

According to a few authors (Freyberger, 1977; Sifneos, 1988), secondary alexithymia may be associated with or explained by sociocultural, developmental and/or psychodynamic factors, in contrast to primary alexithymia which is associated with innate characteristics.

2.1.4.2.1. Psychodynamic factors

Psychoanalytic theorists account for alexithymia by disturbance in early infant-mother interactions. McDougall (cited in Nemiah, 1977) claims that the source of ego defects in the realm of fantasy formation and for experiencing feelings is accounted for in mother-child relationships. She claims that a mother who either over-indulges or restricts her

child's instincts prohibits him/her from developing a mental representation of the mother as a compensation for her temporary absence. The earliest image of the mother is a prototype of fantasy, and the failure to develop this image damages the capacity to form fantasy as a symbol and expression of instinctual drives.

Other accounts claim that psychological conflicts may predispose the child to use defense mechanisms such as denial, affecting their ability to fantasize and to use a concrete way of thinking. Denial is an extreme defense against all affect and fantasy which differs quantitatively from repression, in that repression results in a selective effect of specific individual emotions. Clearly, it is unlikely that any empirical research has been done in this area.

2.1.4.2.2. Sociocultural factors

Sociocultural mechanisms have been proposed to explain alexithymia. Prince (1987) claims that alexithymic characteristics are socially learned behaviours associated with particular cultural groups, where there are constraints on the expression of emotion; this suggests that alexithymia is adaptive. The association between alexithymia and social class has had conflicting results. Lesser et al., (cited in

Parker et al., 1989) found an association between alexithymia and lower social class, whereas Pierloot and Vinck (cited in Parker et al., 1989) found no such association.

Other accounts of alexithymia and the distinction between primary and secondary alexithymia have been offered, as illustrated below.

2.1.4.3. Alexithymia as a coping response

Greenberg and Dattore (1983) have a different interpretation of primary and secondary alexithymia; primary alexithymia is a life-long dispositional factor that can lead to somatic illness, whilst secondary alexithymia is seen as a coping mechanism resulting from a medical illness or other stress. Ahrens and Deffner (1986) similarly describe alexithymia as a coping response to specific situations such as chronic illness, severe psychological or physical trauma. Greenberg and Dattore (1983) comment that this distinction is theoretical as virtually all of the research has utilized people who already demonstrate somatic symptoms. It is only possible to determine whether the characteristics noted are the cause or result of illness by longitudinal studies with a pre-morbid alexithymic measure.

The above accounts of alexithymia are possible explanations. However, it is still not clear whether alexithymia is an organic condition, a communicative impairment, a personality trait, a coping response or a form of psychic defence. With the formulation of a reliable and valid assessment instrument, an essential component of research, research on alexithymia can begin to unravel these issues.

2.1.5. Research

2.1.5.1. Instruments to measure alexithymia

Once Sifneos (1973) introduced the concept of alexithymia, he devised an instrument to assess its occurrence, the Beth Israel Psychosomatic Questionnaire (BIQ). The researcher fills out the 17 item questionnaire after interviewing the individual with alexithymia. There have been variable results, with some reports of high inter-rater reliability of 0.76, whilst others claim it is unreliable and dependent on the interviewer's training. Little is known about the BIQ's validity and so the questionnaire can only really claim "face validity" as it is based on Sifneos' clinical observations and theoretical explanations. Two other instruments include the Minnesota Multiphasic Personality Inventory (MMPI) Alexithymia scale and the Schalling Sifneos Personality Scale (SSPS). The former

correlates with the BIQ, but the SSPS does not correlate with the MMPI Alexithymia scale or the BIQ. These instruments similarly have not yielded satisfactory reliability and validity ratings.

Projective tests, such as the Thematic Apperception Test (TAT) and the Rorschach and content analysis of various verbal responses have been used to establish the existence of alexithymic characteristics, with variable results.

2.1.5.2. The need for a reliable and valid assessment instrument

Given the inadequacy of assessment instruments for alexithymia, there has been criticism of existing research and a call for a reliable and valid instrument.

Todarello et al., (1989) investigated the relationship between alexithymia and cancer, using the SSPS to measure alexithymia. They found that patients whose mammographic examination was positive were significantly more alexithymic. They conclude their study by recognising the need for more refined tools for the measurement of alexithymia.

Research on alexithymia has been criticised on

methodological grounds, particularly the inadequacy of assessment instruments (for example, Von Rad & Lolas, 1982; Taylor et al., 1985; Parker et al., 1989; Lesser & Lesser, 1983). Taylor (1984) reviews the concept of alexithymia and critically evaluates the different methods to measure alexithymia. He states "... methods of measuring alexithymia that are not only reliable and valid but also easy to use must be developed before sound research studies can be carried out" (p.731)

Lesser (1985) reviews two papers on the measurement strategies of alexithymia. She/he concludes that the validity of results using instruments such as the BIQ, MMPI or the SSPS must be questioned and "highlights the problem of interpreting studies conducted with inadequately standardized measurement instruments" (p.83). Lesser (1985) concludes, "the most fruitful future investigation should focus ..... on accurate recognition and measurement of alexithymia, and once recognized, on appropriate treatment modalities" (p.86).

2.1.5.3. The Toronto Alexithymia Scale (TAS)

Research into alexithymia has taken an important step forward with the formulation of the Toronto Alexithymia Scale (TAS), the first questionnaire with

adequate reliability and validity. The TAS is a 26-item self report measure, using a 5-point likert scale (ranging from "strongly disagree" to "strongly agree"). Half of the items are positively keyed and half negatively keyed to control for acquiescent responding. The TAS has been demonstrated to have internal consistency, good test-retest reliability, and construct and criterion validity (Bagby et al., 1986; Bagby et al., 1988; Bagby et al., 1990; Kauhanen et al., 1991; Taylor et al., 1988; Taylor et al., 1990). The TAS has a stable factor structure theoretically congruent with the alexithymia construct and with the clinical observation that alexithymics are not psychologically minded, do not communicate their emotional experiences, have impoverished fantasy lives and an externally oriented thinking style, are prone to developing "functional" somatic symptoms, and lack the ego capacities generally considered necessary for psychodynamic psychotherapy (Taylor et al., 1990). TAS scores were not related to sociodemographic variables or intelligence, which provides further support for the validity of the alexithymia construct (Taylor et al., 1990; Parker et al., 1989).

The Revised Toronto Alexithymia Scale (TAS-R), used in this thesis, has subsequently eclipsed the TAS, (refer to section 3.2.1.)

2.1.5.4. Research using the Toronto Alexithymia Scale (TAS)

Several studies using the TAS will be mentioned. Alexithymia has been described as a personality construct hypothesized to occur especially in psychologically immature personalities who somatize their feelings (Taylor, 1987). It has been suggested that such personalities would use immature ego defenses, where somatization is used rather than an affective response. Wise et al., (1991) tested this hypothesis using the TAS, and found in their study of mildly depressed psychiatric outpatients, that alexithymia was strongly associated with immature ego defenses, inhibition, acting out, withdrawal, regression, projection, undoing, passive aggression and consummatory behaviour. Alexithymia was not associated with depression. Taylor (cited in Wise et al., 1991) claims that alexithymics use primitive defenses, but notes that this may be due to innate alexithymic characteristics that limit other ego adaptive responses.

As mentioned in section 2.1.5.3., Parker et al., (1989) investigated the demographic profile of alexithymics using the TAS. He found that alexithymia is not associated with age, gender, educational level, socioeconomic status and intelligence.

Parker et al., (1992) investigated the relationship between conjugate lateral eye movements (CLEMS) and alexithymia, and found that alexithymia is associated with left cerebral lateralization, supporting the hypothesis that alexithymic characteristics reflect a variation in brain organisation.

2.1.6. Therapy

Numerous authors have commented on how difficult individuals with alexithymia are to treat in therapy, particularly psychodynamic therapy. Krystal (1979, p.17) describes alexithymia as, "possibly the most important single factor diminishing the success of psychoanalysis and psychodynamic psychotherapy". Taylor (1984) claims that alexithymics who are being treated with psychoanalysis present major difficulties for their therapists. He adds that typical neurotics will communicate symbolic material with ease in the form of dreams, thoughts, feelings or fantasies, but the nonsymbolic style of the alexithymic yields little insight and evokes countertransference feelings of dullness, boredom, frustration and sleepiness. Pierloot and Vinck (cited in Sifneos, 1983) note that alexithymics had a higher drop out rate in dynamic psychotherapy. When they were in a situation requiring an awareness of fantasy and an ability to verbalise, they were unable to perform, reacted with hyperactive

autonomic responses, and left treatment in frustration.

Sifneos (1983) suggests that more attention needs to be given to the nature of the patient's character and psychopathology in determining the type of therapeutic intervention. Therapeutic intervention, supportive or dynamic, can then be allocated appropriately. In his opinion, supportive therapies are most effective with individuals with primary alexithymia. Examples of techniques used are: active reassurance, environmental manipulation, psychotropic medication and behavioural modification. According to Sifneos (1983), secondary alexithymia, due to developmental, sociocultural or psychodynamic factors, is best treated with dynamic therapies which attempt to deal with the psychological conflicts underlying the patient's problems and the associated defense mechanisms. Sifneos (1988) estimates that there are 20 per cent of patients whose alexithymic difficulties may be due to excessive utilization of denial and repression, and for whom psychoanalysis may offer some hope; it is unclear how Sifneos (ibid) can state this without any evidence to support his claim. Furthermore, it is unclear why Sifneos (1988) should suggest dynamic therapy for all secondary alexithymia, which according to him includes sociocultural and developmental factors.

Most therapy, however, has not made these distinctions between alexithymic groups, but has treated it as a unitary phenomenon.

Burns (1986) offers such an example. Even though his case example is descriptive and uncontrolled it is worth mentioning, as it is one of the few studies that claim any measure of success with alexithymics. The study consisted of a group of heterogenous somatising inpatients who attended individual therapy and group therapy. Group therapy comprising a "large number of people" was the therapeutic milieu in which patients made progress. The common mode of entry for patients into group activity was via their expression of indignation or anger on behalf of other patients who were too shy to express themselves. Their activity in the group and absorption into the therapeutic unit was in most cases associated with a marked reduction in both symptoms and complaining behaviour and an expanding repertoire of emotional expression.

The unit, thus observed that somatizing patients can, and do, utilise a challenging psychotherapy setting both to explore and develop their emotional range by experience rather than reflection and interpretation.

Burns (1986) refers to Merskey's (1967) remark that

chronic complaining about pain contains a critical component that serves to "punish" the recipient. The large group, with its hierarchy and varied roles, allowed the patient a legitimate and acceptable manner of expressing and channelling this anger and aggression. The annoyance of peers and staff, elicited by the somatizing patients, seemed to embody a correct perception of the covert message in the patient's complaining, that is, an attack on the hearer. Burns (1986, p.282) concludes,

> "The strong countertransference feelings generated by these patients may have contributed to the failure of their individual or small group treatments. They simply became too irritating to be successfully helped to explore their problems. The large group setting and milieu have, therefore, three advantages in their treatment. First, the patient's impact is diffused between many therapists.... Secondly, a well functioning milieu therapy should provide both patients and staff with an effective emotional support system to deal with the strong countertransference issues raised.... Thirdly, ... the structure offers the patient an opportunity to utilise his assertive and aggressive impulses in a socially acceptable manner. This provides both cathartic and social learning benefits".

This review suggests, contrary to earlier material, that alexithymics may well benefit from psychodynamic therapy, and provides clues for therapeutic intervention.

2.2. CHRONIC PAIN (CP)

2.2.1. Introduction

The relief of pain is probably the most common demand made by patients upon the physician (Engel, 1959). France et al., (1988) estimate that one-third of all Americans suffer from some form of CP. The area of pain is controversial with variable definitions of pain and its various forms, such as acute and chronic pain (CP) as well as chronic organic pain (COP) and chronic non-organic pain (CNOP). These distinctions will be clarified as best as possible, and as pain is so frequently linked to depression, the relation between pain and depression discussed and CP conceptualised systemically.

2.2.2. Definitions of pain

Although numerous definitions of pain exist, they will not be elaborated upon in detail. Several definitions will be offered to illustrate the diversity of definitions of pain.

A subcommittee on taxonomy for the International Association for the Study of Pain (cited in France et al., 1988, p.4) define pain as, "an unpleasant sensory and emotional experience associated with actual or potential tissue damage, or described in terms of such damage". They add that this definition attempts to

account for early observations and concepts of pain, such as the subjective nature of pain, past learnt experiences and emotional and physical meanings.

Hardy (cited in France et al., 1988) states that pain perception is based on a neurophysiological process involving specialized neural receptors and conductive pathways of pain. While the receptor mechanism and pathway systems seem to be relatively similar and constant for all individuals, reaction to pain is influenced by past experience, social setting and psychological factors, all of which account for the wide variability seen in responses to pain.

Several authors stress the subjective nature of pain, which is particularly relevant to this thesis. Szasz (1975) describes pain as one component of a person's private experience which cannot be simultaneously shared and reported by anyone other than the person experiencing the pain. In collection of information regarding the patient's pain, the physician is totally dependent upon the report of the patient. Sternbach writes, (1974, p.107) "... pain cannot be measured directly or observed independently of the subjective experience of it. Therefore, its presence must be inferred from the patient's complaints of pain or from overt behaviour".

A distinction between acute pain and CP is necessary.

2.2.3. Acute pain

Acute pain is best understood in terms of its contrast to CP. CP is distinct from acute pain, in terms of its medical and psychological characteristics, the mechanisms responsible for maintaining the pain, and strategies of treatment (Lindegger, 1988). Melzack and Wall (1973, p.53) describe acute pain as the combination of tissue damage, pain and anxiety. "It is a transitional period between coping with the cause of the injury and preparing for recovery". France et al., (1988) contrast acute pain with CP; acute pain is of recent onset and relatively short and variable duration, and the pain, when associated with tissue damage or injury diminishes as the healing occurs. CP persists despite the intervention of medical treatment and the passing of time, and patient and doctor are unable to give a satisfactory explanation for the presence and continuation of the pain. They add that CP occurs most commonly in headache, back pain, arthritis, cancer and certain psychiatric disorders.

2.2.4. Chronic pain (CP)

There is considerable disagreement on issues such as the definition and classification of CP, criteria for the identification of CP patients for treatment and

research purposes and characteristics of CP patients (Lindegger, 1988). Furthermore, much of the research is fraught with methodological difficulties. Hence, CP remains poorly understood.

Lindegger (1988) illustrates how variable the criteria for the definition of CP are. Definitions include: i) temporal criteria; ii) benignity; iii) constant vs intermittent nature of pain; iv) presence of demonstrable organic pathology; v) pain which is disproportionate to demonstrable organic pathology; vi) presence or absence of psychopathology; vii) presence of distinct psychosocial features; viii) failure of conventional medical or surgical procedures and ix) specific behavioural features.

Lindegger (1988, p.41) incorporates the three most commonly used criteria described above, and defines CP as,

> "any condition in which pain is the primary symptom, and the pain has been present for at least three months. Further, the pain must have failed to respond to conventional medical or surgical treatment".

2.2.5. Pain and depression

In many cases, the presentation of pain and depression is very similar with symptoms of apathy, tiredness, anhedonia, sleeplessness and retarded movements. The

two disorders are often closely inter-related and any study of pain needs to include reference to depression. Tauschke et al., (1990) suggest that of all psychological patterns depression seems to have the strongest association with pain. Webb (cited in Lehmann, 1985) describes CP as a motivational affective phenomenon, with depression the commonly associated affect whereas acute pain is a sensory phenomenon with anxiety being the primary associated affect.

Numerous studies have attempted to ascertain the cause and effect relationship between pain and depression but this remains controversial. There have been numerous methodological difficulties, which include lack of control groups (Gamsa, 1990), causational assumptions on the basis of correlational data (Tauschke et al., 1990), a wide range of methods used to measure depression including measures of depressive symptoms or measures of depressed mood (Haythornthwaite et al., 1991), invalid and unreliable measures of pain and finally, the use of retrospective data which is inherently unreliable (Romano & Turner, 1985). Most of the studies set out below have these methodological flaws.

Rudy et al., (1988) comment that three general models

for conceptualizing the relationship between CP and depression have been proposed, but that there is little empirical work to test the relationships predicted. There are in fact, four models which are discussed below.

2.2.5.1. Biological models

Rudy et al., (1988) report that non-specific biological models hypothesize that similar neurochemical mechanisms may be involved in both pain and depression. For example, abnormalities in the levels, turnover or ratio of biogenic amines and endogenous opioids have been postulated to contribute to both depression and CP (Ward et al., 1982). This is discussed further in the work of Beutler et al., (1986), in section 2.4.5.

2.2.5.2. Pain as a variant of depressive disease

Engel (1959) in his seminal work on CP introduced the term "psychogenic pain" and described the pain-prone patient. One of the possible characteristics of the pain prone patient may be depression, where the experience of pain serves to attenuate the guilt and shame of the depression. In some instances the pain may protect the patient from more intense depression or suicide. Other characteristics of the pain prone patient include a strong aggressive drive which is not

fulfilled, with pain being expressed instead; a prominence of guilt; a history of suffering and defeat; intolerance of success and the development of pain upon a real or threatened loss.

Although Rudy et al., (1988) include Engel's (1959) work in this model, Blumer and Heilbronn (1982) state that Engel (1959) did not view CP as a disorder related to depression but provided the elements to do so.

Blumer and Heilbronn (1982) claim that CP in the absence of demonstrable organic pathology should be viewed as a variant of masked depression. This syndrome is termed the pain-prone disorder, which includes a number of characteristics. Patients have a need to view themselves and their family in idealized terms; there is a premorbid history of hard work; a family history of depression, alcoholism and CP; a tendency to deny their conflicts and difficulty expressing their feelings, especially those of anger.

Subsequently Blumer and Heilbronn's work (1982) has been questioned on conceptual and methodological grounds (Turk et al., 1987). However, Von Knorring (1975) and Von Knorring et al., (1983) lend support to Blumer and Heilbronn's (1982) argument in their

finding that 57 per cent of patients with depression reported pain as a symptom, but once again the etiological relationship is unclear.

Aspects of this model overlap with the fourth model in section 2.2.5.4.

2.2.5.3 Depression as a secondary reaction to a chronic physical condition.

Generally these models, which are highly behavioural in nature, propose that depression is due to diminished activities, a reduction in social rewards and a decline in the perceptions of control over reinforcement contingencies and personal mastery that results from CP.

Rudy et al., (1988) comment on the incidence of depression among pain patients which has varied in studies from 10% to 100%. However, the majority report depression as a concomitant of pain in over 50% of CP patients sampled. Doan and Wadden (1989) found that in a sample of heterogenous CP patients 27% of the patients had Beck Depression Inventory scores suggestive of moderate to severe depression, 39% scored in the mildly depressed range and 34% scored as non-depressed.

In criticism of the previous two models, Gamsa and Vikis-Freibergs (1991) criticise this debate which has centred on whether psychological disturbance is a cause or consequence of CP. Dichotomies are unlikely to provide adequate explanations. They suggest that, ".... pain is the outcome of a complex interplay of influences. These influences include psychological factors which may operate both as risk factors in, and as consequences of, pain" (p.275). These complexities are accounted for in the systemic model discussed in section 2.2.6.

A less frequent account of depression and pain focuses on the difficulty in expressing emotion or the experience of anger, which will be the fourth model.

2.2.5.4. Pain and depression as a consequence of the difficulty in expressing emotion.

Several authors have linked difficulties in expressing emotion, especially anger, to pain and depression. This has been mentioned in section 2.2.5.2. Beutler et al., (1986) propose that CP and depression are independent phenomena but also share psychological and biological processes that are activated in response to certain stressors. This is discussed in detail in section 2.4.5.

2.2.6. Systems model of chronic pain (CP)

A useful way to conceptualize CP is with a systemic model. Ranga Rama Krishnan et al., (1988a) offer such a model, making the important point that the CP syndrome is not only the interaction of organic, psychological and socio-environmental factors, but is also influenced by the sequelae of CP, which are also organic, psychological and socioenvironmental. The dynamic nature of these factors is important. For example, a psychological factor that may influence the experience and maintenance of pain could be a personality type which may predispose a person to be particularly vulnerable to CP. Alexithymia has been described as a personality trait, and CP patients have a high incidence of alexithymia (Mendelson, 1982). The alexithymic personality, therefore, may make the individual more vulnerable to CP. The sequelae of CP can be an alteration in the individual's functioning, leading to helplessness and despair, which then exacerbates the CP. Alternatively, the experience of pain may result in secondary gains from adopting a sick role. The sick role may in turn exacerbate the CP.

Pain is often associated with psychiatric disorders such as generalized anxiety (Large, 1980) or affective disorders (Von Knorring, 1975; Von Knorring et al.,

1983). These disorders can be one of the organic, psychological and/or social factors in the systemic model.

Socio-environmental factors play a role in CP. Poverty and powerlessness amongst lower socio-economic groups is often associated with CP (Ranga Rama Krishnan et al., 1988a). Factors in the environment may be reinforcing for the CP patient; the family may accept the individual's sick role and take over his/her responsibilities and duties or there may be monetary compensation for CP. Loss of self-esteem and inactivity may be the sequelae which exacerbates the pain state.

There are numerous accounts of the organic factors associated with CP. The gate control theory (Melzack, 1973) is particularly illustrative of the interaction of organic and psychological factors that bring about the experience of pain.

Ranga Rama Krishnan et al., (1988b, p.38) concisely summarise the gate control theory:

> "...the signal from noxious stimulation as it passes from the peripheral nerves into the spinal cord is modified by a "gating" mechanism located in the dorsal horn of the spinal cord. If the gate is open, information from the periphery is

transmitted to the brain; if the gate is partially open or closed, less information or no information reaches the interpreting centres in the brain. The gate is controlled by afferents coming in from the periphery and descending pathways from the brain stem".

2.2.7. Chronic organic pain (COP) vs chronic non-organic pain (CNOP)

The distinction between COP and CNOP is unclear and controversial. "Chronic pain" can be defined as a term which fulfils many of the characteristics of a "non-organic type of pain", for example, "pain with no demonstrable pathology"; "has not responded to conventional surgical or medical treatment" (Pilowsky, 1983). Distinctions are made between COP and CNOP, the former being predominantly organic pain and CNOP being predominantly psychological pain. However, this distinction tends to be refuted; dichotomizing COP and CNOP can be misleading as all pain has a psychological component. Furthermore, it is not always clear that a diagnosis of CNOP does not have an etiological organic factor, so the term "chronic non demonstrable pain" is perhaps more appropriate.

France et al., (1988, p.8) stress that COP and CNOP should be conceptualised on a continuum, with varying degrees of organic and psychological factors which interact. They offer a diagram (Table 1) classifying pain based on predominant etiological factors.

Table 1

Pain classification based on predominant etiological factors.

ORGANIC		PSYCHOLOGICAL
Neuropathies	Low Back	Depression
Trigem. neuralgia	Atypical facial	Conversion Disord.
Cancer	TMJ	Psychogenic pain Disord.
Thalamic synd.	Chronic pelvic	Somatization
Central pain	Myofascial pain	Disorder
Deafferent pain	Headache	Hypochondrias.
Arthritis	Causalgia	Dementia
Phantom Limb		Generalized anxiety disorder
Peripheral nerve injury		

Note. From Chronic Pain (p.8) by France et al., 1988, Washington D.C: American Psychiatric Press Inc.

This study follows the view that there is a distinction between COP and CNOP, and includes this as one of the independent variables in the design. Given the controversy surrounding the concepts of CNOP and COP, a loose distinction will be made for the purpose of this study.

CNOP will refer to any condition in which pain is the primary symptom, the pain has been present for at least three months and the pain has failed to respond to conventional medical or surgical treatment (based on Lindegger's, 1988, definition of CP). COP will refer to any condition in which pain is the primary symptom, the pain has been present for at least three months and the pain has responded to conventional medical or surgical treatment (it is important to note that a response to treatment is of a relative nature, and does not imply cure. i.e. there are organic and psychological factors contributing to the pain).

For the purpose of this research, pain will be classified according to Table 1, with full recognition that CNOP and COP are not dichotomous phenomena, but are continuous, with varying degrees of organic and psychological factors.

2.3. DEPRESSION

2.3.1. Introduction

A review of the literature reveals that "depression", like alexithymia and CP, has numerous clinical and research definitions. Carson and Carson, (1984, p.349) write, "Research and clinical applications of the term "depression" are varied which reflects the diverse theoretical approaches as well as the heterogeneity of depressive clinical manifestations". A distinction between depression as a mood, an affect and a disorder will be made and the various etiological models outlined.

2.3.2. Depression as a mood, an affect and a disorder

Depression covers a wide range of states. It exists as a normal mood, which every individual will experience at some time of their life. Mood refers to "the internal emotional state of an individual" (Kaplan & Sadock, 1988, p.138). Depression as an affect refers to the "external expression of emotional content" (ibid, p.138) which is the basic feeling and sadness which is part of life and experienced in states of grief and disappointment (Friedman & Katz, 1974). There are pathological conditions of mood and affect, the most serious being the mood disorders.

Depressive disorders are heterogeneous in terms of

their signs, symptoms, clinical course, genetic and biological involvement, psychological factors and their response to treatment. The many types of depression include reactive, endogenous, neurotic, psychotic, situational, major, minor, primary, secondary, trait and state depression (Carson & Carson, 1984).

The most common classification system for the diagnosis of depression is DSM-III-R (1987, p.228) which classifies a Major Depressive Disorder as, "one or more Major Depressive Episodes without a history of either a Manic Episode or an unequivocal Hypomanic Episode". A Major Depressive Episode includes a Major Depressive syndrome as part of the diagnostic criteria (p.222). The symptoms of a Major Depressive Syndrome are a group of mood and associated symptoms that occur together for a minimal duration of time including, depressed mood; loss of interest or pleasure in activities; weight loss or gain, insomnia or hypersomnia; psychomotor agitation or retardation; loss of energy; feelings of worthlessness; diminished ability to concentrate and recurrent thoughts of death.

The diversity of the theoretical approaches to depression are illustrated in the polarity of

biological and psychological accounts with apparently incompatible definitions. Relevant aspects of these models will be outlined.

2.3.3. Etiological models of depression

2.3.3.1. Biological models

Biochemical hypotheses regarding depression are linked to the presumed or demonstrated biochemical alterations produced by clinically effective biophysical therapies, such as antidepressant medication, lithium salts and electroconvulsive therapy. For example, the MAO-inhibiting and tricyclic classes of antidepressants are believed to relieve depression by increasing the availability of the neurotransmitters, norepinephrine and serotonin at the synaptic cleft. As discussed in sections 2.2.5.1 and 2.4.5. certain biological processes in depression are believed to be similar to CP.

2.3.3.2. Psychodynamic theory

In section 2.1.4.2.1. psychodynamic theorists have accounted for alexithymia in terms of object loss which is very similar to their account of depression.

One of the earliest accounts of depression or melancholia was given by Freud (cited in Arieti and Bemporad, 1978) in his classic paper "Mourning and

Melancholia", where he suggested that melancholia resembled mourning with similar attributes such as dejection, constricted interest and lowered activity. This was precipitated by the loss of a love object, such as a person, pet or ideal. This loss may not have occurred in reality but may have occurred intrapsychically.

Most of the psychodynamic theorists developed their work on the basis of Freud's theory, emphasising that a loss during childhood years or loss in the form of inadequate parenting prevents the development of essential coping skills and sensitizes the individual to situations in adulthood that are reminiscent of the initial traumatic experience. This increases the individual's vulnerability to depression.

Early trauma is proposed as an etiological factor in alexithymia and the communication of depression and perception of pain. Traub-Werner (1990) suggests that if the mother or primary caretaker repeatedly fail to validate the child's somatic experiences, if the child's bodily perceptions are "repeatedly disallowed, disavowed or metaphorized, the result could be a faulty integration of the somatic experience into the ego's structure" (p.141). If this should happen the child may as an adult display a distinct cognitive

set, that is concrete and devoid of symbolic language, especially for the area of affect and perception of pain.

One of the major drawbacks of psychodynamic theory is that it lacks empirical support. The hypothesized connections between childhood experience and adulthood are based on uncontrolled clinical observation and retrospective studies which are unreliable. Ideally, longitudinal studies are required but these are time consuming and costly.

2.3.3.3. Emotional expression

As mentioned in section 2.2.5.4., and elaborated upon in section 2.4.3., difficulties in emotional expression have been linked to depression.

Weissman et al., (1971) respond to various findings that the expression of overt anger in depressives is impaired. They found that whilst anger was not impaired, the direct communication of needs, wishes or feelings was. Anger was secondary to a frustrated wish to be cared for or an impasse in a close relationship.

Grinker (1964) describes the communication in general, of depressives as repetitive, exhausting, monotonous and eventually anger arousing. He claims that the

depressive's ego is exhausted by internal stress, resulting from unresolved conflicts and inadequate problem solving techniques. The apparent communication deficits evident in the highly stereotyped perception, cognition and behaviour is defensively determined, with the depressive defensively blocking informational inputs which she/he might be obliged to act upon but feels unable to do so.

There are numerous accounts of depression, as there are for CP. A systems model incorporates the diversity of these models, recognising the interrelations among psychosocial and biological variables in the etiology and maintenance of depression. Alexithymia or alexithymia like processes could be incorporated into such a model.

2.3.3.4. An alternative model

Although the aforementioned models contribute towards an understanding of depression, a more comprehensive model is needed; one that would take into account the interrelations among biological, psychological and social variables. Hirschfeld and Cross (1983) note that the current belief about depression is that it results from a combination of biological and psychosocial factors including biochemical, genetic, general health, neuroendocrine, familial, personality,

social factors and life events.

Zubin & Steinhauers' (1981) vulnerability model of schizophrenia could be used as a framework for understanding depression. The depressed individual could be viewed as having an inborn vulnerability to depression (genetic, neuroendocrine, biochemical factors) and an acquired vulnerability (familial, personality, social factors, learning experience, developmental factors). Alexithymia could be a component in the inborn or acquired vulnerability (if such a distinction does in fact exist). Whether or not the individual crossed his/her threshold of vulnerability would depend on life events which could be triggering factors or moderating variables.

Life events refer to:

> "changes which occur in an individual's social matrix and which may be considered social stressors in as much as they cause disruptions in the person's customary life pattern and necessitate adaptation. Life events may be recent (6 to 12 months prior to onset of the depressive episode) and include such events as marriage, change in residence, death of a significant other, ... Or they may be remote, such as the death of or separation from a parent during childhood" (Hirschfeld & Cross, 1983, p.382).

Kaplan and Sadock (1988) report that the most robust data indicates that the loss of a parent before age 11 and the loss of a spouse at onset of illness are correlated with Major Depression.

Hirschfeld and Cross (1983) comment on the importance of personality in the etiology of depression. Personality has been emphasized in various theories of depression, although there is disagreement regarding the terminology and the etiology of the characteristics themselves. For example, the psychodynamic theorists have described qualities such as interpersonal dependency and orality. They (ibid, p.382) define personality as, ".. relatively enduring traits, the response sets or characteristic modes of behaviour which an individual exhibits and which may be constitutional in origin (temperament) or acquired during development (character)".

Social factors "refer to the interpersonal resources such as family ties and social relationships and to the life circumstances which may influence an individual's vulnerability to stress" (ibid, p.382).

In Hirschfeld and Cross' (1983) review of the research on the role of personality, life events and social factors, they found that these factors can be influential in depression, but none of the conditions of this model are necessary or sufficient causes for depression. This argument points towards a systemic model as discussed, to account for the inter-relations of many variables.

In the final section of the literature review, the links between alexithymia, CP and depression will be reviewed.

2.4. ALEXITHYMIA, CHRONIC PAIN (CP) AND DEPRESSION

2.4.1. Introduction

There is extensive research that links alexithymia to CP, more so than alexithymia to depression. However, CP is usually linked in some respect to depression (either as related entities or independent phenomena with shared etiological factors). It is therefore feasible to assume that there may be a relationship between alexithymia, CP and depression. This chapter elaborates on past research that has made the link between alexithymia, CP and depression. In so doing the inadequacy of this research, with particular reference to the inadequacy of the assessment instruments, is highlighted.

2.4.2. Alexithymia and chronic pain (CP)

Alexithymia and CP were discussed in section 2.2.5.2. but this section will refer more specifically to research which has attempted to establish a relationship empirically.

Papciak et al., (1986-87) studied 208 outpatients who presented with pain as a primary symptom. Alexithymia correlated with age, but not with severity of pain. They suggest that chronicity of pain (presumably as a consequence of being older), rather than severity, is associated with alexithymia and that alexithymia may

play a role in CP syndromes emerging in reaction to persistent pain or illness. However, the etiological relationship is purely speculative. They recognise the shortcoming of the MMPI alexithymia scale that was used - it cannot measure central alexithymic traits. Doody and Taylor (1983) in an earlier study found that this scale did not measure the capacity for fantasizing or the ability to verbally express feelings.

Further studies linking alexithymia to CP have used instruments that are neither reliable nor valid; Acklin and Bernat (1987) found that patients with low back pain (LBP) exhibit alexithymic characteristics on the Rorschach, and are distinguishable from depressives. Sifneos (1973), using the BIQ found that there was a significant prevalence of alexithymic characteristics in a group of patients suffering from a variety of disorders such as colitis, asthma, peptic ulcer and arthritis as compared to patients in a neurotic control group. Mendelson (1982) identified a 47% incidence of alexithymia in inpatients attending a CP clinic using an MMPI alexithymia scale. No difference between the alexithymic and non-alexithymic groups were found on measures of depression. Postone (1986) found that CP patients were significantly more alexithymic than the psychoneurtoic controls using the

BIQ.

Other studies have focused on alexithymia and its relation to physical illness which may or may not be associated with CP. An association has been considered between alexithymia and physical illness by several authors (Kleiger & Jones, 1980; Heiberg, 1980). Kleiger and Dirks (1980) claim that alexithymia negatively influences the course of treatment with chronic respiratory illness and as mentioned in 2.1.5.2., Todarello et al., (1989) found a relationship between alexithymia and cancer, using the SSPS in their study of 200 women. They claim that their data does at least confirm the hypotheses proposed by Weiner (1982) which regards alexithymia as one of several possible "general onset situations or risk factors that seem to increase the susceptibility to disease which is specifiable by other variables". Poulsen (1991) claims that her study of patients (evaluated with the TAS) with rheumatic disease confirms the theory that chronic disease is associated with a high prevalence of alexithymia. This is one of the few studies that have used an instrument that has demonstrated reliability and validity.

Several other authors describe alexithymia as a risk factor in illness (Postone, 1986; Weiner, 1982) or as

a maintaining factor. Alexithymia traits probably do not predict illness or predispose towards illness (Greenberg & Dattore, 1983), but may be important in the psychomaintenance of the illness (Dirks, cited in Chaturvedi, 1988). Chaturvedi (ibid) comments on the complexity of studying alexithymia as it is difficult to operationalize and no sufficiently reliable and valid instruments for measuring it have been developed. Greenberg and Dattore's (1983) longitudinal study examined whether alexithymic characteristics lead to psychosomatic illness (using the MMPI alexithymia scale). Their findings did not conclude that alexithymia is a cause of illness but they do not refute the idea that alexithymia can result from the stress of disease or that it may lead to a decreased response to treatment and a prolonged course of illness.

2.4.3. Alexithymia and depression

Alexithymia and depression share similar symptoms. Controversy exists as to whether alexithymia and depression are distinct or overlapping constructs. Some of the research which has attempted to clarify this is discussed.

Bagby et al., (1986) found a high correlation of 0.60 ($p < 0.001$) between scores on the TAS and the Beck

Depression Inventory in a sample of undergraduate university students. However, the study lacked generalizability as the sample consisted of university students. Haviland et al., (1988) suggests that the association between alexithymia and depression can be explained in that alexithymic features may emerge as a response to stressful situations and depression. Parker et al., (1991) using factor analysis found that alexithymia and depression are separate constructs that may correlate closely, but can be measured independently. They refer to this alexithymic group as a subgroup of patients with depressive disorders. Some people in this alexithymic subgroup experience CP as their predominant symptom and are diagnosed as having a psychogenic pain disorder. Blumer and Heilbronn (1982) have commented on this (in section 2.2.5.2.). Wise et al., (1991) found a lack of correlation, between depression using the Hamilton Depression Rating Scale (HDRS) and alexithymia, using the TAS.

Clearly, the results are so variable that further investigation into the relationship between alexithymia and depression is needed.

2.4.4. Alexithymia, chronic pain (CP) and depression

Several authors have linked difficulties in expressing emotion, especially anger, to pain and depression.

Engel (1959) (in section 2.2.5.2.) has described CP patients as having a prominence of guilt and a strong unfulfilled aggressive drive (which originated in childhood), where the experience of pain takes the place of emotion when threatened with loss. For both Engel (1959) and Freud (cited in Arieti & Bemporad, 1978), the concept of aggression turned inwards is a key psychodynamic explanation of depression. Blumer and Heilbron (1982) have claimed that CP is a variant of masked depression with difficulty in expressing feelings an etiological factor. The work of Beutler et al., (1986) is pivotal to this area of study.

2.4.5. The work of Beutler et al., (1986)

Beutler et al., (1986) propose that CP and depression are independent phenomena but share psychological and biological processes. These may be activated in response to precipitating stressors. These authors suggest that the shared processes at the psychological level include the inability to modulate or express intense feelings which make the individual more at risk for CP and depression. At the biological level, this may lead to a deactivation of the immune system as manifested by impaired endogenous opioid and natural killer cell production. The assumption that follows from this is that psychotherapy resulting in the expression of emotion will have a beneficial

effect on depression and CP. "Therapeutically induced arousal of affect may facilitate the resolution of conflicting emotions and may also reactivate biological systems that assist in warding off pain and depression" (p.752).

Beutler et al., (1986) review the research on the psychological and biological links to the pain-depression relation.

2.4.5.1. Psychological foundations

Beutler et al., (1986) distinguish between two lines of research, "personality constructs" and "process similarities" which overlap. The former refers to research which seeks to find correlations between pain or depression and various cognitive and emotional states. The latter refers to research which compares similarities among methods of information processing or psychological defence in patients with pain and depression. Some of the research referred to has been mentioned in earlier sections.

2.4.5.1.1. Personality constructs

"Personality constructs" is an embracing term for research that attempts to define the personality variables that are common to patients with CP and depression. It is criticised for being correlational

and uncontrolled. The most frequently cited personality constructs of pain and depression is the conflict over the expression of anger and somatoform anxiety. Beutler et al., (1986) suggest that although these factors emerge as common elements in pain and depression, one cannot assume that CP patients have this single personality pattern. This personality pattern should however, be linked more broadly to a vulnerability to disease and stress.

2.4.5.1.2. Process similarities

Common processes of dealing with intense emotions have been linked to patients with CP and/or depression. "The avenues available for expressing emotional conflict and the conditions that evoke pain are variables that affect the coping process and thus, indirectly, contribute to the pain-depression relation" (p. 753). Furthermore, patterns of emotional constraint have been linked to disease proneness. Beutler et al., (1986) refer to Hollaender and Florin's (1983) research where they found that expressions of anger, joy and fear were less frequent and intense in asthmatic children than in normal control subjects. Levy, Herberman, Maluish, Schlien and Lippman (cited in Beutler et al., 1986) found that prolonged levels of diminished emotional arousal, as evidenced by depression and apathy, are associated

with the deteriorated biological status of breast cancer patients and the associated low levels of natural killer cell activity. Conversely, patients who were more agitated about their disease had higher levels of natural killer cell activity than the more passive and accepting group.

Beutler et al., (1986) make a very important point; the link between patients who have difficulties in expressing emotion and patients with CP and/or depression is often cited as evidence that CP and depression are psychological equivalents (Blumer & Heilbronn, 1982). However, it is more accurate to conclude that the two conditions share a common pattern of disturbance in the process of expressing or blocking intense affect.

2.4.5.2. Biological foundations

Beutler et al., (1986) propose that pain and depression may also share similar biological mechanisms. They cite various studies (for example, Hameroff et al., 1982; Turkington, 1980; Ward et al., 1979) which found that antidepressant medication has a positive effect on pain that is relatively independent of its effect on depression. This suggests that the underlying central nervous system structures might be similar, might utilize similar neural

transmitters or might be located in proximal relation to one another. Von Knorring et al., (1984) provide further support for this with their observation that serotonin and other neurotransmitters are depleted in both pain and depressive syndromes. These possibilities have emphasized that stress-induced disturbances of the immune system might have a similar impact on depression and pain. Endogenous analgesics are mediating factors that may block nerve impulses in both conditions.

Beutler et al., (1986) stress that they are not concluding that difficulties in the expression of emotion lead to depression, disease and CP, but that these individuals are at increased risk.

The work of Beutler et al., (1986) has paved the way for exploring these relationships. Further work will be improved by the reliable and valid measurement of alexithymia or alexithymic like processes.

2.4.6. The need for further studies

This literature review has attempted to examine the research linking alexithymia or alexithymic like processes, depression, CP and chronic physical illness (usually closely associated with CP). Most of the research has been methodologically flawed. However,

these studies suggest that there may be a link between alexithymia, depression and CP. Using the TAS-R, the most refined instrument to date on alexithymia, (described in section 3.2.1) this thesis will attempt to establish whether individuals with alexithymia suffer from depression and/or CP, particularly CNOP.

2.4.7. Aims

As so little is known about alexithymia, due to the prolonged delay in the formulation of a reliable and valid assessment instrument, it is initially necessary to identify the biographical characteristics of alexithymics and to identify alexithymic population groups. Further research into the etiology and treatment of alexithymia can then follow. The aims of this thesis are:

(i) to establish whether there is a significant difference in alexithymia scores between groups on certain biographical variables, such as age, sex, race, marital status, education and income.

(ii) to ascertain whether CNOP patients are more alexithymic than COP patients and patients with NO PAIN. This is in keeping with the hypothesis that alexithymics express their emotions somatically, where it would seem likely that patients with CNOP would be

more alexithymic than those with COP.

(iii) to establish whether elevated levels of alexithymia are a characteristic of patients who are experiencing clinical levels of depression as opposed to normals.

(iv) to examine whether alexithymic scores will be jointly determined by elevated levels of depression and CP (i.e. an interaction).

2.4.8. Hypotheses

The hypotheses of this research are set out below.

Hypothesis 1: there is a significant difference in alexithymic scores, between groups on certain biographical variables, such as age, sex, race, marital status, education and income.

Hypothesis 2: there is a significant difference in alexithymic scores between subjects with chronic non-organic pain (CNOP), chronic organic pain (COP) and subjects without any pain (NO PAIN).

Hypothesis 3: there is a significant difference in levels of alexithymia of patients who are experiencing

clinical levels of depression as opposed to non-depressed subjects.

Hypothesis 4: the levels of an individual's alexithymic scores (dependent variable) will be jointly determined by their levels of depression and the organicity of their pain.

3. METHODOLOGY

3.1. Subjects

Questionnaires were distributed as widely as possible among adult subjects to maximize the generalizability of the study. CP subjects were drawn primarily from adult out-patients attending a pain clinic in a general hospital and from neurologists' surgeries. Depressed patients were drawn primarily from a psychiatric and general hospital. "Normals" were drawn primarily from general practitioners' patients, hospital staff and university students older than eighteen. However, there was considerable overlap in these categories where, for example, several subjects with depression were drawn from hospital staff and university students. The biographical characteristics of the sample are outlined in section 4.1. A total of 190 questionnaires were distributed and 107 (56%) were returned.

3.2. Instruments

3.2.1. The questionnaire

The questionnaire distributed to subjects was comprised of a covering letter, 14 short questions refering to biographical data, mental state and pain (compiled by the researcher), the 20 question Zung self-rating scale for depression and the 23 question

Revised Toronto Alexithymia Scale (Appendix 1).

The covering letter (Appendix 1) explained the research in terms of the University of Natal Medical School's Ethics Committee requirements, and asked respondents to forward the questionnaire to the researcher in the attached self-addressed, stamped envelope.

The first six questions covered biographical characteristics: age, sex, race, marital status, education and income.

Questions 7 and 8 were designed to gather a broad indication of whether the mental health of the sample was "normal", neurotic or psychotic.

Questions 9 to 13 were designed to establish degrees of pain organicity.

Question 14 was designed to assess patient's perception of how psychological or physical their pain was. However, this question was not analysed as it became evident that perception of pain would be a separate study in itself.

Question 15 established whether subjects had felt

depressed in the last six months and if they had, were asked to fill out Zung's self-rating scale (question 16).

Zung's Self Rating Depression Scale (SDS) (1965)

The Zung Self Rating Depression Scale (SDS) has been widely used since 1965, as an indicator of levels of depression. It cannot be used diagnostically. The scale consists of twenty items covering affect, physiological and psychological depressive concomitants or equivalents. Items are rated on a four-point scale, half-positively and half-negatively towards depressive content. It is constructed so that the less depressed patient will have a low score on the scale and the more depressed patient, a higher score. The scale correlates 0.70 with the MMPI Depression scale and differentiates diagnostic categories at statistically significant levels (Becker, 1974). Lindegger (1986) reports a Cronbach-alpha of 0.8532, which was virtually replicated in this study (in section 3.2.2.).

Becker (1974) claims that the SDS confounds state and trait depression which is distinguished by Spielberger (cited in Becker 1974); a depressive state refers to the here and now feeling status of the individual. By contrast, an affective trait refers to a lower

threshold for experiencing depressive states. Affective states fluctuate whereas dispositional traits are relatively common.

Zung (1965) identifies cutoff points where normal moods are distinguished from clinical states. Only a minority meet the criteria for depressive conditions as defined by DSM-III-R (1987), which again illustrates how variable clinical criteria are. For the purpose of this thesis, depression will refer to levels of depression in the Zung scale.

Question 16 was comprised of the Revised Toronto Alexithymia scale (TAS-R).

Revised Toronto Alexithymia Scale (TAS-R)

The Revised Toronto Alexithymia Scale (TAS-R) consists of 23 items where respondents are to indicate on a 5-point likert scale whether they strongly disagree, moderately disagree, neither disagree nor agree, moderately agree and strongly agree (Appendix 1).

Taylor et al., (1992) report on the psychometric properties of the scale. The scale can claim item homogeneity with a mean inter-item correlation of 0.16. Internal reliability was confirmed (ibid) with a Cronbach-alpha coefficient of 0.82, which was

confirmed in this study (section 3.2.2). The TAS-R has a two factor structure with a Pearson-product moment correlation of 0.28 ($p < 0.01$), in contrast to the four-factor structure of the TAS. Factor 1 consists of items assessing the ability to distinguish between feelings and the bodily associations associated with emotional arousal and the ability to describe feelings to others (this corresponds to the first two factors of the TAS). Factor 2 consists of items assessing externally oriented thinking which seems to reflect the pensee operatoire component of the alexithymia construct, even though items assessing daydreaming and imaginal activity were omitted (due to high correlations with social desirability scales and/or low item-total correlations).

Evidence of convergent and discriminant validity was confirmed on the basis of correlations with measures of other constructs that overlap conceptually (in the former case) or are unrelated conceptually (in the latter case) with the alexithymia construct. Criterion validity was confirmed indicating that the TAS-R scores can discriminate between alexithymic and nonalexithymic patients.

3.2.2. Reliability

Reliability of the SDS and the TAS-R was tested with Cronbach-alpha. The SDS has a Cronbach-alpha of 0.8608, which confirms Lindegger's (1986) result of 0.8532. A Cronbach-alpha of 0.8299 confirms the result of Taylor et al., (1992), 0.82 (section 3.2.1.).

3.3. Procedure

Permission to distribute the questionnaires was obtained from the Ethics and Professional Standards Sub-Committee of the University of Natal and the Superintendent and Heads of Department of the various hospitals.

Questionnaires with self-addressed and stamped envelopes attached, were distributed among an adult population (refer to section 3.1.) by hospital staff members or myself. Participants were asked if they would fill out the questionnaire; no other instructions were given, as the questionnaire was self-explanatory. Participants returned the questionnaires by post or via the nursing staff in the hospitals and clinics.

3.4. Data analysis and statistics

3.4.1. Scoring

The data from the questionnaires was coded manually and then entered into the computer. For most of the questions, respondents allocated themselves to a particular category, by marking the relevant response, for example, male or female, depending on their sex. There were a few exceptions.

For example, for question 8, "If YES, what are you being treated for?" (following question 7, asking whether they are having any psychiatric/psychological treatment) respondents were allocated to one of five groups depending on their response. Those who did not know what they were being treated for fell into category one; those suffering from depression, anxiety eating disorders etc. were placed in the neurotic category; any-one with schizophrenia was placed in the psychotic category; category four was for subjects with both neurotic and psychotic disorders and category five was the non-applicable group who were not having any psychiatric or psychological treatment.

For most of the pain questions, subjects allocated themselves to categories, which was then used to classify respondents into further categories. This is discussed in detail below (section 3.4.2; refer to

Table 2).

On the depression scale (question 15), actual respondents' scores were used for analysis, but they were also analysed according to categories to facilitate statistical processing. Respondents with scores below 30 were placed in category 1, the group with the lowest depression scores who are not clinically depressed; scores 30-44 in category 2, the group which begins to distinguish clinical levels of depression; scores 45-59 in category 3, the group with high depression scores and 60-80 in category 4, the group with very high depression scores. Categories and actual scores on the scale correlate with levels of depression, i.e. the higher category represents a higher score which represents a higher level of depression.

For six questionnaires, where there was missing data on the SDS or the TAS, the subject's average score was used.

3.4.2. Data analysis

The analysis of the data will be set out according to each hypothesis.

Hypothesis 1: Data was analysed using nonparametric tests, Kruskal-Wallis and Mann Whitney, depending on the number of groups. In some cases, when there were more than three groups this data was further analysed with Duncan statistics (as SPSS does not have a follow-up from Kruskal Wallis). The application of Duncan statistics was necessary as several questions had categories with a large discrepancy in number of subjects. Kruskal Wallis would give a significant difference amongst all groups, which would not always prove to be the case when analysed further with Duncan statistics. Hence Duncan statistics were a more rigorous test, clarifying exactly which groups were significantly different. Correlation analysis was performed.

Hypothesis 2: Categorising subjects according to CNOP and COP categories was fraught with difficulties. It must be stressed again that any classification of pain in general and CNOP and COP in particular is controversial (reflected in section 2.2.7). The rationale for the method of pain classification for these groups is as follows.

(i) Given that the experience of pain can only be reported subjectively (section 2.2.2), it seemed important to obtain a patient's report on pain

experienced, rather than to obtain a report given by a medical practitioner. Furthermore, as the researcher collected data over twelve months it became clear that most pain patients had been to numerous doctors and had acquired a range of diagnoses. Consequently, it would be impossible to assess which description or diagnosis was the most accurate, without becoming extremely time consuming.

(ii) Patients were classified as either a CNOP or COP patient according to their answers to the questions given in Table 2.

Table 2

Classification of CNOP and COP patients according to their responses to questions 9 to 12a.

	CNOP	COP
9. Do you experience any pain?	YES	YES
10. How long have you been in pain?	> 3 MTHS	> 3 MTHS
11. Has your pain been your most important symptom/complaint in the last three months?	YES	YES
12. Do you know what is the cause of your pain?	NO or YES	YES
12a. If YES, what is the cause?	PSYCHOL or ORGANIC & PSYCHOL	ORGANIC

In order to fall into the CNOP category subjects were required to answer affirmatively to questions 9 (Do you experience any pain?) and 11 (Has your pain been your most important symptom/complaint in the last three months?); > 3 months to question 10 (How long have you been in pain?); either an affirmative or negative answer to question 12, (Do you know what is the cause of your pain?) reflecting the uncertainty these patients often exhibit, and to 12a, (If YES, what is the cause of your pain?) a cause which could be attributed to a psychological or an "in between" cause. The cause of the pain was classified according to the criteria given in Table 1. (section 2.2.7.).

For the classification of COP, subjects were similarly required to answer affirmatively to question 9 and 11 and > 3 months to question 10. Question 12 required an affirmative answer. For question 12a, a cause which could be attributed to organic factors, (classified according to Table 1) was necessary for a COP classification.

Question 13 was not used in the classification as it became clear that this question was not helpful in distinguishing the two types of pain. Most respondents reported that their pain had responded to surgical treatment or medication, but in many cases it was

temporary relief offered by analgesics, rather than a cure of the underlying condition. The question would therefore yield false positives in the COP category.

Data was analysed with Kruksal Wallis and further analysed with the Duncan procedure as there were a far greater number of subjects in the NO PAIN group than in the other two groups.

Hypothesis 3: The independent variable, depression, and the dependent variable, alexithymia, was tested with Kruskal-Wallis, Duncan statistics and various correlations.

Hypothesis 4: Two way analysis of variance was used to analyse whether there was an interaction between depression and pain. It is anticipated that such an interaction will occur.

The results of these hypotheses are set out in the next chapter.

4. RESULTS

4.1. Introduction

The response rate of 56% was high. There were 107 subjects of which 30 subjects were having psychiatric or psychological treatment for a variety of neurotic problems and a further 2 were schizophrenic. The remaining 75 subjects were not in psychiatric or psychological treatment. The numbers of subjects with depression and pain are shown in Tables 9 and 10.

4.2. Hypotheses

4.2.1. Hypothesis 1: there is a significant difference in alexithymic scores between groups on certain biographical variables, such as age, sex, race, marital status, education and income.

Alexithymia and age

Alexithymia scores and number of subjects in each age category are shown in Table 3.

Table 3

Number of subjects (n) and mean (standard deviation) alexithymia scores for subjects according to age group.

Ages	n	Alexithymia scores
18 - 29	42	
M		59.9
SD		12.1
30 - 49	42	
M		58.6
SD		17.9
50 +	23	
M		60.7
SD		13.4

There were no significant differences noted in alexithymia scores between the three age groups (Kruskal-Wallis).

Alexithymia and sex

Alexithymia scores and number of subjects in each gender category are shown in Table 4.

Table 4

Number of subjects (n) and mean (standard deviation) alexithymia scores for subjects according to sex.

Sex	n	Alexithymia scores
Male	31	
M		61.7
SD		13.6
Female	76	
M		58.7
SD		15.2

There were no significant differences in alexithymic scores between males and females (Mann-Whitney).

Alexithymia and race

Alexithymia scores and number of subjects in each race category are shown in Table 5.

Table 5

Number of subjects (n) and mean (standard deviation) alexithymia scores for subjects according to race.

Race	n	Alexithymia scores
White	85	
M		58.7
SD		14.0
Black	5	
M		71.8
SD		10.6
Indian	12	
M		62.8
SD		19.3
Coloured	5	
M		54.4
SD		15.8

There were no significant differences in alexithymic scores between the various racial groups (Kruskal-Wallis).

Alexithymia and marital status

Alexithymia scores and number of subjects in each

marital status category are shown in Table 6.

Table 6

Number of subjects (n) and mean (standard deviation) alexithymia scores for subjects according to marital status.

Marital status	n	Alexithymia scores
Single	43	
M		61.0
SD		12.7
Married	52	
M		59.9
SD		14.7
Divorced	12	
M		53.0
SD		20.7

There were no significant differences in alexithymic scores according to marital status (Kruskal-Wallis).

Alexithymia and education

Alexithymia scores and number of subjects in each educational category are shown in Table 7.

Table 7

Number of subjects (n) and mean (standard deviation) alexithymia scores for subjects according to educational category.

Education	n	Alexithymia scores
Std 8 & less	26	
M		66.2
SD		14.4
Std 9 or 10	35	
M		63.3
SD		12.2
Tech Training	5	
M		64.2
SD		14.8
College Diploma	16	
M		52.8
SD		14.2
University degree	5	
M		51.8
SD		15.1
Post-graduate	20	
M		50.7
SD		14.0

Based on the Kruksal-Wallis ($p = 0.001$) and then the Duncan procedure ($p < 0.05$), alexithymia scores of the subjects in the Std 8 and below group, and those in the Std 9 or matric group were significantly different from the College Diploma and Postgraduate qualification, with the latter having the lower scores.

A significant but low correlation of - 0.33 (about 11% of variance) exists between education and alexithymia ($p = 0.01$), Pearson correlation coefficient).

Alexithymia and income

Alexithymia scores and number of subjects in each income category are shown in Table 8. Five subjects failed to complete this question.

Table 8

Number of subjects (n) and mean (standard deviation) alexithymia scores for subjects according to income.

Income	n	Alexithymic scores
< R800	29	
M		64.8
SD		11.8
R800-1500	28	
M		60.9
SD		13.2
R1500-3000	31	
M		57.0
SD		13.8
R3000+	11	
M		44.9
SD		16.4
Non-applicable	3	
M		57.3
SD		20.2

Note. This table excludes the 5 subjects who did not respond to this question.

Based on the Kruksal-Wallis ($p = 0.0086$) and then the Duncan procedure ($p < 0.05$), results show significant differences between the lowest income group (< R800) and the higher income groups (R1500-3000 and R3000+), and between the R3000+ group and the R800-1500 and the R1500-3000 groups. The lower income groups score higher in alexithymia than the higher income groups.

There is a low correlation of -0.27 ($p = 0.01$, Pearson's correlation coefficient) between alexithymia and income.

4.2.2. Hypothesis 2: there is a significant difference in alexithymic scores between subjects with CNOP, COP and NO PAIN.

On the basis of the classification system (Table 2), mean alexithymia scores and number of subjects are shown in Table 9.

Table 9.

Number of subjects (n) and mean (standard deviation) alexithymic scores for subjects according to pain classification.

Pain	n	Alexithymic scores
No Pain	74	
M		55.7
SD		14.9
COP	14	
M		69.9
SD		9.0
CNOP	19	
M		67.1
SD		11.1

Although the Kruskal-Wallis test reveals a difference between groups ($p = 0.0001$), the Duncan procedure confirms no significant difference ($p < 0.05$) in alexithymia scores between COP and CNOP groups. However, there are significant differences (using Duncan procedure) between the CNOP and NO PAIN groups ($p < 0.05$) and COP and NO PAIN groups ($p < 0.05$).

4.2.3. Hypothesis 3: there is a significant difference in levels of alexithymia in patients who are experiencing clinical levels of depression as opposed to non-depressed subjects.

Mean alexithymia scores and number of subjects following allocation according to Zung depression scores (as outlined in 3.4.1.) is shown in Table 10.

Table 10.

Number of subjects (n) and mean (standard deviation) alexithymia scores for subjects allocated to different categories according to Zung depression scores.

Zung scores	n	Alexithymia scores
< 30	9	
M		51.2
SD		13.3
30 - 44	61	
M		55.6
SD		14.1
45 - 59	27	
M		65.1
SD		12.6
60+	10	
M		76.3
SD		7.1

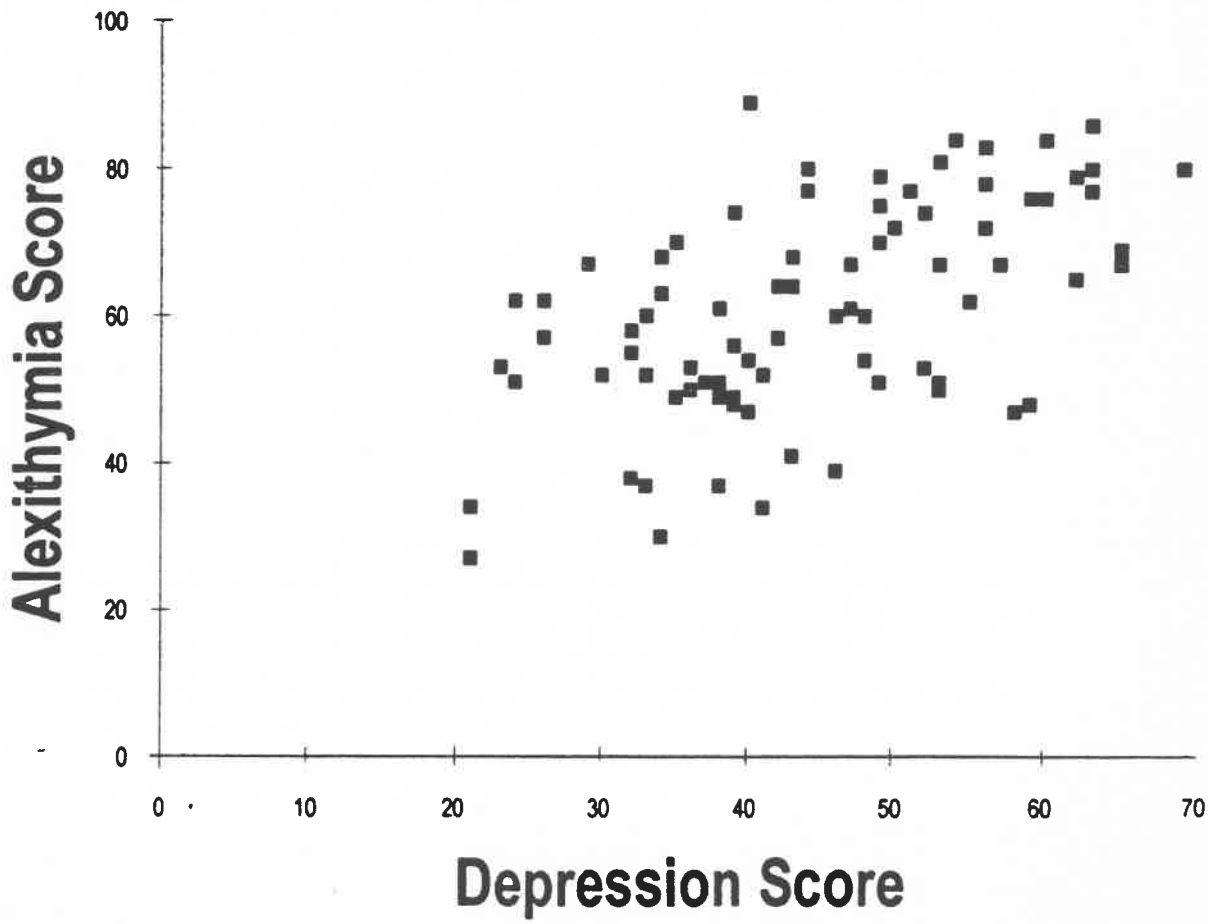
Following Kruskal-Wallis' confirmation of difference between groups ($p < 0.00001$), Duncan procedure showed significant differences ($p < 0.05$) in alexithymia scores between the highest Zung depression group (60+) and each of the other 3 groups, and between the

45 - 59 group and the lower 2 groups (<30 and 30 - 44).

Figure 1 is a plot of alexithymia scores against Zung depression scores. There was a correlation between depression and alexithymia of 0.59, about 35% of the variance ($p = 0.001$, Pearson's correlation coefficient).

Figure 1.

The correlation between depression and alexithymia of 0.59 ($p = 0.001$, Pearson's correlation coefficient).

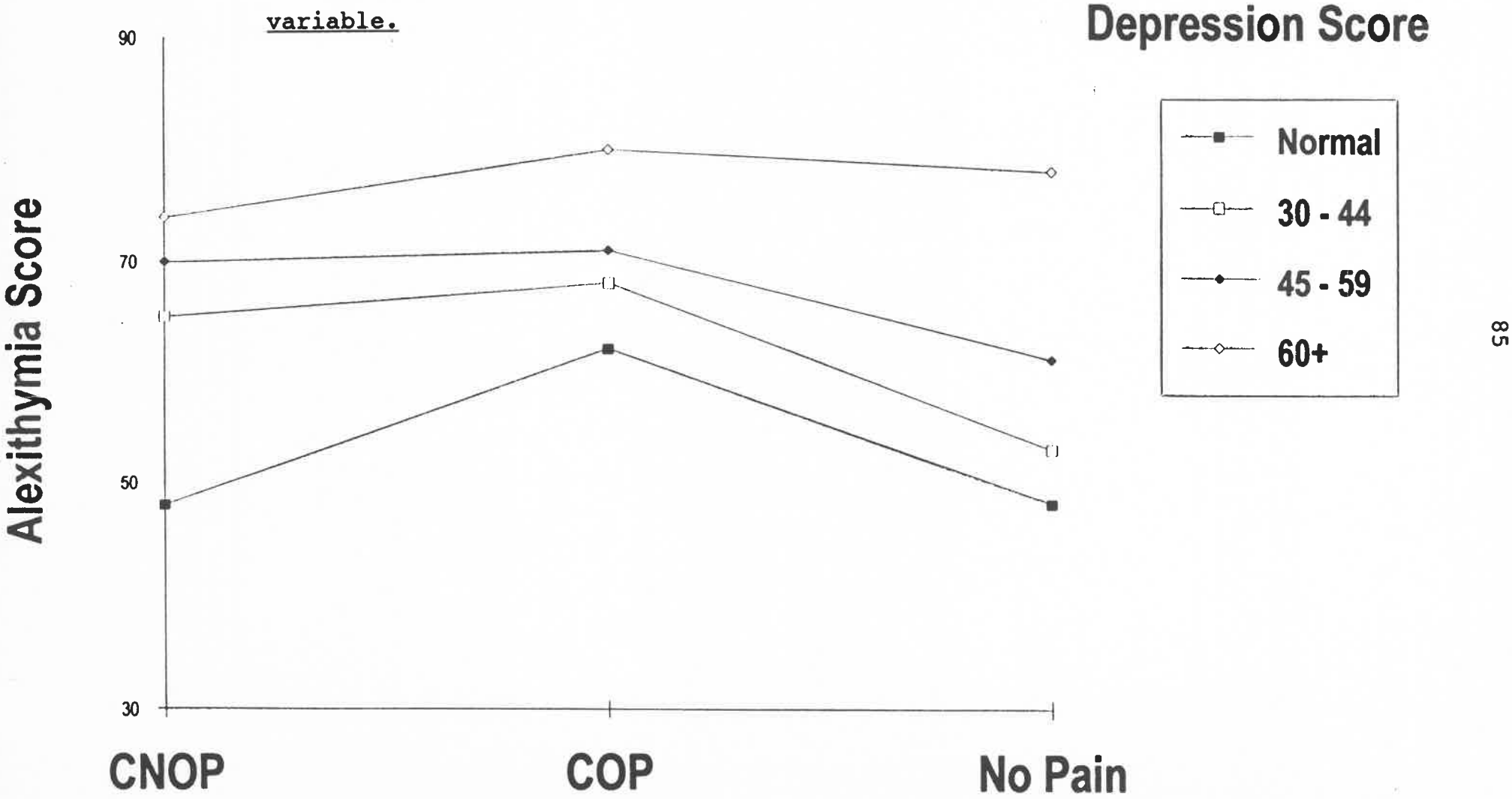


4.2.4. Hypothesis 4: the levels of an individual's alexithymic scores (dependent variable) will be jointly determined by their levels of depression and the organicity of their pain.

Two way analysis of variance reveals that there is no interaction between pain and depression ($F = 0.646$, $p = 0.693$). Results are plotted in Figure 2.

Figure 2.

The analysis of variance with pain and depression as independent variables and alexithymia as the dependent variable.



5. DISCUSSION

5.1. Introduction

The extent to which the hypotheses of this thesis have been successfully explored will be reviewed, each hypothesis discussed and areas for future research outlined.

5.2. Generalizability

For any conclusions to be drawn a comment on the generalizability of the study is necessary. The questionnaires were distributed amongst a cross-section of a "normal", psychiatric and pain population so than any conclusions drawn could be generalized as much as possible. Analysis of question 8, "If YES, what are you being treated for?", in response to, "Are you having any psychiatric/psychological treatment?", revealed that a wide range of psychopathology is represented in the sample. Limitations in generalizability will be mentioned, although there will always be elements of sample bias that are unforseen.

5.3. Hypotheses

Hypothesis 1: there is a significant difference in alexithymic scores, between groups on certain biographical variables, such as age, sex, race,

marital status and education.

No significant differences in alexithymia scores were found in the age, gender and marital status groups. No conclusions can be drawn regarding racial groups as the distribution amongst these groups was unequal. The sample was predominantly white which limits the generalizability of this study to one racial group.

Alexithymia scores are inversely related to education levels. The correlation of -0.33 between these groups is low, but could suggest that the ability to express one's emotions is related to intelligence or verbal literacy. This is an area that needs further investigation. The correlation of -0.27 between alexithymia and income is not surprising given the preceding results, as education and income would probably correlate.

Past research that has investigated alexithymia and demographic characteristics has produced variable results. These have been of "dubious validity and generalizability because many of the studies used psychometrically poor scales to measure the alexithymia construct" (Parker et al., 1989). One such example is Pierloot and Vincks' (1977) study (mentioned in section 2.1.2.), which reported

elevated levels of alexithymia among lower levels of intelligence, education and socio-economic status. However, Parker et al., (1989) using the TAS, found that there was no correlation between age, gender, educational level, socioeconomic status and intelligence. This was based on data from a sample of 101 male and female adults recruited from the passenger lounges at the Central Railway Station and International Airport of a major Canadian city. However, this group lacks generalizability in that the sample would only include a majority of relatively mobile and healthy subjects. They noted that their base rate for alexithymia was 19%, less than their unpublished data of incidences of alexithymia in anorexia nervosa patients (74%) and patients with substance use disorders (49%). A possible explanation for the weak correlation between education, income and alexithymia in this study is that this sample of subjects includes those with mental and physical health problems unlike the unpublished data of Parker et al., (1989) which had higher alexithymia scores.

A recent study by Bourke et al., (1992), found that alexithymia correlated negatively with education in an anorexic group of women matched by age and education, but was unrelated to duration of illness, amount of weight loss, levels of depression and of general

psychoneurotic pathology.

Hypothesis 2: there is a significant difference in alexithymic scores between subjects with chronic non-organic pain (CNOP), chronic organic pain (COP) and NO PAIN.

No significant difference in alexithymia scores was established between subjects who fulfil the criteria for CNOP and COP. This could be due to the criteria themselves that do not accurately distinguish CNOP and COP groups. This is potentially a flaw in this study. Lindegger (1988) reviews a cross-section of the pain literature and illustrates how this definitional problem has hindered research in the past and still needs to be resolved. Furthermore, Blumer and Heilbron (1982) describe CP as a poorly understood and vaguely defined entity. However, individual questions which broadly indicate "organicity" have not yielded any differences in alexithymic scores either. For example, question 12 reveals no significant difference in alexithymic scores between those who know the cause of their pain and those who do not. On the other hand, it is possible that these results are correct and that there is no difference in alexithymia levels between CNOP and COP groups, but this cannot be established from this data. The results do reveal however, that alexithymia is elevated in CP groups as a whole

compared to NO PAIN groups. The CP group would include the COP and CNOP group, who would have had pain as a primary symptom which had been present for at least three months and whose pain had failed to respond to treatment or had responded to treatment with varying degrees, but still experienced pain. Instead of finding support for the hypothesis that alexithymics express their emotions somatically by finding higher levels of alexithymia amongst a CNOP group than a COP group, it is feasible to suggest that there is variable somatic expression of emotions along the CP continuum for both COP and CNOP, which is higher than in the NO PAIN group. Possibly all CP groups are alexithymic and the degree of "organicity" is insignificant.

As we have seen in the literature review alexithymia has been postulated as a causal factor, risk factor, maintaining factor and a coping response to CP and physical illness (Kleiger & Jones, 1980; Heiberg, 1980; Weiner, 1982; Greenberg, 1983; Postone, 1986). Criticisms of these studies have been based on the methodology used. This is the first study to establish that there is a relationship between alexithymia and CP, using a valid and reliable instrument, although alexithymia has been linked to CP in research using inadequate methodology (Acklin & Bernat, 1987;

Sifneos, 1973; Mendelson, 1982 - as mentioned in section 2.4.2.).

Hypothesis 3: there is a significant difference in levels of alexithymia in patients who are experiencing clinical levels of depression as opposed to non-depressed subjects.

The results confirm the hypothesis that depression is associated with alexithymia, and is correlated. As with alexithymia and CP the etiological relationship is unclear and could only be explored with longitudinal studies.

A possible psychodynamic explanation for the association between alexithymia and depression is that anger (which alexithymics cannot express) has turned inwards against the self, resulting in depression. This would suggest that alexithymia may play an etiological role in depression which is associated with CP. Sivik and Hosterey (1992) in their study of low-back pain patients, identified several main characteristics, including alexithymia, denial of aggression and depressivity using the Thematic Apperception Test (TAT). Bagby et al., (1986) found a correlation of 0.60 using the TAS and Beck Depression Inventory (as mentioned in section 2.4.3.).

These research findings conflict with previously reported results; Wise et al., (1991), using the TAS, found that alexithymia was not associated with depression, but their study lacked generalizability as their sample consisted of mildly depressed psychiatric outpatients; Mendelson (1982) found no difference between alexithymic and non-alexithymic groups on measures of depression, using the MMPI.

Hypothesis 4: the levels of an inidividual's alexithymic scores (dependent variable) will be jointly determined by their levels of depression and the organicity of their pain.

There is no interaction between the pain and depression groups. Therefore, subjects are not more likely to have elevated alexithymia scores if they experience CP and elevated levels of depression. This would suggest that CP or elevated levels of depression are independently associated with elevated alexithymia scores.

5.4. An integration

The results of this thesis indicate that amongst a group of mostly white subjects with a cross-section of mental and physical health, elevated levels of alexithymia are associated with lower education, lower income, CP and elevated levels of depression. As

stated above, the relationship of these variables is unclear and could only be understood with longitudinal studies. At best, it can be stated that these variables are correlational and the etiological relationships speculated upon.

A feasible explanation is offered by Blumer and Heilbronn (1982). They draw these characteristics together in their study of the individual with a pain-prone disorder (although their defining criteria of CP patients is "in the absence of demonstrable organic pathology" and could therefore include a different sample group). They propose that alexithymia is a dynamic of patients with CP, where CP is a form of masked depression. Alexithymics have unmet infantile needs which they deny. As these needs assert themselves after a significant loss or disappointment or advent of an injury, they experience conflict with the idealized image of self, resulting in guilt and depression. As a consequence they need to implicate a physical problem for their inadequacy. Thus CP is ultimately an expression of psychic pain. These alexithymics have operative thinking and are oriented toward physical work and mechanical action (which would correlate with lower education and lower income groups).

Another possible explanation for these research findings is the theory of Beutler et al., (1986), as discussed in section 2.4.5., where they suggest that those who suppress and inhibit the expression of intense emotions are at increased risk for depression, CP and disease susceptibility. Possibly low education and income together with alexithymic like processes and other factors such as intelligence or sociocultural factors contribute towards these processes, all of which contribute towards a cycle of helplessness exacerbating depressed and CP states.

Beutler et al., (1986) review existing research (Merskey, 1976; Mahrer, 1980; Udelman, 1975; 1978; 1981) and suggest that there could be therapeutic benefits if emotions can be expressed, especially those of anger. Endogenous opioids and other substances which function as analgesics may be activated, which may play a role in preventing disease, pain and depression. They (ibid) add that the expression of emotion must be aimed at a suitable target and expressed in a manner that offers hope of an appropriate interpersonal response. This is illustrated in Burns' (1986) study in section 2.1.6., where his alexithymic group showed a marked reduction in symptoms, once they had the opportunity to express their emotions, especially anger, in an environment

which appears to have provided both a suitable target and the hope of an appropriate interpersonal response.

The permutations and explanations for elevated levels of alexithymia, low education, low income, elevated levels of CP and depression are numerous. A systems account could accommodate these variables, with recognition of their inter-dependence. Warnes (1986, p.96) states,

> "It is quite possible that alexithymia is one of several mediating processes between stress and disease along with genetic susceptibility, developmental variables, context and reaction to untoward life events, coping dispositions, psychosocial support and sociocultural factors".

Alexithymia could thus be viewed as one of many factors that plays a role (causal, maintaining, or as an adaptive coping response) in the individual's vulnerability to CP and depression. The role of education and income has been speculated upon.

5.5. Limitations of this study

Several of the limitations of this study have been discussed in an earlier section of this thesis, but they will be summarised and limitations of the questionnaire design mentioned.

Certain flaws in the questionnaire design are

apparent. In question 4, (Are you single/ married/divorced?), a category for "widowed" was omitted. In question 6, (Do you earn under R800 per month/between R800 and R1500 per month/between R1500 and R3000 per month/above R3000 per month?), a category of non-applicable, for those who do not earn at all, was also omitted. Three respondents wrote "non-applicable" on the questionnaire, so this category was added when the data was analysed. Although it is unlikely that these omissions would have made any difference to the results of the study, they were nonetheless oversights in the questionnaire design.

Although these questionnaires were distributed as widely as possible, and a reasonable cross-section of age groups, sex and marital status and education were analysed, limitations in generalizability exist, as the sample was predominantly white and fell in the lower to middle income groups. Furthermore, there are always elements of sample bias that are unforseen.

The most important criticism of this thesis is that the criteria for distinguishing between CNOP and COP is potentially flawed and consequently, conclusions can only be made about CP and NO PAIN groups.

5.6. Suggestions for future research

This study has linked the occurrence of alexithymia with CP and depression, which raises many questions concerning the etiological relationships of these conditions. However, many other areas need to be explored such as the occurrence of alexithymia in different population groups, the nature and cause of alexithymia and how it should be treated. A few suggestions for future research are proposed.

The inverse correlation between alexithymia, education and income raises questions about the nature of alexithymia, which is not fully understood. Is it a sociocultural phenomenon related to lower social class and a particular communicative style that is learned in the family and the social group? This could be explored by using an instrument to distinguish family communication styles and class and to measure differences in levels of alexithymia.

The association between alexithymia, CP and/or depression needs to be investigated with longitudinal studies, to establish whether alexithymia precedes CP and/or depression and whether it may play an etiological role. This would clearly have implications for treatment of CP and/or depression. Possibly alexithymia is a learned sociocultural phenomenon and

teaching a different communicative style at an early age would be a useful point of intervention.

It would be interesting to establish whether chronicity of CP and/or depression is related to alexithymia. For example, does the experience of CP for several years, contribute towards depression and alexithymia. Again, longitudinal studies with premorbid measures of alexithymia and depression would be necessary. Individuals with CP would be more likely to be of an older age, and therefore one would expect age to correlate with alexithymia. However, this correlation has not been established.

There are numerous possibilities for future research and many attempts have been made. However, progress has been limited due to poor methodology. It seems appropriate to therefore stress this as a basis for any research. There is the need for investigations to be based on reliable and valid methods of measuring alexithymia (Taylor, 1984; Lesser, 1985), unlike most of the studies in the past. The definitional issues around CP, particularly CNOP and COP need to be clarified and homogenous groups compared (a difficulty that this research has not been able to resolve). Attention to the details of CP and depression, such as the type, duration or intensity of symptoms may yield

a clearer picture of the alexithymia, CP, depression relation. Finally, research ...."needs to be closely linked to testing hypotheses derived from theoretical models of the CP-depression association, so that models can be either substantiated and refined or rejected" (Romano & Turner, 1985, p.31).

6. CONCLUSION

Alexithymia has been researched for the past fifteen years. The nature or mechanisms of alexithymia, the identity of alexithymic groups, the role alexithymia plays in the etiology, the maintenance and recovery of mental and physical states has been speculated upon. One of the major drawbacks in any of the studies published, is that a reliable and valid instrument for the measurement of alexithymia has been lacking. Studies have therefore been too lacking in rigorous methodology to claim any accurate conclusions. This however, has changed with the advent of the Toronto Alexithymia Scale (TAS) and the Revised Toronto Alexithymia Scale (TAS-R), the first instruments that have had adequate reliability and validity. Research into alexithymia is therefore really just beginning.

This thesis has attempted to empirically establish alexithymic groups; individuals with chronic pain (CP), particularly chronic non-organic pain (CNOP), and individuals with clinical levels of depression. Additionally, it has attempted to identify the differences in alexithymia scores between groups on certain biographical variables, such as age, sex, race, marital status, education and income.

A total of 107 questionnaires from a wide range of subjects were analysed. Significant differences in alexithymic levels were noted in the income and education groups, where there was an inverse relation between levels of alexithymia and levels of education and income. There was a significant difference in alexithymia levels in the chronic pain (CP) and NO PAIN groups with subjects with chronic pain (CP) having higher alexithymia scores, but there was no difference in alexithymia scores between the chronic organic pain (COP) and chronic non-organic pain (CNOP) group. (This is possibly due to an inadequate set of criteria for distinguishing them). Alexithymia correlated positively with depression.

There are many possible explanations for these results; CP and depression may share similar biological and psychological processes, such as alexithymia (Beutler et al., 1986); CP may be a variant of masked depression which is partly brought on by alexithymic processes (Blumer & Heilbron, 1982). As these results only provide correlational information, it is only the identification of certain groups that can be assumed, rather than etiological assumptions made. Once alexithymic groups are identified research into alexithymia can begin to establish a clearer picture of the mechanisms of

alexithymia, the role it plays in "illness" and most importantly, how therapeutic interventions can be implemented.

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APPENDIX A

Psychology Department,
University of Natal
P.O. Box 375
PIETERMARITZBURG
3200

Dear Participant,

The Department of Psychology is doing research on pain, depression and various aspects of emotion. I would appreciate it if you could spend about 15 to 20 minutes answering the following questions, although you are under no obligation to do so. You should not experience any side-effects, discomfort or complications, and you are free to withdraw at any time without suffering any disadvantage.

If you decide to participate, your assistance will contribute towards a greater understanding in these areas. Your answers will be treated as strictly confidential.

Please could you forward this questionnaire in the attached self-addressed, stamped envelope. Thank-you very much for your time and co-operation.

Yours sincerely,

(Ms) C. Cholajda
Intern Psychologist

Please place an X over the appropriate answer, or fill in your answer: (Once again, your answers will be treated confidentially, and are only used for research purposes.)

1. How old are you? 18-29 30-49 50+

2. Are you male female

3. Are you white black indian coloured

4. Are you single married divorced widowed

5. Have you completed
   - Std 8 and below
   - Std 9 or matric
   - Technical training
   - College diploma
   - University degree
   - Postgrad. qualification

6. Do you earn
   - under R800 per month
   - between R800 and R1500 per month
   - between R1500 and R3000 per month
   - above R3000 per month

7. Are you having any psychiatric/psychological treatment?

   YES NO

8. If YES, what are you being treated for?

9. Do you experience any pain? YES NO

If YES, please answer questions 10, 11, 12 and 13.
If NO, please go to question 15.

10. How long have you been in pain?

11. Has your pain been your most important symptom/complaint in the last three months?

YES NO

12. Do you know what is the cause of your pain?

YES NO

If YES, what is the cause of your pain?

13. Has your pain responded to any surgical treatment or medication?

YES NO

14. Please rate on the scale from 1 to 5, whether you think your pain is:

(i) - completely physical

(ii) - mostly physical, but also psychological

(iii) - half physical, half psychological

(iv) - mostly psychological, but also physical

(v) - completely psychological

1.	2.	3.	4.	5.
PHYSICAL				PSYCHOLOGICAL

15. Have you felt depressed in the last six months, (for example, feelings of hopelessness or sadness, a lack of enjoyment in activities that you have enjoyed before?)

YES NO

If your answer is YES, please answer the following questions. Rate yourself on each of the following statements by placing a number in the brackets.

1 = SELDOM 2 = SOME OF THE TIME
3 = OFTEN 4 = MOST OF THE TIME

( ) i. I feel downhearted and sad.

( ) ii. Morning is when I feel best.

( ) iii. I have crying spells or feel like crying.

( ) iv. I have trouble sleeping at night.

( ) v. I eat as much as I used to.

( ) vi. I still enjoy sex.

( ) vii. I notice that I am losing weight.

( ) viii. I have trouble with constipation.

( ) ix. My heart beats faster than usual.

( ) x. I get tired for no reason.

( ) xi. My mind is as clear as it used to be.

( ) xii. I find it is difficult to do things I used to.

( ) xiii. I am restless and can't keep still.

( ) xiv. I feel hopeful about the future.

( ) xv. I am more irritable than usual.

( ) xvi. I find it easy to make decisions.

( ) xvii. I feel that I am useful and needed.

( ) xix. I feel that others would be better off if I were dead.

( ) xx. I still enjoy the things I used to.

16. Using the scale provided as a guide, indicate how much you agree or disagree with each of the following statements by marking the appropriate number with an X. Give only one answer for each statement.

1 = STRONGLY DISAGREE
2 = MODERATELY DISAGREE
3 = NEITHER DISAGREE NOR AGREE
4 = MODERATELY AGREE
5 = STRONGLY AGREE

1. I wish I were not so shy	1	2	3	4	5
2. I often get confused about what emotion I am feeling	1	2	3	4	5
3. I seem to make friends as easily as others do	1	2	3	4	5
4. Knowing the answers to problems is more important than knowing the reasons for the answers	1	2	3	4	5
5. It is difficult for me to find the right words for my feelings	1	2	3	4	5
6. I have physical sensations that even doctors don't understand	1	2	3	4	5
7. I'm able to describe my feelings easily	1	2	3	4	5
8. I prefer to analyze problems rather than just to describe them.	1	2	3	4	5
9. When I'm upset, I don't know if I am sad, frightened or angry	1	2	3	4	5
10. I am often puzzled by sensations in my body	1	2	3	4	5
11. I prefer to just let things happen rather than to understand why they turned out that way	1	2	3	4	5

12. I have feelings that I can't quite identify	1	2	3	4	5
13. Being in touch with emotions is essential	1	2	3	4	5
14. I find it hard to describe how I feel about people	1	2	3	4	5
15. People tell me to describe my feelings more	1	2	3	4	5
16. I don't know what's going on inside me	1	2	3	4	5
17. I often don't know why I'm angry	1	2	3	4	5
18. I prefer talking to people about their daily activities rather than their feelings	1	2	3	4	5
19. I prefer to watch "light" entertainment shows rather than psychological dramas	1	2	3	4	5
20. It is difficult for me to reveal my inner mst feelings, even to close friends	1	2	3	4	5
21. I can feel close to someone even in moments of silence	1	2	3	4	5
22. I find examination of my feelings useful in solving personal problems	1	2	3	4	5
23. Looking for hidden meanings in movies or plays distracts from their enjoyment	1	2	3	4	5

Once again, thank-you for your co-operation.

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Summaries of ALEX
By levels of AGE

Variable	Value	Label	Mean	Std Dev	Cases
For Entire Population			59.5701	14.4726	107
AGE	1	18-29 YRS	59.9286	12.0737	42
AGE	2	30-49 YRS	58.5952	17.9145	42
AGE	3	50+ YRS	60.6957	13.3534	23

Total Cases = 107

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Summaries of ALEX
By levels of SEX

Variable	Value	Label	Mean	Std Dev	Cases
For Entire Population			59.5701	14.4726	107
SEX	1	MALE	61.7097	13.6044	31
SEX	2	FEMALE	58.6974	15.2228	76

Total Cases = 107

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Summaries of ALEX
By levels of RACE

Variable	Value	Label	Mean	Std Dev	Cases
For Entire Population			59.5701	14.4726	107
RACE	1	WHITE	58.6941	14.0255	85
RACE	2	BLACK	71.8000	10.5688	5
RACE	3	INDIAN	62.8333	19.2724	12
RACE	4	COLOURED	54.4000	15.8209	5

Total Cases = 107

Summaries of ALEX
By levels of MARITAL

Variable	Value	Label	Mean	Std Dev	Cases
For Entire Population			59.5701	14.7726	107
MARITAL	1	SINGLE	61.0000	12.6679	43
MARITAL	2	MARRIED	59.9038	14.7333	52
MARITAL	3	DIVORCED	53.0000	20.6926	12

Total Cases = 107

Summaries of ALEX
By levels of EDUC

Variable	Value	Label	Mean	Std Dev	Cases
For Entire Population			59.5701	14.7726	107
EDUC	1	STD8	66.1538	14.4158	26
EDUC	2	STD9URMAT	63.2857	12.2055	35
EDUC	3	TECHTR	64.2000	14.8391	5
EDUC	4	COLLDIP	52.8125	14.1855	16
EDUC	5	UNIVDEG	51.8000	15.0892	5
EDUC	6	POSTGRAD	50.7000	14.0267	20

Total Cases = 107

Summaries of ALEX
By levels of INCOME

Variable	Value	Label	Mean	Std Dev	Cases
For Entire Population			58.9902	14.5122	102
INCOME	1	UNDER 800	64.8276	11.8082	29
INCOME	2	800-1500	60.8929	13.2059	28
INCOME	3	1500-3000	56.9677	13.8311	31
INCOME	4	3000+	44.9091	16.4162	11
INCOME	5	NA	57.3333	20.2320	3

Total Cases = 107
Missing Cases = 5 OR 4.7 PCT.

Summaries of ALEX
By levels of PSYTRE ARE YOU IN PSYCHOLOGICAL TREATMENT?

Variable	Value	Label	Mean	Std Dev	Cases
For Entire Population			59.5701	14.7726	107
PSYTRE	1	YES	65.9375	14.2581	32
PSYTRE	2	NO	56.8533	14.0122	75

Total Cases = 107

Summaries of ALEX
By levels of EXPPAIN DO YOU EXPERIENCE PAIN?

Variable	Value	Label	Mean	Std Dev	Cases
For Entire Population			59.4369	14.9625	103
EXPPAIN	1	YES	62.7302	14.0256	63
EXPPAIN	2	NO	54.2500	15.0891	40

Total Cases = 107
Missing Cases = 4 OR 3.7 PCT.

Summaries of ALEX
By levels of LENGPAIN HOW LONG HAVE YOU BEEN IN PAIN

Variable	Value	Label	Mean	Std Dev	Cases
For Entire Population			59.3131	14.9325	99
LENGPAIN	1	<3MTH	61.0000	13.8022	5
LENGPAIN	2	>3MTH	63.0545	13.4688	55
LENGPAIN	3	NA	53.8205	15.0366	39

Total Cases = 107
Missing Cases = 8 OR 7.5 PCT.

Summaries of ALEX
By levels of MUSTNB IS PAIN THE MUST IMPORTANT?

Variable	Value	Label	Mean	Std Dev	Cases
For Entire Population			58.9109	14.6213	101
MUSTNB	1	YES	66.7500	10.9110	36
MUSTNB	2	NO	55.6923	14.3214	26
MUSTNB	3	NA	53.8205	15.0366	39

Total Cases = 107
Missing Cases = 6 OR 5.6 PCT.

Summaries of ALEX
By levels of CAUSPA DO YOU KNOW THE CAUSE?

Variable	Value	Label	Mean	Std Dev	Cases
For Entire Population			59.4369	14.9625	103
CAUSPA	1	YES	62.5833	14.5819	48
CAUSPA	2	NO	65.6875	12.2622	16
CAUSPA	3	NA	53.8205	15.0366	39

Total Cases = 107
Missing Cases = 4 OR 3.7 PCT.

Summaries of ALEX
By levels of WHATCAUS WHAT IS THE CAUSE?

Variable	Value	Label	Mean	Std Dev	Cases
For Entire Population			59.5354	14.6527	99
WHATCAUS	1	ORGANIC	62.5417	14.4703	24
WHATCAUS	2	BOTH	61.1250	13.8606	16
WHATCAUS	3	PSYCH	71.6667	7.5171	6
WHATCAUS	4	NA	56.3208	14.7984	53

Total Cases = 107
Missing Cases = 8 OR 7.5 PCT.

Summaries of ALEX
By levels of RESPTR HAS YOUR PAIN RESPONDED TO TREATMENT?

Variable	Value	Label	Mean	Std Dev	Cases
For Entire Population			59.4706	15.0324	102
RESPTR	1	YES	59.9355	13.2038	61
RESPTR	2	NO	62.0000	13.3590	29
RESPTR	3	NA	53.9286	19.3597	12

Total Cases = 107
Missing Cases = 5 OR 4.7 PCT.

Summaries of ALEX
By levels of PAINPER WHAT IS YOUR PERCEPTION OF PAIN?

Variable	Value	Label	Mean	Std Dev	Cases
For Entire Population			58.9307	14.7860	101
PAINPER	1	COMPPHYS	60.8182	12.9094	55
PAINPER	2	MOSTPHYS	60.7692	16.4932	13
PAINPER	3	HALFPHYS	66.7143	13.3755	7
PAINPER	4	MOSTPSY	62.2857	14.0629	7
PAINPER	5	COMPPSY	69.0000	.0000	1
PAINPER	6	NA	53.8205	15.0566	17
PAINPER	7	?	84.0000	.0000	1

Total Cases = 107
Missing Cases = 6 OR 5.6 PCT.

Summaries of ALEX
By levels of PAIN# STRUNGER CRITERION FOR CHRONIC PAIN

Variable	Value	Label	Mean	Std Dev	Cases
For Entire Population			59.5701	14.4726	107
PAIN#	1.00	CNUP	67.1579	11.1119	19
PAIN#	2.00	CUP	69.8571	8.9859	14
PAIN#	3.00	NO PAIN	55.6725	14.8795	74

Total Cases = 107

Summaries of ALEX
By levels of HEALTH

Variable	Value	Label	Mean	Std Dev	Cases
For Entire Population			59.5701	14.4726	107
HEALTH	1.00	NORMAL	51.2222	13.2852	9
HEALTH	2.00	ZUNG 30 - 44	55.6066	14.0941	61
HEALTH	3.00	ZUNG 45 - 59	69.1111	12.6075	27
HEALTH	4.00	ZUNG 60+	76.3000	7.1188	10

Total Cases = 107

```
This procedure was completed at  1:11:04
NPAR TESTS K-W=ALEX BY RACE(1,4)/M-W=ALEX BY SEX(1,2)/
K-W=ALEX BY AGE(1,3)/K-W=ALEX BY MARITAL(1,3)/K-W=ALEX BY EDUC(1,6)/
K-W=ALEX BY INCOME(1,5)/M-W=ALEX BY PSYTRE(1,2)/M-W=ALEX BY EXPPAIN(1,2)/
K-W=ALEX BY LENGPAIN(1,3)/K-W=ALEX BY MOSTNB(1,3)/K-W=ALEX BY CAUSPA(1,3)/
K-W=ALEX BY WHATCAUS(1,4)/K-W=ALEX BY RESPIR(1,3)/K-W=ALEX BY PAINPER(1,6)/
K-W=ALEX BY PAIN(1,3)/K-W=ALEX BY PAIN#(1,3)/K-W=ALEX BY HEALTH(1,4).

 ***** WORKSPACE allows for    2864 cases for NPAR TESTS *****
```


- - - - - Kruskal-Wallis 1-way ANOVA

ALEX
by RACE

Mean Rank	Cases		
51.94	85	RACE = 1	WHITE
79.50	5	RACE = 2	BLACK
63.29	12	RACE = 3	INDIAN
41.20	5	RACE = 4	COLOURED
	107	Total	

CASES	Chi-Square	Significance	Corrected for Ties Chi-Square	Corrected for Ties Significance
107	5.6768	.1284	5.6818	.1282

- - - - - Mann-Whitney U - Wilcoxon Rank Sum W Test

ALEX
by SEX

Mean Rank	Cases		
58.21	31	SEX = 1	MALE
52.28	76	SEX = 2	FEMALE
	107	Total	

U	W	Corrected for Ties Z	Corrected for Ties 2-tailed P
1047.5	1804.5	-.8966	.3699

- - - - - Kruskal-Wallis 1-way ANOVA

ALEX
by AGE

Mean Rank	Cases		
53.82	42	AGE = 1	18-29 YRS
52.62	42	AGE = 2	30-49 YRS
56.85	23	AGE = 3	50+ YRS
	107	Total	

CASES	Chi-Square	Significance	Corrected for Ties Chi-Square	Corrected for Ties Significance
107	.2783	.8701	.2785	.8700

- - - - - Kruskal-Wallis 1-way ANOVA

ALEX
by MARITAL

Mean Rank	Cases		
55.62	43	MARITAL = 1	SINGLE
55.33	52	MARITAL = 2	MARRIED
42.46	12	MARITAL = 3	DIVORCED
	107	Total	

CASES	Chi-Square	Significance	Corrected for Ties Chi-Square	Corrected for Ties Significance
107	1.8717	.3923	1.8733	.3919

- - - - - Kruskal-Wallis 1-way ANOVA

ALEX
by EDUC

Mean Rank	Cases		
67.65	26	EDUC = 1	STD8
62.57	35	EDUC = 2	STD9URMAT
60.70	5	EDUC = 3	TECHTR
41.13	16	EDUC = 4	COLLDIP
37.80	5	EDUC = 5	UNIVDEG
33.92	20	EDUC = 6	POSTGRAD
	107	Total	

CASES	Chi-Square	Significance	Corrected for Ties Chi-Square	Significance
107	20.4232	.0010	20.4410	.0010

- - - - - Kruskal-Wallis 1-way ANOVA

ALEX
by INCOME

Mean Rank	Cases		
63.31	29	INCOME = 1	UNDER 800
53.77	28	INCOME = 2	800-1500
47.40	31	INCOME = 3	1500-3000
25.91	11	INCOME = 4	3000+
52.33	3	INCOME = 5	NA
	102	Total	

CASES	Chi-Square	Significance	Corrected for Ties Chi-Square	Significance
102	13.6097	.0087	13.6229	.0086

- - - - - Mann-Whitney U - Wilcoxon Rank Sum W Test

ALEX
by PSYTRE ARE YOU IN PSYCHOLOGICAL TREATMENT?

Mean Rank	Cases	
67.80	32	PSYTRE = 1 YES
48.11	75	PSYTRE = 2 NO
	107	Total

U	W	Corrected for Ties Z	2-tailed P
758.5	2169.5	-3.0053	.0027

- - - - - Mann-Whitney U - Wilcoxon Rank Sum W Test

ALEX
by EXPPAIN DO YUU EXPERIENCE PAIN?

Mean Rank	Cases	
58.65	63	EXPPAIN = 1 YES
41.53	40	EXPPAIN = 2 NO
	103	Total

U	W	Corrected for Ties Z	2-tailed P
841.0	1661.0	-2.8366	.0046

- - - - - Kruskal-Wallis 1-way ANOVA

ALEX
by LENGPAIN HOW LONG HAVE YOU BEEN IN PAIN

Mean Rank	Cases	
54.90	5	LENGPAIN = 1 <3MTH
57.16	55	LENGPAIN = 2 >3MTH
39.27	39	LENGPAIN = 3 NA
	99	Total

CASES	Chi-Square	Significance	Corrected for Ties Chi-Square	Significance
99	9.0101	.0111	9.0192	.0110

- - - - - Kruskal-Wallis 1-way ANOVA

ALEX
by MUSTNB IS PAIN THE MUST IMPORTANT?

Mean Rank	Cases	
67.44	36	MUSTNB = 1 YES
43.90	26	MUSTNB = 2 NO
40.55	39	MUSTNB = 3 NA
	101	Total

CASES	Chi-Square	Significance	Corrected for Ties Chi-Square	Significance
101	17.8244	.0001	17.8421	.0001

- - - - - Kruskal-Wallis 1-way ANOVA

ALEX
by CAUSPA DO YOU KNOW THE CAUSE?

Mean Rank	Cases		
58.65	48	CAUSPA = 1	YES
59.91	16	CAUSPA = 2	NO
40.58	39	CAUSPA = 3	NA
	103	Total	

CASES	Chi-Square	Significance	Corrected for Ties Chi-Square	Corrected for Ties Significance
103	9.1962	.0101	9.2048	.0100

- - - - - Kruskal-Wallis 1-way ANOVA

ALEX
by WHATCAUS WHAT IS THE CAUSE?

Mean Rank	Cases		
56.65	24	WHATCAUS = 1	ORGANIC
52.41	16	WHATCAUS = 2	BOTH
74.92	6	WHATCAUS = 3	PSYCH
43.44	53	WHATCAUS = 4	NA
	99	Total	

CASES	Chi-Square	Significance	Corrected for Ties Chi-Square	Corrected for Ties Significance
99	8.6741	.0340	8.6832	.0338

- - - - - Kruskal-Wallis 1-way ANOVA

ALEX
by RESPTR HAS YOUR PAIN RESPONDED TO TREATMENT?

Mean Rank	Cases		
51.58	31	RESPTR = 1	YES
67.29	29	RESPTR = 2	NO
40.54	42	RESPTR = 3	NA
	102	Total	

CASES	Chi-Square	Significance	Corrected for Ties Chi-Square	Significance
102	14.0291	.0009	14.0426	.0009

- - - - - Kruskal-Wallis 1-way ANOVA

ALEX
by PAINPER WHAT IS YOUR PERCEPTION OF PAIN?

Mean Rank	Cases		
54.79	33	PAINPER = 1	COMPPHYS
55.69	13	PAINPER = 2	MOSTPHYS
65.71	7	PAINPER = 3	HALFPHYS
57.21	7	PAINPER = 4	MOSTPSY
74.00	1	PAINPER = 5	COMPPSY
40.60	39	PAINPER = 6	NA
	100	Total	

CASES	Chi-Square	Significance	Corrected for Ties Chi-Square	Significance
100	8.6326	.1246	8.6403	.1243

- - - - - Kruskal-Wallis 1-way ANOVA

ALEX
by PAIN# STRONGER CRITERION FOR CHRONIC PAIN

Mean Rank	Cases			
20.24	19	PAIN# =	1	CNUP
76.82	14	PAIN# =	2	CUP
45.51	74	PAIN# =	3	NU PAIN
	107	Total		

CASES	Chi-Square	Significance	Corrected for Ties Chi-Square	Significance
107	18.3074	.0001	18.3234	.0001

- - - - - Kruskal-Wallis 1-way ANOVA

ALEX
by HEALTH

Mean Rank	Cases			
37.89	9	HEALTH =	1	NORMAL
45.57	61	HEALTH =	2	ZUNG 30 - 44
65.17	27	HEALTH =	3	ZUNG 45 - 59
89.75	10	HEALTH =	4	ZUNG 60+
	107	Total		

CASES	Chi-Square	Significance	Corrected for Ties Chi-Square	Significance
107	23.6911	.0000	23.7118	.0000

Correlations:

	ALEX	PAINPER	DEPRLEV	WHATCAUS
ALEX	1.0000	.2767	.6685**	.2025
PAINPER	.2767	1.0000	.3463	.6175**
DEPRLEV	.6685**	.3463	1.0000	.3014
WHATCAUS	.2025	.6175**	.3014	1.0000

N of cases: 37 1-tailed Signif: * - .01 ** - .001

" . " is printed if a coefficient cannot be computed

- - - - - - - - - - U N E W A Y - - - - - - - - - -

Variable ALEX
By Variable AGE

Analysis of Variance

Source	D.F.	Sum of Squares	Mean Squares	F Ratio	F Prob.
Between Groups	2	74.4500	37.2250	.1679	.8457
Within Groups	104	23057.7743	221.7094		
Total	106	23132.2243			

Tests for Homogeneity of Variances

Cochrans C = Max. Variance/Sum(Variances) = .4976, P = .023 (Approx.)
Bartlett-Box F = 3.363, P = .035
Maximum Variance / Minimum Variance 2.202

- - - - - - - - - - U N E W A Y - - - - - - - - - -

Variable ALEX
By Variable AGE

Multiple Range Test

Duncan Procedure
Ranges for the .050 level -

2.81 2.95

The ranges above are table ranges.
The value actually compared with Mean(J)-Mean(I) is..
10.5288 * Range * Sqrt(1/N(I) + 1/N(J))

No two groups are significantly different at the .050 level

This procedure was completed at 0:53:52
UNEWAY VARIABLES=ALEX BY SEX(1,2)/RANGES=DUNCAN/STATISTICS=3.

- - - - - - - - - - - U N E W A Y - - - - - - - - - - -

Variable ALEX
By Variable SEX

Analysis of Variance

Source	D.F.	Sum of Squares	Mean Squares	F Ratio	F Prob.
Between Groups	1	199.7977	199.7977	.9148	.3410
Within Groups	105	22932.4266	218.4041		
Total	106	23132.2243			

Tests for Homogeneity of Variances

Cochrans C = Max. Variance/Sum(Variances) = .5560, P = .416 (Approx.)
Bartlett-Box F = .517, P = .472
Maximum Variance / Minimum Variance 1.252

No Range Tests performed with fewer than three non-empty groups.

This procedure was completed at 0:54:13
ONEWAY VARIABLES= ALEX BY EDUC(1,6)/RANGES=DUNCAN/STATISTICS=3.

- - - - - - - - - - - U N E W A Y - - - - - - - - - - -

Variable ALEX
By Variable EDUC

Analysis of Variance

Source	D.F.	Sum of Squares	Mean Squares	F Ratio	F Prob.
Between Groups	5	4323.4593	864.6919	4.6435	.0007
Within Groups	101	18808.7650	186.2254		
Total	106	23132.2243			

Tests for Homogeneity of Variances

Cochrans C = Max. Variance/Sum(Variances) = .1893, P = 1.000 (Approx.)
Bartlett-Box F = .223, P = .953
Maximum Variance / Minimum Variance 1.528

- - - - - - - - - - - U N E W A Y - - - - - - - - - - -

Variable ALEX
By Variable EDUC

Multiple Range Test

Duncan Procedure
Ranges for the .050 level -

2.81 2.95 3.05 3.12 3.18

The ranges above are table ranges.
The value actually compared with Mean(J)-Mean(I) is..
9.6495 * Range * Sqrt(1/N(I) + 1/N(J))

(*) Denotes pairs of groups significantly different at the .050 level

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- - - - - - - - - - - U N E W A Y - - - - - - - - - - -

Variable ALEX
(Continued)

Mean	Group	Grp 6	Grp 5	Grp 4	Grp 2	Grp 3	Grp 1
50.7000	Grp 6						
51.8000	Grp 5						
52.8125	Grp 4						
63.2857	Grp 2	*		*			
64.2000	Grp 3						
66.1538	Grp 1	*		*			

This procedure was completed at 0:54:38
ONEWAY VARIABLES=ALEX BY INCOME(1,5)/RANGES=DUNCAN/STATISTICS=3.

- - - - - - - - - - - O N E W A Y - - - - - - - - - - -

Variable ALEX
By Variable INCOME

Analysis of Variance

Source	D.F.	Sum of Squares	Mean Squares	F Ratio	F Prob.
Between Groups	4	3405.6302	851.4075	4.6227	.0019
Within Groups	97	17865.3600	184.1790		
Total	101	21270.9902			

Tests for Homogeneity of Variances

Cochrans C = Max. Variance/Sum(Variances) = .3457, P = .058 (Approx.)
Bartlett-Box F = .644, P = .632
Maximum Variance / Minimum Variance 2.936

- - - - - - - - - - - O N E W A Y - - - - - - - - - - -

Variable ALEX
By Variable INCOME

Multiple Range Test

Duncan Procedure
Ranges for the .050 level -

2.81 2.96 3.05 3.12

The ranges above are table ranges.
The value actually compared with Mean(J)-Mean(I) is..
9.5963 * Range * Sqrt(1/N(I) + 1/N(J))

(*) Denotes pairs of groups significantly different at the .050 level

- - - - - - - - - - - U N E W A Y - - - - - - - - - - -

Variable ALEX
(Continued)

Mean	Group	Grp 4	Grp 3	Grp 5	Grp 2	Grp 1
44.9091	Grp 4					
56.9677	Grp 3	*				
57.3333	Grp 5					
60.8929	Grp 2	*				
64.8276	Grp 1	*	*			

This procedure was completed at 0:55:02
UNEWAY VARIABLES= ALEX BY PSYTRE(1,2)/RANGES=DUNCAN/STATISTICS=3.

- - - - - - - - - - - U N E W A Y - - - - - - - - - - -

Variable ALEX
By Variable PSYTRE ARE YOU IN PSYCHOLOGICAL TREATMENT?

Analysis of Variance

Source	D.F.	Sum of Squares	Mean Squares	F Ratio	F Prob.
Between Groups	1	1850.9626	1850.9626	9.1325	.0032
Within Groups	105	21281.2617	202.6787		
Total	106	23132.2243			

Tests for Homogeneity of Variances

Cochrans C = Max. Variance/Sum(Variances) = .5259, P = .702 (Approx.)
Bartlett-Box F = .118, P = .732
Maximum Variance / Minimum Variance 1.109

No Range Tests performed with fewer than three non-empty groups.

This procedure was completed at 0:55:18
UNEWAY VARIABLES=ALEX BY EXPPAIN(1,2)/RANGES=DUNCAN/STATISTICS=3.

- - - - - - - - - - U N E W A Y - - - - - - - - - -

Variable ALEX
By Variable EXPPAIN DO YOU EXPERIENCE PAIN?

Analysis of Variance

Source	D.F.	Sum of Squares	Mean Squares	F Ratio	F Prob.
Between Groups	1	1759.4271	1759.4271	8.4315	.0045
Within Groups	101	21075.9127	208.6724		
Total	102	22835.3398			

Tests for Homogeneity of Variances

Cochrans C = Max. Variance/Sum(Variances) = .5365, P = .604 (Approx.)
Bartlett-Box F = .256 , P = .613
Maximum Variance / Minimum Variance 1.157

No Range tests performed with fewer than three non-empty groups.

- - - - - - - - - - U N E W A Y - - - - - - - - - -

Variable ALEX
By Variable LENGPAIN HOW LONG HAVE YOU BEEN IN PAIN

Analysis of Variance

Source	D.F.	Sum of Squares	Mean Squares	F Ratio	F Prob.
Between Groups	2	1960.7130	980.3565	4.7316	.0110
Within Groups	96	19890.5800	207.1935		
Total	98	21851.2929			

Tests for Homogeneity of Variances

Cochrans C = Max. Variance/Sum(Variances) = .3696, P = .868 (Approx.)
Bartlett-Box F = .123 , P = .884
Maximum Variance / Minimum Variance 1.187

- - - - - - - - - - - U N E W A Y - - - - - - - - - - -

Variable ALEX
By Variable LENGPAIN HOW LONG HAVE YOU BEEN IN PAIN

Multiple Range Test

Duncan Procedure
Ranges for the .050 level -

2.81 2.96

The ranges above are table ranges.
The value actually compared with Mean(J)-Mean(I) is..
10.1782 * Range * Sqrt(1/N(I) + 1/N(J))

(*) Denotes pairs of groups significantly different at the .050 level

- - - - - - - - - - - U N E W A Y - - - - - - - - - - -

Variable ALEX
(Continued)

Mean	Group	Grp 3	Grp 1	Grp 2
53.8205	Grp 3			
61.0000	Grp 1			
63.0545	Grp 2	*		

- - - - - - - - - - - O N E W A Y - - - - - - - - - - -

Variable ALEX
By Variable MUSTNB IS PAIN THE MUST IMPORTANT?

Analysis of Variance

Source	D.F.	Sum of Squares	Mean Squares	F Ratio	F Prob.
Between Groups	2	3492.1660	1746.0830	9.5620	.0002
Within Groups	98	17886.0321	182.5105		
Total	100	21378.1980			

Tests for Homogeneity of Variances

Cochrans C = Max. Variance/Sum(Variances) = .4109, P = .575 (Approx.)
Bartlett-Box F = 1.922 , P = .147
Maximum Variance / Minimum Variance 1.899

- - - - - - - - - - - O N E W A Y - - - - - - - - - - -

Variable ALEX
By Variable MUSTNB IS PAIN THE MUST IMPORTANT?

Multiple Range Test

Duncan Procedure
Ranges for the .050 level -

2.81 2.95

The ranges above are table ranges.
The value actually compared with Mean(J)-Mean(I) is..
9.5528 * Range * Sqrt(1/N(I) + 1/N(J))

(*) Denotes pairs of groups significantly different at the .050 level

- - - - - - - - - - U N E W A Y - - - - - - - - - -

Variable ALEX
(Continued)

Mean	Group	Grp 3	Grp 2	Grp 1
53.8205	Grp 3			
55.6923	Grp 2			
66.7500	Grp 1	*	*	

This procedure was completed at 0:56:15
UNEWAY VARIABLES= ALEX BY CAUSPA(1,3)/RANGES=DUNCAN/STATISTICS=3.

- - - - - - - - - - U N E W A Y - - - - - - - - - -

Variable ALEX
By Variable CAUSPA DO YOU KNOW THE CAUSE?

Analysis of Variance

Source	D.F.	Sum of Squares	Mean Squares	F Ratio	F Prob.
Between Groups	2	1994.4920	997.2460	4.7851	.0104
Within Groups	100	20840.8478	208.4085		
Total	102	22835.3398			

Tests for Homogeneity of Variances

Cochrans C = Max. Variance/Sum(Variances) = .3838, P = .665 (Approx.)
Bartlett-Box F = .422 , P = .656
Maximum Variance / Minimum Variance 1.504

- - - - - - - - - - - - U N E W A Y - - - - - - - - - - -

Variable ALEX
By Variable CAUSPA DO YOU KNOW THE CAUSE?

Multiple Range Test

Duncan Procedure
Ranges for the .050 level -

2.81 2.95

The ranges above are table ranges.
The value actually compared with Mean(J)-Mean(I) is..
10.2080 * Range * Sqrt(1/N(I) + 1/N(J))

(*) Denotes pairs of groups significantly different at the .050 level

- - - - - - - - - - - - U N E W A Y - - - - - - - - - - -

Variable ALEX
(Continued)

Mean	Group	Grp 3	Grp 1	Grp 2
53.8205	Grp 3			
62.5833	Grp 1	*		
63.6875	Grp 2	*		

This procedure was completed at 0:56:32
UNEWAY VARIABLES=ALEX BY WHATCAUS(1,4)/RANGES=DUNCAN/STATISTICS=3.

- - - - - - - - - - U N E W A Y - - - - - - - - - -

Variable ALEX
By Variable WHATCAUS WHAT IS THE CAUSE?

Analysis of Variance

Source	D.F.	Sum of Squares	Mean Squares	F Ratio	F Prob.
Between Groups	3	1688.0374	562.6791	2.7621	.0463
Within Groups	95	19352.5888	203.7115		
Total	98	21040.6263			

Tests for Homogeneity of Variances

Cochrans C = Max. Variance/Sum(Variances) = .3249, P = .423 (Approx.)
Bartlett-Box F = 1.022 , P = .382
Maximum Variance / Minimum Variance 4.096

- - - - - - - - - - U N E W A Y - - - - - - - - - -

Variable ALEX
By Variable WHATCAUS WHAT IS THE CAUSE?

Multiple Range Test

Duncan Procedure
Ranges for the .050 level -

2.81 2.96 3.05

The ranges above are table ranges.
The value actually compared with Mean(J)-Mean(I) is..
10.0924 * Range * Sqrt(1/N(I) + 1/N(J))

(*) Denotes pairs of groups significantly different at the .050 level

- - - - - - - - - - - O N E W A Y - - - - - - - - - -

Variable ALEX
(Continued)

Mean	Group	Grp 4	Grp 2	Grp 1	Grp 3
56.3208	Grp 4				
61.1250	Grp 2				
62.5417	Grp 1				
71.6667	Grp 3	*			

This procedure was completed at 0:57:01
ONEWAY VARIABLES= ALEX BY RESPTR(1,3)/RANGES=DUNCAN/STATISTICS=3.

- - - - - - - - - - - O N E W A Y - - - - - - - - - -

Variable ALEX
By Variable RESPTR HAS YOUR PAIN RESPONDED TO TREATMENT?

Analysis of Variance

Source	D.F.	Sum of Squares	Mean Squares	F Ratio	F Prob.
Between Groups	2	2940.7551	1470.3775	7.3213	.0011
Within Groups	99	19882.6567	200.8349		
Total	101	22823.4118			

Tests for Homogeneity of Variances

Cochrans C = Max. Variance/Sum(Variances) = .4012, P = .467 (Approx.)
Bartlett-Box F = .516, P = .599
Maximum Variance / Minimum Variance 1.354

- - - - - - - - - - - U N E W A Y - - - - - - - - - - -

Variable ALEX
By Variable RESPTR HAS YUUR PAIN RESPONDED TU TREATMENT?

Multiple Range lest

Duncan Procedure
Ranges for the .050 level -

2.81 2.95

Ihe ranges above are table ranges.
The value actually compared with Mean(J)-Mean(I) is..
10.0209 * Range * Sqrt(1/N(I) + 1/N(J))

(*) Denotes pairs of groups significantly different at the .050 level

- - - - - - - - - - - U N E W A Y - - - - - - - - - - -

Variable ALEX
(Continued)

Mean	Group	Grp 3	Grp 1	Grp 2
53.9286	Grp 3			
59.9355	Grp 1			
67.0000	Grp 2	*		

This procedure was completed at 0:57:25
ONEWAY VARIABLES=ALEX BY PAINPER(1,6)/RANGES=DUNCAN/STATISTICS=3.

- - - - - - - - - - O N E W A Y - - - - - - - - - -

Variable ALEX
By Variable PAINPER WHAT IS YOUR PERCEPTION OF PAIN?

Analysis of Variance

Source	D.F.	Sum of Squares	Mean Squares	F Ratio	F Prob.
Between Groups	5	1777.9425	355.5885	1.7185	.1379
Within Groups	94	19449.8175	206.9130		
Total	99	21227.7600			

Tests for Homogeneity of Variances

Cochrans C = Max. Variance/Sum(Variances) = .2612, P = .250 (Approx.)
Bartlett-Box F = .342 , P = .889
Maximum Variance / Minimum Variance 1.632

- - - - - - - - - - O N E W A Y - - - - - - - - - -

Variable ALEX
By Variable PAINPER WHAT IS YOUR PERCEPTION OF PAIN?

Multiple Range Test

Duncan Procedure
Ranges for the .050 level -

2.81 2.96 3.05 3.12 3.18

The ranges above are table ranges.
The value actually compared with Mean(J)-Mean(I) is..
10.1714 * Range * Sqrt(1/N(I) + 1/N(J))

No two groups are significantly different at the .050 level

This procedure was completed at 0:58:05
ONEWAY VARIABLES=ALEX BY PAIN#(1,3)/RANGES=DUNCAN/STATISTICS=3.

- - - - - - - - - - - O N E W A Y - - - - - - - - - - -

Variable ALEX
By Variable PAIN# STRONGER CRITERION FOR CHRONIC PAIN

Analysis of Variance

Source	D.F.	Sum of Squares	Mean Squares	F Ratio	F Prob.
Between Groups	2	3697.7675	1848.8837	9.8940	.0001
Within Groups	104	19434.4568	186.8698		
Total	106	23132.2243			

Tests for Homogeneity of Variances

Cochrans C = Max. Variance/Sum(Variances) = .5202, P = .009 (Approx.)
Bartlett-Box F = 2.929 , P = .054
Maximum Variance / Minimum Variance 2.742

- - - - - - - - - - - O N E W A Y - - - - - - - - - - -

Variable ALEX
By Variable PAIN# STRONGER CRITERION FOR CHRONIC PAIN

Multiple Range Test

Duncan Procedure
Ranges for the .050 level -

2.81 2.95

The ranges above are table ranges.
The value actually compared with Mean(J)-Mean(I) is..
9.6662 * Range * Sqrt(1/N(I) + 1/N(J))

(*) Denotes pairs of groups significantly different at the .050 level

- - - - - - - - - - - O N E W A Y - - - - - - - - - - -

Variable ALEX
(Continued)

Mean	Group	Grp 3	Grp 1	Grp 2
55.6757	Grp 3			
62.1579	Grp 1	*		
69.8571	Grp 2	*		

This procedure was completed at 0:58:22
ONEWAY VARIABLES=ALEX BY HEALTH(1,4)/RANGES=DUNCAN/STATISTICS=3.

- - - - - - - - - - - O N E W A Y - - - - - - - - - - -

Variable ALEX
By Variable HEALTH

Analysis of Variance

Source	D.F.	Sum of Squares	Mean Squares	F Ratio	F Prob.
Between Groups	3	5213.3442	1737.7816	9.9890	.0000
Within Groups	103	17918.8796	173.9697		
Total	106	23132.2243			

Tests for Homogeneity of Variances

Cochrans C = Max. Variance/Sum(Variances) = .3597, P = .295 (Approx.)
Bartlett-Box F = 1.239, P = .152
Maximum Variance / Minimum Variance 3.920

- - - - - - - - - - U N E W A Y - - - - - - - - - -

Variable ALEX
By Variable HEALTH

Multiple Range Test

Duncan Procedure
Ranges for the .050 level -

2.81 2.95 3.05

The ranges above are table ranges.
The value actually compared with Mean(J)-Mean(I) is..
9.3266 * Range * Sqrt(1/N(I) + 1/N(J))

(*) Denotes pairs of groups significantly different at the .050 level

- - - - - - - - - - U N E W A Y - - - - - - - - - -

Variable ALEX
(Continued)

Mean	Group	Grp 1	Grp 2	Grp 3	Grp 4
51.2222	Grp 1				
55.6066	Grp 2				
65.1111	Grp 3	*	*		
76.3000	Grp 4	*	*	*	

This procedure was completed at 1:18:10
ANOVA ALEX BY HEALTH(1,4) PAIN#(1,3)/STATISTICS=3.

'ANOVA' PROBLEM REQUIRES 1780 BYTES OF MEMORY.

* * * C E L L M E A N S * * *

ALEX
BY HEALTH
PAIN# STRONGER CRITERION FOR CHRONIC PAIN

TOTAL POPULATION

59.57
(107)

HEALTH

1	2	3	4
51.22	55.61	65.11	76.30
(9)	(61)	(27)	(10)

PAIN#

1	2	3
67.16	69.86	55.68
(19)	(14)	(74)

HEALTH	PAIN# 1	2	3
1	48.00 (1)	62.00 (2)	48.17 (6)
2	64.89 (9)	68.75 (4)	52.22 (48)
3	70.00 (5)	71.29 (7)	60.60 (15)

4	73.50	80.00	77.80
	(4)	(1)	(5)

* * * A N A L Y S I S O F V A R I A N C E * * *

ALEX
BY HEALTH
PAIN# STRONGER CRITERION FOR CHRONIC PAIN

Source of Variation	Sum of Squares	DF	Mean Square	F	Signif of F
Main Effects	7499.696	5	1499.939	9.487	.000
HEALTH	3801.928	3	1267.309	8.016	.000
PAIN#	2286.391	2	1143.196	7.231	.001
2-way Interactions	612.748	6	102.125	.646	.693
HEALTH PAIN#	612.748	6	102.125	.646	.693
Explained	8112.444	11	737.495	4.665	.000
Residual	15019.780	95	158.103		
Total	23132.224	106	218.229		

107 Cases were processed.
0 Cases (.0 PCT) were missing.

This procedure was completed at 1:18:53
FINISH

R E L I A B I L I T Y A N A L Y S I S - S C A L E (D E P R E S S)

		MEAN	STD DEV	CASES
1.	D1	2.2951	.9371	61.0
2.	D2	2.5410	1.1630	61.0
3.	D3	1.7213	.8781	61.0
4.	D4	2.2623	1.1958	61.0
5.	D5	2.3770	1.2802	61.0
6.	D6	2.5082	1.3244	61.0
7.	D7	1.5574	.9041	61.0
8.	D8	1.5902	1.0063	61.0
9.	D9	1.5902	.9197	61.0
10.	D10	2.3115	1.2042	61.0
11.	D11	2.3279	1.1507	61.0
12.	D12	2.3279	1.1651	61.0
13.	D13	2.1639	1.0982	61.0
14.	D14	2.2787	1.1851	61.0
15.	D15	2.1475	.9804	61.0
16.	D16	2.3934	1.1730	61.0
17.	D17	2.2295	1.1887	61.0
18.	D18	2.1803	1.2716	61.0
19.	D19	1.4918	.9420	61.0

R E L I A B I L I T Y A N A L Y S I S - S C A L E (D E P R E S S)

		MEAN	STD DEV	CASES
20.	D20	2.2623	1.1676	61.0

OF CASES = 61.0

STATISTICS FOR SCALE	MEAN	VARIANCE	STD DEV	# OF VARIABLES
	42.5574	136.4175	11.6798	20

ITEM MEANS	MEAN	MINIMUM	MAXIMUM	RANGE	MAX/MIN	VARIANCE
	2.1279	1.4918	2.5410	1.0492	1.7033	.1125

R E L I A B I L I T Y A N A L Y S I S - S C A L E (D E P R E S S)

ITEM-TOTAL STATISTICS

	SCALE MEAN IF ITEM DELETED	SCALE VARIANCE IF ITEM DELETED	CORRECTED ITEM-TOTAL CORRELATION	SQUARED MULTIPLE CORRELATION	ALPHA IF ITEM DELETED
D1	40.2623	121.7301	.6678	.7023	.8476
D2	40.0164	126.5164	.3268	.4375	.8594
D3	40.8361	128.9060	.3381	.4623	.8583
D4	40.2951	120.5115	.5513	.5358	.8503
D5	40.1803	120.3503	.5137	.4877	.8519
D6	40.0492	122.9142	.4001	.4864	.8571
D7	41.0000	129.4667	.2981	.3296	.8595
D8	40.9672	133.1322	.0979	.1932	.8665
D9	40.9672	129.1656	.3064	.4699	.8593
D10	40.2459	125.0552	.3678	.4572	.8580
D11	40.2295	118.1464	.6775	.6431	.8453
D12	40.2295	120.3464	.5756	.5898	.8494
D13	40.3934	124.1426	.4523	.5989	.8544
D14	40.2787	121.9044	.5009	.5021	.8524

R E L I A B I L I T Y A N A L Y S I S - S C A L E (D E P R E S S)

ITEM-TOTAL STATISTICS

D15	40.4098	131.2126	.1889	.4225	.8633
D16	40.1639	122.1393	.4976	.5466	.8526
D17	40.3279	121.9907	.4956	.5069	.8527
D18	40.3770	119.5388	.5489	.5713	.8503
D19	41.0656	124.6623	.5166	.4933	.8526
D20	40.2951	118.5448	.6493	.6233	.8463

R E L I A B I L I T Y A N A L Y S I S - S C A L E (D E P R E S S)

RELIABILITY COEFFICIENTS 20 ITEMS

ALPHA = .8608 STANDARDIZED ITEM ALPHA = .8588

RELIABILITY ANALYSIS - SCALE (ALEXITHY)

		MEAN	STD DEV	CASES
1.	A1	2.8229	1.4436	96.0
2.	A2	2.7604	1.4707	96.0
3.	A3	2.3854	1.4391	96.0
4.	A4	2.8333	1.4043	96.0
5.	A5	3.1875	1.4676	96.0
6.	A6	2.0729	1.4527	96.0
7.	A7	2.8125	1.4460	96.0
8.	A8	2.2396	1.1675	96.0
9.	A9	2.5313	1.6089	96.0
10.	A10	2.3542	1.4215	96.0
11.	A11	2.3958	1.3415	96.0
12.	A12	2.9688	1.3876	96.0
13.	A13	1.7500	1.0260	96.0
14.	A14	2.6458	1.4866	96.0
15.	A15	2.4375	1.3903	96.0
16.	A16	2.7500	1.6026	96.0
17.	A17	2.4375	1.4350	96.0
18.	A18	2.9583	1.2642	96.0
19.	A19	3.3333	1.4630	96.0

RELIABILITY ANALYSIS - SCALE (ALEXITHY)

		MEAN	STD DEV	CASES
20.	A20	3.3542	1.4795	96.0
21.	A21	1.8854	1.1413	96.0
22.	A22	2.0000	.9841	96.0
23.	A23	2.7083	1.4136	96.0

RELIABILITY ANALYSIS - SCALE (ALEXITHY)

OF CASES = 96.0

STATISTICS FOR SCALE	MEAN	VARIANCE	STD DEV	# OF VARIABLES
	59.6250	215.1842	14.6692	23

ITEM MEANS	MEAN	MINIMUM	MAXIMUM	RANGE	MAX/MIN	VARIANCE
	2.5924	1.7500	3.3542	1.6042	1.9167	.1878

R E L I A B I L I T Y A N A L Y S I S - S C A L E (A L E X I T H Y)

ITEM-TOTAL STATISTICS

	SCALE MEAN IF ITEM DELETED	SCALE VARIANCE IF ITEM DELETED	CORRECTED ITEM-TOTAL CORRELATION	SQUARED MULTIPLE CORRELATION	ALPHA IF ITEM DELETED
A1	56.8021	204.1604	.2167	.2869	.8307
A2	56.8646	192.4130	.5051	.4803	.8179
A3	57.2396	206.0999	.1697	.1969	.8326
A4	56.7917	200.0825	.3305	.3882	.8257
A5	56.4375	188.5855	.6064	.5791	.8131
A6	57.5521	196.5236	.4063	.5548	.8224
A7	56.8125	195.7961	.4275	.4782	.8214
A8	57.3854	205.6920	.2427	.4066	.8286
A9	57.0938	192.6964	.4455	.4480	.8205
A10	57.2708	195.2522	.4509	.4863	.8204
A11	57.2292	203.5890	.2559	.1962	.8286
A12	56.6563	195.9543	.4455	.5061	.8207
A13	57.8750	210.6789	.1159	.3878	.8323
A14	56.9792	186.0627	.6635	.5601	.8103

R E L I A B I L I T Y A N A L Y S I S - S C A L E (A L E X I T H Y)

ITEM-TOTAL STATISTICS

A15	57.1875	197.9855	.3902	.3940	.8231
A16	56.8750	187.1632	.5804	.6213	.8132
A17	57.1875	194.2592	.4716	.4865	.8195
A18	56.6667	199.2561	.4015	.4206	.8228
A19	56.2917	200.8193	.2948	.3345	.8274
A20	56.2708	193.5259	.4230	.4117	.8193
A21	57.7396	205.0367	.2706	.4988	.8276
A22	57.6250	207.3526	.2422	.4745	.8284
A23	56.9167	204.2456	.2213	.3559	.8303

R E L I A B I L I T Y A N A L Y S I S - S C A L E (A L E X I T H Y)

RELIABILITY COEFFICIENTS 23 ITEMS

ALPHA = .8299 STANDARDIZED ITEM ALPHA = .8240

This procedure was completed at 0:45:11
FINISH