Correlations Between Biopsychosocial Variables and Low Back Pain

Dissertation / Doctoral Thesis

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1. ABSTRACT

This paper examines the correlations between biopsychosocial variables and low back pain. 160 German females and 110 German males (*N* = 270; all pain patients from out-patient practices) completed a battery of questionnaires assessing their pain experience (chronicity and intensity), comorbidities, and causal attributions about disease onset. Findings reported here support previous research in that some biopsychosocial factors are significantly associated with low back pain. Specifically, the significant relationships found for chronic back pain were: higher age, lower education, and higher depression. For high intensity back pain, significant relationships were found for females, participants with lower education, and higher depression. When analyzing the participant sample together, present and past comorbidities were significantly associated with pain chronicity and present comorbidities were also significantly associated with high pain intensity. However, analyses revealed no significant associations between comorbidities (present or past) for the *back* pain group. Older (> 48 years of age), *back* pain patients reported significantly more present comorbidities. Analyses of the causal attributions about disease onset revealed that back pain patients considered "Constantly being stressed out" to be particularly relevant to the onset of heart disease. Shown through the consistently higher means (and consistently lower standard deviations), all participants considered the personality variables of the causal attribution items to be important for the onset of heart disease. Participants seemed to be less certain which personality factors play a role in the onset of low back pain.

2.1. INTRODUCTION

The question, 'What is pain?', has perplexed philosophers, physicians and lay persons since the earliest recorded history. A tremendous amount of effort has been expended in an attempt to understand the mechanisms behind pain, especially chronic pain. Despite advances in modern medicine, 75% of the population in western industrial nations will suffer in their lives at least once from back pain (Pfingsten, Kaluza, Hildebrandt, 1999). In Germany, the number one reason for paying worker's compensation for men is back pain. For women back pain is the number two reason for paying worker's compensation. A German cost-of-illness study calculated that back pain costs more than 18 Billion Euros annually (as cited in Pfingten, Kaluza, & Hildebrandt, 1999). Approximately 90% of back pain patients find relief through rest and relaxation, analgesic drugs, and physical therapy. Sixty percent of back pain patients are able to return to work after a week; only 10% of patients with acute back pain are incapacitated longer than six weeks (Waddell, 1987). Nevertheless approximately 70% of back pain patients experience new episodes of back pain that last longer and are more painful than the first episodes. The high rate of recurrent back pain episodes together with therapy resistant back pain pose the greatest questions in research and treatment.

The general purpose of this study is to identify the mechanisms involved in low back pain and to determine how these mechanisms are related to chronicity and intensity of the pain experience. To accomplish this, the study reported here addresses three topics: (1) the Biopsychosocial Model, (2) the role of comorbidities, and (3) causal attributions about disease onset. The first step in this process is to test whether biopsychological and social factors found in the literature prove to be relevant here. For example, the relationships between depression, job dissatisfaction, psychological "overlay" (Waddell, 1987) and low back pain will be examined. Just as risk factors may be relevant, protective factors may also play a role in preventing acute back pain patients from becoming chronic back pain patients. In particular, the construct, Sense of Coherence, will be analyzed. The next step involves an analysis of other diseases, or comorbidities. Are some individuals more prone to acquiring diseases than others? Is there some validity to the pain prone personality? Are there

differences between individuals with and without comorbidities regarding the psychosocial factors listed above (e.g., Sense of Coherence, depression, job satisfaction, etc.). The third step is to examine the causes behind disease acquisition. What are the causal attributions about low back pain onset? Do these explanations correlate with potentially fatal diseases such as cancer and heart disease?

The purpose of this next section is to give the reader an overview of the historical developments in the field of pain research. Theories that will be discussed below include the biomedical model, the gate control theory (Melzack 1999; Melzack & Wall, 1965), operant learning mechanisms and chronic pain (Fordyce, 1976). These theories form the basis of the Biopsychosocial Model. Comorbidities as a research factor will be examined and a review of the literature covering pain and personality will be presented. Beliefs about disease onset, causal attribution theories, and coping strategies are the content of this section. Gender differences, the role of the family, prevention and treatment conclude the review of the literature in the field of pain research. But before beginning with the theoretical background, pain will be defined and its function and its processing discussed and epidemiological data presented.

2.1.1. Definition of pain

According to the International Association for the Study of Pain, pain is defined as "an unpleasant sensory and emotional experience associated with actual or potential tissue damage or described in terms of such damage" (Merskey, 1986). In comparison to earlier definitions, this definition of pain addresses the psychological component of the pain experience instead of focussing on the purely sensory perception. In addition, this definition suggests that although tissue damage can be an important part of the pain experience, it is not necessary to have tissue damage to feel pain. Further, this definition views emotions as an integral component of the pain experience, and not just as a reaction to pain.

2.1.2. Function of pain

Pain typically leads people to change their activity level and alter their movements. Such pain behaviors are an important component of the pain experience. For people who experience chronic pain, the emotional reactions are more likely to be anxiety and depression. These emotional reactions to pain are also an integral part of the pain experience.

Pain not only hurts and can disrupt our daily activities, it also provides low-level feedback about the functioning of our bodily systems, feedback that we use, often

unconsciously, as a basis for making minor adjustments, such as uncrossing our legs, or rolling over while sleeping, or avoiding the countertop corner.

Pain is the symptom most likely to lead an individual to seek medical treatment. However, the correlation between pain and the severity of the symptom can be weak. For example, patients with similar bodily symptoms may see themselves as differently impaired. The objective functional restrictions could be minimal while the subjective impairments are judged to be high. Further the objective and subjective impairments correlate only moderately with pain intensity (Waddell, Newton, Henderson, Somerville & Main, 1993).

Pain has medical and psychological significance. When patients are asked what they fear most about illness and its treatment, most reported being in pain. The dread of not being able to reduce one's own suffering arouses more anxiety than the prospect of surgery, the loss of a limb, or even death (Taylor, 1999).

2.1.3. Pain Processing

Pain is fundamentally a psychological experience. The degree to which it is felt and how incapacitating it is depends in large part on how it is interpreted. Pain is also heavily influenced by the context in which it is experienced. For example, many athletes who have injured themselves on the playing field stayed in the game, apparently oblivious to their injury. This may be due to the sympathetic arousal that seems to diminish pain sensitivity (Fillingham & Maixner, 1996; Zillman, de Wied, King-Jablonski & Jenzowsky, 1996). In contrast individuals with no family and few social contacts may experience their pain more acutely. It appears that pain has a cultural aspect as well. Although there are no racial or ethnic differences in the ability to discriminate painful stimuli, members of some cultures report pain sooner and experience pain more intensively than members of other cultures (Zatzick & Dimsdale, 1990).

2.1.4. Acute Versus Chronic Pain

Clinically, pain is divided into two groups: acute and chronic pain. Acute pain typically results from some specific injury that produces tissue damage, such as a wound or a broken bone. Acute pain typically disappears when the tissue damage is repaired. Acute pain is usually short in duration, and with treatment and the passage of time pain decreases. Further, the location, pattern, and description of the pain suggest the somatic cause and, thus, help the practitioner in determining appropriate interventions (Gatchel & Gardea, 1999).

Chronic pain typically begins with an acute episode, but unlike acute pain, it does not decrease with treatment and the passage of time. Chronic pain can further be defined into three categories. Chronic benign pain typically persists for six months of longer and is relatively intractable to treatment. The pain varies in severity and may involve any of a number of muscle groups. Chronic low back pain and myofascial pain syndrome are examples. Recurrent acute pain involves a series of intermittant episodes of pain that are acute in character, but chronic because the condition can persist over years. For example with migraine headaches, temperomandibular disorder, and trigeminal neuralgia. Chronic progressive pain persists longer than six months and increases in severity over time. Typically, it is associated with malignancies or degenerative disorders such as skeletal metastatic disease or rheumatoid arthritis. Chronic pain is not necessarily present at all times, but the fact that it is chronic forces sufferers to organize their lives around their pain.

The distinction between acute and chronic pain is important for several reasons. First, acute and chronic pain present different psychological profiles, that is, chronic pain often carries an overlay of psychological distress that complicates diagnosis and treatment. Depression is common among chronic pain patients and may exacerbate pain and pain related behaviors (Kröner-Hedwig et al., 1996). Second, most of the pain control techniques are more effective controlling acute pain, but are less successful with chronic pain management. Third, chronic pain involves the interaction of physiological, psychological, social, and behavioral components, which makes it

much more complicated. The interaction of these components evolves over time making chronic pain a syndrome (Flor et al., 1990).

Chronic pain may derive from a predisposition to respond to an injury with a specific bodily response, such as altering one's posture. This response may then be exacerbated by stress. The chronic back pain that may result can be aggravated by inadequate coping, further exacerbating the pain syndrome and leading to pain behaviors that occur in the process of attempting to cope with pain, e. g., taking time off from work, discontinuing an exercise program. Because of their pain, chronic pain sufferers become more susceptible to stress, further compromising their ability to cope. By the time a pain patient is adequately treated, this complex, dynamic interaction of physiological, psychological, social, and behavioral components is often tightly integrated, making it difficult to modify (Flor et al., 1990).

Chronic pain can entirely disrupt a person's life forcing them to leave their jobs, abandon their leisure activities, and withdraw from their families and friends. Often, income is reduced and work related goals and personal aspirations are set aside, and with it, a loss in self-esteem (Karoly & Ruehlman, 1996). Some chronic pain patients receive compensation because their pain resulted from an injury, such as an automobile accident. Compensation can actually increase the intensity of the pain, the amount of disability experienced, the degree to which pain interferes with life activities, and the amount of distress that is reported (Turk & Okifuji, 1996). Chronic pain patients tend not to communicate well with their families and sexual relationships almost always suffer. Ironically, when spouses remain supportive, such positive attention may inadvertently maintain or increase expression of pain and the experience of disability (Turk, Kerns & Rosenberg, 1992).

Many chronic pain patients are clinically depressed and a large number have contemplated or attempted suicide. Frequently, chronic pain patients consume large quantities of pain-killers and these drugs may be only partially effective and they usually have undesirable side effects such as problems with concentration and addiction.

2.1.5. Classification of Low Back Pain

Further classification of pain can be applied to low back or lumbar spine pain. These types are: Transient back pain is defined as an episode in which back pain is present on no more than 90 consecutive days and does not recur over a 12-month observation period. In recurrent back pain, pain is present on less than half the days in a 12-month period, and occurring in multiple episodes over the year. In patients with chronic back pain (see above), pain is present on at least half the days in a 12-month period in single or multiple episodes. Acute back pain is pain that is not recurrent or chronic (as defined above) and whose onset is recent and sudden. The first onset is an episode of back pain that is the first occurrence of back pain in a person's life. A flare-up is defined as a phase of pain superimposed on a recurrent or chronic course. A flare-up refers to a period (usually a week or less) when back pain is markedly more severe than is usual for the patient (Von Korff, 1994).

Lumbar spine pain can be further classified by onset and duration. Acute pain implies immediate onset, with a duration of 0 to 6 months; Subacute pain means slow onset, with a duration of 0 to 6 months; The duration of chronic pain is longer that 6 months, regardless of onset. Recurrent pain shows intervals during which no symptoms are present, but pain reappears (Olmarker, K. & Hasue, M., 1995).

Classification by location and distribution: Local pain refers to lower lumbar or lumbosacral pain (lumbago). Referred pain is pain experienced at the area that shares a common embryologic origin with the region involved. It is usually located to the inguinal or buttock region or the anterior, lateral, or posterior thigh. In some cases, however, it might be distributed even below the knee. Radicular pain is pain that is distributed along the dermatomal distribution of a spinal nerve root and is caused by a direct affection of the nerve tissue. It is commonly experienced along the course of the sciatic nerve, depending on the spinal level of the involved nerve root. Sciatica: It literally means "related to the hip". It is defined as a local affection of the sciatic nerve in the thigh (Olmarker, K. & Hasue, M., 1995).

2.1.6. Biomechanics of Low Back Pain.

Identifiable disease processes, such as ankylosing spondylitis and rheumatoid arthritis, account for only a small minority of low back pain patients. There is growing evidence that, for the majority, the underlying cause of back pain is mechanical. Adams (1996) proposes that although many influences must be involved, mechanical loading may be one of the primary factors leading to low back pain.

Where does back pain come from? All spinal tissues, except the central region of the intervertebral discs, have the potential to be painful, due to innervation of these tissues and structures. Pain provocation studies, however, suggest that the most common origin of severe and chronic back pain is the posterior annulus fibrosus and the adhering longitudinal ligament (Adams, 1996). In a substantial minority of low back pain patients, the apophyseal joints and sacroiliac joints are also painful.

Mechanical failure. Adams (1996) hypothesizes that pain can be caused by mechanical failure. The most common mechanisms are as follows: ligaments of the neural arch are most easily damaged by forward bending movements; the apophyseal joint surfaces by torsion and backward bending; the vertebral body by compression, and the disc by asymmetrical bending and compression, or following compressive damage to the vertebral body (Adams, 1996). In each of the mechanisms described above, damage can occur during a single loading cycle simulating some incident such as a fall, or by the process of accumulating "fatigue failure" in which the forces remain relatively low. Pain may possibly arise in the absence of structural failure if high stress concentrations are generated within the posterior annulus or apophyseal joints. This can occur as a result of lordotic postures, especially if they are held for long periods, and it may possibly occur as a result of excessive or unbalanced muscle activity.

Effects of age and degeneration on spinal mechanics. Age related changes in disc mechanics appear to be due to change in biochemical composition. With increasing age, the discs' collagens and proteoglycans undergo quantitative and qualitative changes, which may be related to nutritional compromise, or due to degradative

enzymes (Adams, 1996). The most important mechanical consequence of these changes is the 10-15% reduction in the water content of the nucleus pulposus because it reduces the ability of the nucleus to behave like a pressurized fluid. Further, all spinal tissues become weaker and less extensible with age. One manifestation of this is the reduced range of movement demonstrated by degenerated spines.

Muscle dysfunction. It is widely suspected that back pain can lead to abnormal muscle function, which, in turn leads to the recurrent or chronic problems in muscle and underlying tissues. For example, pain may inhibit normal spinal movements, causing muscle atrophy and a reduction in joint mobility. Anatomically, spinal muscles are required to protect the underlying spine from excessive bending, and since this protection is reduced by poor mobility, the end result of the pain may be an increased risk of bending injuries to the intervertebral discs and ligaments. Further, unilateral pain may cause an imbalance in muscle activity, leading to asymmetry in spinal posture and movement. Adams (1996) proposes that small changes in lumbar curvature can lead to high and potentially painful stress concentrations in the intervertebral discs and apophyseal joints. Adams (1996) admits, however, that these hypotheses are difficult to prove.

Variability. Research has shown that individuals who do a lot of bending and lifting due to occupational requirements are much more likely to develop back pain than those who do no lifting. Nevertheless, the majority of individuals in high risk occupations remain unaffected, suggesting that risk factors exist that predispose certain individuals to back problems. Risk factors from a biomechanical perspective include a long back, heavy body, poor range of movement, easily fatigued muscles, and familial predisposition.

Sources of low back pain complaints. As with all human disease, the diagnosis and treatment of low back problems begin with the history, followed by the clinical workup, selected imaging modalities for confirmation, and a treatment protocol. Questioning the patient allows concepts to form regarding the involved anatomy. For

example, low back pain alone is more common in annular tears and in facet degenerative and subluxation syndromes, whereas sciatica points to disc protrusion or stenosis within the vertebral canal. Serious disc lesions are preceded by numerous and worsening bouts of low back pain. Low back pain that suddenly is transformed into only leg pain probably represents a contained disc that has become a noncontained disc (Cox, 1999).

Five common sources of sciatica have been suggested by McCarron and Laros (1987):

- Herniated disc
- Annular tears
- Myogenic disease (muscle-related)
- Spinal stenosis
- Facet joint arthropathy

Herniated disc. The disc performs its role as a shock absorber very efficiently as long as it is watertight. Due to the aging process and or excessive "wear and tear", the disc may partially lose this property, i.e., cracks develop in the annulus fibrosus through which the fluid of the nucleus can escape. This condition is termed a herniated or ruptured disc. It happens most commonly as a result of chronic flexion movements (forward bending), during which the nucleus moves toward the back and fluid can escape there. The fluid may then compress the nerve roots, i.e., the sciatic nerve which exits from the lumbar region, where pressures on the vertebral column are most intense. This situation, combined with chronic or sudden extreme tension on the posterior longitudinal ligaments, can result in chronic back pain.

Annular tears. In some patients with low back pain and unilateral or bilateral radiation to the lower extremities, the pain arises from within the disc. The pain-sensitive structures responsible for the radiating pain to the lower extremity are located somewhere inside the disc, probably in the external parts of the anulus fibrosus and in the longitudinal ligaments (Cox, 1999).

A patient's painful symptoms can be reproduced by injecting a contrast medium into the disc demonstrating an anular tear, and then the symptoms can be relieved by injecting a local anesthetic. This anesthetic does not need to extend beyond the disc margins to relieve low back or leg pain, thus supporting the existence of discogenic pain (Cox, 1999).

Tears in the periphery are caused by trauma rather than by biochemical degradation; they develop independently of nuclear degeneration and are responsible for discogenic low back pain (Cox, 1999).

Myogenic disease (muscle-related). Most chronic muscle strains are actually the result of degenerative or herniated discs. There is no such thing as chronic muscle strain; most are actually degenerative or herniated discs causing secondary muscle spasms.

Spinal stenosis. Stenosis is an abnormal narrowing of the body or ligamentous structures of the vertebral canal. Normal persons have sufficient room in the canal and lateral recesses for molding and gliding; hence, movement produces no clinical symptoms. However, if the size of the canal is narrowed by bony or ligamentous proliferations, symptoms appear.

Spinal stenosis may be the most important element in determining symptoms, and their severity, response to treatment, and prognosis. A patient can have a large disc protrusion and also a large diameter vertebral canal and lateral recess, and therefore, have no symptoms, whereas the same disc protrusion can cause severe motor and sensory findings in a patient with a stenotic canal. Therefore, disc protrusion size is not as important as the size of the canal it bulges into.

Facet joint arthropathy. Arthrosis is the degeneration of the structure of the joint. Arthrosis of the facets is rare in patients under age 30, and it is found progressively more frequently and is more severe as patients age (Cox, 1999).

2.1.7. Social-Epidemiological Data

Low back pain is a condition that is currently taking a significant toll on the health care systems of most industrialized countries, as well as on the personal lives of individuals who suffer from it. It has been estimated that 1 in 25 people will change his or her work because of low back pain or will retire early due to disability stemming from low back pain (Taylor, 1999). Back pain has been found to be the most expensive benign condition in industrialized countries, while representing the primary cause of disability in individuals under the age of 45 years (Garofalo & Polatin, 1999), in the U.S. (Taylor, 1999) and in Germany (Basler, 1999). Further, it is estimated that $16 billion are spent annually in the U.S. alone for the treatment of muscoskeletal pain, of which approximately one-half is consumed by surgical treatment (Garofalo & Polatin, 1999).

In the United States 6.8% of the adult population has been found to have back pain at any given time. The prevalence of low back pain rises after age 25 to a peak in the 55- to 64-year age range, with a falling prevalence after age 65. Consideration of the specific age of onset shows that 11% of persons are afflicted at less than 20 years of age; 28% at 20 to 29 years, 25% at 30 to 39 years, 20% at 40 to 49 years, 11% at 50 to 59 years, and 5% at more than 60 years of age (Deyo & Tsui-Wu, 1987).

The demographic prevalence shows regionally that the Northeastern United States has a 38% higher rate of low back pain than the Western states. There are no known differences between, for example, Northern versus Southern Germany. In the U.S. men and women are afflicted similarly, with white men having the highest prevalence and black men the lowest. Less educated persons have a 50% increased incidence over better-educated persons (Deyo & Tsui-Wu, 1987).

2.1.8. Cross-Cultural Comparisons

Cross-cultural comparisons in pain treatment centers in several countries including the United States, Germany, Mexico, Japan, Italy and New Zealand indicate similar levels of sick-leave due to low back pain, however, it seems that American patients

reported the greatest level of impairment in all areas of life functioning (Garofalo & Polatin, 1999). Other cross-cultural comparisons have revealed that experiences of back problems were reported less frequently in Hong Kong than in Britain (Lau et al., 1996). Although Hong Kong respondents tended to be shorter, weighed less, and engaged in heavy lifting less often than British respondents, these differences did not fully account for the varying incidence rates. It has been postulated that cultural differences may explain a higher threshold for reporting pain symptoms.

A survey in a Nordic sample of more than 2,000 individuals found that 60-65% of 30 to 50 year old men and women reported a lifetime prevalence of low back problems, and 44-54% reported low back pain in the last year (Leboef-Yde, Klougart, & Lauritzen, 1996). However, sick leave in Scandanavia due to low back pain has been estimated at 25-40%, compared to the United States in which sick leave due to low back pain has been reported to be as high as 90% (Mayer, 1991). This finding is interesting in that the costs of medical resources and disability payments are reportedly greater in Northern European countries in which the socialized economies do not emphasize the distinction between compensatory and non-compensatory pain to the extent done in the U.S. (Mayer & Gatchel, 1988).

2.1.9. Occupational Settings

Despite the overarching influence of low back pain, both on a social and individual level, there is still no identifiable cause for its onset or its reoccurrence. Nevertheless, new technology has furthered the understanding of low back pain and the identification of pain sources, as well as neurophysiological mechanisms of nociception from a biomedical perspective. The objective of this section is to provide a cogent discussion of the factors that are believed to contribute to its high incidence in Western industrialized countries and in particular occupational settings. Although there is general agreement among researchers and clinicians that exposure to a combination of occupational risk factors may render certain individuals more susceptible to musculoskeletal pain, it is clear that there is no single causal relationship (Garofalo & Polatin, 1999). In addition, psychological and social factors

will be examined with respect to their potential impact on the expression of back pain.

A number of researchers have targeted the work environment to better assess the rate of low back pain according to vocation and to advance the understanding of the contributing mechanisms of low back pain among different occupational subsets. Burton, Tillotson, Symonds, Burke, and Mathewson (1996) surveyed low back pain in a large sample of U.S. police officers; the aim of the study was to identify potential risk factors that may contribute to the high rate of first-onset and recurrent back problems. The most important factor for first onset low back pain in this subset was wearing body armor, weighing approximately 8.5 kilo. However, the chronicity of the condition appeared to be more attributable to psychological factors such as the stressful nature of being a police officer rather than to physical demands of the job alone.

There has been general agreement that nursing is a high-risk profession for low back pain. Among the 1,616 female nurses surveyed, 60% of the respondents reported a life-time prevalence of low back pain, 45% reported low back pain in the preceding 12-month period (Smedley, Egger, Cooper, & Coggon, 1995). Occupational risk factors associated with low back pain in nurses include frequency of lifting and manually moving patients.

A number of studies have examined prevalence rates of low back pain in various industrial settings in which heavy labor is merely one component of the individual's daily routine. Hildebrandt, Bongers, Dul, Van Dijk, & Kemper (1996) found in a sample of 400 steel workers that the most common symptoms were low back, neck, and shoulder pain. The majority of the workers contributed their back problems to heavy and frequent lifting (Hildebrandt, Bongers, Dul, Van Dijk, & Kemper, 1996).

2.2. BIOPSYCHOSOCIAL MODEL OF PAIN:

Historical Developments In Pain Research

2.2.1. The Biomedical Model

The history of pain research is relatively short. In the last 30 years pain has become an independent research phenomenon. Contemporary understanding of pain in the medical field still reflects the strong influence of Descartes, who propagated the concept that the body and mind were separate entities. According to Descartes, pain is defined as a straight-through sensory projection system that moved injury signals from damaged tissue to the brain, where the mind could recognize them (Chapman, Nakamura, & Flores, 1999). This perspective went unchallenged for almost two centuries, and it still exerts considerable subtle influence. Until the 1960s, researcher and physicians based their model of pain on the assumption that tissue trauma activates specific receptors and that signals of tissue trauma follow specific pain pathways through the spinal cord to a pain center in the brain.

The biomedical model perspective views pain as a sensory experience that signals tissue damage. The transmission of tissue damage information from the periphery to the cerebral cortex causes the experience of pain. Simply said, pain involves (1) the *transduction* of tissue trauma into neural signals, (2) the *transmission* of such signals to the dorsal horn of the spinal cord and from there to the thalamus, (3) the central *registration* of the sensory information in somatosensory cortex, and (4) the *modulation* of the signals along the way is an important feature of pain and a mechanism for pain relief. In addition, the biomedical model recognizes pain states that originate with trauma to neurological pathways (neuropathic pain). A chronic pain state is neuropathic when the brain interprets signals originating from the abnormal firing of damaged nerves as a true sensory experience.

Basic Assumptions: The classical biomedical perspective views pain as a mechanistic sensory experience that signals tissue damage. It takes place in a passive nervous system either through immediate tissue trauma or neuropathy. Alleviating the pain includes: removing the cause of tissue trauma or tissue sensitization in the periphery, blocking or interrupting pathways carrying noxious signals, and activating

endogenous modulation mechanisms by pharmacological or neurosurgical means. This model assumes that a neural message signals pain from the moment trauma activates the sensory end organ, and the awareness of pain depends on the activation of specific regions of the brain. According to the biomedical model, pain is the sensory end product of an essentially passive information transmission process that operates as a biologically adaptive mechanism.

Basic Mechanisms: Transduction. The transduction of tissue trauma into neural signals depends on sensory end organs known as nociceptors. The free nerve endings of thinly melinated A-delta fibers function as thermal and/or mechanical nociceptors, conducting impulses from 4 to 44 meters per second. In addition, certain C-fibers that conduct slowly (0.5-1 meter per second) act as polymodal nociceptors, responding to various high-intensity mechanical, chemical, and thermal stimuli. Both types of fibers are found in the skin and deep tissue. Repetitive stimulation of these receptors produces pain (Chapman, Nakamura & Flores, 1999).

Nociceptors innervate skin, muscle, fascia, joints, tendons, blood vessels, and visceral organs. From a sensory perspective, these tissues group into cutaneous, deep, and visceral types. Nociception appears to serve somewhat different functions in the three types of tissues, and the quality of the pain that ensues from their activation varies across types. Most cutaneous pain is well localized, sharp, pricking pain sensations of short duration whereas C fibers typically generate burning sensations. Deep-tissue pain usually seems diffuse and dull or aching in quality, although deep tissues can produce sharp pains under certain conditions (e.g., muscle rupture). Visceral pain is very diffuse, often referred to the body surface, persists after the orginal stimulus has ceased, and is frequently associated with a queasy quality that patients describe as "sickening". Severe visceral pain typically produces profuse sweating, nausea, and vomiting (Chapman, Nakamura & Flores, 1999).

Sufficient stimuli for nociception differ across tissue types. Cutaneous receptors detect injurious stimuli from the surrounding environment, and so they respond to

severe mechanical and thermal events such as cutting, burning, or freezing. Nociceptors in deep tissue such as muscle detect overuse and strain, deep mechanical injury include tearing, cramping, and ischemia. Visceral nociceptors respond to pathological change. A hollow viscous needs to identify and transduce distention, stretch, and isometric contraction. A solid organ needs to signal distention of the capsule that contains it and inflammation (Chapman, Nakamura, & Flores, 1999). The peripheral origins of pain vary markedly, depending on whether the nociceptors involved lie in superficial or deep tissues.

Sensitization of Nociceptors: Sensitization of nociceptors is an important factor in clinical pain states. As nociceptors become sensitized, pain thresholds diminish (allodynia), and painful qualities of subsequent noxious stimuli increase (hyperalgesia). Such alterations may reflect changes in the transduction process, central changes that facilitate the transmission of noxious messages, or both. Sensitization of nociceptors can result from either inflammation or repetitive stimulation of nociceptors. Enhanced sensitivity is usually adaptive because it allows for recuperation and repair (Chapman, Nakamura, & Flores, 1999). A key feature of sensitization is that it can awaken nociceptors that are otherwise "sleeping" nociceptors (Flor, Birbaumer, & Turk, 1990).

Basic Mechanisms: Transmission. The central transmission of noxious signals takes place in the spinal cord. Nociceptive afferents enter the spinal cord primarily through the dorsal route, terminating principally in lamina I (the marginal zone) but also in laminae II (the substantia gelatinose) and V of the dorsal horn (Cox, 1999). The spinal and medullary dorsal horns are much more than simple relay stations; these complex structures participate directly in sensory processing, performing local abstraction, integration, selection, and appropriate dispersion of sensory impulses (Chapman, Nakamura, & Flores, 1999). Upon entry, nociceptive afferents synapse with projection neurons that convey information to higher centers, facilitory interneurons that relay input to projection neurons, and inhibitory interneurons that modulate the flow of nociceptive signals to higher centers (Cox, 1999). Similar neural processing occurs in the spinal cord and the medullary dorsal horn.

The spinal cord contains a complex network of interneurons. These networks not only relay signals to higher levels of the central nervous system but also modulate signal transmission and initiate motor reflexes. Peripheral trauma can sensitize dorsal horn nociceptive neurons, making them sensitive to normal inputs. The exaggerated response of transmission cells in the spinal cord is central sensitization. Persistent central sensitization could cause chronic pain (Chapman, Nakamura, & Flores, 1999).

There are two principal types of projection neurons in the spinal cord: nociceptive specific and multireceptive neurons. The former convey only tissue trauma signals; the latter respond to stimuli of increasing intensity. Ascending tracts include spinothalamic, spinoreticular, spinomesencephalic, spinocervical, and postsynaptic dorsal cord tracts (Flor, Birbaumer, & Turk, 1990). In biomedical thinking, the spinothalamic tract is clearly the most important. Lesions of the anterolateral quadrant of the spinal cord result in a loss of pain sensation below the segmental level of the lesion on the contralateral side of the body (Cox, 1999).

Central Registration: The thalamus is a gateway and relay center for afferent input coming to the brain; therefore, it is the key structure in central registration (Chapman, Nakamura, & Flores, 1999). It consists of several functionally distinct nuclei that are reciprocally connected to many parts of the limbic system and the cortex (Willis & Westlund, 1997). Medial and ventrobasal thalamic nuclei relay noxious signals to the primary and secondary somatosensory cortices where refined localization and discrimination occur. From a biomedical perspective, the appreciation of pain occurs in these cortical areas.

Recent work acknowledges the existence of spinoreticular, spinomesencephalic, and spinolimbic pathways as nociceptive pathways (Willis & Westlund, 1997), but to date neurophysiologists do not link them to appreciation of pain sensation. Chapman and Stillman (1996) suggest that spinolimbic and spinoreticular pathways play a major role in the emotional component of pain and that this determines the aversive quality of the pain experience.

Basic Mechanisms: Modulation. Modulation refers to the neural activity that leads to control of the nociceptive transmission pathway. Input from the frontal cortex and hypothalamus activate cells in the midbrain, which control spinal nociceptive transmission cells by means of cells in the medulla. The activity of this modulatory system is one reason why people with apparently severe injury may deny significant levels of pain (Turk & Flor, 1999).

To date, researchers are far from understanding all the complexities of the human mind and consciousness. It is assumed that there are specific pathways in the CNS that control pain transmission, and there is evidence that these pathways can be activated by psychological factors. (The importance of psychological factors in the pain experience will be described below in Melzack and Wall's (1965) gate control theory.) The midbrain, periaqueductal gray matter, and adjacent reticular formation that project into the spinal cord via the rostroventral medulla are involved in the modulation of nociceptive signals (Fields, 1987). This pathway inhibits spinal neurons that respond to noxious stimuli.

In addition to the biogenic-amine-containing neurons, endogenous opiods peptides are present in all regions involved in pain modulation (Turk & Flor, 1999). The opiods-mediated analgesia system can be activated by electrical stimulation or by opiate drugs such as morphine. It can also be activated by nociception, stress, and suggestion. Opiods produce analgesia by direct action on the CNS and activate the nociceptive modulating system. Opiod receptors have two distinct functions: chemical recognition and biological action. Researchers hypothesize that the brain can synthesize molecules that would act at these highly specific receptor sites. A number of endogenous opiod peptides that are similar to morphine have been identified at the receptor level (Turk & Flor, 1999).

The action of the endogenous opiods is also modifiable by learning processes. Current research focuses on the classical (respondent) conditioning in humans of

stress-induced analgesia to improve the efficacy of treatment for acute and chronic pain patients (Turk & Flor, 1999).

Basic Mechanisms: Perception. The final physiological process involved with pain is perception. Today, it still remains unresolved how the neural activity of the nociceptive transmission neurons produces a subjective experience. How this comes about is obscure, and it is not even clear in which brain structures the activity occurs that produces the perceptual event. There are inherent limitations to understanding pain because it is a subjective experience. It follows then, that pain cannot be predicted. In some individuals, innocuous stimuli produce excruciating pain. In other situations, patients with severe injuries deny any significant pain.

To understand this variability, it is helpful to distinguish between *pain detection threshold* and *pain tolerance*. *Pain threshold* is a property of the sensory system and is dependent on the stimulus. It is highly reproducible across individuals and in the same individual at different times. In contrast, *pain tolerance* is not particularly reproducible; no two individuals react to nociception and pain in quite the same way. This distinction helps clarify the variability of pain. Pain tolerance is a manifestation of a person's reaction to noxious stimuli and is highly dependent on psychological variables such as behavioral, affective and cognitive factors (Turk & Flor, 1999) discussed below. Not only does it vary between different individuals in the same situation, but also the same individual may react differently in different situations.

Several factors may account for this variability. There may be injury to the nociceptive transmission system or to the activity of the modulatory system that lowers pain intensity. There may be abnormal neural activity, producing hypersensitivity that can result from self-sustaining processes set in motion by injury that may persist beyond the time it takes for the original injury to heal. This self-sustaining process may even create a situation in which pain is experienced without noxious stimulus produced by an active tissue-damaging process (e.g., neuropathic pain).

If pain were simply a sensation, these neural pain mechanisms would probably be sufficient to explain most of the clinically observable variability. However, pain is more than a sensation. The close association of the nociceptive sensory system with the function of protection of the body from damage is unique among sensory systems. It is essential for understanding pain patients that the desire to escape from or terminate the sensation be considered. If it is not unpleasant, it is not pain.

Summary and critique of the biomedical perspective. The biomedical model of pain argues that nociception, transmission of noxious signaling, modulation, and sensory registration of pain are biologically predetermined processes. This model has been criticized because pain is considered to be predominantly a product of rigid, unidirectional, straight-through (allbeit modulated) information transmission. It is not at all clear who or what interprets the signals that complete their journey from periphery to cortex (Chapman, Nakamura & Flores, 1999). Further, this model cannot explain how a sensory experience can contribute so powerfully to suffering; why pain hurts is still unclear.

2.2.2. Gate Control Theory

In the 1960s, a series of important developments changed our understanding of pain. The gate control theory (Melzack & Wall, 1965) proposed that pain was different from other sensations in that pain can be modified. Pain was considered to be a multi-dimensional phenomenon that could be influenced by afferent and efferent mechanisms. The discovery of opiate receptors by Pert and Snyder (1973) proved to be essential in fundamental research. This discovery led to the determination of a number of endogenous opiates – analgesics produced by the body – and to an increase in research addressing nociception and analgesics. Other advances were registered in the areas of pain measurement, primarily questionnaires for subjective pain experience, and in the field psychophysiology in which methods were developed to measure psychophysiological phenomenon (e.g., evoked responses).

The gate control theory of pain (Melzack & Wall, 1965) explains the specificity of pain, different types of pain, and which role psychological factors play in pain processing. The central assumption of the gate control theory is that different parts of the central nervous system are involved in the pain experience. They differentiate between three systems related to the processing of nociceptive stimulation – sensory-discriminative, motivational-affective, and cognitive-evaluative – that are all thought to contribute to the subjective experience of pain. The gate control theory proposes that a mechanism in the dorsal horn substantia gelatinose of the spinal cord acts as a spinal gating mechanism that inhibits or facilitates transmission of nerve impulses from the body to the brain on the basis of the diameters of the active peripheral fibers, as well as of the dynamic action of brain processes (Turk & Flor, 1999). It was postulated that the spinal gating mechanism was influenced by the relative amount of excitatory activity in afferent large-diameter (myelinated) and small-diameter fibers (unmyelinated nociceptor) converging in the dorsal horns. Further, it was hypothesized that activity in A-beta (large diameter) fibers tends to inhibit transmission of nociceptive signals (closes the gate), while A-delta and C (small diameter) fibers primary afferent activity tends to facilitate transmission (opens the gate). The hypothetical gate is proposed to be located in the dorsal horn, and it is at this point that sensory input is modulated by the balance of activity between large-diameter (A-beta) and of small-diameter (A-delta and C) fibers.

Melzack and Wall (1965) postulate that this spinal gating mechanism is influenced not only by peripheral afferent activity but also by efferent neural impulses that descend from the brain. According to their model, a specialized system of large-diameter, rapidly conducting fibers (called the central control trigger) activate selective cognitive processes that then influence, by way of descending fibers, the modulating properties of the spinal gating mechanism. Melzack and Wall hypothesize that the brainstem reticular formation functions as a central biasing mechanism inhibiting the transmission of pain signals at multiple synaptic levels of the somatosensory system. Simply said, this theory assumes that the sensations are not directly transmitted from the peripheral nerve endings to the brain, instead these sensations are modified up through the spinal cord, and that these sensations are

influenced by downward pathways from the brain. The experience of pain is then interpreted.

In the absence of pain, the peripheral nerve endings pick up sensations from our physical activities such as walking or touching. Without pain, the spinal gating mechanism is closed and sensations are transmitted through the neural pathways to the spinal column, through the spinal column to the brain. When the peripheral nerves are exposed to injurious stimuli, the patterning of stimulation may be so intense that they reach a certain threshold and the brain interprets the stimulus as painful. Then the gate will open and sensations of pain will be transmitted up the spinal column to the brain.

The gate control theory proposes that the large-diameter fibers play an important role in pain by inhibiting synaptic transmission in dorsal horn cells. When large fiber input is decreased, mild stimuli that are not typically painful trigger severe pain. Loss of sensory input to this complex neural system, such as in neuropathies, tend to weaken inhibition and lead to persistent pain. It is proposed that factors such as herniated disc material or tumors may exert pressure on these neural structures and also lead to persistent pain. Emotional stress and medication that affect the reticular formation may also alter the biasing mechanism and thus the intensity of pain (Turk & Flor, 1999).
Emotions, in general, can alter the pain experience through the central control mechanism. For example, anxiety or fear can exacerbate the experience of pain, while laughter or positive experiences tend to mute it (Cogan, Cogan, Waltz & McCue, 1987).

According to the gate control theory, the experience of pain is an ongoing sequence of activities that even in the early stages is modifiable through a variety of excitatory and inhibitory influences as well as the integration of ascending and descending nervous system activity. The result of this process is the expression of pain, both verbally and behaviorally, and attempt by the individual to terminate the pain. In this model, considerable potential for shaping of the pain experience is implied, because

the gate control theory invokes continuous interaction of systems such as: sensory-physiological, affect, cognition, and behavior (Turk & Flor, 1999).

The gate control theory integrates peripheral stimuli with cortical variables, such as mood and anxiety, in the perception of pain. This model postulates that both somatic and psychogenic factors have either a potentiating or moderating effects on pain perception. According to Melzack and Wall (1965) pain is not understood to be the result of depression or vice versa, but rather the two are seen as evolving simultaneously. Any significant changes in mood or in the pain experience will alter the other factors.

Since this model emphasizes the dynamic role of the brain in pain processes and perception, psychological variables such as beliefs, past experience, and other cognitive activities have been integrated in more current research and therapy. Previously, psychological processes were considered to be simply reactions to pain. The gate control theory suggested that many factors modulated the *input*.

Summary and critique of the gate control theory: Some of the physiological details of the gate control model have been challenged, and it has been charged with being incomplete. For example, large-diameter fibers may under certain conditions (e.g., inflammation) increase rather than decrease pain perception (Turk & Flor, 1999). Further, although the gate control theory provides a physical basis for the role of psychological variables in pain, it does not describe them in detail. The original gate control theory has been displaced by numerous, and more elegant models of modulation over the past three decades. Currently, the dominant model is the Diffuse Noxious Inhibitory Control (DNIC) concept (DeBroucker, Cesaro, Willer, & Lebars, 1990). Briefly, this model focuses on counterirritation, a phenomenon by which an additional noxious stimulus reduces the pain caused by the initial noxious stimulus.

Nevertheless, the gate control theory of pain has been fundamental in the conceptualization of the pain experience. The gate control theory acknowledges that

pain experiences differ in their qualities, that the overall patterning is important in how pain is interpreted, and that psychological processes are described by the central control mechanism. It helps explain the different kinds of pain that individuals experience and integrates the importance of sensory, affective, and evaluative components of the model. The gate theory has been helpful in clinical settings by suggesting techniques of pain control and explaining the way in which pain control methods work (Taylor, 1999).

2.2.3. Vulnerability-Diathesis-Stress Model

In this model, the pathogenesis of chronic pain originates in the neurobiological and psychosocial predisposing factors that precede the onset of pain. The neurobiological factors that predispose individuals to develop chronic pain are undoubtedly diverse. They include genetic factors that underlie individual differences in physiology or structure that make an individual more likely to develop chronic pain (e.g., scoliosis). These neurobiological factors also include physiological and structural abnormalities resulting from prior disease or injury (e.g., musculoskeletal pathology). Since few prospective studies have been conducted that directly address neurological predisposing factors in the development of chronic pain, our understanding of these factors is based on animal models, clinical observations, and studies of patients already suffering from chronic pain.

The psychosocial factors that predispose individuals to develop chronic pain are also likely to be diverse. Such factors probably include pain relevant personality traits (e.g., somatization, hypervigilance) and psychopathology, and especially mood, anxiety, and substance abuse disorders (Dworkin, 1997). Physical and sexual abuse and other traumatic events (e.g., emotional abuse and neglect) occurring before the onset of pain, especially during childhood, also appear to be risk factors for the development of chronic pain (Linton, 1997). In addition, the individual's prior experiences with pain may also be a psychosocial predisposing (or protective) factor, although it is possible that such experiences results in, for example, central sensitization or increased descending inhibition and could therefore be considered neurobiological predisposing factors (Dworkin & Banks, 1999). Other pain relevant

attitudes, beliefs, and behaviors that are a consequence of the individual's socialization experiences and that develop during childhood and adolescence are very likely to be psychosocial predisposing factors. For example, modeling of responses to pain and illness by significant others in childhood and adolescence is commonly thought to be an important influence on how an adult responds to a painful injury or illness (Dworkin & Banks, 1999). Unfortunately, however, virtually all existing studies of this question make it impossible to distinguish the relative effects of shared genetic and environmental influences.

In the vulnerability-diathesis-stress model of chronic pain proposed by Dworkin and Banks (1999), the neurobiological and psychosocial predisposing factors that precede the onset of pain constitute the vulnerability component of the model. This vulnerability is conceptualized as a continuum to which both the neurological and psychosocial predisposing factors contribute. Individuals therefore range from low to high in their vulnerability to the development of chronic pain.

2.2.4. Stress.

Selye (1950) described stress as a biological response to a wide range of stressors. They include physical injury, infection, and other pathology, as well as psychological stressors such as the loss of a job or death of a spouse. A current definition of stress is that homeostasis is being threatened. That is, a disruption by stressors of physiological processes such as blood sugar level and body temperature that are normally maintained at a fixed, delicately balanced set point (Chrousos, 1992).

The disruption of homeostasis by a stressor, either physical or psychological, activates programs/automatic processes of neural, hormonal, and behavioral activity aimed at restoring homeostasis. The particular programs that are activated are selected from a genetically determined repertoire of programs, which are modifiable by events such as earlier exposure to stress, and are influenced by the extent and severity of the perceived stress (Melzack, 1999).

It is proposed that the interacting neural and hormonal factors that contribute to homeostasis are so complex, that it is not surprising that the programs to reinstate homeostasis occasionally go awry. Melzack (1999) proposes that the consequence is a variety of stress-related disorders, which include several chronic pain syndromes. Recent research has been directed towards testing the hypothesis that stress may produce the conditions that give rise to some forms of chronic pain.

The underlying principle of this hypothesis is the neuromatrix theory of pain (Melzack, 1999). It proposes that pain is a multidimensional experience produced by characteristic "neurosignature" patterns of nerve impulses generated by a widely distributed neural network – the "body-self neuromatrix" – in the brain. What is interesting about this theory is that the neurosignature patterns may be triggered by sensory inputs, but they may also be generated independently of them. Pain resulting from noxious sensory inputs has been well researched (Melzack & Wall, 1996), in contrast, chronic pain syndromes, which are often characterized by severe pain associated with little or no discernible injury or pathology, are poorly understood. The neuromatrix theory provides the conceptual framework that helps researchers better understand pain from both noxious sensory inputs and pain with no pathology, as in chronic pain syndromes.

According to the neuromatrix theory, pain is produced by the output of a widely distributed neural network in the brain rather than directly by sensory input evoked by injury, inflammation, or other pathology (Melzack, 1999). The primary mechanism, the neuromatrix, generates the neural pattern that produces pain and it is genetically determined and modified by sensory experience. Its output pattern is determined by multiple influences, of which the somatic sensory input is only a part, that converge on the neuromatrix (Melzack, 1999).

Further, Melzack (1999) emphasizes that pain disrupts the body's homeostatic regulation systems, thereby producing stress and initiating complex programs to restore homeostasis. Pain, therefore, is no longer a purely perceptual phenomenon.

By recognizing the role of the stress system in pain, the scope of the puzzle of pain is greatly expanded.

Due to the complexity of the stress-regulation system, a schematic presentation of the major components will be given below. For more details regarding programs involving the cortisol system, the immune system, the homeostatic regulation please refer to, among others, Melzack (1999), Sapolsky (1992), Chrousos (1992).

Mechanisms: When injury occurs, sensory information is projected rapidly to the brain, and, in parallel with the neuromatrix activities that usually lead to pain perception, the stress system initiates the complex sequence of events to restore biological homeostasis. Activities of the injured tissues produce cytokines, which are complex molecules produced by the interaction of transformed white blood cells (macrophages) and injured tissue. These cytokines are released within seconds after injury and take part in producing a local inflammatory response. Within minutes, cytokines enter the blood stream and travel to the brain. The cytokines, together with the perception of pain – a stressor – rapidly begin a sequence of activities aimed at the release and utilization of glucose for necessary actions such as the repair or tissues and "fight or flight" responses to survive the threat to the body-self (Melzack, 1999).

Cytokines that penetrate the hypothalamus activate the hypothalamic-pituitary-adrenal (HPA) system, in which the cortisol releasing hormone (CRH → ACTH) is set free. Cortisol plays a powerful role in the stress response. At the same time, the autonomic system is activated. During the stress response, the sympathetic system predominates and produces readiness of the heart, blood vessels, and other viscera for complex action programs to respond appropriately to the stressor and to reinstate homeostasis (Chrousos, 1992).

As the stress response continues, it has a powerful impact on other systems. The immune system is suppressed, and major portions of the limbic system, which play a role in emotional, motivational, homeostatic, and cognitive processes, are activated.

Furthermore, the endogenous opiods, such as endorphins, are released within minutes. Their initial function may be primarily to inhibit or modulate the release of cortisol (Chrousos, 1992; Sapolsky, 1992). This highly simplified description does not include multiple neural and hormonal systems that take part in the stress response.

According to Melzack (1999), the stress and the pain-perception systems possess overlapping mechanisms. Injury produces information that feeds into the body-self neuromatrix that generates the output patterns that comprise the neurosignature for the perception of the extent and severity of the injury and concurrently activate the appropriate action patterns to be chosen from the available pool (Melzack & Wall, 1996). This output, together with information generated by the neuromatrixes that receive inputs from the other sensory and cognitive systems, acts on the stress regulation mechanisms that are part of the system and determines whether or not pain will be experienced or suppressed (Melzack & Wall, 1996). To prove their point, Melzack and Wall (1996) explain that individuals who undergo severe injury may not feel any pain for as long as hours, even days, afterward. Because the stress system requires about 1-4 minutes to be activated, the endorphin and other opiod substances released by stressors cannot be the determinant of the complete suppression of pain after injury. Rather, the neuromatrixes that generate sensory-discriminative and evaluative information regarding the state of the body and the circumstances of injury determine the initial activation of suppression of the pain, inflammation processes, and immune systems (Sapolsky, 1992).

Prolonged activation of the stress-regulation systems produces breakdown of muscle, bone, and neural tissue. Excessively long or intense activation of these systems, therefore, can have disastrous consequences (Melzack, 1999). They may provide the conditions for the onset of chronic pain syndromes, including fibromyalgia, and osteoporosis.

Implications of stress regulation: Psychological contribution to pain. Cortisol is released by either psychological stress or physical injury. Sapolsky (1992) has proposed that the cumulative release of pulses of cortisol is a major determinant of

pathology. All psychological stresses may contribute to the neuronendocrine processes that give rise to pain syndromes, and psychological therapies that control stress ultimately affect cortisol release and, therefore, influence the development of chronic pain. A decrease in cortisol output by psychological therapy may not by itself be sufficient to produce a major reduction in pain, but it should be part of multiple therapies that can have additive effects in decreasing the destructive effects of cortisol.

Each kind of stressor can produce physiological effects that are combined with the effects of other stressors. Sapolsky (1992) emphasizes that the stress effects of an injury can vary in severity and pattern as a function of other stresses, such as loss of self-esteem, employment, or other security symbols. Individual variation in response to injury or other stresses may be influenced by the enhancement of a given stress by (1) other concurrent stress, (2) the cumulative effect of prior stresses (determined partly by their pattern of appearance), (3) the kinds of concurrent or prior stress – that is, psychological or physical, and (4) the severity and duration of the stresses (Melzack, 1999).

The neuromatrix theory provides a reasonable mechanism whereby psychological stresses may provide that basis for chronic pain. Stressors have destructive effects on muscle, skeletal, and hippocampal neural tissue, which may become the immediate basis of pain or provide a basis for the devastating effects of later minor injuries in which the severity of pain is disproportionately far greater than would be expected from the injury (Melzack, 1999).

Melzack (1999) proposes further that psychological stress alone could become a cause of chronic pain because it produces substances that have destructive effects on body tissue. Prolonged stressful events can leave a "memory" etched into bone, muscle, and nerve tissue, just as an injury sculpts a neuronal pattern into the neuromatrix. Stress, like pain however, is a subjective experience. Threatening sensory or cognitive events may or may not be perceived as stressors, just as the sensory input from an injury may or may not be perceived as pain. Even when pain

is experienced, it may be a stressor if it implies danger and threat to survival of the self, physically or psychologically. In contrast, a major injury may evoke little or no stress if it is perceived as a successful escape from danger, such as a battlefield.

Predictors of Chronic Pain: A further important feature of chronic pain that implicates the stress system is the fact that the severity of pain during an injury or infection is a major predictor of the occurrence of subsequent persistent pain. Malenfant, Forget, Papillon, Amsel, Frigon, & Choinière (1996) found that patients with severe burns who suffer the most intense pain in the initial stages of recovery and healing are the ones most likely to have persistent pain that continues over years after full healing has occurred. Katz, Jackson, Kavangagh, and Sandler (1996) observed that patients reporting intense pain during the first 2 days after a chest operation are much more likely to report persistent chest pain 1½ years post-operative than patients who were pain-free after the operation. These and other studies seem to show, that severe pain, which is a powerful stressor, is a major determinant of chronic pain that remains after healing has occurred, when there are no obvious physical causes of the severe pain suffered by the patients. It is reasonable to assume, then, that the initial pain and stress produced changes in both the perceptual and stress systems that contributed to the abnormal output patterns of the body-self neuromatrix.

Summary: In summary, the neuromatrix theory of pain proposes that the neurosignature for pain experience is determined by the synaptic architecture of the neuromatrix, which is produced by genetic and sensory influences (Melzack, 1999). Melzack proposes further that the neurosignature is also modulated by sensory inputs and by cognitive events, such as psychological stress. It may also occur because stressors, physical as well as psychological, act on stress-regulation systems, which may produce lesions of muscle, bone, and nerve tissue, thereby contributing to the neurosignature patterns that give rise to chronic pain. Simply said, the neuromatrix, as a result of homeostasis-regulation patterns that have failed, produces the destructive conditions that may give rise to many of the chronic pain types that so far have been resistant to treatments developed primarily to manage pain that are triggered by sensory inputs. The stress regulation system, with its

complex, delicately balanced interactions, is an integral part of the multiple contributors which give rise to chronic pain.

Melzack (1999) argues that since the neuromatrix of pain places genetic contributions and neural-hormonal mechanisms of stress on a level of equal importance with the neural mechanisms of sensory transmission described in the biomedical model above, this model has important implications for research and therapy. Further, Melzack (1999) proposes that interdisciplinary pain clinics should expand to include specialists in endocrinology and immunology in order to reveal the underlying mechanisms of chronic pain.

2.2.5. Behavioral Factors

Operant learning mechanisms: A change in thinking began as Fordyce (1976) described the role of operant factors in chronic pain. The operant approach is markedly different than the biomedical model described above. This approach emphasizes the behavioral manifestations of pain, not the pain per se. It is suggested that when an individual is exposed to a stimulus that causes tissue damage, the immediate response is withdrawal and attempts to escape from the noxious sensations. This may be accomplished by avoidance of activity believed to cause or exacerbate pain, seeking help to reduce symptoms, and so forth. These behaviors are observable and consequently subject to the principles of operant conditioning.

The operant view proposes that acute "pain behaviors", such as limping to protect a wounded limb from producing additional nociceptive input, may come under the control of external contingencies of reinforcement and thus develop into a chronic pain problem (Turk & Flor, 1999). Pain behaviors (e.g., inactivity, complaining, grimacing) may be positively reinforced directly, for example, by attention from a spouse or health care providers. Pain behavior may also be maintained by avoiding undesirable activities such as work, or by escaping from noxious stimulation through the use of drugs or rest. In addition, "well behaviors" such as activity and work, may not be sufficiently reinforced, and the pain behaviors may therefore be maintained.

The pain behavior originally elicited by organic factors may exist purely, or in part, in response to reinforcing environmental factors (Taylor, 1999). This operant perspective suggests that pain behaviors may persist long after the initial cause of the pain is resolved.

This model does not focus on the initial cause of pain, instead, it emphasizes the subjective experience and the communicative function of pain behaviors. In this model, however, the psychological factors are treated as secondary, as reactions to sensory stimulation – as in the biomedical model – rather than as being directly involved in the perception of pain per se. Although operant factors undoubtedly play a role in the maintenance of disability, they do not explain the entire experience of pain. This model has been criticized for its reliance on motor pain behaviors, and its failure to consider cognitive and emotional aspects (Turk & Flor, 1999). Nevertheless, this model may prove useful in areas addressing verbal-subjective and physiological responses to pain since recent research suggests that these areas are subject to operant learning.

Respondent Learning Mechanisms: Factors contributing to chronicity that have previously been conceptualized in terms of operant learning may also be initiated and maintained by respondent conditioning. Fordyce and colleagues (Fordyce, Shelton, & Dundore, 1982) proposed that avoidance behavior does not necessarily require intermittent sensory stimulation from the site of bodily damage, environmental reinforcement, or successful avoidance of aversive social activity to account for the maintenance of protective movements. Avoidance of activities has been shown to be related more to anxiety about pain than to actual reinforcement (Linton, 1985).

At the onset of acute pain, fear of motor activities that the patient expects to result in pain may develop and motivate avoidance of activity. Nonoccurance of pain is a powerful reinforcer for reduction of activity, and thus the original respondent conditioning may be followed by an operant learning process whereby the nociceptive stimuli and the associated responses need no longer be present for the avoidance behavior to occur (Turk & Flor, 1999). Reduction of movement may be

useful in acute pain states to accelerate the healing process. Over time, however, anticipatory anxiety related to activity may develop and act as a conditioned stimulus (CS) for sympathetic activation (conditioned response, CR) that may be maintained after the original unconditioned stimulus (US, e.g., injury) and unconditioned response (UR; pain and sympathetic activation) have subsided.

Since many activities that are normally pleasurable or neutral may elicit or exacerbate pain in the acute phase, these activities are experienced as aversive and are consequently avoided. Over time, the number of activities seen to elicit or to exacerbate pain increases so that more and more activities will be avoided (stimulus generalization). Fear of pain may become conditioned to an expanding number of situations, including simple motor behaviors, as well as work, leisure, and sexual activity. In addition to avoidance learning, pain may be exacerbated and maintained due to the anxiety-related sympathic activation and muscle tension that may occur in anticipation of pain and also as a consequence of pain (Flor, Birbaumer, & Turk, 1990). This is an example in which the psychological factors may directly affect nociceptive stimulation and need not be viewed as merely reactions to pain.

Rachman and Arntz (1991) described the persistent avoidance of specific activities on hand from over and under predictions of pain. The prediction of pain promotes pain-avoidance behavior, and over predictions of pain promote excessive avoidance behavior. When the result of pain-avoidance is successful (i.e., no pain), then the over predictions remain unchecked and the process continues unchanged. In constrast, by repeatedly engaging in behavior that produces significantly less pain than was predicted, adjustments in subsequent predictions follows. Thus, the predictions become more accurate. These increasingly accurate predictions will be followed by increasingly appropriate avoidance behavior. When appropriate, all avoidance may be eliminated. These observations emphasize the importance of physical therapy to help patients progressively increase their activity levels and muscle mass despite fear of injury and discomfort associated with renewed use of deconditioned muscles.

From the conditioning perspective, the patient may have learned to associate increases in pain with all kinds of stimuli that were originally associated with nociceptive stimulation (stimulus generalization). Sitting, walking, engaging in social interaction, sexual activity, or even thoughts about these activities may increase anticipatory anxiety and concomitant physiological and biochemical changes. Subsequently, patients may display maladaptive responses to many stimuli and reduce the frequency of performance of many activities other than those that initially induced pain. the physical abnormalities often observed by chronic pain patients (e.g., distorted gain, decreased range of motion, etc.) may actually be secondary changes initiated in behavior through learning. As the pain symptoms persist, more and more situations may elicit anxiety and anticipatory pain and depression because of the low rate of reinforcement obtained when behavior is greatly reduced. In chronic pain, the anticipation of suffering or prevention of suffering may be sufficient for the long-term maintenance of avoidance behaviors (Turk & Flor, 1999).

Social learning mechanisms: Based on Bandura's (1969) concept of social learning, the acquisition of pain behaviors may occur by means of observational learning and modeling processes. That is, individuals can acquire responses that were not previously in their behavioral repertoire by observing others performing these activities. From this perspective it is believed that children acquire attitudes about health and health care, the perception and interpretation of symptoms, and appropriate responses to injury and disease from their parents and social environment. Dependent upon their learning experience then, they will tend to either minimize or overreact to symptoms. Due to the importance of the social environment, different responses to pain have been documented across different cultures (Taylor, 1999). The observation of others in pain is an event that captivates attention. This attention may have survival value, may help an individual to avoid experiencing more pain and to learn what to do about acute pain.

Expectancies and actual behavioral responses to nociceptive stimulation are based, at least partially, on prior learning history. The mechanisms behind social learning

may contribute to the understanding of the marked variability in response to objectively similar degrees of physical pathology found in clinical settings.

Summary and critique: According to Fordyce (1976), it was important to pay attention to the observable signs of pain and suffering, i.e., pain behavior, especially in pain therapy. He assumed that long after an injury had healed, pain behavior could persist due to the learning processes that had taken place at the time of injury. According to Fordyce (1976), the best way to treat chronic pain is the systematic extinction of pain behaviors and the reinforcement of healthy behaviors, i.e., behavior that is incompatible with pain behavior. This theoretical model had a large effect on pain therapy at the time and reflected a rise in the popularity of behavioral modification in the 1960s and the establishment of behavioral medicine as independent research field.

Behavioral medicine is defined as an interdisciplinary field because it addresses the integration of behavioral and biomedical sciences in the research of disease and health, and the treatment of disease. Behavioral medicine led to an increase in behavioral research with diseases that were previously considered to be caused purely by organic disorders. More than 2000 pain clinics, most of them interdisciplinary, opened in the USA between 1970 and 1990.

2.2.6. Affective Factors

Pain has been defined differently by various authors depending on their perspective. Pain can be an emotion, a sensory perception, or a behavior. According to the International Association for the Study of Pain, pain is defined as "an unpleasant sensory and emotional experience associated with actual or potential tissue damage or described in terms of such damage" (Merskey, 1986, p. 217). In comparison to earlier definitions, this definition of pain addresses the psychological component of the pain experience instead of focussing on the purely sensory perception. In addition, this definition suggests that although tissue damage can be an important part of the pain experience, it is not necessary to have tissue damage to feel pain.

Further, this definition views emotions as an integral component of the pain experience, and not just as a reaction to pain.

The affective components of pain include many different emotions, but they are primarily negative in quality. Chronic pain patients frequently report symptoms of depression, anxiety and anger.

There are several issues and controversies regarding the role of negative emotion in pain. These include: (1) the prevalence of negative emotion in patients with chronic pain, (2) causal relationships between pain and negative affect, and (3) models incorporating negative emotion and pain.

Prevalence of negative emotion in chronic pain: Depression has received the most empirical investigation. Methodological problems including differences in the definition of depression, populations sampled, and measurement account for a variability in prevalence, with estimates ranging from 10% - 100%. Despite this variability in the absolute prevalence of depression, the estimates have almost universally indicated higher rates of depression in patients with chronic pain when compared to the general population (Robinson & Riley, 1999). Since depression estimates of 30-54% are typical in clinical settings, depression is decidedly a significant issue.

Anxiety, too, has received considerable attention in the literature (Robinson & Riley, 1999), and suffers from similar methodological problems as depression (cf. above). In addition, the range of diagnostic criteria (e.g., Diagnostic and Statistical Manual of Mental Disorders (DSM-IV), American Psychiatric Association, 1994; International Classification of Mental and Behavioral Disorders (ICD-10), World Health Organization, 1993) for anxiety disorders is much broader. Nevertheless, Atkinson, Slater, Patterson, Grant, and Garfin (1991) compared patients with low back pain to a matched sample of pain-free men and found that the chronic pain group had a significantly higher lifetime prevalence rate of major anxiety disorder (31% vs. 14%). Other studies (e.g., Gaskin, Greene, Robinson, & Geisser, 1992) found that patients

with a variety of chronic pain conditions report anxiety levels significantly greater than published norms and that a significant amount of the variance in pain reports can be explained by anxiety. In clinical populations, anxiety appears to play an important role.

Recent attention has been given to a anxiety-related concept termed "fear-avoidance". According to Waddell and colleagues (Waddell, Newton, Henderson, Somerville, & Main, 1993), this construct is based on learning theory models of the acquisition and maintenance of pain behaviors (cf. above). The pain behavior in particular is "avoidance of painful activities". This avoidance of activity is postulated to result in chronic pain syndromes characterized by a cycle of decreased activity, deconditioning, loss of self-efficacy, fear, and negative affect, leading to further avoidance of pain-related activity (Robinson & Riley, 1999). The questionnaire evolving from this construct (PASS. Pain Anxiety Symptoms Scale; McCracken, Gross, Sorg, & Edmands, 1993) addresses the issues of fearful appraisals and cognitive, behavioral, and physiological components of pain-related anxiety.

Anger, in comparison to depression and anxiety, has received far less attention in the pain literature. Little evidence exists as to whether the prevalence of anger is a clinically significant factor in patients with chronic pain. Unlike other negative affective states, anger lacks a specific diagnosis. Some data (cf. Gaskin et al., 1992) indicate that patients with chronic pain report levels of anger significantly higher than the published norms on the State-Trait Anger Expression Inventory (Spielberger, 1985). Although the scope of anger as a diagnostic entity remains unclear, the consequences of anger in patients with pain appear to be significant and to contribute to treatment obstacles and other negative affective experiences (Robinson & Riley, 1999).

Measurement of negative emotion in patients with chronic pain. Some controversy exists as to whether the assessment of negative emotion in patients with chronic pain poses methodological problems due to the overlap with somatic symptoms. Some researchers (Novy, Nelson, Frances, & Turk, 1995) argue that patients with

chronic pain may have artificially high levels of depression because of the somatic symptoms of the pain condition. Geisser, Roth, & Robinson (1997) examined this issue with the Beck Depression Inventory (BDI). Discriminant analyses showed that the BDI, with and without somatic items, could predict classification of depression through structured interviews of the DSM-IV. These results suggest that the concerns about overlap of somatic symptoms in depression and chronic pain are not empirically founded.

The same concerns raised about overlap and the assessment of depression apply to the assessment of anxiety, though there have been no direct tests of the hypotheses that chronic pain symptoms artificially increase the diagnosis of anxiety. Practically no studies have addressed the issue of overlap in chronic pain and anger.

In addition to the symptom overlap issue in chronic pain and negative affect, researchers have questioned whether depression, anxiety, and anger correlate strongly enough with each other to represent a higher order factor that has been termed in the literature "psychological distress" (Brown, Robinson, Riley, & Gremillion, 1996). A number of studies have employed measures of more than one type of negative affect invariably show a correlation between the affective measures (e. g., Brown et al., 1996, Gaskin et al., 1992). Regression analyses with other variables have shown that both within and across studies, separate variance is accounted for by each of the negative affect constructs, although the variance accounted for is rarely impressively large for any single affect construct. The experience of chronic pain has far-reaching effects, including concerns for health, loss of avocational and vocational involvement, financial stressors, loss of role identity, and legal complications, all of which can differentially affect an individual and manifest itself in varying amounts of depression, anxiety and anger. Negative emotion, like pain, is multivariate, with the expectation that its components are likely to intercorrelate (Robinson & Riley, 1999). The utility of assessing each component separately in comparison to an approach that measures a global psychological distress construct is not yet fully understood (Robinson & Riley, 1999).

Negative emotion as a predictor of pain. As mentioned above, a number of studies have shown a concurrent relationship between negative emotion and levels of pain in correlational designs (Brown et al., 1996; Gaskin et al., 1992). Other studies have shown that negative affect is a significant negative predictor with respect to spine surgery outcome (Hasenbring, Marienfeld, Kuhlendahl, & Soyka, 1994), multidisciplinary treatment for pain, and conservative therapies. Others have suggested that negative affect may mediate the relationship between pain, impairment, and disability (Banks & Kerns, 1996). Further, studies have shown that fear-avoidance alone accounted for a 66% correct classification of which acutely injured back pain patients would become chronic at a 12-month follow-up. To summarize, these studies demonstrate that the co-occurrence of negative affect, whether causally linked to injury status or a consequence of injury, are predictive of a number of key variables, including treatment efficacy (Robinson & Riley, 1999).

2.2.7. Cognitive Factors

Not only do patients with pain tend to believe that they have limited ability to exert any control over their pain, but pain patients often have negative expectations about their own ability to control motor skills without pain (Turk & Flor, 1999). These negative appraisals of the situation and personal efficacy may reinforce the experience of demoralization, inactivity, and overreaction to nociceptive stimulation. These cognitive appraisals and expectations are postulated to have an effect on behavior that leads to reduced activity and that may contribute to increased psychological distress and physical deconditioning.

Reesor and Craig (1988) showed that the primary difference between chronic pain patients who were referred because of the presence of many "medically incongruent" signs and those who did not display these signs was *maladaptive thinking*. Interestingly, there were no significant differences between these groups on the number of surgeries, compensation, litigation status, or employment status. These maladaptive cognitive processes may amplify or distort the experience of pain. Further, Turk and Rudy (1992) argue that the cognitive activity of chronic pain

patients may contribute to the exacerbation, attenuation, or maintenance of pain, pain behavior, affective distress, and dysfunctional adjustment to chronic pain.

The injury that may have initiated the original report of pain plays an increasingly smaller role over time, although secondary problems associated with deconditioning may exacerbate and serve to maintain the problem. Inactivity leads to increased focus on the preoccupation with the body and pain, and these cognitive-attentional changes increase the likelihood of misinterpreting symptoms, of overemphasizing symptoms, and of perceiving oneself as disabled (Turk & Flor, 1999). Reduction of activity, fear of reinjury, pain, losing one's job, and an environment that supports the "pain patient" role can all impede alleviation of pain, successful treatment, and reduction of disability.

According to cognitive theories, all individuals respond to situations in part based on their subjective representations, called schemata. In chronic pain patients, individuals also respond to injury based on their idiosyncratic schemata. When confronted with new stimuli – an injury – the individual engages in what is termed "meaning analysis" that is guided by the schemata that best fits the attributes of the stimuli (Cioffi, 1991). Incoming stimuli are interpreted, labeled, and acted on, based on the patients' schemata.

Further, beliefs about the meaning of pain and one's ability to function despite discomfort are important aspects of the cognitive schemata about pain. These representations are used to construct causal, covariational, and consequential information about the pain symptoms (Turk & Flor, 1999). For example, a cognitive schemata that one has a very serious debilitating condition, and that disability is a necessary aspect of pain, that activity causes further pain, and that pain is an acceptable excuse for neglecting responsibilities, then the most likely response will be maladaptive (Turk & Flor, 1999). As described above, through stimulus generalization, pain patients may increasingly avoid activities, and as a consequence they become more physically deconditioned and more disabled.

Factors that may effect an individual's ability to cope with pain include beliefs, appraisals, and expectations about pain, the ability to cope, the social support system, the employer, and the health care provider. These factors also influence patients' investment in treatment and acceptance of responsibility for treatment outcome.

To conclude, interrelated sets of cognitive variables, including thoughts about the controllability of pain, attributions about one's own ability to use specific coping responses, expectations concerning the possible outcomes of various coping efforts, and common erroneous beliefs about pain and disability influence the experience of pain (Turk & Flor, 1999).

2.2.8. Summary: Biopsychosocial Model of Pain

Historically, the biopsychosocial model began with Melzack and Wall's (1965) proposed Gate Control Model. For the first time, pain was seen as more than a simple alarm message arriving at the cerebral cortex like a radio signal exciting a beeper. Melzack and his colleagues speculated about the nature and location of higher order processes in the brain. To review, they hypothesized that the neospinothalamic projection system mediates pain sensation, whereas reticular and limbic structures determine the motivation and negative effect necessary to initiate action. Unspecified neocortical processes match input with prior experience and with the neospinothalamic, reticular, and limbic systems (Chapman, Nakamura & Flores, 1999). This elaboration represented and encouraged a growing openness for discussion of the brain's influence on the body.

The current biopsychosocial model of chronic pain involves three major factors (1) integrated action, (2) reciprocal determinism, and (3) evolution (Turk, 1996). According to Turk (1996) integrated action emphasizes that chronic pain does not occur in a vacuum, that is, biological, psychological, and social elements are integrated to color the pain experience. For example, a male patient's site of injury (lower back), emotional state (anger for having to work overtime), and the manner in which he expresses his pain to others based on social and cultural beliefs (men

don't cry), all affect how he will give meaning to his pain. The second factor, reciprocal determinism, proposes that biological, psychological, and social factors can influence each other. To expand on the example above, the physiological changes, stemming from pain (increased sympathetic arousal), can affect emotional aspects of the pain experience (increased anger); and these consequently influence his response to the environment (hostility towards family members), which can further alter his physiological state. Finally, the concept of evolution states that the patient's pain experience is not a static condition, instead it is constantly changing to adapt to the new biological, psychological and social circumstances. An important aspect of this model, is that when we observe, for example, a man in pain at any given point, we are receiving only a snapshot of his experience. His condition will most likely change, after he has had some relief from the pain through analgesic medication, after he receives comfort from his family, and after he has talked to his supervisors.

From what we understand today, the biological factors are most important in initiating, continuing, and modulating pain in the acute stages. Psychological factors seem to play a role in shaping the individual's understanding of resulting physiological cues and determining consequent behavior. Psychological factors include beliefs about cause of onset, as well as a sense of coherence (Antonovsky, 1987). Both of these factors will be addressed in detail below. The social and cultural context further influence how an individual acts based on memories and prior learning. For example, Richard (1988) showed that children of chronic pain patients visit the school nurse more often and complain more frequently than children of healthy individuals.

The biopsychosocial model has spawned research in many different areas. Currently in research and in the clinical setting attention has been turned toward the cognitive aspects of behavior change. Most multidisciplinary pain clinics include cognitive-behavioral techniques such as reframing of certain painful stimuli into more benign sensory information rather than interpreting the stimuli as signals of progressing disease; and identifying, challenging, and altering automatic thoughts about pain

(e.g., my pain will prevent me from enjoying my life). These techniques illustrate the power of altering beliefs in order to sustain behavioral change over time.

The goals of cognitive-behavioral treatment for chronic pain are (1) to develop and bolster patients' beliefs that they can function adaptively in everyday life and manage pain, and (2) to teach patients skills for effectively handling future pain-related challenges (Bradley, 1996). Often patients believe that their ability to enjoy life is destroyed by pain; they may think that they may never enjoy their hobby again, or that they have lost their ability to perform everyday tasks. Few patients, however, realize that their thoughts, emotions, and behaviors constantly influence the pain experience. By increasing their awareness of and increasing their ability to influence (both positively and negatively) their pain through thinking and behavior, patients can feel more in control over their lives, and they may be better able to see alternatives other than becoming increasingly disabled.

Summary and critique. The biopsychosocial model focuses on the dynamic interplay between the biological, psychological, and social dimensions that seem to perpetuate chronic pain (Turk, 1996). Whereas biological factors are believed to initiate a physical disturbance, psychological factors appear to influence pain perception and experience, and social factors mediate the behavior exhibited by the patient in response to the pain. Stress can indeed influence hormonal levels and immune function by way of biological pathways (Andersen, Kiecolt-Glaser, & Glaser, 1994; Flor, Turk, & Birbaumer, 1985; Turk, 1996), contributing to chronic illness, as well as to emotional distress. As a result, the patient may have decreased motivation to participate in exercise or work behavior that would prevent atrophy of an injured area (Gatchel, 1996). The subsequent decrease in physical capacity may then pose a threat to the emotional well-being of an individual and may lead to further psychological distress (Taylor, 1999). The symptoms may acquire the significance of demonstrating a sense of helplessness with the intent of securing assistance or release from responsibilities from an external source (i.e., amplification of physical and/or emotional symptoms as a result of secondary gain issues.

An important difference between the biopsychosocial model and the biomedical model is that the biopsychosocial model does not try to cure the pain. The pursuit of a cure has left many patients (and physicians) frustrated and disappointed. Instead, the biopsychosocial model focuses on enabling patients to cope more effectively with their pain and improve daily functioning. This could mean that patients are frequently confronted with pain as they maintain their pre-pain activity level, instead of letting pain become the determining factor which could lead to increased inactivity in an attempt to avoid pain. Although these cognitive-behavioral treatments have provided relief for some patients, they have failed to have a universal positive effect. This implies that the biopsychosocial interventions can and must be optimized in order to help more patients better cope with their pain. Another critique regarding the biopsychosocial model is that it deals in generalities and offers no explanation of how psychosocial factors affect the brain and the body. Because current understanding of psychological and social influences is based primarily on overt behaviors, it is possible only to make assumptions about the mental processes that evoke these behaviors. Further, some researchers in this field claim that no solid theoretical basis exists to explain the mechanisms behind chronic pain. Although psychologists have broken free of the biomedical model, they have failed to provide a better theory (Chapman, Nakamura, & Flores, 1999).

2.3. COMORBIDITIES

2.3.1. Personality Factors

Pain and personality factors pose a complex field for researchers and clinicians alike. Patients with neurotic tendencies may worry about minor painful physical complaints long after the tissue pathology has healed. Patients with recurring or long-standing depression may seem hopeless and helpless when is comes to taking an active part in their own treatment. Patients with personality disorders (e.g., borderline) may demand immediate attention or may suddenly break off all clinical contacts. To deal with such challenges, physicians and researchers have developed and refined a number of approaches to better understand and assess personality factors in chronic pain.

The conceptual background for the assessment of personality factors and chronic pain can be divided into three main groups: (1) Psychodynamic theories, (2) trait theories, and (3) biopsychosocial theories. Psychodynamic theories maintain that deep-rooted unresolved personality conflicts can either serve as the basis for persistent pain or complicate the management of chronic pain. Freud was one of the first to recognize the connection between pain and underlying emotional conflicts. According to his theories, persistent pain is viewed as an emotional response to an actual loss or injury. Because he viewed perceived loss as critical to the development of persistent pain, Freud saw parallels between chronic pain and mourning. An important component of Freud's model of pain was his concept of "conversion", that is, the idea that emotional pain can be expressed through physical mechanisms. Further, Freud postulated that individuals with personality attributes that did not allow for the expression of emotional pain through emotional symptoms are likely to "convert" their emotional pain to somatic pain symptoms. The specific pain symptoms that would occur in such cases were considered to be symbolic of the underlying emotional issues (Weisberg & Keefe, 1999).

An historically important article "Psychogenic Pain and the Pain-prone Patient" (Engel, 1959) further discussed the various meanings of persistent pain. Engel (1959) argued that although pain may have a physical basis, the individual's

interpretation of that pain is a psychological phenomenon. According to Engel (1959), pain may serve several important functions. One, pain may provide a means of absolving one of guilty feelings by turning the suffering inward. Second, a focus on pain may enable an individual to displace attention from aggressive feelings that she or he is unable to express directly. Finally, persistent pain may be related to a lifelong history of suffering and defeat. Engel (1959) also found relationships between persistent pain and psychiatric diagnoses like hysteria, depression, and hypochondria. Today, Engel's (1959) perspectives are important for two reasons. First, they underscored the notion that pain is a complex phenomenon. Second, they maintained that psychodynamic formulations are important not only in understanding pain due to hysteria or psychiatric disorders, but also in understanding how people adjust to pain due to injuries or disease (Weisberg & Keefe, 1999).

Trait theories. According to trait theorists, long-standing personality traits or dispositions can have a strong influence on how an individual responds to the onset, persistence, and treatment of pain. In the 1970s, Sternbach (1974) began to question whether, for example, neuroticism predisposed individuals to chronic pain, or the reverse. Persistent pain is an ambiguous phenomenon that has no obvious meaning and does not serve a protective function. Sternbach argued that, in response to such an ambiguous situation, the meaning that a person ascribes to her or his pain could be very important since it reflects the individual's personality and past experiences.

Biopsychosocial models. The biopsychosocial model maintains that personality traits and dispositions interact with biological factors to determine how one responds to pain. For example, an individual's back "goes out". The biological event may initiate and maintain a psychological reaction, such as anger or frustration. The psychological response may, in turn, be affected by the person's social environment. That is, family and friends may be overly solicitous and provide excessive attention or sympathy to the reported pain. These social responses may lead the individual to avoid getting up or taking on previous responsibilities, which can result in muscle weakness and deconditioning – biological factors that can perpetuate the back pain.

When viewed from this perspective, a single event (a back "goes out") can precipitate a cascade of biological, psychological, and social responses that can interact to exacerbate and maintain pain. This model is especially relevant to understanding chronic pain.

Another model which has been applied to the understanding of pain is the diathesis-stress-model. According to this model, illness develops as an interaction between an underlying biological or genetic substrate (diathesis) and the expression of that substrate under certain conditions (stress). This model has been successfully applied to medical illnesses such as diabetes, ulcerative disease, and heart disease, as well as to psychiatric conditions that include depression, substance abuse and schizophrenia (Weisberg & Keefe, 1999). Further, the diathesis-stress model has been proposed as an explanation for why some individuals develop chronic pain disorders while others do not (Flor & Turk, 1984). According to the diathesis-stress model, chronic pain disorders are a function of the interaction between the individual's premorbid biological and psychological predispositions (diathesis) and the challenges or stressors (stress) that the individual faces as the result of pain. The diathesis includes the individual's personality strengths and vulnerabilities, and the stress includes the biochemical and nociceptive changes that occur at onset of the pain disorder. Flor, Turk, & Birbaumer (1985) argued that poor coping resources for managing stressful situations, coupled with depressed mood, place individuals at risk for developing excessive muscle-tension responses to pain. In fact, studies with low back patients have shown that only those individuals who are depressed, worried, and emotionally affected by their pain are likely to show high levels of low back muscle tension in response to stress (Flor, Turk, & Birbaumer, 1985).

Empirical studies examining the relationship between personality and chronic pain.
The body of research examining the relationship between personality and chronic pain can be divided into two basic categories: (1) studies that examine personality traits and (2) studies that examine personality disorders.

Personality traits and chronic pain. Numerous studies have examined personality traits in patients who have chronic pain. This following section reviews the instruments used, the descriptive studies identifying common personality traits, and predictive studies examining the relationship of personality traits to treatment outcome and the development of chronic pain.

Instruments. Over the last five decades, psychologists have developed a number of standardized psychological test instruments for assessing personality traits. The basic features of these instruments are that they are self-administered paper-and-pencil tests with standardized scoring based on a normative population. These tests are psychometrically strong, showing good evidence of reliability and validity. Much of the research on personality traits in chronic pain patients has used the first versions of the Minnesota Multiphasic Personality Inventory (MMPI, Hathaway & McKinley, 1943). When scoring the MMPI, the interpreter looks at the pattern of the three validity scales and the ten clinical scales (e.g., Hypochondriasis, Hysteria, Depression). Another standardized test that has been used in personality assessment of pain patients is the NEO-PI (Neuroticism, Extroversion, Openness Personality Inventory; Costa & McCrae 1985). It is a self-report inventory developed to assess a variety of interpersonal and intrapsychic functions in healthy individuals. The five domains of personality (e.g., Neuroticism, Extroversion, Openness, Agreeableness, Conscientiousness) were empirically derived through factor analyses and demonstrate good reliability and validity across a variety of populations.

Descriptive studies. Descriptive studies that have attempted to identify personality traits that are common in patients having chronic pain have used the MMPI. One of the first studies conducted by Hanvik (1951) found significant differences between low back pain patients with pain of physical origin versus pain of psychological origin on the scales impulsivity (Pd), anxiety (Pt), odd thinking (Sc). He then developed a Low Back Pain Scale to distinguish between functional and organic pain. This scale received considerable attention, since it purported to provide a simple means of discriminating functional from organic pain and led to increased use of the MMPI in low back pain assessment. Hanvik (1951) also described the "conversion-V"

personality structure, i.e., elevated scores on Hypochrondriasis (Scale 1) and Hysteria (Scale 3), with lower scores on Depression (Scale 2) form a "V" pattern on the first three scales of the MMPI. Sternbach (1974) in his classic text *Pain Patients: Traits and Treatments* found four common patterns on the MMPI: (1) Hypochondriasis (Hs) and Hysteria (Hy) reflecting excessive somatic concerns; (2) "reactive depressive" show elevated scores on Hypochondriasis (Hs), Hysteria (Hy) and Depression (D), reflects patients that are often depressed and anxious, and, according to Sternbach (1974), benefit the most from treatment; (3) conversion V, described above; (4) "Psychopathic deviate" reflects patients who tended to be angry and manipulative and often acted out, using their physical symptoms to get needs met. Although subsequent studies have shown mixed support of the personality traits described above, these studies provided the foundation for current research and treatment.

More recent research used the NEO-PI (cf. Wade, Dougherty, Hart, and Cook, 1992) to differentiate between the four subgroups previously found by Sternbach (1974) have determined that only Neuroticism could differentiate any of the MMPI subgroups. In general, Wade and colleagues (1992) concluded that chronic pain patients and normal, well-adjusted adults show similar responses on the NEO-PI. There is no conclusive evidence that chronic pain patients fit into any one profile, as previously believed, although some traits may be common between individuals.

Predictive Studies. These studies attempt to predict the outcome of treatments for chronic pain. The research design of these studies involves administering a personality test (e.g., MMPI) prior to treatment, and then following up with patients at a later time (e.g., immediately after surgery, or 6 months later). One early study (Wiltse and Rocchio, 1975) that used the MMPI to predict the outcome of low back surgery found that high scores on the Hypochrondriasis (Hs) and Hysteria (Hy) scales predicted poorer surgery outcome. Waring, Weisz, and Bailey (1976) found contrary results; their results indicated that the MMPI was not useful in predicting surgical outcomes.

Most recent research has focused on the question as to which comes first, the personality traits that lead to chronic pain or chronic pain that leads to certain changes in personality traits. Despite repeated attempts to gain a better understanding of the temporal relationship between chronic pain and personality, the relationship remains enigmatic. Bigos et al. (1991) conducted a large predictive study of acute back pain in an industry setting (Boeing aircraft manufacturing plant) over 4 years. Data analyses revealed that individuals who scored high on the MMPI scale Hysteria (Hy) were twice as likely to develop back pain than participants scoring low on the Hysteria scale. However, the overall elevations of the scores on the MMPI scales were lower than those found in other studies of chronic pain. Other important predictors of the onset of back pain found in this study included Hanvik's Low Back Pain scale and job dissatisfaction.

Hansen, Biering-Sorenson, and Schroll (1995) also conducted a prospective study. They gave the MMPI to a cohort of Danish men and women (N = 400) over a 20-year period. The MMPI was given to participants at age 50 and 60 during a routine health examination. Participants were questioned about the presence or absence of back pain over a 10-year interval. Results indicated that MMPI scale elevations on Hypochrondriasis (Hs), Hysteria (Hy), Depression (D), at ages 50 and 60 were associated with low back pain during the following decade. In addition, elevated MMPI scores at age 60 were associated with back pain during the preceding decade, from ages 50-60. These results are interesting in that they suggest a relationship between certain personality traits and the development of pain complaints. However, this study does not provide a definitive test of which factor came first, back pain or MMPI scale elevations.

Gatchel, Polatin, and Mayer (1995) compared the ability of the MMPI, the Structured Clinical Interview for DSM-III-R (SCID; Spritzer, Williams, Gibbon, & First, 1988), and the Structured Clinical Interview for DSM-III-R Personality Disorders (SCID-II; Spitzer et al., 1988) to predict subsequent pain and disability. 421 participants were recruited within 6 weeks of pain onset and completed a battery of tests including MMPI, SCID, SCID-II. At a one-year follow-up, participants' work status was

classified as either "working/in school", "not working due to pain", "not working, unrelated to pain". Data analyses revealed that participants who initially scored higher on MMPI Hysteria (Hy) scale were much more likely to be disabled from work than subjects scoring low on this scale. Other important predictors were gender, filing for worker's compensation, and initial scores on pain intensity. Regression analyses revealed that the MMPI Hysteria scale scores, combined with gender, worker's compensation, pain intensity correctly classified outcomes for over 90% of the patients treated. Contrary to other studies, Gatchel and colleagues (1995) found that depression and substance abuse were not predictive of outcome. Further, Gatchel and colleagues (1995) assert that their study supports the belief that the chronicity of pain disability results in pathology, rather than psychopathology resulting in chronic pain disability.

Summary and critique. Psychological tests can provide a reliable and standardized method of assessing personality traits in patients with chronic pain. Past research has most often used the MMPI and descriptive studies using it have identified a number of common profiles in diverse populations of patients experiencing chronic pain. However, the predictive utility of the MMPI has been questioned for two primary reasons. First, the results of predictive studies of personality traits in chronic pain patients have been inconsistent. Although some studies found a relationship between certain personality traits (e.g., hypochondriasis, hysteria, and depression) and treatment outcome, other studies have not found evidence for such relationships. Second, some researchers suggest a relationship between high scores on the scales measuring hypochondriasis and hysteria and neuroticism (Love & Peck, 1987).

2.3.2. Personality Disorders and Chronic Pain.

The DSM-IV lists two criteria that must be satisfied in order to classify personality traits severe enough to be a disorder. First, the individual's intrapsychic and interpersonal functioning must be significantly different from that of his or her society or culture. Second, these characteristics must be inflexible and pervasive (Weisberg & Keefe, 1997). Personality disorders, by definition, develop during

childhood and become apparent in adolescence or early adulthood (American Psychiatric Association, 1994). They reflect long-standing patterns of maladaptive behaviors, thoughts, and emotions with symptoms severe enough to interfere with the individual's daily functioning.

Diagnostic methods for assessing personality disorders. Traditionally, diagnosis of personality disorders was made by a mental health professional who conducted a clinical interview with the patient. Because this method fails to show sufficient interrater reliability, structured and semistructured interviews have been developed to assist in the diagnosis of both clinical disorders and personality disorders in psychiatric research. Current research relies on the advantages of the semistructured interview, the advantages are increased interrater reliability and construct validity through structured questions, i.e., the structured questions have been demonstrated to have high factor loadings on specific DSM criteria. In addition, the clinician can ask follow-up questions, depending on the patient's response to the structured question. The primary disadvantages of these semistructured interviews is that they are time inefficient and psychologically invasive in that they inquire about the entire range of cognitive, behavioral, interpersonal, and intrapsychic function. Briefer, less invasive measures are currently being developed and their utility is being tested (Weisberg & Keefe, 1999). In addition to the semistructured interviews mentioned above (e.g., SCID, SCID-II), the Semistructured Interview for DSM-IV Personality Disorders (SIDP-IV; Pfohl, Blum & Zimmerman, 1995) and the Personality Disorder Evaluation (PDE; Loranger, Lehmann-Susman, Oldham, & Russakof, 1985) have been used in recent psychiatric research.

Descriptive Studies. It is important to bear in mind that the overall base rates for all of the personality disorders among the U.S. population are unknown but believed to be relatively low, i.e., 0.5% for paranoid and avoidant personality disorder to 2-3% for histrionic and antisocial personality disorder. The first large-scale study of personality disorders in chronic pain patients was conducted at the University of Miami Comprehensive Pain and Rehabilitation Center (Fishbain, Goldberg, Meagher, Steele, Rosomoff, 1986). 283 chronic pain patients were interviewed with semi-

structured interview measures that yielded DSM-III personality disorder diagnoses. Results indicated that 59% of chronic pain patients met criteria for a personality disorder diagnosis. Dependent personality disorder was the most frequent disorder (17.4%), followed by passive-aggressive (14.9%) and obsessive-compulsive personality disorders (6.7%). This study was important in that it was the first to use rigorous operational criteria to make DSM-III Axis I (clinical disorders) and Axis II (personality disorders) diagnoses in chronic pain patients (Weisberg & Keefe, 1997).

Another research team (Polatin, Kinney, Gatchel, Lillo, & Mayer, 1993) interviewed 200 chronic pain sufferers at the time of entry into a comprehensive pain and rehabilitation program. The measure used was the Structured Clinical Interview for DSM-III-R (SCID and SCID-II, Spitzer et al., 1988). This instrument evaluated the current and lifetime incidence of both Axis I (clinical disorders) and Axis II (personality disorders) disorders. Results showed that 98% of the chronic pain patient met criteria for at least one lifetime Axis I diagnosis. 97% of the patients met the criteria for a somatoform pain disorder, followed by depression as the most common lifetime (64%) and current (45%) Axis I diagnosis. This study also examined the incidence of Axis I disorders that developed after the onset of pain and found that all of the patients with somatoform pain disorders and 29% of the patients with major depression developed these disorders after pain onset. Data analyses of Axis II revealed that 51% of patients met criteria for one personality disorder and 30% met criteria for more than one personality disorder. Paranoid personality disorder was the most common Axis II diagnosis (33%), followed by borderline (15%), avoidant (14%) and passive-aggressive (12%) personality disorders.

Predictive Studies. Gatchel and his colleagues (Gatchel, Polatin, Mayer, & Garcey, 1994) examined the role of clinical (Axis I) and personality (Axis II) disorders by comparing pretreatment SCID and SCID-II diagnoses of 152 patients who returned to work versus those who did not return to work following a functional restoration program. Results were consistent with other studies (cf. Fishbain et al., 1986; Polatin et al., 1993) in that 58% of patients met criteria for Axis II personality disorder. The

most common personality disorders found in both those patients who returned to work and those that did not return to work were paranoid personality disorder, passive-aggressive personality disorder, and borderline personality disorder. There were no significant differences between the groups on any of the personality disorders. More important, there were no significant differences in the prevalence of either Axis I or Axis II disorders between the patients who successfully returned to work and those who did not return to work. The authors interpret these results to mean that if treatment addresses both clinical psychiatric symptoms and personality issues, psychopathology need not interfere with successful treatment outcome, concluding returning to work (Weisberg & Keefe, 1999).

Summary and critique. In addition to the pain prone personality discussed above, other individual differences may prove to be important. Specifically, certain chronic pain patients have physiological stereotype responses to stress that aggravate particular groups of muscles exacerbating the pain. For example, patients suffering from myofascial pain dysfunction syndrome (a set of disorders in which the chronic pain originates within the head or neck muscles) show increased activity in particular facial muscles in response to stress (Gerber & Hasenbring, 1999). These and other findings suggest the important relationship between stereotypic bodily responses and stress. Pain management focuses on teaching patients to recognize sources of stress and help them learn to cope with it in more productive ways.

Based on the review of the literature addressing pain and personality, several conclusions can be made. First, the assessment of personality seems to be useful in identifying personality traits and personality disorders that may potentially influence the course and treatment of chronic pain. Pain treatment programs are likely to improve when they address individual personality differences in treatment decisions. Second, personality disorders occur at a higher rate in the chronic pain population than in the general population (Weisberg & Keefe, 1999). Studies have shown that chronic pain patients have psychiatric comorbidities that include both clinical symptoms, such as depression and anxiety, and personality traits. It is important to note that a causal relationship between personality traits or disorders and chronic

pain has yet to be established. Researchers and clinicians have posited that certain personality styles, such as histrionic, dependent, and depressive traits, predispose an individual to the development of chronic pain. This hypothesis has been more recently replaced with the diathesis-stress model (Weisberg & Keefe, 1999), in which certain traits that are normally under control of the individual's defensive structure become exacerbated under the stress of an acute injury, and that, when poorly managed, result in a personality disorder. Further large-scale prospective studies will be able to further our understanding of the relationship between personality traits and disorders and chronic pain.

2.3.3. Negative Emotion and Chronic Pain.

As described above, clinical studies have shown that chronic pain and negative emotion are frequently associated, with comorbidity documented to a varying degree depending on the specific pain condition, clinical sample studied, and dimension of negative emotion measured. The temporal relationship between pain and emotion remains unclear since few longitudinal studies have addressed this issue. Nevertheless, the nature of these relationships can generally be expressed in four different statements: (1) negative emotion increases somatic sensitivity; (2) negative emotion causes some pain; (3) negative emotion can result from the experience of chronic pain; (4) pain and negative emotion are concomitant constructs because of similar biological foundations (Banks & Kerns, 1996; Fishbain, Cutler, Rosomoff, & Rosomoff, 1997).

Negative emotion increases sensitivity. As described above in the Gate Control Theory, Melzack and Wall (1965) hypothesized that an individual's physiological perception of pain is modulated by his or her emotions and cognition, with depression and pain modulated by a similar process in the periaqueductal grey area of the dorsal horn. Some mood induction studies have supported this hypothesis, for example, Salovey & Birnbaum (1989) found increased reporting of aches and pains and decreased tolerance for experimentally induced pain. Depressed patients also tend to interpret events negatively and are more likely to interpret a given sensation as painful (Pennebaker, 1982). Other researchers, for example, Geisser, Gaskin,

Robinson, & Greene (1993) failed to find a relationship between the BDI and pain threshold or pain tolerance in patients with arthritis and fibromyalgia, using a cold pressor paradigm.

Pain is caused by negative emotions. Some researchers have argued that chronic pain is due to an underlying depressive disorder, particularly when adequate physical findings are absent. Blumer and Heilbronn (1981), based on associations between past or family history of depressive disorder, proposed that chronic pain reflects a manifestation of a muted depressive state. They defined a pain-prone disorder that was associated with specific clinical, psychodynamic, and genetic characteristics.

It has been speculated that anxiety, and, to a lesser extent, anger may be responsible for the development and maintenance of muscoskeletal disorders. For example, Flor and Turk (1989) propose that increased somatic reactivity such as increased skeletal muscle tension may lead to pain. Further, Burns, Wiegner, Derleth, Kiselica, and Pawl (1996) found that anger-induced stress produced increased muscle tension, which predicted greater pain intensity in chronic back pain patients. This effect was specific to anger in that a measure of depression that was significantly correlated with pain was not associated with increased muscle reactivity.

Negative affect occurs as a result of chronic pain. This statement implies that negative emotion is a frequent psychological reaction to chronic pain. This suggestion has intuitive appeal, considering the physical and social limitations chronic pain patients experience. This implies the existence of unique cognitive and/or behavioral responses to pain that have a tendency to develop into depressive symptomatology (Robinson & Riley, 1999). A number of cognitive responses have been implicated in the risk for negative emotion in chronic pain patients, including attributional style, catastrophizing cognition, negative self-image, and beliefs about pain (Robinson & Riley, 1999). Consistent with behavioral models of depression, increased pain and related somatic symptoms reduce the ability to engage in activities that had been sources of positive reinforcement in the past. In addition, these activities are now accompanied by pain and are, therefore, aversive. The

chronic pain patient then reduces his or her range of instrumental activities further because of physical impairment or fear of pain or further injury.

Pain and negative emotion are concomitant. This hypothesis posits that pain and negative emotion occur simultaneously because of similar biological mechanisms. Serotonin and norepinephrine are believed to play a role in the development of depression and the modulation of pain. Frequently cited evidence for common biological mechanisms is that individuals suffering from either pain or depression respond well to the administration of tricyclic antidepressants. Further, depressed individuals and chronic pain patients frequently share other physiological markers, such as reduction of REM sleep, an increase of plasma cortisol, a pathological dexamethasone test, and low 5-hydroxyidoleacetic acid (5-HIAA) levels in the cerebrospinal fluid (Robinson & Riley, 1999).

Longitudinal studies. Longitudinal studies allow temporal and directional inferences to be made about the nature of the relationship between negative emotion and pain. Atkinson and colleagues, (1991) studied lifetime prevalence and premorbid risk of psychiatric disorder in a sample of male chronic low back patients attending a primary care clinic at a Veterans Administration Medical Center, USA. Psychiatric disorders were assessed using the NIMH Diagnostic Interview Schedule, a DSM-III-based structured interview. They found that 32% had experienced a previous major depressive disorder and 31% had experienced a major anxiety disorder. Date of onset between the psychiatric disorders and the chronic pain were then compared. For major depressive disorder, results showed that 42% experienced onset of depression before the onset of pain, 58% after onset of chronic pain. With regard to major anxiety disorder, 47% experienced onset of anxiety before the onset of pain and 53% after onset of pain. Limitations of the study are the retrospective nature of the design and possible sample bias. Atkinson and colleagues (1991) conclude that this study supports the posit of postpain mood disorder, although other findings suggest that psychiatric disorders precede the onset of chronic pain.

Leino and Magni (1993) examined the relationship between distress and muscoloskeletal symptoms (low back pain, neck shoulder pain) in 607 Finnish industry workers at three different times at five-year intervals. In general, they found that emotional distress scores from an earlier assessment were positively related to self-report of musculoskeletal symtpoms. Distress was predictive for men only. None of the muscoskeletal symptoms were predictive of later depressive symptoms when the direction of the analysis was reversed. A limitation of the study is that the measure of emotional distress was not a clinical measure but a composite of 7 items representing depressive symptoms. The strengths of this study are that it is prospective in nature and that is does not suffer from the inherent sampling bias of clinical populations.

Magni, Morschi, Rigatti-Luchini, and Merskey (1994) tested for a directional effect of depressive symptoms on muscoloskeletal pain and vice versa using epidemiological data from a general population collected by the U.S. National Center for Health Statistics. Chronic pain was defined as pain that was experienced for most of a day for at least 1 of the past 12 months. Depression was assessed using the CES-D. Results showed that participants reporting chronic pain were 2.85 times more likely to report depression at follow-up 8 years later, whereas the risk ratio for the prediction of chronic pain from depressive symptoms was 2.14 times. These findings suggest that there is no definitive antecedent. The authors (Magni et al., 1994) speculate that depression may be more predictive of some pain conditions and that certain pain conditions may be more likely to predict depressive symptoms. A limitation of this study include the inability to determine whether depression or pain symptoms were continuous or intermittent over the eight-year duration of the study.

An exhaustive review of longitudinal studies is not possible here, nevertheless results seem to indicate that the causal path is not unidirectional (Robinson & Riley, 1999). Pain precedes negative emotion for some individuals, negative emotion precedes pain for other individuals. The complex nature of the human experience suggests that the nature of pain and emotion are probably not entirely direct but are mediated

by a number of biological and psychosocial variables. In addition, these mediators are most likely to be bidirectional in nature.

Mediators between pain and negative mood. Given the probability that the relationship between pain and negative emotion are not entirely direct, Robinson and Riley (1999) examined the variables that promote their comorbidity. It is also likely that the influence of these variables may differ in degree across dimensions of negative emotion (depression, anxiety, and anger). The constructs repeatedly found in the chronic pain literature that are known to be associated with pain and emotion that may account, in part, for the comorbidity of pain and negative emotion will be briefly described below.

Somatization. Somatization is defined as the predisposition to amplify physiological sensations or the misclassification of symptoms of emotional arousal. It has been proposed that with chronic pain, there may be a sensitizing effect to physiological events that heightens bodily awareness. It has been proposed that chronic pain patients blur painful and nonpainful experiences and interpret a wide variety of experience in terms of pain (Robinson & Riley, 1999). Geisser, Gaskin, Robinson, & Greene (1993) found that a measure of somatic focus mediated the relationship between depression and the sensory component of the McGill Pain Questionnaire. These researchers did not find this relationship for the affective or evaluative components of pain. In general, the authors conclude that somatization is related to both pain symptoms and depressive complaints.

Catastrophizing. Catastrophizing is a cognitive process characterized by negative expectations about future outcomes and lack of confidence (Beck, 1976). Related to chronic pain, catastrophizing is thought to be a unique aspect of pain-related negative cognition and is typically measured with the Coping Strategies Questionnaire (Riley & Robinson, 1997). There has been some debate as to whether catastrophizing and depression are distinct constructs, however, cognitive models of depression view negative cognition as distinct from, but related to, symptoms of depression (Beck, 1976).

Social / Interpersonal Factors. The increased risk for the occurrence of negative emotion in chronic pain patients may be due, in part, to the effects of a patient's social interactions and interpersonal relationships. The occurrence and severity of depression that occurs in physical disorders such as chronic pain are associated with lack of social support, marital dissatisfaction and conflict (Robinson & Riley, 1999). Trief, Carnike, and Drudge (1995) examined the relationship between family environment and depression in 70 low back pain patients. Results showed that depression was associated with the perception of social support and the quality of the family environment. These authors conclude that low social support is linked to and may be a risk factor for depression in chronic pain (Trief et al., 1995).

Summary and Critique. It appears that pain is strongly associated with negative emotion. There are literally thousands of published articles on the relationship between chronic pain and emotion. There is evidence that negative emotions co-occur with pain conditions and that individuals with chronic pain have on average greater levels of negative emotions. There is no conclusive data as to causality. Despite the large body of literature, this area shows some weaknesses. For example, the dimensions of negative emotion have both shared and distinct components. Depression and anxiety share an emotional-distress component but differ on physiological arousal. Most studies consider depression; only a few have considered anger. Further, there is a lack of theory-driven models with empirically testable, falsifiable relationships. To conclude, some researchers (Robinson & Riley, 1999) suggest the existence of relatively homogeneous subtypes of chronic pain patients. It is possible that patients could be characterized into subtypes based on their experiences of depression, anxiety, and anger. The existence of subtypes may explain inconsistencies in the literature and lack of closure regarding the relationship between pain and emotion.

An area that has received little attention is the relationship between pain and other physical diseases, or comorbidities. As discussed above, comorbidities are typically described as psycho-pathological in nature, the primary focus of research has been the relationship between depression and chronic pain. The question to be addressed in this study is whether chronic pain is associated with other diseases.

2.4. CAUSAL ATTRIBUTIONS ABOUT DISEASE ONSET

This third section examines the relationship between psychological and social factors and disease onset from a lay person's perspective. This field of research examines constructs including causal attribution theories, attribution theories, coping strategies and beliefs about illness, and self-blame. These constructs will be discussed below based on research addressing chronic pain, in general, and back pain, as well as, cancer and heart disease due to the extensive research addressing these two most life-threatening diseases in the Western world.

2.4.1. Subjective Theories

Subjective theories are defined as a complex aggregate of concepts, which parallel research theories in structure and function. This means that the subjective theories are implicitly logical in form (Groeben, 1988). Further, "subjective" theories are influenced by "objective" (i.e., research) theories and vice versa. This exchange is advantageous for both types of theories.

2.4.2. Attributions

Past research has been directed towards the ideas sick individuals have about onset of their diseases. Empirical evidence has shown that patients make attributions about causes for their illness. Turnquist, Harvey and Andersen (1988) found that most patients (between 69% and 95% of the patients) reported causal attributions for onset of disease. These causal attributions help sick individuals cope on an emotional level (Verres, 1989). According to Filipp (1990), patients suffering from a (chronic) disease are not the only ones who have particular ideas about onset and treatment of diseases; healthy individuals also have their theories about how diseases develop, albeit different theories.

Bar-On (1987) illustrated the importance of both causal attributions for illness and feelings of control in the recovery of myocardial infarction patients. Participants were asked why they thought they had a heart attack, and what health measures they planned to take as a result of the attack. A follow-up several months later measured the work and social functioning of the participants. Bar-On (1987) found that

patients who attributed the cause of their myocardial infarction to modifiable factors under their personal control (such as stress or smoking) were more likely to have initiated active plans for their recovery (e.g., changing jobs or exercising) and to have returned to work and resumed other activities. In contrast, patients who attributed their myocardial infarction to factors beyond their personal control (e.g., back luck, fate) were less likely to have generated active plans for recovery or to have returned to work. These results seem to indicate that the recovery process is enhanced when illness conditions are perceived as being modifiable and under one's personal control. Further, Bar-On (1987) suggests that these kinds of perceptions may be more important predictors of successful rehabilitation than physical indicators.

2.4.3. Coping

Although most patients suffer from adverse psychological reactions as a result of a disease, most do not seek professional help. Instead, they draw on their internal and social resources for solving problems and alleviating psychological distress. Maes, Leventhal and DeRidder (1996) hypothesized that coping with a diagnosis is much like coping with any other severely stressful event. The appraisal of a chronic disease as threatening and challenging leads to the initiation of coping efforts (Lazarus & Folkman, 1984). Mercado, Carroll, Cassidy and Coete (2000) assessed coping styles in patients with neck and low back pain in the general population. They differentiated between passive and active coping, and found that these coping styles were related to particular psychosocial variables. Results showed an association between passive coping with being married, greater pain severity, depression, and poor health. Active coping was associated with female gender, higher education, less depression, good health and frequent exercise. While researching the relationship between chronic low back pain, depression and coping activities, Atkinson, Slater, Patterson, Grank and Garfin (1991) found that chronic low back pain patients used different coping strategies when attempting to manage pain exacerbations than when confronting more general life stressors. An increased rate of passive-avoidant coping responses was associated with the combination of chronic low back pain and concurrent depressed mood, rather than with chronic low back pain alone. Results suggest that

although some patients may selectively employ passive-avoidant coping activities in response to pain exacerbations, these are likely a function of depressed mood.

Dunkel-Schetter, Feinstein, Taylor, & Falke (1992) asked cancer patients to identify the aspect of their cancer they found to be most stressful and how they intended to deal with these problems. Results showed that fear and uncertainty about the future was most common (41%), followed by limitations in physical abilities, appearance, and lifestyle (24%), and then pain management (12%). Patients' answers to coping strategies could be categorized into five groups: social support / direct problem-solving (e.g., "I talked to someone to find out more about the situation"), distancing (e.g., "I didn't let it get to me"), positive focus (e.g., "I came out of the experience better than I went in"), cognitive escape / avoidance (e.g., "I wished that the situation would go away"), behavioral escape / avoidance (e.g., excessive sleeping, drinking). In comparison to coping strategies employed in other stressful events, health problems lead to more emotion-focused coping, perhaps because a threat to one's health is an event that must be tolerated but is not necessarily amenable to direct action (Taylor, 1999). Health problems also lead people to seek social support (Taylor, 1999).

Taylor (1999) addressed the issue of which coping strategies facilitate psychological adjustment. She found that the use of avoidant coping is associated with increased psychological stress and thereby may be a risk factor for adverse responses to illness. In contrast, research has found lower psychological distress to be associated with positive, confrontative responses to stress; with beliefs that one can personally direct control over an illness (Taylor, Heigeson, Reed, & Skokan, 1991). It appears that individuals who employ multiple strategies may cope better with the stress of chronic disease than do those who engage in a predominant coping styles (Taylor, 1999). The rationale behind this hypothesis is that the individuals are better able to match the coping strategy to the particular problem at hand. This area appears complex and requires further research.

2.4.4. Beliefs

Beliefs about chronic illness: When adjusting to a new diagnosis of a chronic illness, patients must somehow integrate their illness into their lives. Virtually all chronic illnesses require some alteration of activities and some degree of management. One problem that can arise in adjustment to chronic illness is that patients adopt an inappropriate model for their disorder, most notably, the acute model (Tayler, 1999). For example, hypertensive patients may believe incorrectly that if they feel all right, they no longer need to take their medication because their hyptertension is under control; accordingly they may fail to monitor their condition closely. Taylor (1999) emphasizes how important it is for health care providers to probe the patients' comprehension of their illness to check for significant gaps and misunderstandings in their knowledge that may interfere with self-management.

Beliefs about the cause of the illness: According to research, long term adjustment is related to two primary beliefs: perceptions of the cause of the illness and beliefs about whether the illness can be controlled. Individuals suffering from both acute and chronic illness often develop theories about where their illness came from (Downey, Silver, & Wortman, 1990). Examples of such theories include stress, physical injury, disease-causing bacteria, and God's will. Where patients place the blame for their illness seems to be the most important factor. Do they blame themselves, another person, the environment, or a quirk of fate?

Beliefs about the controllability of the illness. This area of research addresses whether patients who believe they can control their illness are better off than those who do not see their illness as under their control. For example, many cancer patients believe that they can prevent a recurrence of the disease through good health habits or even sheer force of will. They may believe that by complying with treatments and physicians' recommendations, they achieve vicarious control over their illness. These control-related beliefs may or may not be accurate.

Interestingly, interventions that attempt to instill feelings of control are often highly successful in promoting good adjustment and in reducing physiological arousal and

emotional distress caused by illness and its treatment. Could feelings of control have the same beneficial effect when they are self-generated by patients attempting to deal with chronic illness?

The literature indicates that belief in control and a sense of efficacy with respect to the disease and its treatment are generally adaptive. For example, cancer patients who believed that they had control over their illness were better adjusted to their cancer than were patients without such beliefs (Thompson, Sobolew-Shubin, Galbraith, Schwankovsky, & Cruzen, 1993). Similar reports have been reported for patients suffering from various diseases including rheumatoid arthritis, and patients with spinal cord injuries. Thus, control appears to be helpful not only in coping with acute disorders but also with the long term debilitation that may result from chronic illness. There is some evidence that the experience of control or self-efficacy may prolong life. Kaplan, Ries, Prewitt, Eakin (1994) studied patients with chronic obstructive pulmonary disease and found that those with high-efficacy expectations lived longer than those without such expectations. However, not all studies have found that feelings of control are adaptive in adjusting to chronic conditions .

2.4.5. Blame.

In chronic pain patients, self-blame is reported often. Patients frequently perceive themselves as having brought on their illness through their own actions. In some cases these perception are correct. Poor health habits such as smoking, improper diet, or lack of exercise can promote diseases such as heart disease, stroke, and cancer. In some cases, self-blame may be groundless, e.g., by a genetically based defect.

The consequences of self-blame are not known. Some researchers suggest that self-blame can lead to guilt, self-recrimination, or depression. Self-blaming patients may be poorly adjusted to their illness because they focus on things they could have or should have done to prevent it. Other researchers have found that the self-blame may be adaptive. Perceiving the cause as self-generated may represent an effort to assume control over the disorder; such feeling can be adaptive in coping with and

coming to terms with the disorder. These contradictory findings seem to suggest that self-blame is adaptive under certain conditions but not others. Research does suggest that blaming another person for one's disorder is maladaptive (Turnquist et al., 1988). For example, some patients believe that their disorders were brought on by stress caused by family members, ex-spouses, or colleagues at work. Blame of this other person or persons may be tied to unresolved hostility, which can interfere with adjustment to the disease. The effects of other attributions is not well documented. Specifically, no relationships have been found between luck and adjustment or between attribution to environmental factors and adjustment.

Empirical Study of Particular Interest: The following study is particularly relevant since it serves as a model for assessing causal attributions for the diseases cancer and heart disease. Schmidt-Rathjens (1998) examined the subjective relevance of 16 psychological factors in the onset of these diseases. N = 1,430 participants rated the influence of personality characteristics on cancer and heart disease. Factor analyses showed a clear distinction between the items loading on Factor 1 "cancer" and the items loading on Factors 2 and 3 "heart disease". Participants considered both depression, loss, and resignation (Factor 2) and constant stress and dissatisfaction (Factor 3) to be important for the onset of heart disease, while a combination of all the hypothetical causal factors seemed to be relevant for cancer (Factor 1).

In the next step, Schmidt-Rathjens (1998) tested whether being afflicted with either cancer or heart disease had an effect on causal attributions. According to subjective theories, it was expected that disease sufferers would make more differentiated cognitions, i.e., choose more relevant factors, than healthy controls. The distinction between the healthy controls (n = 131), cancer (n = 86), and heart disease (n = 201) sufferers was made through self-reports. Varimax loadings conformed to expectations for the cancer group; cancer patients selected more personality factors relevant to disease onset than healthy controls. The varimax loadings for the heart disease group did not conform to expectations; there was no difference in the number of personality factors between the heart disease group and healthy controls.

3 (healthy, cancer, heart disease) x 2 (relevance ratings for cancer and heart disease) ANOVAs showed that, with the exception of Item 11 ("Constantly ignoring one's own needs."), significant differences were found for all of the items regarding their relevance for onset of cancer or heart disease, i.e., the possible causes of cancer or heart disease were rated differently. Participants scored the personality items for the onset of heart disease higher and more consistently (recognizable through the lower standard deviations), than for cancer (Schmidt-Rathjens, 1998). These differences may be attributed to the fact that the risk factors for heart disease, e.g., stress, are better researched than those for cancer, in particular because the etiology of cancer is generally complex and not well understood.

In this study, the 16 personality factors formulated by Schmidt-Rathjens (1998) will be used to identify causal attributions for cancer, heart disease, as well as for back pain.

2.5. THE ROLE OF GENDER AND FAMILY; PREVENTION AND TREATMENT

2.5.1. Gender Differences.

Both clinicians and researchers have largely ignored potential or actual differences in how men and women report symptoms or respond to therapeutic interventions. Most studies have treated men and women as equals and have not asked questions about whether these are between gender group differences or within gender group differences in treatments. In fact, in the study of new pharmaceutical agents, samples are restricted to male participants because of concerns about exposing women of childbearing years to drugs that might produce teratogenic effects (Miaskowski, 1999)

Pain perception. The majority of the studies done in this area have attempted to determine if men and women have different pain thresholds and different levels of pain tolerance. Pain threshold is defined as a minimum amount of stimulation that reliably evokes a report of pain in an individual. Pain tolerance is defined as the time that a continuous stimulus is endured by an individual or the maximally tolerated stimulus intensity that a person can endure (Miaskowski, 1999). Studies using experimental pain (e.g., cold pressure pain) seem to show that women exhibit lower pain thresholds than men. In addition, in most of the studies women exhibited less tolerance to noxious stimuli than men did. These findings are often interpreted to mean that women are more sensitive to painful stimuli than men.

Miaskowski (1999) presents plausible explanations to explain the gender differences in responses to experimentally induced pain. One explanation involves the hormonal status of the participant. Several studies reported that a woman's phase in the menstrual cycle or her reproductive status (i.e., pregnant, not pregnant) can affect pain intensity ratings (Miaskowski, 1999). Another possible explanation is that the gender of the individual performing the experiments plays a role pain reports. Some studies found that men reported less pain in front of a female experimenter than a male experimenter, while female participants were not influenced by the gender of the experimenter (Miaskowski 1999). Although the data is not conclusive, it does seem to suggest gender differences.

Perceptions about the pain experience. Bendelow (1993) conducted a qualitative study addressing the relationships between perceptions of pain and various social characteristics of the individual, including the influence of gender. Results showed that significantly more women than men thought that anxiety, fear, and depression affected their perception of pain. Another finding of Bendelow's study (1993) was that 66% of the females and 33% of the males believed that women were better able to cope with pain than men. Both men and women expressed the view that the combination of female biology and the reproductive role serve to equip women with a "natural" capacity to endure pain both physically and emotionally. Further, Bendelow (1993) suggested that children are socialized to think about pain and react to painful events in certain ways. For example, boys are actively discouraged from expressing emotions.

It has been suggested that several chronic pain problems have specific gender distributions. Unruh (1996) reviewed numerous studies in this area and found that women tend to suffer more often from migraine headaches (3:1), rheumatoid arthritis (3:1), Carpal tunnel syndrome (2:1) Temporomandibular joint disorder (2:1 to 8:1), while men tend to suffer more than women from cluster headaches (8:1).

Several epidemiological (retrospective) studies have also indicated that women report greater pain with the same pathology, greater number of painful sites and are more likely to develop a chronic pain syndrome after equivalent trauma (Miaskowski, 1999). Numerous factors influence that data. For example, women report more doctor visits, and more return visits than men. Miaskowski (1999) argues that these difference may be due to a willingness to report pain, in general, and to report health care visits, in particular.

Finally, Miaskowski (1999) examined whether health care professionals respond differently to men's and women's reports of pain and pain behaviors. Studies seem to suggest that women receive less pain medication than men in various patients' populations (e.g., abdominal surgery, coronary artery bypass graft). She suggested that women may be taken less seriously than men and may, in fact, receive more

sedative medication to inhibit their expressive behavior (Miaskowski, 1999). Although there is evidence that a gender bias in how health care professionals respond to men's and women's reports of acute and chronic pain, additional research is warranted.

2.5.2. The Role of the Family.

It seems obvious that when an individual suffers from chronic pain, that this will have an effect on the daily lives of each family member. The reverse is also true, i.e., family norms also influence how members deal with the issue of chronic pain. It is within the family that healthy or unhealthy behaviors are encouraged, and that habits are formed.

Research demonstrates the importance of the family and close social environments with respect to health and illness. As cited in Fydrich and Flor (1999): (1) most sicknesses are treated at home; (2) patient compliance behavior is related to the attitudes of close friends and family; (3) children are more likely to show healthy habits when their parents partake in similar activities; and (4) treatment efficacy is related to positive family support.

Family dynamics can also negatively influence the pain experience. Fydrich and Flor (1999) in their review found that stressful situations like continuous fighting may negatively influence the course – and possibly play a role in the onset – of an acute or chronic disease. In 78% of the families of chronic pain patients, another family member also suffers from pain, patients report more pain and show more functional limitations in the presence of a solicitous partner than alone.

Fydrich and Turk (1999) address four issues regarding the interaction between pain and families: (1) Can the family influence the onset of chronic pain?; (2) Does the family play a role in the chronification and the course of a chronic pain syndrome?; (3) How does chronic pain influence the every day lives of family members?; (4) How can inclusion of family members increase the efficacy of diagnosis and treatment of chronic pain patients?

The Role of the Family in the Onset of Chronic Pain. Although a review of the literature suggests that in families with chronic pain patients, more pain symptoms are reported by family members than in comparison groups, no conclusive evidence proves a familial causality. This issue is complicated with various contributing factors including biological, psychological, and social variables (cf. above). In addition, these studies are afflicted with methodological problems such as retrospective collection of data, selection biases (e.g., clinical samples), inadequate or missing comparison groups, and poor reliability due to self-reports.

The Role of the Family in the Chronification of Chronic Pain. Based on the operant model discussed above, it is assumed that particular pain behaviors (e.g., non-verbal behaviors including sighing, rubbing the painful area) in connection with pain will increase or decrease depending on whether they are reinforced (i.e., through attention, being relieved of responsibilities, pain avoidance). For example, in chronic back pain, inactivity is negatively reinforced through pain avoidance . This inactivity is often supported by family members and can lead to painful muscular atrophy. This cycle of pain-inactivity-muscle atrophy may represent an important factor in the chronification of pain (Fydrich & Flor, 1999). Familial support does not necessarily have to be disadvantageous. Paulsen and Altmeier (1995 as cited in Fydrich & Flor, 1999) found a relationship between the type of social support and pain. They report that a partner's behavior that relieves the pain patient of responsibilities, or supports rest and relaxation can lead to more overt pain behaviors. On the other hand, perceived support in the form of emotional support, belonging, partner dependability, social integration and respect is related to less overt pain behaviors in the presence of the partner. Further, Jamison and Virts (1990 as cited in Fydrich & Flor, 1999) propose that familial support is a predictor for better treatment efficacy. At the 12 months follow-up, patients supported by their families reported a lower pain intensity, required less medication, and were more active than the comparison group. The results from these studies suggest the importance of the type of social support received. An emotionally supportive partner and close familial ties may enable some pain patients to better cope with their pain (Fydrich & Flor, 1999).

The Effect of Chronic Pain on the Family. Chronic pain is clinically defined as a pain experience lasting six months or more, often years. The aspect of time, then, is, in and of itself, a stress factor that affects family members. Kerns, Dworkin, Romano, Thorn, & Williams (1999) and Flor (1999) found that the partners of pain patients report more stress symptoms, and were more likely to fulfill the criteria for a depressive disorder. Maruta and Osborne (1978 as cited in Fydrich & Flor, 1999) suggest that as pain patients may experience reduced libido, partners may become dissatisfied because of it. Financial difficulties can also be expected, especially when chronic pain causes loss of vocation. This area of research, however, is empirically not well supported. It is also important to keep in mind that a large portion of the family members of chronic pain patients demonstrate effective coping strategies and that not all families of chronic pain patients show physical or mental health problems (Fydrich & Flor, 1999).

The Role of the Family in Diagnosis and Treatment. Due to the interaction of family and chronic pain discussed above, it seems important to include familial aspects in diagnosing pain patients. In addition to the behavioral analyses well established in behavioral therapy, Fydrich and Flor (1999) have formulated the following questions: How has family life changed since pain onset?; Which positive and negative aspects have emerged due to the changes?; What kind of resources (psychological, social, financial) are available and how can they be used in the therapy?; How would family members like to change the current situation?

Summary. Although causal statements about the relationship between chronic pain and families cannot be made based on current empirical data, Fydrich & Flor (1999) emphasize that the family plays an important role in the development and course of chronic pain. In addition, families are negatively affected – financially, psychologically, and physically – when a family member has chronic pain. To increase therapeutic effectiveness, these authors recommend involving the family especially during the diagnostic processes.

2.5.3. Prevention

The essential component of any effective treatment modality is a comprehensive understanding of a disorder's etiology. Consequently, a shift from curative efforts toward preventative strategies is beginning to dominate the current direction of research with regard to low back pain (Garofalo & Polatin, 1999). There are three levels of prevention recognized by treatment teams: primary, secondary, and tertiary. Whereas primary prevention techniques try to reduce the incidence of the disorder prior to onset, secondary prevention strives to implement a comprehensive intervention early in the development of the disorder. Tertiary prevention generally involves eliminating or reducing the disability and suffering stemming from the disorder. Past preventive strategies designed to reduce low back problems in the workforce have focused on: (1) reducing low back pain episodes believed to be initiated by work; (2) reducing low back pain-related medical leave; (3) prolonging the tenure of employees with low back pain.

In general, prevention consists of identifying individuals at high risk for low back pain, predicting who will remain disabled for a long period of time after such an injury, ensuring effective treatment algorithms to get patients back to work as soon as possible, and to identify workplace modifications to prevent injury and enable early work return (Garofalo & Polatin, 1999).

2.5.4. Treatment

The usual course of a low back pain episode is usually benign. Fifty-percent of the patients suffering from low back pain will return to full functioning within two weeks; 70% will recover in 1 month; and approximately 90% will recover within three to six months (Mayer & Gatchel, 1988). Nevertheless, 30 to 70% of individuals suffering from low back pain will experience a recurrence of three or more episodes. Patients remaining symptomatic after six months have a much poorer prognosis, with diminished levels of occupational and social functioning (Mayer, 1991). The majority of this group will remain disabled after one and two years. Chronicity of pain is less dependent on medical than on psychosocial factors (Garofalo & Polatin, 1999).

Treatment begins with the patient seeking immediate relief for his or her pain from a primary care provider. Both spontaneous recovery and early intervention account for the majority of successful outcomes. Immediately following an injury, passive treatment modalities (e.g., medication, bed rest) are typically directed toward controlling the pain symptoms (Polatin, Kinney, Gatchell, Lillo & Mayer, 1993). It is at this point that the majority of patients experience full relief of their pain and a complete recovery from injury. However, for the patients whose pain symptoms do not subside, the processes of deconditioning and disability reinforced by secondary gain are beginning (Garofalo & Polatin, 1999). The desire to avoid pain leads to reduced physical activity and results in joints becoming progressively stiffer and muscles becoming weaker.

When pain persists beyond the initial four weeks, secondary care programs are recommended to correct early deconditioning. Mobilization, strengthening, and work simulation help to develop a greater endurance to fatigue and pain, and may facilitate the return to work (Garofalo & Polatin, 1999). Psychological intervention is not offered at this time in most treatment clinics.

Physical Therapy. Some evidence indicates that exercise aimed at strengthening back or abdominal muscles and exercise aimed at improving overall fitness can decrease risk of subsequent low back pain, but the effect is modest and of unknown duration. Insufficient evidence is found to recommend that either back education programs or mechanical supports be used routinely to prevent back pain. These conclusions should be generalized cautiously because they are based primarily on studies conducted in the work place and not in the clinical setting. Although no evidence shows that smoking cessation, weight loss, or attention to psychological risk factors can prevent the development of low back pain, recommendations to address these factors may influence the severity or course of low back pain development (Lahad, Malter, Berg et al., 1994).

Tertiary care is recommended for the 5-8% of patients who did not benefit from secondary care and remain symptomatic and disabled beyond six months after

injury. Factors associated with poor treatment outcome in secondary care include noncompliance, financial benefits, psychological distress and inhibited functioning (Polatin et al., 1993). Tertiary care tends to offer a higher level of intensity than either primary or secondary care and effects improvements in a subgoup of chronic pain patients that seems treatment resistant. At this stage of treatment, cognitive-behavioral techniques are combined with physical and occupational therapy programs (Garofalo & Polatin, 1999). It is important to continue treatment beyond primary care if patients fail to improve within the first few weeks after injury, for there is a decreased likelihood of returning to work as the duration of the low back pain experience increases.

2.5.5. Pain Control Techniques

Most of the techniques used for pain patients has been effective for acute pain patients, but less so for chronic pain patients. Pain control techniques include pharmacological, surgical, physical therapy, relaxation, acupuncture, and mental coping techniques. Although no single pain control technique has been clearly effective in modifying chronic pain, it seems that chronic pain patients profit from pain management programs. The first pain management program was founded in Seattle at the University of Washington by John Bonica, M.D., in 1960. Typically, these programs are interdisciplinary efforts, involving physicians, cognitive-behavioral psychologists, and physical therapists with consultation from neurology, rheumatology, orthopedic surgery, and internal medicine. The goal of such programs is to enable patients to reduce their pain as much as possible and live more active and rewarding lives, even if the pain cannot be entirely eliminated. Today these pain management programs are currently standard in the U.S. and in northern European countries.

The first step in this program involves the evaluation of pain and pain behaviors. Typically, such evaluation begins with a qualitative and quantitative assessment of the pain, including its location, sensory qualities, intensity, duration, as well as onset and history. Functional status is then assessed, where patients provide information about the degree to which their work and family lives have been impaired. Treatment

goals are based on how the patient has coped with the pain in the past. In addition, most patients are evaluated for their emotional and mental functioning.

Pain management programs include several common features. First, the patient is educated about the nature of their condition, the function of pain, and the various aspect of their lives (e.g., social contacts) that are affected by pain. Discussion in group settings includes topics such as medication, and its side effects; assertiveness or social skills training; depression; sleep disturbance; relaxation techniques; posture; weight management; and other topics related to the daily management of pain. Second, pain management programs aim to change maladaptive cognitions that may arise in response to chronic pain. Typical cognitive errors (Beck, 1976) include catastrophizing, developing distorted negative perceptions about their pain, and their inability to overcome and live with it.

There is some evidence that chronic pain patients react more acutely and psychologically more intensely than patients not prone to chronic pain (Taylor, 1999). However, it remains unclear whether these reactions are a cause or a response to the chronic pain experience. These reactions need to be systematically addressed through cognitive and behavioral management techniques (Turk, Kerns and Rosenberg, 1992). Some programs examine patients' preferred coping styles and try to match pain treatment to them in order to maximize the benefits of treatment.

Many pain management programs intervene at the family level, combining family therapy with other interventions. On the one hand, chronic pain patients often withdraw from their families, but on the other hand, efforts by the family to be supportive may inadvertently reinforce pain behaviors (Turk et al., 1992).

The final feature involves relapse prevention and follow-up activities so that patients will not backslide once they are discharged from the outpatient programs. Non-compliance is a common problem among pain patients. Turk & Rudy (1992) estimate the relapse rate following initial successful treatment of chronic pain to be approximately 30 – 60%.

Summary. Pain management programs appear to be successful in helping to control chronic pain. In particular, behavioral interventions are regarded as integral towards reducing reports of pain and disability. Programs designed to manage chronic pain acknowledge the complex interplay between physiological, psychological, behavioral and social factors, representing a truly biopsychosocial approach to pain management. As the importance, complexity, and costs of pain have become increasingly clear, pain is now taken more seriously in the medical management of patients and is now recognized as an important medical issue in its own right rather than an inconvenient symptom it was once regarded to be (Turk, 1999).

2.6. LITERATURE REVIEW: RISK FACTORS OF CURRENT RELEVANCE FOR THIS STUDY

The National Advisory Committee on Health and Disability of New Zealand (as cited in Gatchel & Gardea, 1999) defined the following psychosocial variables as risk factors for chronic back pain: maladaptive attitudes and beliefs concerning back pain, frequent display of pain behaviors, reinforcement of pain behaviors by family members, heightened emotional reactivity, lack of social support, job dissatisfaction, and compensation issues. Without a doubt these psychosocial issues, as well as genetics and biomechanics are of importance and deserve research attention. Nevertheless, this paper cannot address all areas that are interesting, and therefore, focuses on the following risk factors: pain chronicity, pain intensity, higher age, lower education, female gender, no physical exercise habits, smoking habits, depression, job dissatisfaction, and reported comorbidities.

2.6.1. Transition to Chronicity

Perrot (2000) defined risk factors that favor the transition to chronicity of low back pain. These factors include comorbidity, the social circle of the patient and family status, health status, profession, and interval before medical treatment. For surgery outcomes, professional status and age appear to be most important. Interestingly, the initial handicap and the type of management were of little importance. Perrot (2000) emphasized that the effectiveness of both medical and psychosocial approaches remain to be evaluated and defined in prospective studies.

A follow-up study was run on a cohort of 444 patients aged form 16 to 59 who consulted with their general practitioners after back pain onset. Miedema, Chorus, Wevers, and van der Linden (1998) collected data both retrospectively (symptoms, medical treatment received), and prospectively (health outcomes, quality of life scores and work factors). Results suggest that high pain intensity and sick leave were positively associated with chronicity.

Pincus, Burton, Vogel, and Field (2002) proposed that although the biopsychosocial model is generally accepted in low back pain, the unique contribution of

psychological factors in the transition from an acute presentation to chronicity has not been rigorously assessed. Analyses based on twenty-five publications suggest that psychological factors (i.e., distress, depressive mood, and somatization) are implicated in the transition to chronic low back pain.

Sanders (2000) presents a clear review of the known and empirically supported risk factors for chronic, disabling (defined as failure to return to work) low back pain. These risk factors are: (1) Minnesota Multiphasic Personality Inventory (MMPI) Scale-3 elevation, (2) depression, (3) low activity/high pain behavior, (4) negative beliefs/fear of pain. The MMPI Scale 3 focuses on patients' reports of sensory or motor symptoms, denial of problems or social anxiety, and feelings of uneasiness. It has not yet been established which of these factors is involved in predicting disabling low back pain (Sanders, 2000). A possible connection with the construct Neuroticism, has been proposed. Depression is a relevant risk factor when patients exhibit clinically significant levels of mood disturbance. The low activity/high pain behavior factor is exhibited by patients who report sedentary lifestyles, low physical exercise, and/or exhibit significant overt pain behaviors (cf. Hasenbring, 1992) such as limping, verbally complaining, holding or rubbing the affected area, and demonstrating extreme reactions during physical examination. Finally, negative beliefs or fears about pain refers to patients' beliefs about their pain, that it is harmful, disabling, or out of their control, or that increasing activity (e.g., by returning to work) would increase their pain (Sander, 2000).

Other risk factors, all of which have received considerable research attention, include job dissatisfaction, heavy physical labor, age (older than 40), severe psychological stress or abuse, subjective pain intensity, substance abuse, and compensation and unemployment benefits (Sanders, 2000). For this study, subjective pain intensity is of particular interest. The factors that most commonly describes patients who experience high levels of subjective pain intensity during the acute phase appear to have a significantly increased risk for developing chronic, disabling low back pain (Dworkin, 1997). If a cause-and-effect relationship can be established between

subjective pain intensity and chronic, disabling back pain, then it would be critical to initiate aggressive approaches to managing acute low back pain (Sander, 2000).

Gatchel (1996) describes the transition of acute to chronic pain as "a layering of behavioral / psychological problems over the original nociception of the pain experience itself". Gatchel (1996) has proposed a 3-stage model explaining the progression: In Stage 1, the acute phase, the patients' natural response to the injury are predominant. If the pain continues beyond the normal healing period (approximately 2 to 4 months), then behavioral or psychological reactions emerge. Stage 2 is characterized by increased distress, anger, depression or learned helplessness, and somatization. Gatchel (1996) proposed that in this stage the individuals' preexisting personality and psychological characteristics, in addition to their current socioeconomic and environmental conditions, contribute largely to the form of the developed problem; In Stage 3, the patients begin to adopt the sick role, often relieved of their normal responsibilities, social obligations, and compensation payments. In addition, the physical deconditioning exacerbates the situation.

2.6.2. Pain Intensity

Linton, Hellsing and Bryngelsson (2001) investigated the association between psychological factors, physical function, and moderate levels of spinal pain. Data from n = 271 individuals reporting moderate pain from a general population survey were analyzed through a series of discriminant analyses. Results showed that distress, perceived workload, physical function, sexual abuse, and catastrophizing were associated with moderate pain.

Research suggests that high levels of pain are indicative of a chronic and disabling injury (Gatchel & Gardea, 1999). Gatchel, Polatin and Mayer (1995), in a prospective study, followed 421 acute low back pain patients over one year. Results suggest that high levels of self-reported pain and disability, and elevated scores on the MMPI Hysteria Scale (Scale 3) were less likely to have returned to work after one year.

Pain intensity was found to be an important outcome variable in a follow-up study by Von Korff (1994). A sample of 1,128 primary care back pain patients were evaluated 1 year after the initial visit to the doctor. The most clinically significant differentiation measures were levels of pain intensity, pain-related disability, and pain persistence. Also indicative of poor treatment response were number of days in pain, lower education level, and female gender.

Feyer, Williamson and Mandryk, De Silva (1993) reported that patients (n = 45) attending a pain management clinic showed psychological disturbance while blue-collar workers (n = 116) and white-collar workers (n = 164) did not. Further, disability resulting from low back pain was positively and linearly related to severity of pain for subjects drawn from the working groups, irrespective of psychological disturbance. Work dissatisfaction was not related to the presence of, and did not account for disability resulting from low back pain in working subjects.

Mercado and colleagues (2000) analyzed data from N = 655 individuals from the general population and found through regression analyses that passive coping was associated with greater pain severity, being married, depression, and poor general health. In contrast, active coping was associated with female gender, higher education, less depression, good health, and frequent exercise. Their most interesting finding was the strong independent relationship between increased pain severity and the greater use of passive strategies and the lack of a relationship between pain severity and active coping.

2.6.3. Age

Research suggest that age (over 40) increases the risk of chronicity for patients suffering from back sprain (as cited in Gatchel & Gerdea, 1999). Also compare Sanders (2000) above. Cox (1999) stated that degeneration (or arthrosis) is rarely found in patients under 30 years of age, but it can be clinically documented more frequently and the cases are more severe as patients age.

2.6.4. Socioeconomic Status and Education

In the British community, back pain (self-reports) is more likely to be reported by women of low income and low educational qualifications; men from the unskilled manual labor force also report more back pain (Croft & Rigby, 1994).

In a review article examining 64 articles from 1966-2000, Dionne and colleagues (2001) conclude that the current available evidence points indirectly to a stronger association of low education with longer duration and/or higher recurrence of back pain than to an association with onset. Mechanisms that may explain these associations include variations in behavioral and environmental risk factors (e.g., hazardous living environment, smoking, obesity), differences in occupational factors (e.g., heavy manual labor, job dissatisfaction), compromised "health stock" (e.g., small adult vertebral canal among subjects with lower education as a marker of early impairment of growth), differences in access to and utilization of health services, and adaptation to stress.

2.6.5. Gender

Please refer to the section entitled "The Role of Gender and Family" above.

2.6.6. Physical Exercise Habits

Physical exercise is generally associated with better health, and persons who exercise are less often overweight, are less often sick, and live longer than those who do not exercise (Schwarzer, 1996). Physical fitness not only effects longevity, but also the quality of life; active individuals suffer less often from chronic pain, including chronic back pain, and show better general health (Schwarzer, 1996). The influence of physical exercise on coronary heart disease is the best researched field (Schwarzer, 1996) and exercise has shown positive effects in both physiological and psychological functioning of coronary-prone patients (as cited in Taylor, 1999). The effect of physical exercise on infections, cancer, and chronic pain is less well researched.

2.6.7. Smoking Habits

Cigarette smokers have an increased risk of low back pain which may be caused, for example, by disc degeneration and spinal instability. Fogelholm and Alho (2000) hypothesize that the high serum proteolytic activity of cigarette-smokers accesses a previously degenerated neovascularized disc and speeds up the degenerative process. The increase proteolytic activity may also weaken the spinal ligaments resulting in spinal instability.

Brage and Bjerkedal (1996) examined the association between smoking and musculoskeletal pain in 6,681 Norwegian males and females, aged 16-66 years. Statistical analyses showed that current smoking was independently associated with musculoskeletal pain (e.g., back pain) after controlling for gender, age, comorbidity, mental distress, lifestyle factors, and occupation related factors.

2.6.8. Depression

Joukamaa (1994) proposed that depression in low back pain patients is atypical meaning that low back pain sufferers have some symptoms of depression, but not enough to fulfill the diagnostic criteria for any type of depression defined in the DSM-IV. Some of these symptoms include crying spells, hopelessness, irritability, feeling of inadequacy, diminished self-esteem. One type of atypical depression is masked depression meaning that a patient has some symptoms of depression but not depressed mood. Another type of atypical depression is alexithymia, meaning "no words for feelings". It refers to a group of patients who have difficulty in communicating emotion, rarely cry, and tend to focus self-reports on physical symptoms and trivial environmental details. According to Joukamaa (1994) chronic pain can be divided into three components: somatic, depressive, and a role component. The therapeutic procedures should be devised individually to account for the dominance of these three aspects.

Epker and Block (2001) reviewed the major psychosocial risk factors impacting recovery from spine surgery. Among other factors, depression can affect spine surgery outcome. Individuals with depression tend to focus on negative events, to

have a low threshold for induced pain, and tend to report greater functional impairment. Therefore, it is not surprising to find that higher levels of depression are frequently associated with poor surgical outcomes. Epker and Block (2001) further emphasize the importance of examining chronicity of the depressive symptoms in the assessment of depression. For instance, some depressive symptoms such as sleep disturbance, concentration problems, and weight change may be a direct result of the experience of continued pain or physical limitations and, therefore, less important in predicting response to surgical outcome. Alternatively, chronic depression may be more predictive of a poor response to any surgical procedure aimed at pain relief.

2.6.9. Job Satisfaction

There appears to be general agreement among researchers and clinicians that exposure to a combination of occupational risk factors may render certain individuals more susceptible to muskoskeletal pain, however, no single causal relationship is responsible for it. Garofalo & Polatin (1999) propose that psychological and social factors impact the expression of pain.

Krause (1996) investigated the role of psychosocial job factors for prevalence of back and neck pain after taking into account the role of biomechanical factors in N = 1,449 San Francisco bus drivers. At the five year follow-up, back and neck pain risk factors were found to be: female sex, height, weight, years of driving, weekly hours of driving, break time, vehicle type, ergonomic problems, frequency of job problems, psychological demands, job strain, job dissatisfaction, and low supervisor support.

Based on a review of 21 prospective studies, Linton (2001) concludes that there is a clear association between psychological variables and future back pain. There was strong evidence that job satisfaction, monotonous tasks, work relations, demands, stress, and perceived ability to work were related to future back pain problems. Further, the results suggest that eliminating psychosocial risk factors at work could reduce the number of cases of back pain by as much as 40%.

238 workers involved in heavy physical work participated in one prospective study examining the relationship between physical and psychological risk factors. Data collected at baseline and in the 12 month follow-up showed that the history of back pain was the best predictor for the occurrence of a new episode of back pain, and that low job satisfaction was also associated with an increased risk for the occurrence of back pain during follow-up.

Kerr and colleagues (2001) found in a population of 137 back pain sufferers that self-reported risk factors include a physically demanding job, poor workplace social environment and inconsistency between job and education level. Contrary to previous studies, job satisfaction and better co-worker support showed modest associations with low back pain. Physical risk factors included peak lumbar shear force, peak load handled, and cumulative lumbar disc compression. Further they proposed that specific physical and psychosocial demands of work can be precisely identified as independent risk factors for low back pain.

2.6.10. Comorbidities

Somatic Symptoms: In a comparison study between chronic low back pain patients (n = 97) and health controls (n = 49), Bacon and colleagues (1994) found that almost 26% of the chronic pain patients reported a lifetime history of 12 or more somatic symptoms, compared to only 4% of the controls. In the less symptomatic ranges, chronic back pain patients generally reported more symptoms than controls. Major depression and alcohol dependence were significantly associated with increased severity of somatiziation.

N = 294 patients with chronic low back pain selected for surgery were matched by age and gender in a random sample of the Swedish general population. Hägg, Fritzell, Nordwall, & the Swedish Lumbar Spine Study Group (2002) analyzed data in which the general population sample was divided into two subgroups: subjects with and those without back pain. Results showed that surgical candidates with chronic low back pain differed significantly from the control subjects without back pain by demonstrating more general morbidity, smoking, and depressive symptoms, as well

as, self-assessed workload. However, surgical candidates did not differ from the control subjects with back pain in these respects. In a multiple logistic regression analysis, physical disability was the only variable that independently discriminated between all three groups.

Diabetes Mobbs, Newcombe and Nadana (1999) analyzed medical records of 363 patients and found 33 who received a lumbar discectomy had a preoperative diagnosis of diabetes. At the seven-year follow-up, these patients were matched with nondiabetic controls based on age, sex, and similar operative approach. Post-operative results were positive (good to excellent) for 86% (n = 28) of the control patients, and 60% (n = 25) for the diabetic patients. 28% of the diabetic patients had another operation compared to 3.5% of the controls.

Heartburn, Asthma, Ulcer: Xuan, Kirchdoerfer, Boyer and Norwood (1999) proposed that coexisting diseases may have unforeseen yet clinically significant effects on a patient's well-being. They assessed the effects of comorbidity on the results of Quality of Life measures through an analysis of longitudinal data from three double-masked, randomized, placebo-controlled clinical trials dealing with heartburn, asthma, and ulcer. Patients were assigned to subgroups by comorbidity status: Group 1 = no comorbid diseases; and Group 2 = principal disease of heartburn, asthma, or ulcer with comorbidities of chronic obstructive pulmonary disease, asthma, or chronic bronchitis; hypertension; migraine; coronary artery disease, or varicose veins; chronic gastrointestinal conditions; arthritis or back pain; diabetes; or depression. Multivariate analyses of covariance were run and the results suggest that comorbid conditions significantly and extensively affect patients' scores on generic Quality of Life scales. The most important comorbidities in the three trial populations were arthritis or back pain and depression.

2.7. AIM OF THE STUDY

The purpose of this paper is to examine some correlations between biopsychosocial factors and low back pain. Due to the number of interesting variables discussed above, this paper focuses on a few, and they are: Sense of Coherence, depression, job satisfaction, age, gender, education, smoking and physical exercise and their relationship to pain duration (chronicity) and intensity of the low back pain experience.

2.7.1. Biopsychosocial Model

Since the biological model alone can not explain why some patients with obvious pathological anatomical findings experience little pain and other patients with seemingly normal anatomical structures experience more intensive pain, researchers have turned their attention towards psychological factors such as personality, depression, daily stress, and life events; and towards social factors such as physical strain, length of illness, and education, in an attempt to understand chronic back pain. In accordance with a holistic approach to health, the phenomenon low back pain will be examined in this study from a biopsychosocial perspective.

The term, biopsychosocial model, implies a complex relationship between cause and effect. This complicated association may be best expressed in terms of risk and protective factors. Risk factors are generally considered to be influences on a person that increase the probability of becoming ill, in comparison to someone not being exposed to these influences (Blohmke, 1986). Typically, a particular influence becomes a risk factor when it reaches a certain level. For example, blood cholesterol levels over 200 are thought to promote arteriosclerosis. Hypothetically, based upon the biopsychosocial model, the following risk factors for low back pain could be considered:

- biological risk factors include mechanical causes such as lumbar muscles (injured by movement or overuse) or spondylolisthesis (slow subluxation of the posterior facets) and vascular or hematologic causes such as a ruptured disc or degeneration. Age and gender are also important biological variables.

- psychological risk factors include depression, which can both be the result of pain and also can worsen the perception of pain. Depression is correlated with a heightened risk of new onset back pain (Mannion, Dolan & Adams, 1996) and also of new episodes of back pain (Croft, Papageorgious, Ferrs, et.al., 1995). Higher scores obtained on depression scales, i.e., sum scores above a certain "cut-off" point, could represent an equivalent to the cholesterol example above. Another potentially important psychological risk factor may be the psychological "overlay" criteria proposed by Waddell (1987). His evaluation criteria consist of five basic elements including abnormal tenderness, performance of the patient on tests simulating orthopedic maneuvers, performance on distraction tests (where a similar maneuver is performed two different ways or in two different positions), non-physiologic weakness or sensory disturbance, and overall exaggeration of symptoms;
- social risk factors include a evaluation of the factors in the patient's life and work that may affect overall prognosis of the condition. Education and job satisfaction are relevant social factors (Swenson, 1999) and these factors are examined here.

Protective factors potentially play just as an important role in the evaluation of health as risk factors. An early proponent of protective factors was Antonovsky (1987, 1993) and his theory of saluto-genesis provided an alternative to the pathologic perspective. Antonovsky's research focussed on the question of why an individual, despite ever present stressors, remains healthy. According to Antonovsky (1987), health and illness are not two distinct categories, instead he saw them along a continuum ("Health Ease / Dis Ease" (HEDE)-continuum). This saluto-genesis theory implies that a patient has not only symptoms, but is also recognized as an individual with healthy attributes. Further, the central component of Antonovsky's theory of health is the Sense of Coherence. This personality construct is defined as a global orientation in which one possesses a general, underlying feeling of trust that the internal and external environment is predictable and that things will most probably develop as expected. Antonovsky (1987) proposed that the Sense of Coherence can directly affect an individual's health status as well as indirectly mobilize resources.

Table 1: Study Variables

Risk Variables	Protective Variables	Criterion Variables
Higher Age	Lower Age	Pain Chronicity
Lower Education	Higher Education	Pain Intensity
Female Gender	Male Gender	
No Physical Exercise	Regular Physical Exercise	
Smoker	Non-Smoker	
Depression	Low Depression Scores	
Job Dissatisfaction	Job Satisfaction	
Comorbidities	No Comorbidities	
Low SOC*	High SOC*	

* Sense of Coherence

Study Hypotheses:

Length of and Intensity of the Pain Experience

H1 Biopsychosocial Variables: There is a significant relationship between the biopsychosocial risk factors higher age, lower education, female gender, no regular physical exercise, smoking, depression and comorbidities and higher ratings for chronicity and for pain intensity in *back* pain patients.

H2 Predictor Scales: There is a significant relationship between the psychosocial protective factors Sense of Coherence and Job Satisfaction and lower ratings for chronicity and for pain intensity in *back* pain patients. Back pain patients are expected to score higher on the Depression Scale than no *back* pain patients.

H3 Explaining the Variance for Chronicity, Intensity, Chronicity + Intensity: The number of significant correlations between criterion variables and predictor variables is expected to increase when the criterion variables chronicity and intensity are combined.

H4 Exaggerated Pain Experience: Patients experiencing chronic pain and/or high intensity pain will score higher on the items measuring a Exaggerated Pain Reaction than patients with acute pain or low intensity pain.

Group Differences

H5 Group Differences: Mean scores on the psychosocial risk factors (e.g., depression) for the back pain group are higher than for the no back pain group. The reverse is expected for the psychosocial protective factors (e.g., Sense of Coherence, job satisfaction), i.e., mean scores for the back pain group on the psychosocial protective factors is lower than mean scores for the no back pain group.

2.7.2. Comorbidities

The relationship between low back pain and 15 other diseases, or comorbidities, will be examined. It seems logical to assume that chronic, high intensity low back pain patients tend to report more comorbidities, i.e., diseases such as cancer, heart disease, allergies, asthma, and diabetes, than participants without chronic, intense pain. This may suggest a particular vulnerability for disease, or a disease-prone personality, a construct suggested by Friedman and Booth-Kewley (1987). This variable includes both life threatening diseases (e.g., cancer, heart disease) and annoying, but non-life threatening diseases (e.g., allergies, asthma).

H6 Comorbidities: There is a significant relationship between high ratings on chronicity and on pain intensity and self-reported comorbidities (present, past).

2.7.3. Causal Attribution Theories About Disease Onset:

This section addresses the relationship between personality and disease onset (back pain, cancer, heart disease) from a lay person's perspective. The main question addressed here is whether similar attribution patterns exist between back pain, cancer, and heart disease, i.e., do participants attribute "job dissatisfaction" to the onset of chronic back pain? And further, do back pain patients make the same attributions about disease onset as controls? Differences are expected since cancer and heart diseases are life threatening, while back pain is debilitating, but not life

threatening. Differences are also expected when participants themselves are afflicted with, for example, chronic back pain.

H7 Disease Related Attributions: Participants rate personality factors to be responsible for the onset of cancer and of heart disease, less so for the onset of back pain.

H8 Back Pain Patients' Attributions: Low back pain patients attribute personality factors for the onset of back pain lower than no back pain participants.

3. METHOD

3.1. Participants

3.1.1. Means (Gender, Age, Nationality, Level of Education)

160 German females and 110 German males (*N* = 270) with a mean age of 45 years (*SD* = 15.7) participated in this study. The youngest participant was 15 years old, the oldest participant 73 years. All participants were of European origin, spoke the German language, and were recruited from the same semi-rural town of approximately 45,000 inhabitants in southern Germany. 231 participants had not completed their "abitur", 39 participants had completed their "abitur". (Abitur is an approximate equivalent to U.S. high school graduates with a 3.0 G.P.A. or better. These students intend to continue their education at the university level.)

3.1.2. Rate of Return

From the 310 questionnaires delivered to the various medical practices, 270 questionnaires were returned. The response rate for the entire sample was approximately 87%. For the questionnaire "back pain", the return rate was 85.5%; for the questionnaire "no back pain", 89.2%. It is not possible to determine why the return rates varied across the doctors' practices. Hypothetically, these differences could be due to the personality of the doctor (i.e., introverted versus extroverted) and how comfortable he (all of the doctors involved in this study were male) was in asking his employees to recruit patients for the study. The rate of return for this study was extremely high. This may be due to the status/respect doctors in Germany enjoy. None of the participants received reimbursement for questionnaire completion.

Table 2: Rate of Return

	Questionnaires		
Group	Out	In	%
1. Questionnaire "back pain"	180	154	85.5
2. Questionnaire "no back pain"	130	116	89.2
Total			87.4

3.2. Procedure and Recruitment of Participants

Group "back pain"

Data for the first group of participants (*n* = 154), were collected in three of the four orthopedic practices in town. Patients were told that a study about back pain was being run in the practice to better understand the phenomenon of pain, with the hope of eventually being able to offer a more effective therapy. Further, patients were informed that the questionnaire included self reported health behaviors such as exercise and smoking, as well as some personality scales. Patients were recruited in clusters, i.e., patients were randomly (dice-throwing method) recruited from all of the back pain patients receiving an appointment on a particular day. During the data collection phase patients were able to choose freely which orthopedic doctor they would like to see, patients could change doctors during the course of treatment, and they did not have to pay for the examination(s). Patients reporting back pain (in the past or in the present) at the registration desk were asked by office employees to complete the questionnaire in the waiting room upon arrival before they saw the doctor. Patients were reminded that completion or non-completion of the questionnaire had no effect upon their treatment and their answers would be treated confidentially. The exact instructions for completion can be found in the Appendix. Participants completed this questionnaire within an hour without consultation from others or from printed material. Patients completing the questionnaire "back pain" were also rated by their orthopedic doctors on an objective scale identifying the cause of the back pain as well as an exaggerated pain experience. (The complete questionnaire can be found in the Appendix). Office employees were instructed not to give patients who had difficulties with the German language a questionnaire because it was so extensive and time did not allow for a translation by practice personnel. Only three practices were included in this study because the fourth orthopedic doctor was on sick leave at the time of data collection.

Group "no back pain"

The second group (*n* = 116) of participants was recruited through an orthopedic practice, a general practitioner's office and a nose-ear-throat specialist's office. In these practices, patients were told that a study about health behaviors was being run

in the practice to better understand why some individuals remain healthy and some become ill. Participants were also recruited as described above. The only exclusion criterion was acute back pain, i.e., the reason for their present visit was not back pain. The questionnaires were completed within an hour in the waiting room, mostly before treatment began, participants were instructed to complete the questionnaire without consulting a neighbor or printed material. Four questionnaires were returned through the mail because time in the waiting room was not sufficient for completion. For further instructions for completion, please refer to the complete copies of the questionnaire(s) in the Appendix.

Group "causal attributions"
A third group (n = 101) completed only the items assessing causal attributions about disease onset, and questions regarding demographics. Participants were recruited through the snowballing technique, i.e., friends and friends of the researcher's friends were requested to complete the questionnaire. Participants required approximately 15 minutes to complete this questionnaire. Although participants were instructed to complete the questionnaire without consultation from others or from printed material, this was not controlled since none of the questionnaires were completed in the researcher's presence. None of these participants were recruited through a doctor's practice and none of them had seen a doctor about back pain within the last six months. However, due to the number of missing values for all the other scales, and in order not to jeopardize the integrity of the other analyses, this subgroup was not included in the data analyses.

Table 3: Group frequencies

"back pain"	n = 154
"no back pain"	n = 116
Total	N = 270

Although the "no back pain" questionnaire did not include as many detailed questions about the pain experience chronicity and intensity of the pain experience were assessed. However, the questions regarding health behavior (e.g., exercise and smoking) and the personality scales remained the same and in the same order for the questionnaire groups "back pain" and "no back pain group". (The problems involved with order effect will be addressed in the Discussion section of this paper.)

3.3. Questionnaire Materials

Two versions of the questionnaire were distributed to the groups "back pain", "no back pain".

3.3.1. Questionnaire "back pain"

Pain history

The pain history scale includes an assessment of pain duration, i.e., whether the pain is acute or chronic; and a measurement of pain intensity on a 5-point Likert scale.

Comorbidities

The comorbidity scale developed for a large, longitudinal study taking place in the Personality Department at the University of Heidelberg (Amelang & Schmidt-Rathjens, 1993; Amelang, Schmidt-Rathjens & Matthews, 1996) requires participants to rate whether they have had (in the past) a particular disease, or if they are currently suffering from a particular disease, i.e., the participants give "yes" or "no" answers. The following table lists the assessed diseases.

Table 4: Comorbidity Scale: Assessed Diseases

Disease
Cancer
Heart disease
Diseases of the blood
Diabetes
Thymus dysfunction
Diseases of the liver
Allergies
Asthma
Arthritis
Depression
Heartburn
Ulcers
High blood pressure
Digestive complications
Other

General Health

Demographic characteristics such as: Age, gender, weight, height, physical exercise, diet, subjective health status, smoking habits and alcohol consumption were assessed through this scale. For the sake of coherency not all possible variables could be analyzed here. Only age, education, gender, smoking and physical exercise habits were determined to be relevant for the statistical analyses presented here. Age was given on a ratio scale; gender, physical exercise, and smoking habits on a nominal scale.

Causal Attribution Theories

This scale was originally designed to assess the lay person's ideas about the relationship between psychological factors and the onset of cancer and coronary heart disease (Schmidt-Rathjens, 1998). Relevant topics were found in the literature and the items were formulated by members of the Heidelberg research team for health issues. This scale is an adapted version of the scale developed by Schmidt-Rathjens (1998) to include back pain. Based on the review of the literature above, seven of the 16 items are known to be relevant for chronic back pain. For all three illnesses, participants rated the importance of the psychological factor for disease onset on a 5-point Likert scale; -2 = absolutely untrue, +2 completely true. The following table lists the 16 items of this scale, the seven items measuring back pain are marked with an asterisk.

Table 5: Items Assessing Causal Attributions for Disease Onset

Item	Text
1.*	Dissatisfaction with the work situation.
2.	Constantly overtaxed/constant stress.
3.	Disappointed about unattainable (life) goals.
4.*	Constantly recurring family problems.
5.	Recurring anxiety about the future.
6.*	Hopelessness and resignation.
7.	Time pressure and long-term stress.
8.*	Loss of a loved one through death or divorce.
9.	Frequent repression of rage and anger.
10.	Loneliness and isolation.
11.	Constant denial of one's own needs.
12.	Frequent repression of sadness and despondency.
13.*	High self-imposed expectations for achievement.
14.	Continual emotional instability and sensitivity..
15.*	Constant depressed mood.
16.*	Traumatic life events.

* Indicates which items measure back pain.

Sense of Coherence

The Sense of Coherence Scale used in this study is a short form of the German questionnaire (SOC-HD) developed by the Health Psychology Research Team for a large, longitudinal study taking place at the University of Heidelberg University. Based on previous factor analyses, low item test correlations (r_{it} correlations; cf. Amelang & Schmidt-Rathjens, 1993; Schmidt-Rathjens, 1998), and deletion of repetitious items (e.g., "I am an optimistic person." vs. "My outlook on life is generally very optimistic"), the original German version containing 26 items was reduced to the following 9 items in order not to over-tax pain patients participating in this study. (The item test correlations based on a sample of healthy n = 100 male and n = 100 female participants from Schmidt-Rathjens (1998) can be found in the

Appendix). The reliability estimate of the scale is $\alpha = 0.75$. Participants rated the items on a 5-point Likert scale; 0 = absolutely untrue, 4 = completely true.

Table 6: Sense of Coherence Scale (SOC)

Item	Text
1*	"I often don't understand how I got myself into these circumstances."
2*	"I often ask myself: Why is this happening to me?"
3	"I love life."
4	"I believe that I can accomplish any task set before me."
5*	"My life is chaotic, every day inexplicable things happen or I find myself in unexpected predicaments."
6	"With respect to my future, I'm very optimistic."
7 *	"I often don't understand why things turn out as they do."
8	"I am an optimistic person."
9	"In general, I have confidence in the skills and the goals of our politicians."

* indicates negatively poled items.

Depression

This depression scale from von Zerssen (1976) is well established in German speaking countries and is utilized to consistently assess clinical criteria of depression. Based on a sample of healthy men (n = 100) and women (n = 100), Schmidt-Rathjens (1998) selected 11 items that showed an r_{it}-correlation of at least .50 for both sample groups. These same 11 items were used in this study. (The item test correlations for the Depression Scale can be found in the Appendix). The reliability estimate for this scale is $\alpha = 0.87$. Here, participants rated items on a 4-point Likert scale; 0 = absolutely untrue, 3 = completely true.

Table 7: Depression Scale (von Zerssen, 1976)

Item	Text
1	”I’ve been anxious and jumpy lately.”
2	”I feel “down” and out of energy.”
3	“I understand a lot less written text than I used to.”
4	”I would consider killing myself.”
5	“I no longer have any deep relationships.”
6	“I feel like I’m about to fall apart.”
7	“I’m constantly anxious that I’ll say or do something wrong.”
8	“I am less interested in my love relationship(s) than I used to be.”
9	“I often feel simply miserable.”
10	”Even when I try, I can’t think straight.”
11	”I don’t have any emotions anymore.”

Job satisfaction

The job satisfaction scale (Neubauer, 2002) contains six items scored on a 5-point Likert scale; 0 = very infrequently, 4 = very frequently. The items are designed to measure job satisfaction on a continuum, i.e., the first item reflects a positive attitude towards work (see below), the middle items progressively less positive, and the fifth item measures a negative attitude towards the work situation, or job dissatisfaction. The last item assesses the work situation in general. The reliability estimate is $\alpha = 0.83$.

Table 8: Job Satisfaction Scale (Neubauer, 2002)

Item	Text
1	“I’m excited to get back to work after vacations.”
2	“I’m content with my work situation and I hope that it stays that way.”
3	”I don’t have much control over my work situation.”
4	”I’m not ecstatic about my job, but it could be worse.”
5	“I almost ready to quit. My work situation is no longer tolerable.”
6	“In general, how content are you with your job?”

Education

One item measures the educational level of study participants. Six levels of education are assessed through this item. For the statistical analyses, educational level was evaluated on a nominal scale "high" or "low" education. Based on the German educational system, completion of the "Abitur" is an indication that the participants intend to complete a college or university education. The distinction between participants with an "Abitur" (high) diploma versus participants without an "Abitur" (low) diploma has been made for the statistical analyses, since it also reflects the jobs markets available for these two groups.

Questionnaire for the Physician

Exaggerated Pain Experience

Based on Waddell's (1987) evaluation criteria to assess the concept of psychological "overlay" in chronic pain patients, five items were adapted for this study to test for abnormal tenderness, performance of the patient on tests simulating orthopedic maneuvers, performance on distraction tests (where a similar maneuver is performed two different ways or in two different positions), non-physiologic weakness or sensory disturbance, and overall tendency to exaggerate symptoms. Physicians rated patients responses on a nominal scale.

Table 9: Exaggerated Pain Experience

Item	Text
SKIN	"Is the skin sensitive in the lumbar spine area, or does the patient report pain when pressing upon the hip bone, Sacrum, or upper back?"
PRESS	"Does the patient report pain when applying light pressure to the top of the head when standing up?"
LIFT	"Does the patient report more pain when lifting the leg with an extended knee while sitting, than while lying down?"
NERVE	"Does the patient show motoric and sensory deficits that are hard to attribute to one or more nerve roots?"
OVER	„Does the patient tend to exaggerate the pain reaction or overreact?"

(adapted from Waddell, 1987)

Pain Origin Scale

Orthopedic doctors were asked to classify the cause of the patient's low back pain. 12 categories were presented including genetic malformation, herniated disc, post-operative pain, degeneration and psychosomatic causes.

3.3.2. Questionnaire "No Back Pain"

Participants completing the no back pain questionnaire responded to the following scales: pain history (general pain intensity and duration, not necessarily back pain), comorbidity, general health, causal attribution theories, sense of coherence, depression, job satisfaction, education. (Complete copies of both questionnaires can be found in the Appendix.)

3.4. Research Design

The research design is a cross-sectional, questionnaire study, involving self-reports on all of the scales except for the pain origin scale, in which orthopedic physicians responded to the items. As mentioned above, participants were recruited from five different doctors' offices, a procedure involving selection bias. This bias and other methodological problems are inherent in a study of this kind, and may jeopardize the internal and external validity.

3.5. Factors Jeopardizing Internal and External Validity.

While the internal validity allows for the interpretation of the results and is a basic minimum in research, external validity is a question of generalizability. To what populations, settings, treatment variables, and measurement variables can the results be generalized? Ideally, in choosing the research design, both types of validity should be maximized.

In their classic work, Campbell and Stanley (1963) discuss possible extraneous variables that could affect study findings. Only the variables relevant to the research design here will be discussed below.

History: The specific events occurring in the time the study was run.

This study: To control for this factor, this study was run during the winter months of 2001-2002, i.e., two months before and two months after the winter holidays, in order to ensure that the extraneous variable, for example weather, was held to a minimum. Nevertheless, an event like the September 11th incident, occurring two months before the study began, could not be controlled. It may be that participants were more depressed or hopeless in the fall than normal, and that their scores on the Sense of Coherence or depression scale may have been higher or lower if they had completed the questionnaire at another time.

Maturation: The processes within the respondents operating as a function of the passage of time per se (not specific to the particular events), including growing older, growing hungrier, growing more tired, etc.

This study: This factor plays a minimum role here since questionnaires were completed during normal business hours and the average time for filling out the questionnaire was approximately one hour; in addition, controls were equally affected. Nevertheless, this variable may be important with respect to self-reports about the duration and intensity of a pain experience since the reason for the visit to the doctor was pain.

Testing: The effects of filling out a questionnaire.

This study: Variables such as social desirability and issues of socialization (e.g., men do not admit to having pain) may effect scale scores. However, controls, are equally affected by these issues.

Biases: Resulting in differential selection of respondents from the comparison groups.

This study: This selection bias poses a problem in this study and the interpretation of the results. The most critical bias is that data were collected in an out-patient, clinical

setting. In addition, although the same instructions were given to each practice regarding recruitment criteria, there is no way of controlling whether the instructions were followed as given. Further, due to organizational factors, the participants were recruited in clusters, e.g., from all the patients who had an appointment on Thursday, the dice was thrown to determine which patient would be recruited. Or participants may have been more aggressively recruited at the beginning, than at the end of the data collection period. Factors which may compensate for the biases discussed above include: (1) The high rate of return, i.e., very few patients refused to complete the questionnaire; (2) In the German health system, patients are able to choose freely which doctor they would like to see and it is possible to change doctors in the middle of treatment; (3) The doctors' nurses did not know the hypotheses being tested.

The factors described above can affect scores obtained from the questionnaires. These extraneous variables were partially controlled in this study. Where no control was possible, then the interpretations of the results must be made taking into account these weaknesses.

The threats to external validity are referred to as interaction effects, involving the treatment and some other variable. These effects represent a potential specificity of the effects of the treatment to a limited set of conditions. Although no treatment per se is involved here, some of these factors still jeopardize the external validity (i.e., representativeness) of this study's results (Campbell & Stanley, 1963).

Interaction effects of selection biases and the study variables.

This study: Recruiting participants from a mid-sized semi-rural town may affect the representativeness of the sample. Since this town does not have an institution of higher education and the nearest university is over an hour away, it is relatively uncommon to collect data in waiting rooms in this part of Germany. Some participants may have found the items to be too personal, e.g., on the Depression Scale "I would consider killing myself.", and therefore may not have answered

openly. Some patients may have been concerned that their treatment would be affected by their ratings, although the instructions explicitly stated that treatment would not be affected.

Reactive effects of study arrangements, which would preclude generalization about the effect of the questionnaire upon persons being exposed to it in other settings.

This study: This study did not control for these kinds of effects although it is possible that a participant reported study details to a spouse, and that the spouse also came in the practice and was recruited by office staff.

4. RESULTS

As reported earlier in Aim of the Study, the problem low back pain is addressed from three distinct perspectives: The Biopsychosocial Model; the role of comorbidities, and causal attributions about disease onset. The data were analyzed according to these three organizing principles. However, when it seemed appropriate theoretically, for example, to test whether chronic back pain patients tend report other somatic symptoms (or comorbidities), the data covering comorbidities were combined in the statistical analyses of the biopsychosocial variables and of the causal attributions. The data analyses are divided into the following sections: Biopsychosocial Model (4.1) General description of the experimental and control groups; (4.2) Testing for differences between the experimental and control groups; (4.3) Explaining the variance in chronicity and intensity; (4.4) Exaggerated Pain Experience; (4.5) Comorbidities; (4.6) Causal attributions about disease onset; (4.7) Summary of the main results based on the research hypotheses. All data were analyzed through the SAS Statistical Package. An alpha level of 0.05 was used for all statistical tests reported below.

4.1. General Description of the Experimental and Control Groups

4.1.1. Sample Size, Mean Age, Education, Smoking Habits, Physical Exercise Habits.

The characteristics of the two participant groups, questionnaire "back pain" (experimental group) versus questionnaire "no back pain" (control group), will be described below.

92 German women and 62 German men (n = 154) with a mean age of 47 years (sd = 16.4) completed the questionnaire back pain; 68 German women and 48 German men (n = 116) with a mean age of 41.6 years (sd = 14.2) completed the questionnaire no back pain for a total participant sample of N = 270. The youngest participant was 15 years old, the oldest participant 73 years. A large majority of the participants had not completed their Abitur (n = 231), compared to n = 39 who had (shown in the table below as high education versus low education). N = 110 participants are non-smokers, n = 92 smokers, and n = 63 ex-smokers. 51 % of the

participant sample reported exercising more than 2 hours a week. The following table presents the frequencies for gender and education separately for the back pain group and the no back pain group.

Table 10 Group frequencies (N = 270)

Group	N	Females	Males	High Education	Low Education
Back Pain	154	92	62	26	128
No Back Pain	116	68	48	13	103
Total	270	160	110	39	231

4.1.2. Pain Related Description of the Participant Sample:

Chronicity and Intensity

Chronicity. From the total (*N* = 270), 70 participants reported suffering from chronic pain, i.e., pain duration of six months or more; 57 patients (back pain group) suffer from chronic back pain while data from 13 chronic back pain sufferers were collected from the no back pain questionnaire. A more differentiated picture of chronicity can be found in the table below.

Table 11: Chronicity of the Pain Experience

Group	N	< 1 W[1]	2-11 W[1]	3-6 M[2]	>6 M[2]
Back Pain	143	41	36	9	57
No Back Pain	77	32	26	6	13
Total	220*	73	62	15	70

Note: * Missing values = 50; [1] signifies weeks; [2] signifies months

Note: No mean values can be calculated for this item since it is rated on a nominal scale.

Intensity. Pain intensity was reported on a 5-Point-Likert Scale from 'no pain' to 'unbearable pain'. 43% of the patients reported extreme pain (ratings of 3-4), while almost half of the patients reported moderate pain (ratings of 1-2). The frequencies for intensity can be found below.

Table 12: Intensity of the Pain Experience: 5-Point-Likert Scale,
(0 = no pain, 4 = unbearable pain)

Group	N	Mean(SD)	0	1	2	3	4
Back Pain	146	2.36(0.95)	7	17	49	62	11
No Back Pain	91	2.05(1.07)	10	12	39	23	7
Total	237*	2.24(1.01)	17	29	88	85	18

Note: *Missing values = 33

4.1.3. Predictor Scales

The following table shows the sample sizes, means, standard deviations for the scales used in this study. The Sense of Coherence Scale (SOC) contains 8 items (Item 9 was removed to increase the alpha coefficient of the scale, see below); the Depression Scale contains 11 items; and the Job Satisfaction Scale contains 5 items (Item 3 was also removed to increase the reliability of the scale, see below). Scale items are presented above and as fold-outs in the Appendix.

Assessing Scale Reliability with Coefficient Alpha:. Scale reliability was assessed by calculating coefficient alpha (Cronbach, 1951). In order to increase the reliability of the scales Sense of Coherence and Job Satisfaction, one item from each scale was removed based on extremely low item-total correlations. For the Sense of Coherence Scale, with the removal of item 9 (”In general, I have confidence in the skills and the goals of our politicians”; r_{it} = 0.01), increased the reliability of the scale to 0.75, which is moderate and acceptable (cf. Schmidt-Rathjens, Benz, Van Damme, Feldt, & Amelang, 1997). For the Job Satisfaction Scale, with the removal of item 3 (”I don’t have much control over my work situation.”; r_{it} = 0.14), the reliability increased to 0.83. No single item to total correlation was extremely low for the Depression Scale.

Intercorrelations: Pearson correlation coefficient. Pearson product-moment correlation coefficients were calculated to assess the nature of the relationship between the questionnaire scales. Theoretically, the Sense of Coherence and job satisfaction were expected to act as protective factors, which would imply at least a positive correlation with each other, and a negative correlation with depression.

According to expectations, the Sense of Coherence Scale correlates significantly and negatively with the Depression Scale (r = -- 0.49; $p < 0.0001$). The correlation between the Sense of Coherence Scale and the Job Satisfaction Scale is slight, but positive and significant (r = 0.17; $p < 0.05$). The Depression Scale shows a significant, negative correlation with Job Satisfaction (r = -- 0.25; $p < 0.0005$).

Table 13: Means, Standard Deviations, Intercorrelations, and Coefficient Alpha Reliability Estimates for the Predictor Scales.

Variables	Mean (SD)	1	2	3
Sense of Coherence	2.76 (0.74)	(.75)		
Depression	0.37 (0.42)	-- 0.49[2]	(.87)	
Job Satisfaction	2.89 (0.86)	0.17[3]	--0.25[1]	(.83)

Note: [1] p < 0.0005; [2] p < 0.0001; [3] p < 0.05

Note: Reliability estimates appear on the diagonal, in parentheses.

Note: n = 207. Differences in group sizes due to missing values.

Predictor Scales and Pain Related Description: Logistic regression analyses were run on the binary criterion variables (e.g., chronic versus acute, high intensity versus low intensity) and the predictor scales Sense of Coherence, Depression, Job Satisfaction. Results are presented in the table below.

Table 14: Logistic Regression Analyses: Predictor Scales and Pain Description: Chronicity

Parameter	n	df	Wald Statistic	95% Wald Confidence Limits	
Sense of Coherence	263	1	0.46	0.80	1.66
Depression	258	1	7.01[1]	0.23	0.80
Job Satisfaction	210	1	1.24	0.85	1.78

Note: [1] $p < 0.01$

Table 15: Logistic Regression Analyses: Predictor Scales and Pain Description: Intensity

Parameter	n	df	Wald Statistic	95% Wald Confidence Limits	
Sense of Coherence	263	1	8.14[2]	1.17	2.34
Depression	258	1	10.29[2]	0.19	0.67
Job Satisfaction	210	1	2.32	0.93	1.79

Note: [2] $p < 0.005$

A significant relationship was found between high Depression Scale scores and chronicity (Wald Statistic = 7.01, $p < 0.01$) and between high Depression Scale scores and high intensity (Wald Statistic = 10.29, $p < 0.005$). No significant relationships were found between chronicity and the Sense of Coherence or between chronicity and job satisfaction. Analysis revealed a significant relationship between low Sense of Coherence Scores and high pain intensity (Wald Statistic = 8.14, $p < 0.005$). No significant relationship was found between pain intensity and the Job Satisfaction Scale.

Group Differences on the Predictor Scales: The predictor scales were initially tested for normality. Data collected for all three scales were found not to be normally distributed. Therefore, the mean differences between the two groups were tested through the Kruskal Wallis Test, a procedure for nonparametric data. Mean scores (and standard deviations) for the predictor scale, separate for back pain and the no back pain group can be found below.

Table 16: Mean scores, Standard Deviations for Group (Back Pain versus No Back Pain) on the Predictor Scales

Group	Mean Scores (SD) SOC Scale	Mean Scores (SD) Depression Scale	Mean Scores (SD) Job Satisfaction Scale
Back Pain	2.72 (0.75)	0.42[1] (0.43)	2.88 (0.91)
No Back Pain	2.79 (0.73)	0.31 (0.40)	2.90 (0.80)

Note: $p < 0.05$

Note: Back Pain Group: SOC scale n = 149; Depression scale n = 147; Job Satisfaction scale n = 113.

No Back Pain Group: SOC scale n = 114; Depression scale n = 111; Job Satisfaction scale n = 97.

Significant mean differences were found between the back pain and the no back pain groups on the Depression Scale (Kruskal-Wallis Test = 3.97; $p < 0.05$); this finding conforms to the research hypothesis, back pain patients had significantly higher Depression Scale scores than patients without back pain. Back pain patients also had

lower scores on the Sense of Coherence Scale and on the Job Satisfaction Scale, however, these differences did not reach significance.

On an item basis, significant mean differences were found on the Depression Scale items 8 "I am less interested in my love relationship(s) than I used to be." (Kruskal Wallis Test = 11.73; $p < 0.001$), 10 "Even when I try, I can't think straight." (Kruskal Wallis Test = 6.15; $p < 0.05$), and 11 "I don't have any emotions anymore." (Kruskal Wallis Test = 5.53; $p < 0.05$). No other significant mean differences were found on an item basis for the other two scales. (Please refer to the figures below.)

Figure 1: Sense of Coherence Scale: Mean Scores Between Groups[a]

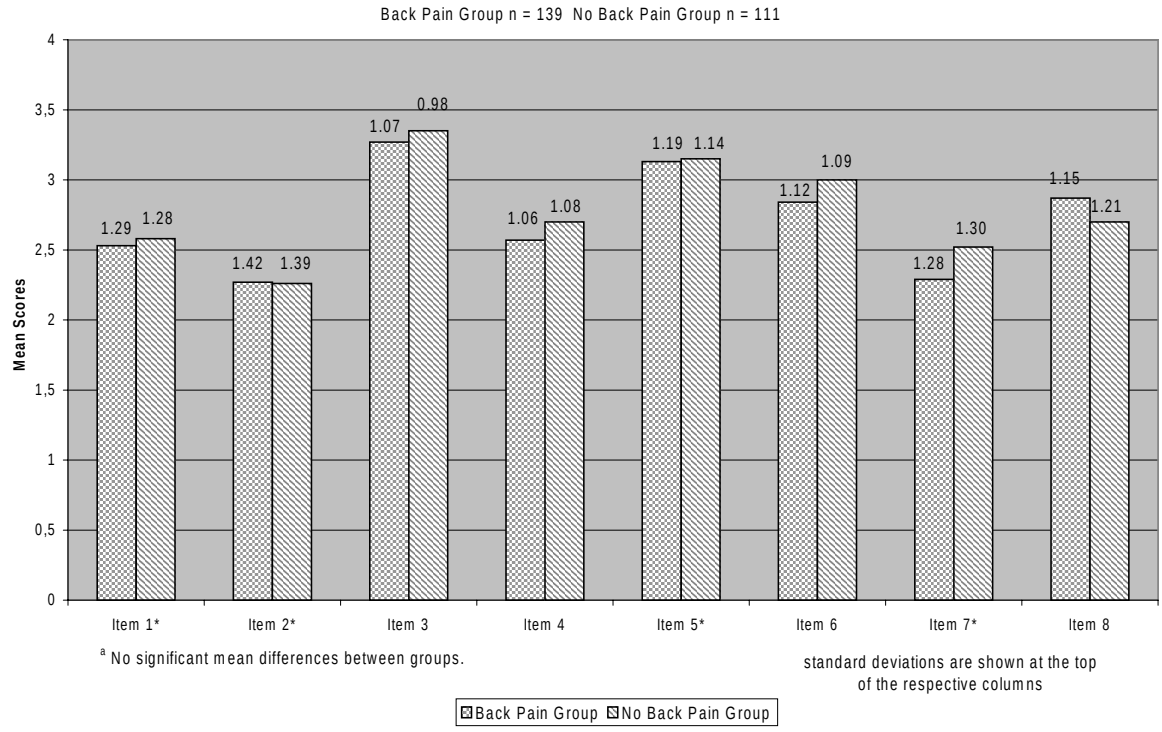
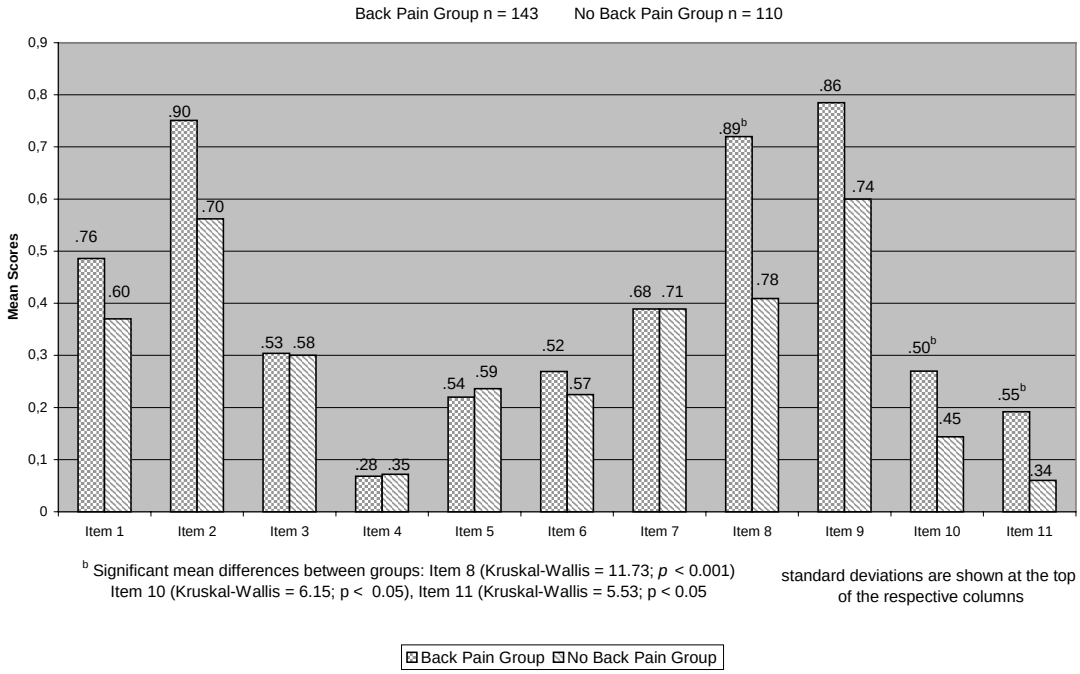


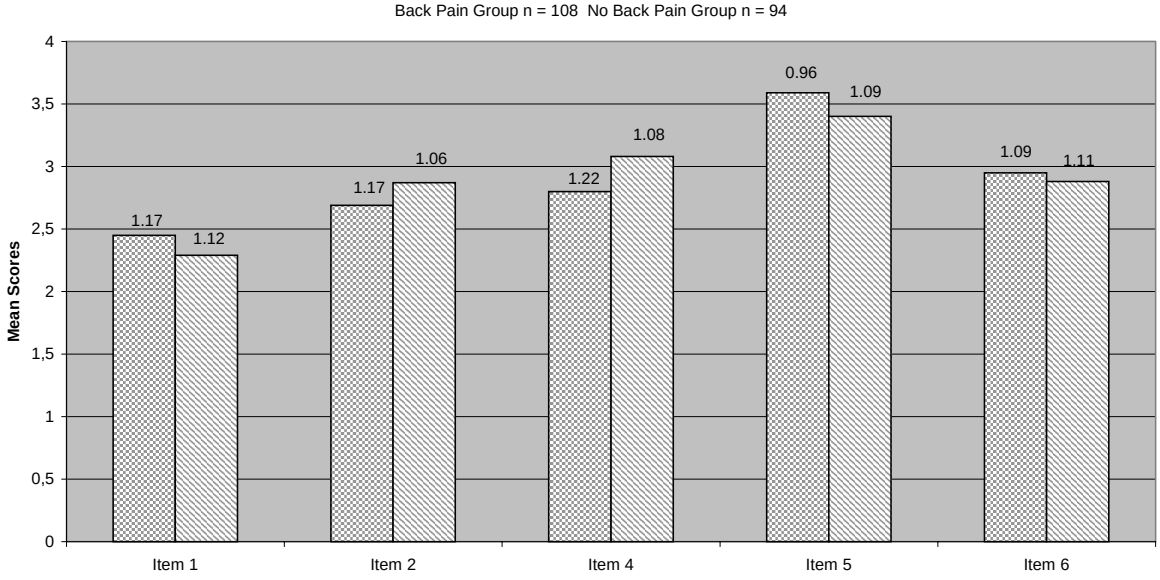
Figure 2: Depression Scale: Mean Scores Between Groups[b]



[b] Significant mean differences between groups: Item 8 (Kruskal-Wallis = 11.73; $p < 0.001$) Item 10 (Kruskal-Wallis = 6.15; p < 0.05), Item 11 (Kruskal-Wallis = 5.53; p < 0.05

standard deviations are shown at the top of the respective columns

Figure 3: Job Satisfaction Scale: Mean Scores Between Groups[a]



[a] no significant differences between groups

standard deviations are shown at the top of the respective columns

4.2. Testing for Differences Within and Between the Experimental and Control Groups

4.2.1. Chi-Square Test of Independence (Back Pain Group and No Back Pain Group, Together)

The predictor variables examined here are: age, gender, education, comorbidities-present, comorbidities-past, smoking, and physical exercise. The criterion variables are chronicity and intensity. Due to the number of Chi-Square Tests that have been calculated, only the significant results are reported in the table below. Significant Chi-Square Tests run between the predictor variables can be found in the Appendix.

Table 17: Summary Table of Significant Chi-Square Tests: Back Pain Group and No Back Pain Group, together

Variables	n	df	Chi-Square	p <
Chronicity and Intensity	212	1	4.40	0.05
Chronicity and Age	220	2	14.25	0.001
Chronicity and Education	220	1	4.55	0.05
Chronicity and Comorbidities, present	220	1	6.39	0.05
Chronicity and Comorbidities, past	220	1	7.62	0.01
Intensity and Gender	237	1	4.42	0.05
Intensity and Education	237	1	5.76	0.05
Intensity and Comorbidities, present	237	1	8.54	0.005

Note: The relationships between chronicity and gender, smoking, and physical exercise were not significant. Intensity and age, smoking, and physical exercise showed no significant relationships.

Interpretation.

For Chronicity and Intensity of the pain experience, most of the patients reported acute, low intensity pain (n = 84). In the acute pain group, 57% of the patients experience low intensity pain and 43% high intensity pain. In the chronic pain group 42% of the patients experience low intensity pain while 58% experience high intensity pain. Statement: Chronic pain patients tend to report higher intensity pain than acute pain patients.

Table 18: Chi-Square Frequency Table: Chronicity and Intensity

Frequency
(Row Pct)

	Low Intensity	High Intensity	Total
Acute	**84** (57%)	**63** (43%)	**147**
Chronic	**27** (42%)	**38** (58%)	**65**
Total	**111**	**101**	**212**

Chronicity and Age: The number of patients reporting acute pain remains relatively constant across the three age groups (32%, 33%, 35%, respectively). Another trend was found for chronic pain. In the chronic pain group, 14% of the patients are under 35 years of age, 24% are between 35 and 48 years of age, 61% of the patients over 48 years old. Statement: Chronic pain is reported more often by older patients (> 48 years of age) than by younger patients.

Table 19: Chi-Square Frequency Table: Chronicity and Age

Frequency
(Row Pct)

	< 35	35 — 48	> 48	Total
Acute	**48** (32%)	**49** (33%)	**53** (35%)	**150**
Chronic	**10** (14%)	**17** (24%)	**43** (61%)	**70**
Total	**58**	**66**	**96**	**220**

Chronicity and Education: A significant relationship was found between chronic pain and education. 94% of the chronic pain sufferers had received a lower education compared to 6% with a higher education. For acute pain, 16% of the participants came from the higher education participants, and 84% from the lower education participants. Statement: Participants with a lower education tend to report chronic pain more than participants with a higher education.

Table 20: Chi-Square Frequency Table: Chronicity and Education

Frequency (Row Pct)	Low Education	High Education	Total
Acute	**126** (84%)	**24** (16%)	**150**
Chronic	**66** (94%)	**4** (6%)	**70**
Total	**192**	**28**	**220**

Chronicity and Comorbidities, present: A significant relationship was found between chronicity and present comorbidities. For chronic pain patients, 64% reported comorbidities while 36% reported not suffering from another illness. 46% of the acute patients reported present comorbidities compared to 54% of the acute patients reported no comorbidities. Statement: Chronic pain patients tend to report present comorbidities.

Table 21: Chi-Square Frequency Table: Chronicity and Comorbidities, present

Frequency (Row Pct)	No Comorbidities	Comorbidities	Total
Acute	**81** (54%)	**69** (46%)	**150**
Chronic	**25** (36%)	**45** (64%)	**70**
Total	**106**	**114**	**220**

Chronicity and Comorbidities, past: A significant relationship was also found between chronicity and past comorbidities. In the chronic pain group, 57% of the participants reported past comorbidities while 43% reported no past comorbidities. 37% of the acute patients also reported suffering from past comorbidities, 63% of the acute patients reported no comorbidities. Statement: Chronic pain patients also tend to report past comorbidities.

Table 22: Chi-Square Frequency Table: Chronicity and Comorbidities, past

Frequency
(Row Pct)

	No Comorbidities	Comorbidities	Total
Acute	**94** (63%)	**56** (37%)	**150**
Chronic	**30** (43%)	**40** (57%)	**70**
Total	**124**	**96**	**220**

Intensity and Gender: There is a significant relationship between pain intensity ratings and gender. 68% of the participants experiencing high intensity pain are women, compared with 32% of the male participants. In the low intensity pain group, 54% of the women reported low intensity pain, while 46% of the men reported low pain intensity. Statement: Women report high intensity pain more than men.

Table 23: Chi-Square Frequency Table: Intensity and Gender

Frequency
(Row Pct)

	Female	Male	Total
Low Intensity	**73** (54%)	**61** (46%)	**134**
High Intensity	**70** (68%)	**33** (32%)	**103**
Total	**143**	**94**	**237**

Intensity and Education: Pain intensity and education are significantly related. In the low education group, participants report almost equally low and high intensity pain (53%, 47%, respectively). In the high education group, 76% of the participants report low intensity pain, 24% report high intensity pain. Statement: Participants with higher education tend to report low intensity pain. *Table 24: Chi-Square Frequency Table: Intensity and Education*

Frequency
(Col Pct)

	Low Education	High Education	Total
Low Intensity	**109** (53%)	**25** (76%)	**134**
High Intensity	**95** (47%)	**8** (24%)	**103**
Total	**204**	**33**	**237**

Intensity and Comorbidities, present: Sixty percent of the participants reporting high intensity pain also report present comorbidities, compared to 40% of the high intensity pain patients who report no comorbidities. This trend is reversed in the low intensity pain group, here 59% of the participants report no present comorbidities, while 41% report suffering from at least one present comorbidity. Statement: Participants reporting high intensity pain also report present comorbidities.

Table 25: Chi-Square Frequency Table: Intensity and Comorbidities, present

Frequency
(Row Pct)

	No Comorbidities	Comorbidities	Total
Low Intensity	**79** (59%)	**55** (41%)	**134**
High Intensity	**41** (40%)	**62** (60%)	**103**
Total	**120**	**117**	**237**

Summary: Chronic pain is significantly associated with high intensity pain. Significant relationships were also found between chronic pain sufferers and higher age, less education and reported comorbidities in the present and past. High intensity pain is significantly associated with the female gender, low education and present comorbidities.

4.2.2. Chi-Square Test of Independence

(Back Pain Group and No Back Pain Group, separate)

The following section examines the relationship between the criterion variables chronicity and intensity and the predictor variables (cf. 4.2.1.) separately for the back pain group and the no back pain group. Only in this section are the results directly related to *back pain*. The following table summarizes the significant chi-square results.

Table 26: Summary Table of Significant Chi-Square Tests:

Back Pain Group and No Back Pain Group, separate

Variables	Group	n	df	Value	p <
Chronicity and Age	Back Pain	143	2	7.33	0.05
Chronicity and Education	Back Pain	143	1	7.46	0.01
Chronicity and Comorbidities, past	No Back Pain	77	1	10.38	0.005
Intensity and Gender	Back Pain	146	1	5.55	0.05
Intensity and Education	Back Pain	146	1	9.77	0.005
Intensity and Comorbidities, present	No Back Pain	91	1	4.75	0.05
Intensity and Comorbidities, past	No Back Pain	91	1	5.48	0.05
Intensity and Smoking	No Back Pain	90	2	8.08	0.05

Note: No significant relationship was found between chronicity and intensity when the groups were examined separately. Chronicity and age show no significant relationship for the no back pain group. No significant relationship was found between chronicity and gender for either group. Chronicity was not related to education for the no back pain group. Smoking and physical exercise habits showed non-significant relationships with chronicity.

Note: No significant relationships were found between pain intensity and age for either group. Intensity and gender did not reach significance for the no back pain group. Education shares no significant relationship with intensity for the no back pain group. No significant relationship between intensity and comorbidities (past or present) exist for the back pain group. Intensity and smoking habits showed no

significant relationships with the back pain group. Physical exercise habits showed no significant relationship with intensity in either group.

Interpretation.

Chronicity and Age: A significant relationship was found between chronic *back* pain and age. 63% of the participants over 48 years of age report chronic back pain, 23% of the participants between 35 and 48 years of age reported chronic back pain, 14% of the patients under 35 years reported chronic back pain. In the acute pain group, 41% are over 48 years of age, 31% between 35 and 48 years, and 28% of the participants under 35 years reported acute back pain. Statement: Back pain patients over 48 years of age are more likely to report chronic pain than younger back pain patients.

Table 27: Chi-Square Frequency Table: Chronicity and Age (Back Pain Group)

Frequency
(Row Pct)

	< 35	35 – 48	>48	Total
Acute	**24** (28%)	**27** (31%)	**35** (41%)	**86**
Chronic	**8** (14%)	**13** (23%)	**36** (63%)	**57**
Total	**32**	**40**	**71**	**143**

Chronicity and Education: The relationship between chronic *back* pain and education is significant. In the high education group, 86% reported acute pain and 14% reported chronic pain. For those participants receiving less education, 55% reported acute pain compared to 45% who reported chronic pain. Statement: Back pain patients with a high education are less likely to report chronic pain relative to back pain patients with a low education.

Table 28: Chi-Square Frequency Table: Chronicity and Education (Back Pain Group)

Frequency
(Col Pct)

	Low Education	High Education	Total
Acute	**67** (55%)	**19** (86%)	**86**
Chronic	**54** (45%)	**3** (14%)	**57**
Total	**121**	**22**	**143**

Chronicity and Comorbidities, past: Eighty-five percent of the chronic pain sufferers tended to report past comorbidities, and 15% of those with chronic pain report no comorbidities. This trend was reversed for the acute pain patients; 64% tended to report no comorbidities, while 36% of them reported comorbidities. Statement: Chronic pain patients tend to report past comorbidities.

Table 29: Chi-Square Frequency Table: Chronicity and Comorbidities, past (No Back Pain Group)

Frequency (Row Pct)	No Comorbidities	Comorbidities	Total
Acute	**41** (64%)	**23** (36%)	**64**
Chronic	**2** (15%)	**11** (85%)	**13**
Total	**43**	**34**	**77**

Intensity and Gender: Sixty-eight percent of the women reported intense *back* pain, compared to 32% of the men. For low intensity *back* pain, 49% of the women reported low intensity *back* pain compared to 51% of the men. Statement: Women with back pain are more likely to report high intensity pain than men.

Table 30: Chi-Square Frequency Table: Intensity and Gender (Back Pain Group)

Frequency Row Pct	Females	Males	Total
Low Intensity	**36** (49%)	**37** (51%)	**73**
High Intensity	**50** (68%)	**23** (32%)	**73**
Total	**86**	**60**	**146**

Intensity and Education: Twenty-one percent of the participants with a higher education reported high intensity *back* pain, compared to 79% who reported low intensity back pain. In the low education group, just over half (56%) of the participants rated high intensity back pain, while 44% rated their *back* pain experience to be of less intense. Statement: Back pain patients with a higher education are more likely to report low intensity back pain than high intensity back pain.

Table 31: Chi-Square Frequency Table: Intensity and Education (Back Pain Group)

Frequency (Col Pct)	Low Education	High Education	Total
Low Intensity	**54** (44%)	**19** (79%)	**73**
High Intensity	**68** (56%)	**5** (21%)	**73**
Total	**122**	**24**	**146**

Intensity and Comorbidities, present: Forty-six percent of the participants reporting comorbidities also reported high intensity pain, 54% of the participants with comorbidities reported low intensity pain. For those participants reporting no comorbidities, 24% reported high intensity pain compared to 76% who reported low intensity pain. Statement: Participants reporting low intensity pain are more likely to report no present comorbidities.

Table 32: Chi-Square Frequency Table: Intensity and Comorbidities, present (No Back Pain Group)

Frequency (Col Pct)	No Comorbidities	Comorbidities	Total
Low Intensity	**41** (76%)	**20** (54%)	**61**
High Intensity	**13** (24%)	**17** (46%)	**30**
Total	**54**	**37**	**91**

Intensity and Comorbidities, past: For the participant group reporting general pain (no back pain group), forty-seven percent of the participants with past comorbidities reported high intensity pain, 53% percent reported low intensity pain. For those participants reporting no comorbidities, 24% had high intensity pain compared to 76% with low intensity pain. Statement: The same trend can be found with comorbidities, past, participants with low intensity pain are less likely to report past comorbidities.

Table 33: Chi-Square Frequency Table: Intensity and Comorbidities, past (No Back Pain Group)

Frequency (Col Pct)	No Comorbidities	Comorbidities	Total
Low Intensity	**42** (76%)	**19** (53%)	**61**
High Intensity	**13** (24%)	**17** (47%)	**30**
Total	**55**	**36**	**91**

Intensity and Smoking: Pain intensity and smoking showed a significant relationship with one another. Fifty-nine percent of the participants who reported high intensity pain were smokers, 28% were non-smokers, and 14% ex-smokers. 28% of the smokers reported less intense pain, 43% were non-smokers, and 18% ex-smokers. Statement: Smokers tend to report high intensity pain.

Table 34: Chi-Square Frequency Table: Intensity and Smoking (No Back Pain Group)

Frequency (Row Pct)	Ex-Smoker	Smoker	Non-Smoker	Total
Low Intensity	**18** (30%)	**17** (28%)	**26** (43%)	**61**
High Intensity	**4** (14%)	**17** (59%)	**8** (28%)	**29**
Total	**22**	**34**	**34**	**90**

Summary: As hypothesized, significant associations were found between chronic *back* pain and higher age and lower education. High intensity *back* pain was also significantly related to the female gender and low education. In the no back pain group, chronic pain and reported comorbidities in the past reached significance, high intensity pain and comorbidities (present and past) also reached significance. Counter to expectations, comorbidities (present nor past) do not have a significant association with *back* pain. High pain intensity and smoking were significantly associated only in the no back pain group.

4.2.3. Combined Variable: Chronicity + Intensity

Chi-Square Test of Independence

Among the chronic pain patients 46% reported low intensity pain while 54% reported high intensity. Compared with the acute pain patients, 33% rated their pain to be of high intensity; and 68% rated their pain to be of low intensity. Statement: Chronic pain patients tend to rate their pain experience to be of high intensity.

As reported above, the relationship between chronicity and intensity is statistically significant (Chi-Square = 10.43, df = 1, *p* (Fischer's Exact Test) < 0.005). Further, there is a nonsignificant, positive correlation between chronicity and intensity, (Pearson correlation, *r* = 0.20; *p* = 0.06).

Table 35 (cf. Table 18): Chi-Square Frequency Table: Chronicity and Intensity

Frequency
(Row Pct)

	Low Intensity	High Intensity	Total
Acute	**84** (57%)	**63** (43%)	**147**
Chronic	**27** (42%)	**38** (58%)	**65**
Total	**111**	**101**	**212**

Chronicity + Intensity formed a new variable in which participants rating high on both chronicity and intensity form the subgroup 1 (n = 38); participants rating low on both chronicity and intensity form the subgroup 2 (n = 135); participants rating either chronicity or intensity high (but not both) form the subgroup 3 (n = 97).

4.2.4. Logistic Regression Analysis: Chonicity + Intensity

Binary response variables (for example chronic or acute pain; high or low intensity pain) were established in this study. Logistic analyses were used to investigate the relationship between the response probability and the predictor variables. Variables with a significant effect on the criterion variable chronicity + intensity were established.

Logistic regression analyses were run on the data from the two extreme groups, Subgroup 1 ("high") and Subgroup 2 ("low"). The predictors variables age (< 35, 35

– 48, > 48 years of age), gender (female, male), education (high, low), comorbidities (present and past; yes, no), smoking (yes, no), physical exercise (< 2 hours weekly, > 2 hours weekly) depression (high, low), job satisfaction (high, low), and Sense of Coherence (high, low) were tested individually with the criterion variable: chronicity + intensity. When the variables reaching significance on an individual level were analyzed simultaneously, curiously, none of the models reached significance. The table below shows the results of the individual analyses. The confidence limits are given on the right hand side and the key to how the variables are coded can be found in the Appendix as a foldout.

Table 36: Logistic Regression Analyses: Combined Variable, Chronicity + Intensity

Parameter	n	df	Wald Stat.	95% Wald Confidence Limits	
Age	173	1	7.01[3]	1.19	3.14
Gender	173	1	4.53[1]	0.19	0.93
Education	173	1	4.19[1]	1.07	20.76
Comorbidities, present	173	1	6.20[1]	1.22	5.41
Comorbidities, past	173	1	3.98[1]	1.01	4.35
Smoking	171	1	1.32	--	--
Physical Exercise	156	1	2.68	--	--
Depression	166	1	10.55[2]	1.05	1.24
Job Satisfaction	136	1	5.41[1]	0.81	0.98
Sense of Coherence	169	1	0.04	--	--

Note: [1] = $p < 0.05$; [2] = $p < 0.005$; [3] = $p < 0.01$

Note: The relationships between the criterion variable Chronicity + Intensity and the biopsychosocial variables physical exercise, smoking habits, Sense of Coherence were not significant.

Summary: Higher age, the female gender, and low education are significantly associated with the combined variable chronicity + intensity. Present and past comorbidities are significantly associated with chronicity and high pain intensity. High scores on the Depression Scale are significantly related to chronicity and high

intensity pain ratings. Significant results were found for low Job Satisfaction Scores and chronicity and high intensity pain reports.

4.2.5. Testing for Significance

Predictor variables (e.g., age, gender, education) that reached significance when tested for significant associations on an individual basis were combined and then tested simultaneously for significance for each of the criterion variables. For the criterion variable chronicity, no combination of predictor variables reached significance when the variables were analyzed simultaneously. The same results were found for intensity, no model reached significance when the predictor variables were analyzed simultaneously. (Results for the combined variable: Chronicity + intensity can be found above.)

4.3. Explaining the Variance in Chronicity and Intensity

4.3.1. Logistic Regression Analysis: Chronicity

Logistic analyses were used to investigate the relationship between the response probability and the predictor variables. Variables with a significant effect on the criterion variable chronicity were established using logistic regression analyses with the model selections 'forward' and 'backward'. The following table shows the significant predictor variables for chronicity.

Table 37: Logistic Regression Analysis: Chronicity

Model Fit Statistic	N	df	Wald Statistic	p <	
Chronicity	270	3	20.51	0.0001	
				95% Wald	
Parameter	N	DF	Wald Statistic	Confidence Limits	
Age	270	1	8.95[2]	1.01	1.05
Education	270	1	4.43[1]	1.07	7.11
Comorbidities, past	270	1	6.26[3]	1.18	3.76

Note: [1] = $p < 0.05$; [2] = $p < 0.005$; [3] = $p < 0.01$

Summary: Significant effects on the criterion variable chronicity were found for higher age, lower education and comorbidities in the past.

4.3.2. Logistic Regression Analysis: Intensity

The results of the logistic analyses are presented in the table below.

Table 38: Logistic Regression Analysis: Intensity

Model Fit Statistic	N	df	Wald Statistic	p <
Intensity	238	3	18.01	0.0005

Parameter	N	DF	Wald Statistic	95% Wald Confidence Limits	
Exercise	238	1	4.59[1]	0.33	0.95
Depression	238	1	4.45[1]	1.01	1.14
Comorbidities, present	238	1	5.61[1]	1.13	3.52

Note: [1] = $p < 0.05$

Summary: Here the variables with a significant effect on the criterion variable intensity were low physical exercise, higher depression scores, and comorbidities in the present. Counter to expectations, none of the variables overlapped for the two criterion variables chronicity and intensity.

4.4. Exaggerated Pain Experience

4.4.1. Chi-Square Test of Independence: (Back Pain Group, n = 154)

As reported above, the physicians were requested to (1) assess the patients' tendency to exaggerate pain reports on five items and (2) report the patients' diagnosis. The items are presented above (cf. Method), a foldout can be found in the Appendix.

Analyses of the first five items of this scale showed one significant relationship between pain intensity and the item skin sensitivity ("skin sensitivity in the lumbar spine area, or reported pain when pressing upon the hip bone, Sacrum, or upper back", Chi-Square = 5.02, $p < 0.05$). 32% of the participants reporting skin sensitivity also reported low intensity pain compared to 68% of the participants with skin sensitivity who reported high intensity pain. For the back pain patient group not reporting skin sensitivity, 54% reported low intensity back pain relative to 46% reporting high intensity back pain. Statement: Back pain patients reporting skin sensitivity also tend to report high intensity pain.

Table 39: Chi-Square Frequency Table: Pain Intensity and Item Skin Sensitivity

Fequency (Col Pct)	No Skin Sensitivity	Skin Sensitivity	Total
Low Intensity	**55** (54%)	**12** (32%)	**67**
High Intensity	**47** (46%)	**25** (68%)	**72**
Total	**102**	**37**	**139**

Note: Frequency missing = 15

4.4.2. Assessing the Effect of the Predictor Variables on Physicians' Diagnoses

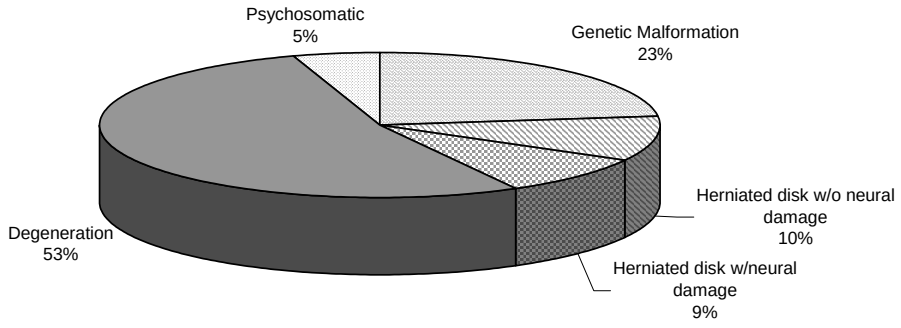
The second part of the items measuring an exaggerated pain reaction included the patients' diagnoses reported by their physicians. Of the 12 possible diagnoses, five diagnoses had a frequency equal to or above the cut-off value of n = 8. The frequencies for five diagnoses are presented in the table below (cf. Figure on the following page).

Table 40: Physicians' Diagnoses for the Cause of Patients' Back Pain

Diagnosis	n
Genetic malformation	39
Herniated disc without neural damage	17
Herniated disc with neural damage	16
Degeneration	92
Psychosomatic origin	8

Figure 4: Physician's Diagnoses of Back Pain Origin

n = 154



The binary variables gender (female, male), education (high, low), comorbidities (present, past; yes, no), smoker (ex-smoker, smoker, non-smoker), regular physical exercise (< 2 hours a week, >= 2 hours a week) were analyzed through 5 (Diagnoses) x 2 (Predictor Variable) Chi-Square Tests of Independence.

Gender: Analyses revealed a small, but significant relationship between gender and the diagnosis "psychosomatic origin" (Chi-square = 5.69, $p < 0.05$). 95% of the females with back pain did not receive the diagnosis "psychosomatic origin" from their physicians, 5% of the female back pain patients did receive this diagnosis for

the cause of their pain. None of the men were diagnosed with the cause of back pain being of psychosomatic origin. Statement: When the cause of back pain is diagnosed as psychosomatic, physicians tend to give this diagnosis more to women, than men. Please refer to the chi-square frequency table below.

Table 41: Chi-Square Frequency Table: Gender and Physicians' Diagnosis "Psychosomatic origin"

Frequency (Col Pct)	Female	Male	Total
Not this Diagnosis	**84**	**62**	**146**
	95%	100%	
Psychosomatic	**8**	**0**	**8**
	5%	0%	
Total	**92**	**62**	**154**

Results showed that gender and the diagnoses genetic malformation, herniated disc without neural damage, herniated disc with neural damage, and degeneration were all non significant.

Education: None of the chi-square tests reached significance for education.

Comorbidities, present: Analyses revealed non significant chi-square associations between present comorbidities and the physicians' diagnoses.

Comorbidities, past: None of the chi-square tests reached significance for past comorbidities.

Smoking: Non significant associations were found between smoking and the physicians' diagnoses.

Physical Exercise: Chi-square analyses revealed a non significant relationship between physical exercise and the physicians' diagnoses.

Summary: Chi-square analyses showed one significant association. When physicians determined the cause of back pain to be of psychosomatic origin, they tended to give

this diagnosis more to women, than men. None of the other Chi-Square analyses reached significance.

The variables measured on an interval scale (age, Sense of Coherence, depression, job satisfaction) were analyzed using factorial ANOVAs with 5 between groups factors(5 diagnoses). The analysis of the physicians' diagnoses and age revealed a significant main effect for "degeneration" ($F(1,148) = 21.40$; $p < 0.0001$). The back pain patients who received this diagnosis are significantly older than those who were not diagnosed with spinal degeneration. This finding conforms to biomedical considerations. Specifically, the nature of the diagnosis "degeneration" is simply wear-and-tear on the spine from years of, for example, bad posture or repetitive movements. None of the other diagnoses showed significant main effects. The following table summarizes the results.

Table 42: ANOVA Summary Table for Physicians' Diagnoses and Age

Source	df	Type III SS	Type III MS	F	R^2
Genetic Malformation	1	676.17	676.17	3.13	0.02
Herniated Disc (w/o)	1	6.60	6.60	0.03	0.00
Herniated Disc (w/)	1	138.66	138.66	0.64	0.00
Degeneration	1	4629.64	4629.64	21.40[1]	0.11
Psychosomatic	1	37.51	37.51	0.17	0.00

Note: $N = 154$; [1] $p < 0.0001$

The factorial ANOVAs for the physicians' diagnoses and the Scales Sense of Coherence ($F(5, 143) = 2.18$; $p < 0.06$), Depression ($F(5,141) = 0.40$; $p < 0.85$), and Job Satisfaction ($F(5, 107) = 0.60$; $p < 0.70$) were all non significant.

4.5. Comorbidities

4.5.1. Frequencies:

The figure on the following page shows the self-reported frequencies for comorbidities, present and past. The most important present comorbidities are allergies (*n* = 46), heart burn (*n* = 36), arthritis (*n* = 34), and digestive complications (*n* = 33). For past comorbidities, the same comorbidities were most important, but in a slightly different order: allergies (*n* = 39), digestive complications (*n* = 31), heart burn (*n* = 27), arthritis (*n* = 23). Like low back pain, these somatic symptoms are definitely irritating and make life uncomfortable, they are, however, not life threatening. The high frequencies are due to the fact that participants were instructed to mark one or more comorbidities, when applicable. No objective control was run to confirm these self-reported diagnoses (i.e., general practitioners were not requested to confirm the diagnoses).

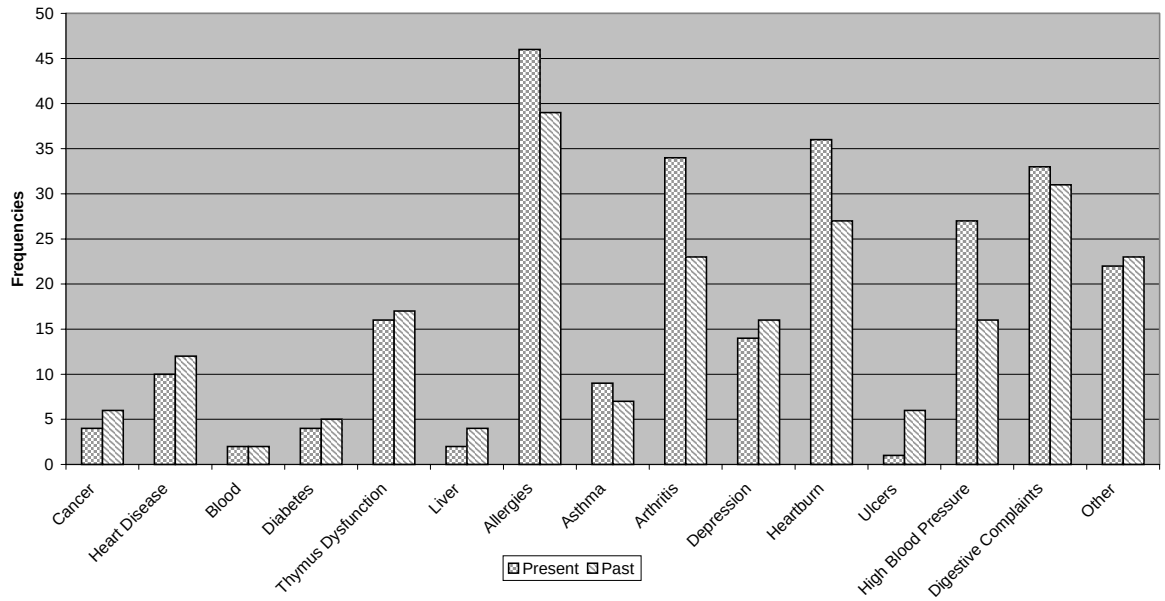
From the back pain group, *n* = 87 participants reported presently suffering from at least one other comorbidity, compared to *n* = 50 participants from the no back pain group. Self-reported comorbidities decline in the past; *n* = 68 past comorbidities for the back pain group, and for the no back pain group, *n* = 48.

Table 43: Frequency Table for Comorbidities in the present and in the past, separate for the back pain group and the no back pain group

Group	n	Present		Past	
		0	1 or more	0	1 or more
Back Pain	154	67	87	86	68
No Back Pain	116	66	50	68	48
Total	270	133	137	154	116

Figure 5: Comorbidites: Frequencies

Self-Reported Comorbidities*, Present n = 260, Past n = 234



* Participants were instructed to mark all comorbidities that applied, i.e., more than one answer was possible.

4.5.2. Chi-Square Test of Independence: Comorbidities, Present and Past (Back Pain and No Back Pain Groups, together)

As reported above (cf. 4.2.1.), significant chi-square results were found between comorbidities, present and chronicity and between comorbidities, past and chronicity. As chronic pain patients tend to report overall more comorbidities, pain patients with high intensity pain tend to report more present comorbidities. These relationships are also significant (cf. above). This trend did not appear for the comorbidities, past and intensity when both groups were analyzed together(nonsignificant association).

The relationship between age and comorbidities in the present is significant (Chi-square Statistic = 10.60, df = 2, $p < 0.005$), but not for comorbidities in the past. In accordance with expectations, pain patients younger than 48 years of age reported less frequently present comorbidities (under 35 years of age, 22%; between 35 and 48 years of age, 27%) than pain patients over 48 years of age (51%). In the group reporting no comorbidities, the number of subjects in each age group remained relatively equal.

Table 44: Chi-Square Frequency Table: Comorbidities, present and Age

Frequency (Col Pct) Col Pct	No Comorbidities	Comorbidities	Total
< 35	47 (35%)	30 (22%)	77
35 – 48	43 (32%)	37 (27%)	80
>48	43 (32%)	70 (51%)	113
Total	133	137	270

The relationships between comorbidities, present and past, and gender were not significant. No significant results were found between comorbidities (present and past) and education, physical exercise and smoking habits.

Summary:

In general, patients with chronic, high intensity pain and older patients (> 48 years of age) tend to report significantly more comorbidities, present and past, than patients with acute pain and younger patients when analyzing the two experimental groups together. Two exceptions to this trend were found for comorbidities in the past with intensity, and past comorbidities with age. No significant relationships were found between comorbidities (present or past) with gender, education, physical exercise, and smoking habits.

4.5.3. Chi-Square Tests of Independence: Comorbidities (Experimental and Control Groups, separate)

As reported above (cf. 4.2.2.), no significant relationships between comorbidities, present and past, and chronicity or between comorbidities, present and past, and intensity were found for the *back* pain group. However, in the no back pain group significant results were found between chronicity and past comorbidities and between intensity and comorbidities in the present and past.

Age and present comorbidities, present showed a significant relationship (Chi-square Statistic = 8.03, df = 2, $p < 0.05$) for the *back* pain group. Eighteen percent of the back pain patients under 35 years of age reported present comorbidities, 23% between the ages of 35 and 48 years, compared to 59% over 48 years of age. In the group of back pain patients with no comorbidities, the frequencies (and percentages) remain relatively equal. Statement: Older patients reported more comorbidities (n = 51) relative to younger ones.

Table 45: Chi-Square Frequency Table: Comorbidities, present + Age

(Back Pain Group)

Frequency (Col Pct)	No Comorbidities	Comorbidities	Total
< 35	21 (32%)	16 (18%)	37
35-48	22 (33%)	20 (23%)	42
>48	24 (36%)	51 (59%)	75
Total	66	87	153

No significant relationships were found for the no back pain group. Age and comorbidities, past showed no significant relationship. No significant results for the predictor variables: gender, education, smoking, physical exercise habits and comorbidities, past or present were found.

Summary: As predicted, significant results were found between higher age and present comorbidities in the back pain group. There were no significant results found between age and comorbidities, past in the back pain group. For the no back pain group, no significant results were found between comorbidities (present or past) and age.

4.5.4. Logistic Regression Analyses for Comorbidities

Logistic analyses were used to investigate the relationship between the response probability and the predictor variables. Variables with a significant effect on the variable comorbidities (present and past) were established using logistic regression analyses with the model selections 'forward' and 'backward'. The following table shows the significant predictor variables for comorbidities (present and past).

Table 46: Logistic Regression Analysis for Comorbidities

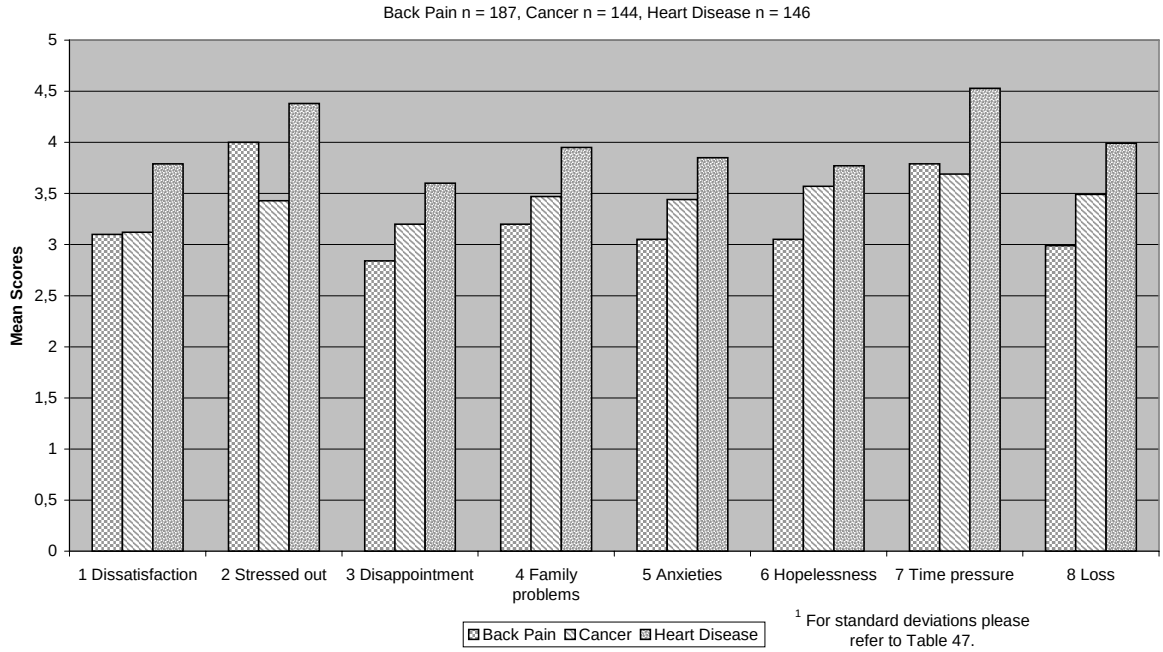
		Present[1]		Past[2]	
Model Fit Statistic	df	Wald Statistic		Wald Statistic	
Comorbidities	8	17.06[3]		9.15[2]	
		Present[a]		Past[b]	
Parameter	DF	Wald Stat.	Conf. Limits	Wald Stat.	Conf. Limits
Age	1	7.71[1]	1.01 1.06	4.75[2]	1.00 1.05
Depression	1	12.47[3]	1.07 1.28	5.59[2]	1.02 1.17

Note: [1] $p < 0.01$; [2] $p < 0.05$; [3] $p < 0.0005$

Note: [a] $n = 241$; [b] $n = 258$

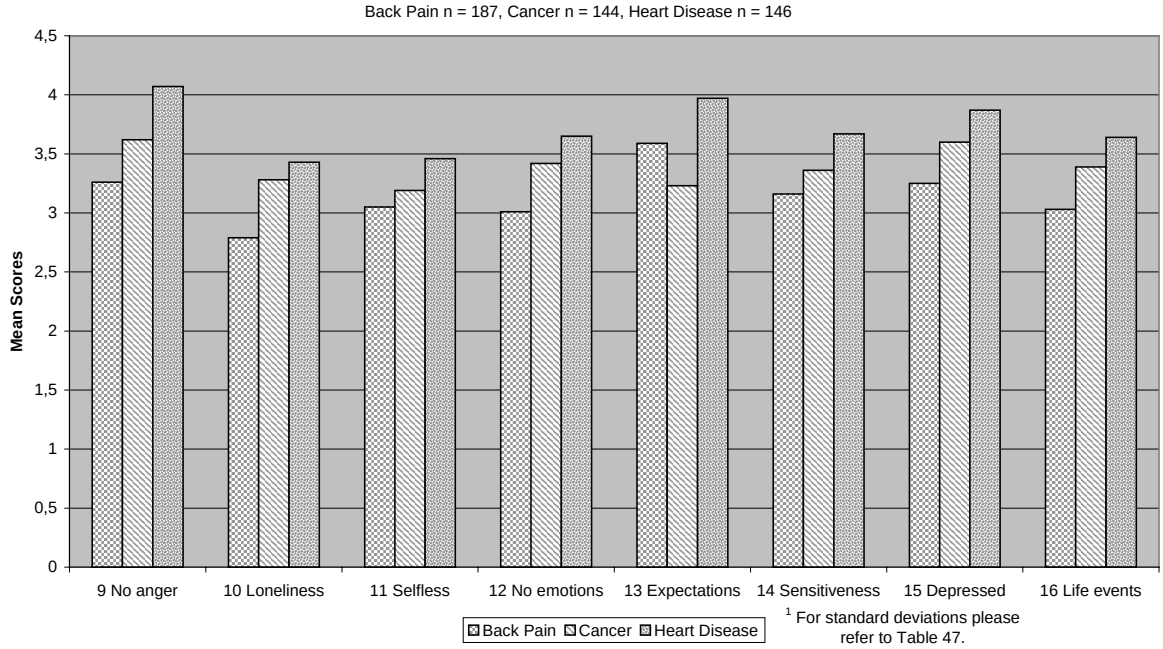
Summary: Significant effects on the criterion variables comorbidities, past and present, were found for higher age and higher Depression Scale scores. Both conform to expectations.

Figure 6: Causal Attributions About Disease Onset: Back Pain, Cancer, Heart Disease
Mean Scores[1] Items 1 - 8



[1] For standard deviations please refer to Table 47.

Figure 7: Causal Attributions About Disease Onset: Back Pain, Cancer, Heart Disease
Mean Scores[1] for Items 9 - 16



[1] For standard deviations please refer to Table 47.

4.6. Items Assessing Causal Attributions for Disease Onset

4.6.1. Means: Item Basis

On the Attribution Scale for disease onset, mean scores were consistently higher (and the standard deviations consistently lower) for heart disease than for cancer or back pain. Cancer, with the exception of items 1, 2, 7, 13, obtained the second highest mean scores while back pain (with the exceptions mentioned above) received the lowest mean scores. In other words, factors such as "Job dissatisfaction", "Recurring anxieties about the future", "Hopelessness and resignation", and "Constant stress and time pressure" are considered to be important variables for the onset of heart disease, more so than for the onset of cancer, or for the onset of back pain. Initially, this finding seems to contradict previous results in which a more differentiated perspective has been propagated. For example, "High expectations for oneself." and "Constant stress and time pressure." relate to heart disease; while "Inability to express fury and anger.", "Loneliness and loss of social contacts.", and "Constantly ignoring one's own needs." relate to cancer. For back pain onset, "Continuous depressed mood." and "Job dissatisfaction." have been important predictors in the past. The following table shows the means scores for each item, separately for each of the three diseases. The two figures on the preceding pages make clear the consistently higher means for heart disease. To enhance clarity, the standard deviations for the causal attribution item scores can be found in Table 47, not in the figures.

Table 47: Causal Attributions: Mean Scores and Standard Deviations reported on an item basis

Item	Mean Score(SD) Back Pain(n=187)	Mean Score(SD) Cancer(n=144)	Mean Score(SD) Heart Disease(n=146)
1* "Dissatisfaction"	3.10 (1.30)	3.12 (1.11)	3.79 (1.04)
2 "Stressed out"	4.00 (1.16)	3.43 (1.13)	4.38 (0.98)
3 "Disappointment"	2.84 (1.18)	3.20 (1.09)	3.60 (0.97)
4* "Family Problems"	3.20 (1.30)	3.47 (1.22)	3.95 (1.01)
5 "Anxieties"	3.05 (1.21)	3.44 (1.16)	3.85 (0.99)
6* "Hopelessness"	3.05 (1.23)	3.57 (1.16)	3.77 (0.95)
7 "Time pressure"	3.79 (1.18)	3.69 (1.14)	4.53 (0.86)
8* "Loss"	2.99 (1.26)	3.49 (1.19)	3.99 (1.02)
9 "No anger"	3.26 (1.20)	3.62 (1.13)	4.07 (0.91)
10 "Loneliness"	2.79 (1.21)	3.28 (1.11)	3.43 (0.97)
11 "Selfless"	3.05 (1.19)	3.19 (1.11)	3.46 (0.99)
12 "No emotions"	3.01 (1.16)	3.42 (1.05)	3.65 (0.94)
13* "Expectations"	3.59 (1.20)	3.23 (1.05)	3.97 (1.02)
14 "Sensitiveness"	3.16 (1.14)	3.36 (1.08)	3.67 (0.99)
15* "Depressed"	3.25 (1.25)	3.60 (1.13)	3.87 (0.96)
16* "Life events"	3.03 (1.23)	3.39 (1.10)	3.64 (0.96)

Note: * items measuring back pain.

4.6.2. Univariate Analyses: Items Assessing Causal Attributions About Onset of Back Pain, Cancer and Heart Disease and Comorbidities, present

Univariate analyses were run to investigate the relationship between the items assessing causal attribution and comorbidities, present (Means and standard deviations for the attribution items can be found in Table 47). There is a significant relationship between present comorbidities and the attributions for back pain items 1 "Job dissatisfaction", 4 "Constant problems in the family", 5 "Recurring anxieties about the future", 8 "Loss of a loved one through death or divorce", 9 "Inability to express fury and anger", 12 "Often not showing emotions such as sorrow and disappointment", 14 "Continuous emotional instability and sensitiveness". Analyses indicate that there is no significant relationship between the causal attributions for cancer and comorbidities in the present. Similarly, analyses showed that no significant relationships exist between causal attributions for heart disease and comorbidities in the present. Results are shown in the table below for all three diseases.

Table 48: Univariate Analyses Assessing the Relationship Between Causal Attribution About Onset of Back Pain, Cancer, Heart Disease and Comorbidities, present

	Back Pain	Cancer	Heart Disease
Parameter	Wald	Wald	Wald
Item 1 "Dissatisfaction"	5.90[1]	0.98	0.38
Item 2 "Stressed out"	0.07	0.00	0.77
Item 3 "Disappointment"	3.54	0.18	1.15
Item 4 "Family problems "	5.26[1]	0.04	0.00
Item 5 "Anxieties"	7.73[2]	0.02	0.52
Item 6 "Hopelessness"	0.93	0.29	1.21
Item 7 "Time pressure"	0.40	0.39	0.00
Item 8 "Loss"	3.91[1]	0.08	0.39
Item 9 "No anger"	5.21[1]	0.01	0.66
Item 10 "Loneliness"	3.32	0.11	0.37
Item 11 "Selfless"	1.95	0.13	3.62
Item 12 "No emotions"	3.86[1]	0.01	0.16
Item 13 "Expectations"	1.28	0.53	2.31
Item 14 "Sensitiveness"	4.19[1]	1.50	0.20
Item 15 "Depressed"	3.19	0.03	0.29
Item 16 "Life events"	1.21	0.07	0.11

Note: [1] $p < 0.05$, [2] $p < 0.01$; df = 1

Note: Back pain n = 232 – 214; Cancer n = 179 – 165; Heart disease n = 185 - 171

4.6.3. Univariate Analyses: Items Assessing Causal Attributions About Onset of Back Pain, Cancer and Heart Disease and Comorbidities, past

The relationship between causal attributions for disease onset (back pain, cancer, heart disease) and comorbidities in the past were assessed through univariate analyses. (Means and standard deviations for the attribution items can be found in Table 47.) In this series of tests, only Item 12 "Often not showing emotions such as sorrow and disappointment" reached significance for the back pain attributions. Items 6 "Hopelessness and resignation" and 8 "Loss of a loved one through death or divorce" reached significance for the heart disease attributions. No significant relationship was found between comorbidities in the past and causal attributions about onset of cancer. The following table presents results of the analyses between causal attributions about disease onset and past comorbidities.

Table 49: Univariate Analyses Assessing the Relationship Between Causal Attribution About Onset of Back Pain, Cancer, Heart Disease and Comorbidities, past

Parameter	Back Pain Wald	Cancer Wald	Heart Disease Wald
Item 1 "Dissatisfaction"	1.81	0.16	0.00
Item 2 "Stressed out"	0.22	0.02	0.00
Item 3 "Disappointment"	0.13	0.67	0.70
Item 4 "Family problems"	3.04	0.69	0.55
Item 5 "Anxieties"	3.49	0.17	0.15
Item 6 "Hopelessness"	0.65	2.70	6.08[1]
Item 7 "Time pressure"	0.04	1.52	0.14
Item 8 "Loss"	0.51	0.51	4.26[1]
Item 9 "No anger"	0.95	0.01	0.01
Item 10 "Loneliness"	3.69	0.19	0.39
Item 11 "Selfless"	1.83	0.00	1.64
Item 12 "No emotions"	5.47[1]	0.01	0.03
Item 13 "Expectations"	0.06	0.10	0.18
Item 14 "Sensitiveness"	3.61	0.00	0.00
Item 15 "Depressed"	2.67	0.99	1.48
Item 16 "Life events"	1.66	0.92	0.74

Note: [1] $p < 0.05$; df = 1

Note: Back pain n = 232 – 214; Cancer n = 179 – 165; Heart disease n = 185 - 171

Summary: In comparison to comorbidities in the past (one significant relationship), causal attributions about back pain and comorbidities in the present showed seven significant relationships. The items "Job dissatisfaction" (Item 1), "Constant problems in the family" (Item 4), "Loss of a loved one through death or divorce" (Item 8) have been shown to be related to back pain in past research. "Recurring anxieties about the future" (Item 5), "Inability to express fury and anger" (Item 9), "Often not showing emotions such as sorrow and disappointment" (Item 12), and "Continuous emotional instability and sensitiveness" (Item 14) have not been associated with back pain onset in the past. Here these items are significantly related to

comorbidities in the present and causal attributions about back pain onset. Only Item 12 "Often not showing emotions such as sorrow and disappointment" reached significance for comorbidities in the past and causal attribution about back pain onset. Significant relationships between comorbidities in the past and causal attributions about heart disease were found for "Hopelessness and resignation" (Item 6) and "Loss of a loved one through death or divorce" (Item 8). These items (6, 8) are most often associated with onset of cancer. No significant relationships were found for causal attribution about cancer onset and comorbidities, present or past.

4.6.4. Principal Components Analysis

Responses to the 16-items assessing causal attribution for disease onset (back pain, cancer, heart disease) were subjected to a principal component analysis using ones as prior communality estimates. The principal axis method was used to extract the components, and this was followed by a varimax (orthogonal) rotation.

Back Pain

Only the first two components displayed eigenvalues greater than 1; these first two components were retained for the rotation. Combined, components 1 and 2 accounted for 65% of the total variance.

Questionnaire items and corresponding factor loadings are presented in the table below. In interpreting the rotated factor pattern, an item was said to load on a given component if the factor loading was .40 or greater for that component, and was less than .40 for the other. Using these criteria, seven items were found to load on the first component, which was subsequently labeled the negative affect component. Three items loaded on the second component, which was labeled the pressure component.

Table 50: Rotated Factor Pattern and Final Communality Estimates from Principal Component Analysis of Causal Attributions for the Onset of Back Pain

	Component		
Items*	**1** 'negative affect'	**2** 'pressure'	h^2
2: Stressed Out	0.17	**0.77**	0.62
3: Disappointment	**0.78**	0.18	0.64
4: Family Problems	**0.73**	0.39	0.69
5: Anxieties	**0.76**	0.35	0.70
6: Hopelessness	**0.85**	0.23	0.78
7: Time Pressure	0.33	**0.71**	0.62
8: Loss	**0.69**	0.41	0.64
10: Loneliness	**0.83**	0.15	0.71
11: Selfless	**0.69**	0.31	0.57
13: Expectations	0.19	**0.81**	0.70

Note: $N = 187$. Communality estimates appear in column headed h^2.
*The full text for the items can be found in the Appendix as a fold-out.
Note: loadings >.50 are emphasized in bold print.

Cancer

Only one component showed an eigenvalue greater than 1 and this component accounted for 70% of the total variance. Items 1 through 15 loaded high on this one component. Since rotation is not possible with one component, no further results will be reported for the items assessing causal attribution for the onset of cancer.

Heart Disease

Eigenvalues greater than 1 were found for the first two components; these components were retained for rotation. Sixty-two percent of the total variance is accounted for by components 1 and 2.

As described above, items and corresponding factor loadings are presented in the table below. Here, too, factor loadings of .40 were used as inclusion criteria. Six items loaded on the first component which was labeled emotional withdrawal. Four items loaded on the second component; it was labeled stress.

Table 51: Rotated Factor Pattern and Final Community Estimates from Principal Component Analysis of Causal Attribution for the Onset of Heart Disease Component

Items*	1 'emotional withdrawal'	2 'stress'	h^2
1: Dissatisfaction	0.22	**0.76**	0.63
2: Stressed Out	0.33	**0.75**	0.67
4: Family Problems	0.26	**0.88**	0.84
5: Anxieties	0.34	**0.78**	0.72
10: Loneliness	**0.73**	0.30	0.62
11: Selfless	**0.73**	0.29	0.62
12: No Emotions	**0.79**	0.29	0.71
13: Expectations	**0.66**	0.26	0.51
14: Sensitiveness	**0.81**	0.22	0.71
15: Depressed	**0.72**	0.39	0.68

Note: $N = 146$. Communality estimates appear in column headed h^2.
*The full text for the items can be found in the Appendix as a fold-out.
Note: loadings >.50 are emphasized in bold print.

Summary: Results from the Principal Component Analysis of *back pain* attributions showed factor loadings on two components: (1) "*negative affect*"(including Items 3 "disappointment", 4 "family problems", 5 "anxieties". 6 "hopelessness", 8 "loss", 10

"loneliness", 11 "selfless"), and (2) "*pressure*" (including Items 2 "stressed out", 7 "time pressure", 13 "expectations".

The Principal Component Analysis of *heart disease* attributions also showed loadings on two components: (1) "*emotional withdrawal*" (including Items 10 "loneliness", 11 "selfless", 12 "no emotions", 13 "expectations", 14 "sensitiveness", 15 "depressed"), (2) "*stress*" (including Items 1 "dissatisfaction", 2 "stressed out", 4 "family problems", 5 "anxieties").

This analysis provided little information regarding causal attributions for the onset of cancer.

Six items overlapped for onset of back pain and of heart disease: Items 2 "stressed out", 4 "family problems", 5 "anxieties", 10 "loneliness", 11 "selfless", 13 "expectations". The items comprising components 1 and 2 for both diseases showed certain similarities; it seems that the items loading on the first component involve loneliness and depression, while the second component is comprised of items assessing stress.

4.6.5. Analyses of Variance: 2 (Group) x 3 (Disease)

2 (GROUP) x 3 (DISEASE) Analyses of Variance with Repeated Measures, Separate for ITEM

a. Statement of the problem: The purpose of this analysis was to determine whether there was a difference between mean scores for back pain, cancer, and heart disease (cf. Table 47) with respect to causal attributions for disease onset on an item basis.

b. Nature of the variables: This analysis involved three variables. One predictor variable was the item, for example item 2: "Disappointment over unaccomplished (life) goals.". Another predictor variable was the group, which was measured on a nominal scale, back pain group and no back pain group. The criterion variable represented the participant's causal attributions to disease onset, which was measured on an interval scale.

c. Statistical test: Two-Group Experimental Design with Repeated Measures.

d. Null Hypothesis: M1 = M2 = M3; In the population, there is no difference between groups (back pain, no back pain), or between diseases (back pain, cancer, heart disease) with respect to their mean attribution scores.

e. Alternative Hypothesis: In the population, there is a difference between back pain patients and no back pain patients on their mean attribution scores. And there is a difference between diseases: cancer and heart disease (because they are life-threatening) and back pain.

f. Obtained statistic: see below.

Testing for Effects

Interaction Effect: With non significant results the null hypothesis is maintained and it can be concluded that there is no ITEM * GROUP interaction. A *significant interaction* was found for only Item 16 "Traumatic life event(s)".

Group Effect (Between Subjects): Nonsignificant results indicate that there were no overall differences between the experimental (back pain group) and control group (no back pain) with respect to their mean attribution scores. Only one item showed a significant group effect. Analyses showed that the back pain group had significantly higher mean scores than the no back pain group ($F(1, 161) = 6.38$, $p < 0.05$) for Item 2 ("Constantly being stressed out.").

Item Effect (Within Subjects): A main effect for ITEM would suggest that there was a significant difference between attribution scores obtained for back pain, cancer, and heart disease. All of the personality variables of the Causal Attribution Scale showed a *significant item effect*. Mean scores and standard deviations are given in parentheses (M(sd)). The reference scale is always back pain, i.e., significant differences are reported for the comparisons: back pain and cancer, back pain and heart disease.

Item 1: Significant mean differences were found between back pain (3.10(1.30)) and the heart disease (3.79(1.04)) for Item 1 ("Job dissatisfaction.").
Item 2 ("Constantly being stressed out."): Significant mean differences were shown between back pain (4.00(1.16)), cancer (3.43(1.13)), and heart disease scale (4.38(0.98)).
Item 3: The same holds true for Item 3 ("Disappointment over unaccomplished (life) goals."), analyses showed significant mean differences between back pain (2.84(1.18)), cancer (3.20(1.09)), and heart disease (3.60(0.97)).
Items 4, 5, 6: Significant mean differences between back pain, cancer, and heart disease were also found for Item 4 ("Constant problems in the family."; back pain 3.20(1.30), cancer 3.47(1.22), heart disease 3.95(1.01), Item 5 ("Recurring anxieties about the future."; back pain 3.05(1.21), cancer 3.44(1.16), heart disease 3.85(0.99), and Item 6 ("Hopelessness and resignation.", back pain 3.05(1.23), cancer 3.57(1.16), heart disease 3.77(0.95).
Item 7 ("Constant stress and time pressure."): This item did not show the same trend; significant differences were calculated between back pain (3.79(1.18)) and heart disease (4.53(0.86)).
Items 8, 9, 10: Significant mean differences were found between back pain, cancer, and heart disease for the Items 8 ("Loss of a loved one through death or divorce.", back pain 2.99(1.26), cancer 3.49(1.19), heart disease 3.99(1.02), 9 ("Inability to express fury or anger.", back pain 3.26(1.20), cancer 3.62(1.13); heart disease 4.07(0.91)), and 10 ("Loneliness ad loss of social contacts.", back pain 2.79(1.21), cancer 3.28(1.11), heart disease 3.43(0.97)).
Item 11 ("Constantly ignoring one's own needs."): This item showed significant mean differences for only back pain (3.05(1.19)) and heart disease (3.46(0.99)).

Items 12, 13: Analyses showed significant mean differences between back pain, cancer, and heart disease for Item 12 ("Often not showing emotions such as sorrow and disappointment.", back pain 3.01(1.16), cancer 3.42(1.05), heart disease 3.65(0.94)), and for Item 13 ("High expectations for oneself.", back pain 3.59(1.20), cancer 3.23(1.05), heart disease 3.97(1.02)).
Item 14: Differences between back pain (3.16(1.14)) and heart disease (3.67(0.99)) reached significance for Item 14 ("Continuous emotional instability and sensitiveness.").
Item 15: The same is true for Item 15 ("Continuous depressed mood."): significant differences were found between back pain (3.25(1.25)) and heart disease (3.87(0.96)).
Item 16 ("Traumatic life event(s)."): Significant differences were found between back pain (3.03(1.23)), cancer (3.39(1.10)), and heart disease (3.64(0.96)).

Contrasts: Mean scores for attributions on back pain are compared with mean scores for cancer and heart disease.

Table 52: ANOVA Summary Table for Causal Attributions for Disease Onset: Item 1 "Job dissatisfaction." (Nonsignificant Interaction)

Source	df	SS	MS	F
Between Subjects	167	384.76		
Group (A)	1	0.82	0.82	0.36
Residual between	166	383.94	2.31	
Within Subjects	336	321.12		
Item (B)	2	48.00	24.00	29.55***
Item*Group (A*B)	2	3.43	1.72	2.11
Residual within	332	269.69	0.81	
Total	503	705.88		

Note: $N = 168$; *** $p < .0001$

Table 53: ANOVA Summary Table for Causal Attributions for Disease Onset: Item 2 "Constantly being stressed out." (Nonsignificant Interaction)

Source	df	SS	MS	F
Between Subjects	162	373.71		
Group (A)	1	14.25	14.25	6.38*
Residual between	161	359.46	2.23	
Within Subjects	326	264.36		
Item (B)	2	56.84	28.42	44.29***
Item*Group (A*B)	2	0.91	0.45	0.71
Residual within	322	206.61	0.64	
Total	488	638.07		

Note: $N = 163$; * $p < .01$, *** $p < .0001$

Table 54: ANOVA Summary Table for Causal Attributions for Disease Onset: Item 3 "Disappointment over unaccomplished (life) goals." (Nonsignificant Interaction)

Source	df	SS	MS	F
Between Subjects	165	367.25		
Group (A)	1	1.94	1.94	0.87
Residual between	164	365.31	2.22	
Within Subjects	332	219.91		
Item (B)	2	33.86	16.23	29.89***
Item*Group (A*B)	2	0.29	0.14	0.25
Residual within	328	185.76	0.57	
Total	497	587.16		

Note: $N = 166$; *** $p < .0001$

Table 55: ANOVA Summary Table for Causal Attributions for Disease Onset: Item 4 "Constant problems in the family." (Nonsignificant Interaction)

Source	df	SS	MS	F
Between Subjects	168	496.14		
Group (A)	1	2.97	2.97	1.01
Residual between	167	493.17	2.95	
Within Subjects	338	240.77		
Item (B)	2	34.10	17.05	27.67***
Item*Group (A*B)	2	0.87	0.43	0.71
Residual within	334	205.80	0.61	
Total	506	736.91		

Note: $N = 169$; *** $p < .0001$

Table 56: ANOVA Summary Table for Causal Attributions for Disease Onset: Item 5 "Recurring anxieties about the future." (Nonsignificant Interaction)

Source	df	SS	MS	F
Between Subjects	164	464.68		
Group (A)	1	4.73	4.73	0.20
Residual between	163	459.95	2.82	
Within Subjects	330	189.29		
Item (B)	2	38.85	19.42	42.18***
Item*Group (A*B)	2	0.32	0.16	0.35
Residual within	326	150.12	0.46	
Total	494	653.97		

Note: $N = 165$; *** $p < .0001$

Table 57: ANOVA Summary Table for Causal Attributions for Disease Onset: Item 6 "Hopelessness and resignation." (Nonsignificant Interaction)

Source	df	SS	MS	F
Between Subjects	164	425.71		
Group (A)	1	0.09	0.09	0.03
Residual between	163	425.62	2.61	
Within Subjects	330	175.60		
Item (B)	2	23.64	11.82	25.47***
Item*Group (A*B)	2	0.70	0.35	0.76
Residual within	326	151.26	0.46	
Total	494	601.31		

Note: $N = 165$; *** $p < .0001$

Table 58: ANOVA Summary Table for Causal Attributions for Disease Onset: Item 7 "Constant stress and time pressure." (Nonsignificant Interaction)

Source	df	SS	MS	F
Between Subjects	165	323.92		
Group (A)	1	3.66	3.66	1.88
Residual between	164	320.26	1.95	
Within Subjects	332	287.28		
Item (B)	2	59.07	29.53	42.56***
Item*Group (A*B)	2	0.59	0.29	0.42
Residual within	328	227.62	0.69	
Total	497	611.20		

Note: $N = 166$; *** $p < .0001$

Table 59: ANOVA Summary Table for Causal Attributions for Disease Onset: Item 8 "Loss of a loved one through death or divorce." (Nonsignificant Interaction)

Source	df	SS	MS	F
Between Subjects	168	487.36		
Group (A)	1	8.11	8.11	2.83
Residual between	167	479.25	2.87	
Within Subjects	332	273.37		
Item (B)	2	66.66	33.33	54.39***
Item*Group (A*B)	2	2.03	1.01	1.65
Residual within	328	204.68	0.61	
Total	500	760.73		

Note: $N = 169$; *** $p < .0001$

Table 60: ANOVA Summary Table for Causal Attributions for Disease Onset: Item 9 "Inability to express fury and anger." (Nonsignificant Interaction)

Source	df	SS	MS	F
Between Subjects	167	359.99		
Group (A)	1	1.46	1.46	0.68
Residual between	166	358.53	2.16	
Within Subjects	336	256.17		
Item (B)	2	44.63	22.32	35.49***
Item*Group (A*B)	2	2.80	1.40	2.23
Residual within	332	208.74	0.63	
Total	503	616.16		

Note: $N = 168$; *** $p < .0001$

Table 61: ANOVA Summary Table for Causal Attributions for Disease Onset: Item 10 "Loneliness and loss of social contacts." (Nonsignificant Interaction)

Source	df	SS	MS	F
Between Subjects	167	431.21		
Group (A)	1	0.04	0.04	0.02
Residual between	166	431.17	2.60	
Within Subjects	336	168.21		
Item (B)	2	15.47	7.73	16.87***
Item*Group (A*B)	2	0.54	0.27	0.59
Residual within	332	152.20	0.46	
Total	503	599.42		

Note: $N = 168$; *** $p < .0001$

Table 62: ANOVA Summary Table for Causal Attributions for Disease Onset: Item 11 "Constantly ignoring one's own needs." (Nonsignificant Interaction)

Source	df	SS	MS	F
Between Subjects	171	483.37		
Group (A)	1	5.27	5.27	1.87
Residual between	170	478.10	2.81	
Within Subjects	336	147.87		
Item (B)	2	11.29	5.64	14.07***
Item*Group (A*B)	2	0.15	0.07	0.19
Residual within	332	136.43	0.40	
Total	507	631.24		

Note: $N = 172$; *** $p < .0001$

Table 63: ANOVA Summary Table for Causal Attributions for Disease Onset: Item 12 "Often not showing emotions such as sorrow and disappointment." (Nonsignificant Interaction)

Source	df	SS	MS	F
Between Subjects	162	350.51		
Group (A)	1	0.67	0.67	0.31
Residual between	161	349.84	2.17	
Within Subjects	326	198.37		
Item (B)	2	31.32	15.66	30.65***
Item*Group (A*B)	2	2.53	1.27	2.48
Residual within	322	164.52	.0.51	
Total	488	548.88		

Note: $N = 163$; *** $p < .0001$

Table 64: ANOVA Summary Table for Causal Attributions for Disease Onset: Item 13 "High expectations for oneself." (Nonsignificant Interaction)

Source	df	SS	MS	F
Between Subjects	165	427.11		
Group (A)	1	7.18	7.18	2.80
Residual between	164	419.93	2.56	
Within Subjects	332	219.16		
Item (B)	2	41.24	20.62	38.34***
Item*Group (A*B)	2	1.51	0.75	1.40
Residual within	328	176.41	0.54	
Total	497	646.27		

Note: $N = 166$; *** $p < .0001$

Table 65: ANOVA Summary Table for Causal Attributions for Disease Onset: Item 14 "Continuous emotional instability and sensitiveness." (Nonsignificant Interaction)

Source	df	SS	MS	F
Between Subjects	164	383.47		
Group (A)	1	1.98	1.98	0.84
Residual between	163	381.49	2.34	
Within Subjects	330	168.81		
Item (B)	2	15.59	7.79	16.78***
Item*Group (A*B)	2	1.78	0.89	1.92
Residual within	326	151.44	0.46	
Total	494	552.28		

Note: $N = 165$; *** $p < .0001$

Table 66: ANOVA Summary Table for Causal Attributions for Disease Onset: Item 15 "Continuous depressed mood." (Nonsignificant Interaction)

Source	df	SS	MS	F
Between Subjects	163	423.45		
Group (A)	1	1.65	1.65	0.63
Residual between	162	421.80	2.60	
Within Subjects	328	166.22		
Item (B)	2	15.60	7.80	17.00***
Item*Group (A*B)	2	1.99	0.99	2.16
Residual within	324	148.63	0.46	
Total	491	589.67		

Note: $N = 164$; *** $p < .0001$

Table 67: ANOVA Summary Table for Causal Attributions for Disease Onset: Item 16 "Traumatic life event(s)."

Source	df	SS	MS	F
Between Subjects	164	402.44		
Group (A)	1	0.21	0.21	0.08
Residual between	163	402.23	2.47	
Within Subjects	330	175.43		
Item (B)	2	18.94	9.47	20.29***
Item*Group (A*B)	2	4.41	2.21	4.73*
Residual within	326	152.08	0.47	
Total	494	577.87		

Note: $N = 165$; * $p < .01$, *** $p < .0001$

Summary:
Concluding remarks for 2 (GROUP) X 3 (DISEASE) ANOVA, with repeated measures.
A significant *interaction effect* was found for Item 16 "Traumatic life event(s)" (F (2,162) = 4.73, p <0.01), which indicates inherent differences in the mean attribution scores between the two groups and the three diseases.

Significant *group* differences between the back pain group and the no back pain group (F(2,160) = 6.38, p < 0.05) were found on Item 2 "Constantly being stressed out": the back pain group had significantly higher item scores. No other significant differences were found between the two groups.

Significant *item* effects were found between the diseases for all of the 16 causal attribution items. The contrasts are reported below:
Item 1: Causal attribution scores for back pain were significantly lower than mean scores for heart disease (F(2,165) = 40.38; p < .0001).
Item 2: Causal attribution scores for back pain were significantly higher than for cancer (F(2,160) = 26.74; p < .0001). Mean scores for back pain were significantly lower than for heart disease (F(2,160) = 15.54; p < .0001).
Item 3: Causal attribution scores for back pain were significantly lower than for cancer (F(2,163) = 14.20; p < .0005). Mean scores for back pain were significantly lower than for heart disease (F(2,163) = 50.05; p < .0001).
Item 4: Causal attribution scores for back pain were significantly lower than for heart disease (F(2,166) = 42.49; p < .0001). Mean scores for back pain were significantly lower than for cancer (F(2,166) = 5.31; p < .05).
Item 5: Causal attribution scores for back pain were significantly lower than for heart disease (F(2,162) = 77.56; p < .0001). Mean scores for back pain were significantly lower than for cancer (F(2,162) = = 17.97; p < .05).
Item 6: Causal attribution scores for back pain were significantly lower than for heart disease (F(2,162) = 44.75; p < .0001). Mean scores for back pain were significantly lower than for cancer (F(2,162) = = 20.11; p < .05).
Item 7: Causal attribution scores for back pain were significantly lower than for heart disease (F(2,163) = 53.06; p < .0001).

Item 8: Causal attribution scores for back pain were significantly lower than for heart disease (F(2,166) = 93.91; p < .0001). Mean scores for back pain were significantly lower than for cancer (F(2,166) = = 23.75; p < .05).
Item 9: Causal attribution scores for back pain were significantly lower than for heart disease (F(2,165) = 56.91; p < .0001). Mean scores for back pain were significantly lower than for cancer (F(2,165) = = 10.42; p < .005).
Item 10: Causal attribution scores for back pain were significantly lower than for heart disease (F(2,165) = 26.85; p < .0001). Mean scores for back pain were significantly lower than for cancer (F(2,165) = = 14.32; p < .0005).
Item 11: Causal attribution scores for back pain were significantly lower than mean scores for heart disease (F(2,169) = 22.45; p < .0001).
Item 12: Causal attribution scores for back pain were significantly lower than for heart disease (F(2,160) = 50.25; p < .0001). Mean scores for back pain were significantly lower than for cancer (F(2,160) = = 17.97; p < .0001).
Item 13: Causal attribution scores for back pain were significantly lower than for heart disease (F(2,163) = 16.42; p < .0001). Mean scores for back pain were significantly higher than for cancer (F(2,163) = 19.73; p < .05).
Item 14: Causal attribution scores for back pain were significantly lower than for heart disease (F(2,162) = 27.26; p < .0001).
Item 15: Causal attribution scores for back pain were significantly lower than for heart disease (F(2,161) = 26.51; p < .0001).
Item 16: Causal attribution scores for back pain were significantly lower than for cancer (F(2,163) = 8.76; p < .005); and lower than for heart disease (F(2,163) = 33.00; p < .0001)

4.6.6. Analysis of Variance: 2 (Group) x 16 (Item)

2(Group) x 16(Item) with Repeated Measures: Separate for DISEASE

a. Statement of the problem: The purpose of this analysis was to determine whether there was a difference between mean scores for the back pain group and the no back pain group with respect to items 1-- 16 for the diseases back pain, cancer and heart disease.

b. Nature of the variables: This analysis involved three variables. One predictor variable was the group (back pain versus no back pain), measured on a nominal scale. Another predictor variable was the item, measured on an interval scale. The criterion variable represented the diseases (back pain, cancer, heart disease), which was measured on a nominal scale.

c. Statistical test: 2 x 16 Analysis of Variance with Repeated Measures.

d. Null Hypothesis: M1 = M2; In the population, there is no difference between groups (back pain, no back pain), or between items (1 – 16) with respect to their mean attribution scores.

e. Alternative Hypothesis: In the population, there is a difference between back pain patients and no back pain patients across the items. And there is a difference between diseases: cancer and heart disease (because they are life-threatening) and back pain.

f. Obtained statistic: see below.

Table 68: 2 X 16 ANOVA Summary Table for Causal Attributions for Disease Onset: Back Pain (Nonsignificant interaction).

Source	df	SS	MS	F
Between Subjects	186	2514.74		
Group (A)	1	9.37	9.36	0.69
Residual between	185	2505.37	13.54	
Within Subjects	2805	2073.99		
Scale (B)	15	211.12	14.07	21.13***
Scale*Group (A*B)	15	14.57	0.97	1.46
Residual within	2775	1848.30	0.66	
Total	2991	4588.73		

Note: $N = 187$; *** $p < .0001$

Table 69: 2 X 16 ANOVA Summary Table for Causal Attributions for Disease Onset: Cancer (Nonsignificant interaction).

Source	df	SS	MS	F
Between Subjects	143	2021.75		
Group (A)	1	5.97	5.96	0.42
Residual between	142	2015.78	14.20	
Within Subjects	2160	924.14		
Scale (B)	15	53.27	3.55	8.73***
Scale*Group (A*B)	15	4.06	0.27	0.67
Residual within	2130	866.81	0.41	
Total	2303	2945.89		

Note: $N = 144$; *** $p < .0001$

Table 70: 2 X 16 ANOVA Summary Table for Causal Attributions for Disease Onset: Heart Disease (Nonsignificant interaction).

Source	df	SS	MS	F
Between Subjects	145	1207.29		
Group (A)	1	0.04	0.04	0.00
Residual between	144	1207.25	8.38	
Within Subjects	2190	1157.75		
Scale (B)	15	165.01	11.00	24.11***
Scale*Group (A*B)	15	7.11	0.47	1.04
Residual within	2160	985.63	0.46	
Total	2335	2365.04		

Note: $N = 146$; *** $p < .0001$

Disease Effect: A significant disease effect was found for back pain ($F(15, 171) = 11.45$, $p < 0.001$), cancer ($F(15, 128) = 6.91$, $p < 0.001$), and heart disease ($F(15, 130) = 14.11$, $p < 0.001$); Participants rated the items lower for back pain than for cancer, and lower for cancer than for heart disease. Participants of both groups scored Item 2 ("Constantly being stressed out.") as statistically significant for heart disease.

Group Effect: No significant differences were found between back pain group mean scores and the no back pain group mean scores on the 16 items for back pain. No significant differences between groups in attributions for onset of cancer. No group differences in causal attribution for heart disease.

Interaction Effect: No significant interaction effects between back pain, cancer, or for heart disease.

Summary:

A significant disease effect was found for back pain, cancer, and heart disease. Participants attributed different personality factors to "cause" each of these three diseases. No significant group effects were found. The analyses revealed no significant interaction effects.

4.7. Summary of the Main Results Based on the Research Hypotheses

H1: Biopsychosocial Factors: Significant relationships were found between chronic back pain and higher age and lower education. The female gender and lower education were significantly related to high intensity back pain in Chi-Squared Tests.

H2: Predictor Scales: Through logistic regression analyses, significant relationships were found between mean scores on the Depression Scale and pain chronicity and pain intensity. Analyses revealed a significant association between the Sense of Coherence Scale and pain intensity. The relationship between the Sense of Coherence and pain chronicity was not significant. No significant results were found between the predictor scale Job Satisfaction and the criterion variables pain chronicity. and pain intensity.

H3: Explaining the Variance for Chronicity, Intensity, and Chronicity + Intensity: Chronicity: Using the logistic regression analyses and testing the variables simultaneously, higher age, lower education, and past comorbidities reached significance for *chronicity*. For *intensity*, the analysis revealed significant results for low reported physical exercise, high scores on the Depression Scale, and present comorbidities. For the *combined variable* (Chronicity + Intensity) none of the variables reached significance when testing the predictors variables simultaneously. When testing for significance on an individual level, significant results were found for high ratings on the combined variable and the following predictors: higher age, female gender, low education, comorbidities (present and past), high Depression Scale scores, and low job satisfaction.

H4: Exaggerated Pain Experience: A significant relationship between the Item "Skin Sensitivity" ("skin sensitivity in the lumbar spine area, or reported pain when pressing upon the hip bone, Sacrum, or upper back") on the Exaggerated Pain Reaction items and high pain intensity was found through Chi-Squared Tests. Physicians' Diagnoses: Chi-square test showed one significant association between gender and the diagnosis "psychosomatic origin". Females were more likely to receive the diagnosis "psychosomatic origin" for their chronic back pain than men. A factorial ANOVA for AGE and the physicians' diagnoses showed a significant main effect for "degeneration". Back pain patients receiving the diagnosis "degeneration" were significantly older than patients receiving any of the other diagnoses. Analyses of the other criterion variables (Sense of Coherence, depression, job satisfaction) revealed nonsignificant effects.

H5: Group Differences: As expected, the back pain patients scored significantly higher than no back pain patients on the Depression Scale. No significant differences were found between the two groups on the Sense of Coherence Scale or on the Job Satisfaction Scale, however, according to expectations, the pack pain group scored lower on both these scales.

H6: Comorbidities: Significant relationships were found for comorbidities (present or past) and chronicity. Taken as a group (N = 270), chronic pain patients tended to report comorbidities in the present and in the past. However, there is no significant relationship between chronicity and comorbidities (present or past) in the *back* pain group. The whole participant sample reporting high intensity pain, also reported significantly more present comorbidities, but not past comorbidities. No significant relationship was found between intensity and present or past comorbidities in the *back* pain group. There is a significant relationship between higher age and comorbidities in the present for the *back* pain group: Older, *back* pain patients report significantly more present comorbidities.

H7: Disease Related Attributions: The personality factors were rated to be most relevant for heart disease; the highest means (and lowest standard deviations) were found on all of the items for heart disease. All of the 16 personality factors showed a significant item effect.

H8: Back Pain Patients' Attributions: A significant group effect was found for Item 2 ("Constantly being stressed out."); the back pain group had significantly higher mean scores than the no back pain group on this item. Back pain patients rated this item to be particularly important for the onset of heart disease. Analyses revealed a significant disease effect for back pain, cancer, and heart disease.

5. DISCUSSION

General Statements

Biopsychosocial Variables: This paper examined some correlations between biopsychosocial variables and low back pain. Low back pain was defined in terms of chronicity and intensity. Findings reported here support previous research in that some biopsychosocial factors are significantly associated with low back pain. Specifically, the significant relationships found for chronic back pain were: higher age, lower education, and higher scores on the Depression Scale. For high intensity back pain, significant relationships were found for females, participants with lower education, and higher Depression Scale scores.

Comorbidities: Comorbidities, present and past, were significantly associated with chronicity for general pain. Analyses revealed a significant association between present comorbidities and intensity for general pain. However, comorbidities were not significantly associated with low *back* pain chronicity or intensity. However, in the no back pain group, present and past comorbidities were significantly related to high intensity pain; past comorbidities were significantly associated with pain chronicity when analyzing the two group separately. According to expectations, older participants (> 48 years of age) and present comorbidities proved to be significantly associated in the *back* pain group.

Causal Attributions About Disease Onset: While analyzing causal attributions about disease onset, back pain patients considered "Constantly being stressed out" to be particularly relevant to the onset of heart disease. Shown through the consistently higher means (and consistently lower standard deviations), all participants considered the personality variables of the causal attribution items to be important for the onset of heart disease. Participants seemed to be less certain which personality factors play a role in the onset of low back pain.

Shortcomings of the study

The problem of causality. Although findings may help us to better understand the complexity surrounding the pain experience, in particular the transition from acute pain to chronic pain, a study of this kind can make no speculations about causality. In fact, nonexperimental research that investigates the relationship between just two variables generally provides relatively weak evidence concerning cause-and-effect relationships. Studies using direct manipulation in a prospective, controlled, and randomized fashion would enable statements about causality to be made.

Reliability.

Reliability of the Tendency to Exaggerate the Pain Reaction: An adaptation of the scale Psychological "overlay" (Waddell, 1993) was used here in this study and it proved to be of little statistical value. That is, this scale was impossible to analyze statistically since the orthopedic doctors, with very few exceptions, responded to the five items measuring skin sensitivity, pressure, leg lifting, nerve association, and the tendency to overreact with a "no" (nominal scale).

Reliability of the Causal Attribution Items: The coefficient alpha estimates calculated for the Causal Attribution items showed extremely high reliability, although it is not possible to recognize any internal (binnen) structure. The items address a wide range of topics from job satisfaction to traumatic life events. Potentially, a response set may have influenced the scores on these items.

Definition of the Outcome Variable. Another concern is the definition and measurement of the outcome variable: low back pain. Consider, for example, the difference between the simple report of having had back pain during the past year with the number of health care visits, further diagnostic tests, sick leave. All of which were variables collected in the study questionnaire. However, not all of these variables were statistically analyzed. Due to time pressure and to economic reasons, information was therefore lost as the two variables chronicity and intensity were defined.

Temporal Aspects. Research suggests (Linton, 2001) that certain factors are important very early on while others may be important at recurrence. A temporal analysis of the data was not possible due to the insufficient numbers of participants here to identify which predictor variables were significant depending on length of the pain experience.

Selection Biases. Data were collected for the experimental group from the orthopedic practices in a semi-rural community. Not only is this participant group biased due to the out-patient setting, but also due to the selection biases that surround group samples (cluster samples). Although the rate of return was high (88%), some potential participants refused to participate, a fact which jeopardizes the generalizability of the findings. Results apply only to individuals who are willing to participate in a study, since data could not collected from those who refused to participate.

Missing Data. Although it is generally well-known that missing data are common in questionnaire research (Hatcher & Stepanski, 1994), participants from this study failed to respond to many items, even through the instructions emphasized the importance of responding to all the questions. This questionnaire was lengthy, and participants experiencing pain may have been simply over-taxed.

Self-Reports. Self-report questionnaires are known to be the most frequently used and the least accurate and most unreliable form of assessment. In particular, this applies to the comorbidity section of this questionnaire study, in which participants are requested to indicate whether they suffer from (present), or had suffered from (past) any of the diseases listed. The construct of neuroticism complicates this issue, since "neurotic" individuals tend to focus their attention on small physical complaints that may not necessary be associated with a clinical diagnosis (McCrae & Costa, 1986). The personality construct Neuroticism was not controlled in this study, nevertheless, it should be considered in the interpretation of the results. Further, the assessment of pain is difficult to make from an objective perspective since it is a subjective experience, and affected by numerous individual differences (discussed above).

There is a great need for an assessment instrument capable of measuring physical complaints of low back pain.

Order Effect: The order of the questionnaire scales was not controlled in this study. The scales remained in the same order for the entire participant population. One possible consequence may be that participants lost concentration at the end of the lengthy questionnaire.

Sense of Coherence As A Protective Factor. Results from Amelang and Schmidt-Rathjens (2000) suggest that this construct may not show unique validity relative to the effects of neuroticism and depression. Therefore, the Heidelberg Sense of Coherence Scale may not be a strong enough construct to act as protective factor against disease. In general, this study fails to thoroughly investigate the protective constructs.

Control group. It could further be questioned whether the control group was sufficiently comparable. The type of pain was not controlled through the questionnaire, nor was the definition between, for example, chronic and acute determined by a health professional. Future research may show that subtypes of pain, e.g., low back pain, temporomandibular disorders or cancer pain, may be correlated with different variables.

Other Areas of Research Deserving Attention. This study failed to adequately assess the cognitive and social processes that are involved in the transition from acute to chronic pain. The studies examining the role social support plays in this area have provided fascinating evidence on how a solicitous spouse can actually contribute to the pain reported. In combining the work from Flor (2001) and her colleagues with cognitive processes may further our understanding of the variables that are critical in the development of chronic pain.

Future research directions.

Research over the last decade has focused on identifying subgroups of people with lower back pain. More recently, greater emphasis has been given to finding predictors and risk factors for lower back pain chronicity, improving self-care strategies, and stimulating self-reliance. A report from the Second International Forum for Primary Care Research on Low Back Pain (1998) predicts that future research will be directed towards expanding on methodological avenues of inquiry.

Few studies have penetrated the reasons why these predictors and risk factors might be important. A challenge for future research is therefore to devise studies that include a theoretical perspective as to why a variable might be important. Based on this observation, the American Pain Society (Kerns, Dworkin, Romano, Thorn, & Williams, 1999) emphasizes the need for developing research models that can help provide a foundation for generating testable models for the development of chronic pain. An example of such a model has been proposed by Kendler, Kessler, Neale, Heath, and Eaves (1991). They studied risk factors of depression, and their work suggests that four domains of variables are likely to be important: genetics, traumatic life events (childhood as well as recent), interpersonal support, and temperament. In addition, since depression is a common comorbidity in chronic pain, such models may also help illuminate the sources of chronicity in pain patients.

Finally, the American Pain Society (Kerns et al., 1999) emphasizes that research on treatment efficacy must now include cost-offset outcome data, standardization of treatment protocols, and component analyses of effective aspects of treatment as well as appropriate patient characteristics. The goal is to clearly define which treatments are appropriate for which patients, for which problems, and at what cost savings.

Meaning and Interpretation of the Results

Length of and Intensity of the Pain Experience

H1 Biopsychosocial Factors: Dionne and her colleagues (2001) found that low education was strongly associated with chronicity and/or high recurrence of back pain in a review of 64 articles. Results found here show that lower education is significantly associated with both the chronicity and the intensity of low back pain. Age appears to play a more significant role in the duration of the pain experience, higher age is significantly associated with chronic back pain. Gatchel and Gerdea (1999) reported similar findings for age. Females report significantly higher back pain intensity than males. These results support the findings that women exhibit lower pain thresholds than men. Plausible explanations to explain gender differences may be the hormonal state of the participant, or that females may be more willing to admit to experiencing pain (Miaskowski, 1999).

H2 Predictor Scales: Published studies have suggested that pain patients suffer from atypical depression (Joukamaa, 1994) and that depression may effect treatment outcomes (Epker & Block, 2001). Therefore, it is not surprising that depression proved to be a significant variable for the chronicity and intensity of pain in this study. A significant relationship was found between the criterion variable intensity and the Sense of Coherence, but not between chronicity and the Sense of Coherence. Analyses of the criterion variables chronicity and intensity did not show significant associations with the Job Satisfaction Scale.

H3 Explaining the Variance for Chronicity, Intensity, and Chronicity + Intensity Combined: Results from the logistic regression analyses make clear, that although chronicity and intensity are significantly related (Chi-Square = 10.43, $p < 0.005$), entirely difference predictor variables significantly effect the respective criterion variables.

Chronicity: Specifically, higher age, lower education and past comorbidities have a significant effect on chronicity when the variables are tested

simultaneously. Significant findings for the predictor variables higher age and lower education conform to expectations. Investigating the relationship between past comorbidities and chronicity is unique to this study. Nevertheless it seems logical that participants who have had illnesses in the past may be more susceptible to chronic pain. Potentially, passing through the 3-stage model proposed by Gatchel (1996) for more than one disease may increase the probability of pain chronicity. Interestingly, although depression reached significance when tested separately with chronicity, it did not have a significant effect on chronicity when tested simultaneously with other variables.

Intensity: Low physical exercise, high Depression Scale Scores and present comorbidities showed a significant effect on intensity when tested simultaneously. The influence of physical exercise on pain intensity is not well researched. It is, however, expected that individuals suffering from high intensity pain are less likely to engage in physical exercise. It is not surprising that depression had a significant effect on pain intensity reports since there is a strong association between depression and the subjective experience of pain (i.e., pain intensity) in the literature (cf. Robinson & Riley, 1999). Although the role of comorbidities in pain intensity reports is not well researched, it seems logical to hypothesize that individuals presently suffering from more than one illness, are more likely to rate higher pain intensity.

Chronicity + Intensity: Although high intensity pain may play a role in the transition from acute to chronic pain, the relationship between these two variables is not well understood. These two variables were combined in the statistical analyses in order to examine which predictor variables would have an effect on chronicity + intensity. Significant results were found between the criterion variable chronicity + intensity and the following predictor variables on an individual basis: (in descending order of importance) depression, higher age, present comorbidities, job dissatisfaction, female gender, low education, and past comorbidities. These findings also conform to expectations and other

published results. It seems logical to conclude that a combined variable would be best described through more predictors. Curiously, when testing these variables for significance simultaneously, none of the predictor variables reached significance.

H4 Exaggerated Pain Experience: For the back pain group, one significant association was found between the items measuring exaggerated pain reports and the criterion variable, intensity. Skin sensitivity ("skin sensitivity in the lumbar spine area, or reported pain when pressing upon the hip bone, Sacrum, or upper back") was significantly associated with high intensity pain in 35% of the participants reporting high intensity pain. This finding shows a slight tendency to exaggerate pain reports in a small portion of the participants.

Physicians' Diagnoses: Physicians were more likely to diagnose the cause of low back pain for females to be psychosomatic than for males. This finding could reflect the gender biases discussed above (cf. Miaskowski, 1999). A factorial ANOVA for AGE and physicians' diagnoses (5 diagnoses) revealed a significant effect for "degeneration". Participants of the back pain group receiving the diagnosis "degeneration" from their physicians were significantly older than the back pain patients receiving one of the other diagnoses. This significant effect is explicable through a biomedical perspective.

H5 Group Differences: According to expectations (e.g., Epker & Block, 2001), back pain patients scored significantly higher on the Depression Scale than no back pain patients. Also consistent with the hypotheses, the back pain patients had lower scores on the Sense of Coherence Scale and the Job Satisfaction Scale, however, these differences were not significant. Kerr and colleagues (2001) also failed to find significant relationships between back pain and job satisfaction.

Significant mean differences were found between the back pain group and the no back pain group on the Depression Scale Items 8 "I am less interested in my love relationship(s) than I used to be.", 10 "Even when I try, I can't think straight.", and 11 "I don't have any emotions anymore.".

H6 Comorbidities: The four most important comorbidities (present and past) in this study are allergies, heart burn, arthritis, and digestive complications. They, like back pain, are not life threatening and it may be difficult to reason theoretically that they could change cognitive processes or behavior. That is a problem for future research.

In this paper, the correlations between the criterion variables chronicity and intensity and the predictor variable comorbidities (present and past) were examined. A significant relationship was found between chronicity and self-reported comorbidities in the present and in the past. Intensity was also significantly related to present comorbidities (but not to past comorbidities). The associations between pain chronicity and intensity were expected. Interestingly, past comorbidities seemingly did not have enough influence to be associated with intensity significantly.

When analyzing the *back* pain patients and the no back pain patients separately, no significant associations between chronicity and intensity and *back* pain were found. In the no back pain group, analyses revealed significant associations between chronicity and past comorbidities, and between intensity and comorbidities, present and past.

H7 Disease Related Attributions: Study participants rated the personality items to be most important for the onset of heart disease shown through the consistently higher mean scores (and the consistently lower standard deviations). In other words, participants rated items that are traditionally associated with onset of back pain, e.g., Item 1 "dissatisfaction with the work situation", Item 4 "constantly recurring family problems", Item 6

"hopelessness and resignation", Item 8 "loss of a loved on through death or divorce", Item 13 "high self-imposed expectations for achievement", Item 15 "continuous depressed mood", Item 16 "traumatic life events", to be most important for the onset of heart disease. In fact, participants rated all of these back pain items, with the exception of Item 13 "high self-imposed expectations", to be more important for the onset of cancer than for back pain. Although, the highest mean scores on all 16 items was also found for heart disease by Schmidt-Rathjens (1998), it was expected that the back pain items would be rated higher. These differences may be attributed to the fact that the risk factors for heart disease, e.g., stress, are better researched than those for cancer or back pain.

Causal Attributions and Comorbidities: A number of causal attribution items are significantly associated with comorbidities. Interestingly, for present comorbidities, all of the significant correlations were found for *back* pain. Significant correlations were found for Items 1 "Job dissatisfaction", 4 "Constant problems in the family", 5 "Recurring anxieties about the future", 8 "Loss of a loved one through death or divorce", 9 "Inability to express fury and anger", 12 "Often not showing emotions such as sorrow and disappointment", and 14 "Continuous emotional instability and sensitiveness". No significant relationships were found between present comorbidities and cancer, or between present comorbidities and heart disease. Past comorbidities showed one significant association with back pain for Item 12 "Often not showing emotions such as sorrow and disappointment". The relationships between heart disease and past comorbidities for Items 6 "Hopelessness and resignation" and 8 "Loss of a loved one through death or divorce" were significant. No significant associations were found between past comorbidities and cancer.

The personality factors for both back pain and heart disease loaded on two, principally similar, factors. The first factor contained items expressing hopelessness or depression, the second factor involved items measuring

problems and stress. However, only three items showed the same loading patterns. This finding supports the hypothesis that participants attribute different personality factors to the onset of each of the three diseases.

H8 Back Pain Patients' Attributions: A significant disease effect was found for back pain, cancer, and heart disease. Participants rated the personality factors lowest for back pain, highest for heart disease, and cancer in the middle. The personality factor "Constantly being stressed out" (Item 2) was rated by the back pain group and the no back pain group to be statistically significant for heart disease. Analyses revealed no group effect and no interaction effect.

6. SUMMARY

This paper examines the low back pain paradigm from three distinct perspectives. First, how are biopsychosocial variables correlated with chronicity and intensity of the low back pain experience? Second, do chronic low back pain patients tend to suffer from other somatic symptoms or comorbidities? Third, how does chronic low back pain develop? An appraisal of the causal attribution theories and beliefs regarding disease onset comprises the final section. 160 German females and 110 German males (N = 270) completed a battery of questionnaires including causal attribution theories about disease onset (Schmidt-Rathjens, 1997), Sense of Coherence (Antonovsky, 1987), depression (von Zerssen, 1976), and job satisfaction (Neubauer, 2002). In addition, for the low back pain sample, physicians completed an objective scale assessing organic causes of pain. Results show significant correlations between the biopsychosocial variables higher age, lower education and higher depression scores and chronic low back pain. Females, low education and high depression scores correlate significantly with high intensity back pain. A small, but significant association was found between female back pain patients and the physicians' diagnosis "psychosomatic origin". Higher age (> 48 years) and present comorbidities proved to be significant variables for back pain patients. Regarding causal attributions about disease onset, the variable „Constantly being stressed out" proved to be significant in the causal attributions for heart disease. Shown through the consistently higher means (and consistently lower standard deviations), all participants considered the personality variables of the causal attribution items to be important for the onset of heart disease. Participants seemed to be less certain which personality factors play a role in the onset of back pain.

7. REFERENCES

Adams, M.A. (1996). Biomechanics of low back pain. *Pain Reviews*, *3*, 15-30.

Akil, H. Mayer, D.J., & Liebeskind, J.C. (1976). Antagonism of stimulation-produced analgesia by naloxone, a narcotic antagonist. *Science*, *191*, 961-962.

Akil, H., Watson, S.J., Young, E., Lewis, M.E., Khachaturian, H., & Walker, J.M. (1984). Endogenous opiods: Biology and function. *Annual Review of Neuroscience*, *7*, 223-255.

Amelang, M. & Schmidt-Rathjens, C. (1993). Persönlichkeit, Stress und Krankheit: Untersuchungen zu den psychometrischen Gütekriterien der Krankheitsprädiktoren von Grossarth-Maticek und Eysenck. [Personality, stress and disease: Statistical evaluation of the predictor variables for disease from Grossarth-Maticek and Eysenck.] *Zeitschrift für Gesundheitspsychologie*, *1*, 160-182.

Amelang, M. & Schmidt-Rathjens, C. (2000). Kohärenzsinn als Prädiktor und Supressor bei der Untersuchung von Gesundheit und Krankheit [Sense of Coherence as a predictor and a supressor variable in the examination of health and illness]. *Zeitschrift für Gesundheitspsychologie*, *8(3)*, 85-93.

Amelang, M., Schmidt-Rathjens, C. & Matthews, G. (1996). Personality, cancer and coronary heart disease: Further evidence on a controversial issue. *British Journal of Health Psychology*, *1*, 191-205.

American Psychiatric Association (1994). *Diagnostic and statistical manual of mental disorders* (4th ed.). Washington, DC: Author.

An, H. S., Silveri, C.P., Simpson, M., et al. (1994). Comparison of smoking habits between patients with surgically confirmed herniated lumbar and cervical disc disease and controls. *Journal of Spinal Disorders*, *7(5)*. 369-373.

Andersen, B. L., Kiecolt-Glaser, J. K., & Glaser, R. (1994). A biobehavioral model of cancer stress and disease course. *American Psychologist*, *49(5)*, 389-404.

Andersson, G. B. J. (1992). Factors in the genesis an prevention of occupational back pain and disability. *Journal of Manipulative Physiological Therapy*, *15(1)*, 43-46.

Antonovsky, A. (1979). *Health, stress, and coping*. San Francisco: Jossey-Bass.

Antonovsky, A. (1987). *Unraveling the mystery of health*. San Francisco, CA: Jossey-Bass.

Antonovsky, A. (1993). The structure and properties of the Sense of Coherence scale: *Social Science and Medicine*, *36*, 725-733.

Atkinson, J.H., Slater, M.A., Patterson, T.L., Grank, I., & Garfin, S.R. (1991). Prevalence, onset, and risk of psychiatric disorders in chronic pain patients: A controlled study. *Pain*, *45*, 111-121.

Bacon, N.M., Bacon, S.F., Atkinson, J.H., Slater, M.A., Patterson, T.L., Grant, I., & Garfin, S.R. (1994). Somatization symptoms in chronic low back pain patients. *Psychosomatic Medicine*, *56(2)*, 118-127. Abstract from MEDLINE, Number: 8008798.

Bandura, A. (1969). *Principles of behavior modification*. New York: Holt, Rinehart & Winston.

Banks, S.M., & Kerns, R.D. (1996). Explaining high rates of depression in chronic pain: A diathesis-stress framework. *Psychological Bulletin*, *119*, 95-110.

Bar-On, D. (1987). Causal attributions and the rehabilitation of myocardial infarction victims. *Journal of Social and Clinical Psychology*, *5*, 114-122.

Basler, H.-D., Franz, C., Kroener-Herwig, B., Rehfisch, H.P. & Seemann, H. (Eds.). (1999). *Psychologische Schmerztherapie [Psychological Pain Therapy]*. Heidelberg: Springer.

Beck, A.T. (1976). *Cognitive therapy and the emotional disorder*. New York: International Universities Press.

Becker, P., Bös, K., Opper, E., Woll, A. & Wustmans, A. (1996). Vergleich von Hochgesunden, Normal-, und Mindergesunden in gesundheitsrelevanten Variablen (GRV). [A Comparison Between High- Normal-, Low Health Participants on Health Related Variables]. *Zeitschrift für Gesundheitspsychologie*, *4*, 55-76.

Bendelow, G. (1993). Pain perceptions, emotions, and gender. *Sociology of Health and Illness*, *15(3)*, 273-294.

Bigos, S.J., Battie, M.C., Spengler, D.M., Fisher, L.D., Fordyce, W.E., Hansson, T.H., Nachemson, A.L., & Wortley, M.D. (1991). A prospective study of work perceptions and psychosocial factors affecting the report of back injury. *Spine*, *16(1)*, 1-6.

Blohmke, M. (1986). Sozialmedizinische Aspekte von Krankheiten [Socialmedical aspects of illness.]. In M. Blohmke (Ed.) *Sozialmedizin*. (2. Edition). Ferdinand Enke Verlag, Stuttgart.

Blumer, D., & Heilbronn, M. (1981). The pain prone patient: A clinical and psychological profile. *Psychosomatics*, *22*, 395-402.

Borkan, J.M., Koes, B., Reis, S., Cherkin, D.C. (1998). A Report from the Second International Forum for Primary Care Research on Low Back Pain: Reexamining Priorities. *Spine*, *23(18)*, 1992-1996.

Bradley, J.J. (1996). *The management of pain*. (2nd Edition). Philadelphia: Lea & Febiger.

Brage, S., & Bjerkedal, T. (1996). Musculoskeletal pain and smoking in Norway. *Journal of Epidemiology & Community Health*, *50(2)*, 166-169.

Brown, F.F., Robinson, M.E., Riley, J.L., & Gremillion, H.A. (1996). Pain severity, negative affect, and microstressors as predictors of life interference in TMD (Temporomandibular disorders) patients. *Cranio*, *14*, 63-70.

Burns, J.W., Wiegner, S., Derleth, M., Kiselica, K., & Pawl, R. (1996). Linking symptom-specific physiological reactivity to pain severity in chronic low back pain patients: A test of mediation and moderation models. *Health Psychology*, *16*, 319-326.

Burton, A.K., Tillotson, K.M., Symonds, T.L., Burke, C., & Methewson, T. (1996). Occupational risk factors for the first-onset and subsequent course of low back trouble: A study of serving police officers. *Spine*, *21(22)*, 2616-2620.

Calais-Germain, B. (1993). *Anatomy of Movement*. Seattle: Eastland Press.

Campbell, D.T., & Stanley, J.C. (1963). *Experimental and Quasi-Experimental Designs for Research*. Boston: Houghton Mifflin Company.

Chapman, C.R., Nakamura, Y., & Flores, L.Y. (1999). Chronic Pain and Consciousness: A Constructivist Perspective. In R.J. Gatchel and D.C. Turk (Eds.), *Psychosocial Factors in Pain: Critical Perspectives*, 18-34. New York: Guilford Press.

Chapmanm C.R., & Stillman, M. (1996). Pathological pain. In L. Kruger (Ed.) *Pain and Touch* (2nd ed.). New York: Academic Press.

Chrousos, G.P. (1992). Regulation and dysregulation of the hypothalamic-pituitary-adrenal axis. *Endocrinology and Metabolism Clinics of North America*. *21*, 833-858.

Chrousos, G.P., & Gold, P.W. (1992). The concepts of stress and stress system disorders. *Journal of the American Medical Association*, *267*, 1244-1252.

Cioffi, D. (1991). Beyond attentional strategies: A cognitive-perceptual model of somatic interpretation. *Psychological Bulletin*, *109*, 25-41.

Cody, R.P. & Smith, J.K. (1997). *Applied Statistics and the SAS Programming Language*, 4th Edition. New Jersey: Prentice Hall.

Cogan, R., Cogan, D., Waltzm, W., & McCue, M (1987). Effects of laughter and relaxation on discomfort thresholds. *Journal of Behavioral Medicine*, *10*, 139.

Costa, P.T., Jr., & McCrae, R.R. (1985). *The NEO personality inventory manual*. Orlando, FL: Psychological Assessment Resources.

Cox, D.C. (Ed.). (1999). *Low Back Pain: Mechanism, Diagnosis, and Treatment*. Baltimore: Williams & Wilkins.

Cox, G.B., Chapman, C.R., & Black, R.G. (1978). The MMPI and chronic pain: The diagnosis of psychogenic pain. *Journal of Behavioral Medicine*, *1*, 437-444.

Croft, P.R., Papageorgious A.C., Ferry, S., et al. (1995). Psychologic distress and low back pain. Evidence from a prospective study in the general population. *Spine 20*, 2731-2737.

Croft, P.R., & Rigby, A.S. (1994). Socioeconomic influences on back problems in the community of Britain [On-line]. *Journal of Epidemiology & Community Health*, *48(2)*, 166-170. Abstract from: PsycINFO, Number: 45740-001.

Cronbach, L.J. (1951). Coefficient alpha and the internal structure of tests. *Psychometrika*, *16*, 297-334.

DeBroucker, T., Cesaro, P., Willer, J.C., & LeBars, D. (1990). Diffuse noxious inhibitory controls in man. Involvement of the spinoreticular tract. *Brain*. *113*, 1223-1234.

Deyo, R.A., Tsui-Wu, Y. (1987). Descriptive epidemiology of low-back pain and its related medical care in the United States. *Spine*, *12(3)*; 264.

Dionne, C.E., Korff M. von, Keopsell, T.D., Deyo, R.A., Barlow, W.E., & Checkoway, H. (2001). Formal education and back pain: A review. *Journal of Epidemiology and Community Health*, *55(7)*, 455-468. Abstract from: PsycINFO, Number: 07459-001.

Downey, G., Silver, R.C., & Wortman, C.B. (1990). Reconsidering the attribution-adjustment relation following a major negative event: Coping with the loss of a child. *Journal of Personality and Social Psychology*, *59*.

Diagnostic and Statistical Manual of Mental Disorders : DSM-IV, (1994). Washington D.C. American Psychiatric Association.

Dunkel-Schetter, C., Feinstein, L.G., Taylor, S.E., & Falke, R.L. (1992). Patterns of coping with cancer. *Health Psychology*, *11*, 79-87.

Dworkin, R. H. (1997). Which individuals with acute pain are most likely to develop chronic pain syndrome? *Pain Forum*, *6*, 127-136.

Dworkin R. H. & Banks, S. M. (1999). A Vulnernability-Diathesis-Stress Model of Chronic Pain: Herpes Zoster and the Development of Postherpeutic Neuralgia. In R. J. Gatchel & D. C. Turk (Eds.) *Psychosocial Factors in Pain: Critical Perspectives*. New York: Guilford Press.

Engel, G.L. (1959). "Psychogenic" pain and the pain-prone patient. *American Journal of Medicine*, *26*, 899-918.

Epker, J., & Block, A.R. (2001). Presurgical psychological screening in back pain patients: A review. *The Clinical Journal of Pain*, *17*, 200-205.

Faller, H. (1993). Subjektive Krankheitstheorien: Determinanten oder Epiphänomene der Krankheitsverarbeitung? [Subjective Theories about Disease: Determinants or a Phenomenon of Disease Acceptance]. *Zeitschrift für Psychosomatische Medizin und Psychoanalyse*, *39*, 356-374.

Feyer, A.-M., Williamson, A., Mandryk, J., de Silva, I., et al. (1993). Role of psychosocial risk factors in work-related low-back pain. *Scandinavian Journal of Work, Environment & Health*, *18(6)*, 368-375. Abstract from PsycINFO, Number: 26171-001.

Fields, H.L. (1987). *Pain*. New York: McGraw-Hill.

Filipp, H.S. (1990). Subjektive Theorien als Forschungsgegenstand [Subjective theories in research]. In R. Schwarzer (Ed.) *Gesundheitspsychologie* [Health Psychology], 247-262. Göttingen: Hogrefe.

Fillingham, R.B., & Maixner, W. (1996). The influence of resting blood pressure and gender on pain responses. *Psychosomatic Medicine*, *58*, 326-332.

Fishbain, D.A., Cutler, R., Rosomoff, H.L., & Rosomoff, R.S. (1997). Chronic pain-associated depression: Antecedent or consequence of chronic pain? A review. *Clinical Journal of Pain*, *13*, 116-137.

Fishbain, D.A., Goldberg, M., Meagher, B.R., Steele, R., & Rosomoff, H. (1986). Male and female chronic pain patients categorized by DSM-III psychiatric diagnostic criteria. *Pain*, *26*, 181-197.

Flor, H. (1999). Verhaltensmedizinische Grundlagen chronischer Schmerzen [Behavioral-medical perspective of chronic pain]. In Basler, H.-D., Franz, C., Kroener-Herwig, B., Rehfisch, H.P. & Seemann, H. (Eds.). *Psychologische Schmerztherapie*. Heidelberg: Springer.

Flor, H., Birbaumer, N., & Turk, D.C. (1990). The psychology of chronic pain. *Advances in Behavior Research and Therapy*, *12*, 47-84.

Flor, H., & Turk, D.C. (1984). Etiological theories and treatments for chronic back pain: I. Somatic models and interventions. *Pain*. *19*, 105-121.

Flor, H., & Turk, D.C. (1989). Psychophysiology of chronic pain: Do chronic pain patients exhibit symptom-specific psychophysiological responses? *Psychological Bulletin*, *105*, 215-259.

Flor, H., Turk, D. C., & Birbaumer, N. (1985). Assessment of stress-related psychophysiological responses in chronic pain patients. *Journal of Consulting and Clinical Psychology*, *35*, 354-364.

Fogelholm, R.R., & Alho, A.V. (2000). Smoking and intervertebral disc degeneration. *Medical Hypotheses*, *56(4)*, 537-539. Abstract from Idealibrary, Number: 0306-9877.

Fordyce, W. E. (1976). *Behavioral methods in chronic pain and illness*. St. Louis: Mosby.

Fordyce, W.E., Shelton, J., & Dundore, D. (1982). The modification of avoidance learning pain behaviors. *Journal of Behavioral Medicine*, *4*, 405-414.

Friedman, H. S. & Booth-Kewley, S. (1987). The "disease-prone personality": A meta-analytic view of the construct. *American Psychologist*, *42*, 539-555.

Fydrich, T. & Flor, H. (1999). Die Rolle der Familie bei chronischen Schmerzen [The Role of the Family with Chronic Pain Patients]. In H.-D. Basler, C. Franz, B. Kröner-Herwig, H.P. Rehfisch, & H. Seemann (Eds.) *Psychologische Schmerztherapie [Psychological Pain Therapy]*. Heidelberg: Springer.

Garofalo, J. P., & Polatin, P. (1999). Low back pain: An epidemic in industrialized countries. In R.J. Gatchel & D. C. Turk (Eds.), *Psychosocial factors in Pain: Critical Perspectives*, 164-174. New York: Guilford Press.

Gaskin, M.E., Green, A.F., Robinson, M.E., & Geisser, M.E. (1992). Negative effect and the experience of chronic pain. *Journal of Psychosomatic Research*, *36*, 707-713.

Gatchel, R. J. (1996). Psychological disorders and chronic pain: Cause-and-effect relationships. In R. J. Gatchel & D. C. Turk (Eds.), *Psychological approaches to pain management: A practioner's handbook*, 33-52. New York: Guilford Press.

Gatchel, R.J., & Gardea, M.A. (1999). Psychosocial issues: Their importance in predicting disability, response to treatment, and search for compensation. *Neurologic Clinics of North American*, *17(1)*, 149-166.

Gatchel, R.J., Polatin, P.B. & Mayer, T.G. (1995). The dominant role of psychosocial risk factors in the development of chronic low back pain disability. *Spine*, *20(24)*, 2702-2709.

Gatchel, R.J., Polatin, P.B., Mayer, T.G. & Garcy, P.D. (1994). Psychopathology and the rehabilitation of patients with chronic low back pain. *Archives of Physical Medicine and Rehabilitation*, *75*, 666-670.

Geisser, M.E., Gaskin, M.E., Robinson, M.E. & Greene, A.F. (1993). The relationship of depression and somatic focus to experimental and clinical pain in chronic pain patients. *Psychology and Health*, *8*, 405-415.

Geisser, M.E., Roth, R.S., & Robinson, M.E. (1997). Assessing depression among persons with chronic pain using the Center for Epidemiological Studies-Depression Scale and the Beck Depression Inventory: A comparative analysis. *Clinical Journal of Pain*, *13*, 163-170.

Gerber, W.-D. (1999). Migräne [Migraine Headaches]. In H.-D. Basler, C. Franz, B. Kröner-Herwig, H.P. Rehfisch, & H. Seemann (Eds.) *Psychologische Schmerztherapie [Psychological Pain Therapy]*. Heidelberg: Springer.

Gerber, W.-D. & Hasenbring, M. (1999). Chronische Gesichtsschmerzen [Chronic Headache Disorders]. In H.-D. Basler, C. Franz, B. Kröner-Herwig, H.P. Rehfisch, H. Seemann (Eds.) *Psychologische Schmerztherapie [Psychological Pain Therapy]*. Heidelberg: Springer.

Graver, V., Ljunggren, A.E., Malt, U.F., Loeb, M., et al. (1996). Can psychological traits predict the outcome of lumbar disc surgery when anamnestic and physiological risk factors are controlled? Results of a prospective cohort study. *Journal of Psychosomatic Research*, *39(4)*, 465-476. Abstract from PsycINFO, Number: 02541-001.

Groeben, N. (1988). Explikation des Konstrukts "Subjective Theorie" [Exploration of the construct "subjective theories"]. In N. Groeben, D. Wahl, J. Schlee & B. Schlee (Eds.) *Das Forschungsprogramm Subjektive Theorien* [Research program subjective theories]. Tübingen: Francke.

Hägg, O., Fritzell, P., Nordwall, A., & the Swedish Lumbar Spine Study Group (2002). Characteristics of patients with chronic low back pain selected for surgery: A comparison with the general population reported form the Swedish Lumbar Spine Study. *Spine*, *27(11)*, 1223-1231.

Hansen, F.R., Biering-Sorenson, F., & Schroll, M. (1995). Minnesota Multiphasic Personality Inventory profiles in persons with or without low back pain: A 20-year follow-up study. *Spine*, *20(24)*, 2716-2720.

Hanvik, L. J. (1951). MMPI profiles in patients with low back pain. *Journal of Consulting Psychology*, *15*, 350-353.

Hasenbring, M. (1992). *Chronifizierung bandscheibenbedingter Schmerzen.* [Chronification of Low Back Pain Due to Disk Problems.] Stuttgart: Schattauer.

Hasenbring, M. (1999). Prozesse der Chronifizierung von Schmerzen [Processes involved in the chronification of pain]. In H.-D. Basler, C. Franz, B. Kroener-Herwig, H.P. Rehfisch, & H. Seemann (Eds.).*Psychologische Schmerztherapie*. Heidelberg: Springer.

Hasenbring, M. Marienfeld, G., Kuhlendahl, D., & Soyka, D. (1994). Risk factors of chronicity in lumbar disc patients: A prospective investigation of biologic, psychologic, and social predictors of therapy outcome. *Spine*, *19*, 2759-2765.

Hathaway, S.R., McKinley, J. (1943). *Minnesota Multiphasic Personality Inventory*. Minneapolis: University of Minnesota Press.

Hatcher, L., & Stepanski, E. (1994). *A Step-by-Step Approach to Using the SAS System for Univariate and Multivariate Statistics*. Cary, NC: SAS Institute Inc.

Hildebrandt, V.H., Bonger, P.M., Dul, J., Van Dijk, F.J., & Kemper, H.C. (1996). Identification of high-risk groups among maintenance workers in a steel company with respect to musculoskeletal symptoms and workload. *Ergonomics*, *39(2)*, 232-242.

International Classification of Mental and Behavioural Disorders : ICD-10 (1993), Geneva: World Health Organization.

Joukamaa, M. (1994). Depression and back pain. *Acta Psychiatrica Scandinavica*, *377*, 83-86.

Kapit, W., Macey, R.I. & Meisami, E. (2000). *The Physiology Coloring Book*. San Francisco: Benjamin/Cummings.

Kaplan, R.M., Ries, A.L., Prewitt, L.M., & Eakin, E. (1994). Self-efficacy expectations predict survival for patients with chronic obstructive pulmonary disease. *Health Psychology*, *13*, 366-368.

Karoly, P., & Ruehlman, L.S. (1996). Motivational implication of pain: Chronicity, psychological distress, and work global construal in a national sample of adults. *Health Psychology*, *15*, 383-390.

Katz, J., Jackson, M., Kavanagh, B.P., & Sandler, A.N. (1996). Acute pain after thoratic surgery predicts long-term post-thoracotomy pain. *Clinical Journal of Pain*, *12*, 50-55.

Keefe, F.J., Dunsmore, J., & Burnett, R. (1992). Behavioral and cognitive-behavioral approaches to chronic pain: Recent advances and future directions. *Journal of Consulting and Clinical Psychology*, *60*, 528-536.

Kendler, K.S., Kessler, R.C., Heath, A.C., Neale, M.C., & Eaves, L.J. (1991). Coping: A genetic epidemiological investigation. *Psychological Medicine*, *21*, 337-346.

Keoph, E., & Mansoor, L. (2001). Investigating the effects of anxiety sensitivity and coping on the perception of cold pressure pain in healthy women. *European Journal of Pain*, *5(1)*, 11-22. Abstract from Idealibrary, Number: 1090-3801.

Kerns, R.D., Dworkin, R.H., Romano, J.M., Thorn, B.E., & Williams, D.A. (1999). Psychosocial Research Special Interest Group: History, Advances, and Challenges. *American Pain Society Bulletin*, *9(4)*, 1-11.

Kerns, R.D., Turk, D.C., & Rudy, T.E. (1985). The West Haven-Yale Multidimensional Pain Inventory. *Pain*, *23*, 345-356.

Kerr, M.S., Frank, J.W., Shannon, H.S., Norman, R.W., Wells, R.P. Neumann, P., Bombardier, C. (2001). Biomechanical and psychosocial risk factors for low back pain at work. *American Journal of Public Health*, *91(7)*, 1069-1075. Abstract from PsycINFO, Number: 07473-006.

Krause, N. (1996). Back and neck pain in San Francisco bus drivers: The role of biomechanical and psychosocial job factors. *Dissertation Abstracts International: Section B: The Sciences & Engineering*, *57(3-B)*, pp.1719. Abstract from PsycINFO, Number: 95017-089.

Kroener-Herwig, B. (1999). Chronischer Schmerz – Eine Gegenstandsbestimmung. [Chronic Pain – A Definition]. In H.-D. Basler, C. Franz, B. Kroener-Herwig, H.P. Rehfisch, & H. Seemann (Eds.). *Psychologische Schmerztherapie*. Heidelberg: Springer.

Kroener-Herwig, B. (1999). Die Schmerzpersönlichkeit – Eine Fiktion? [Pain personality – Fact or fiction?]. In H.-D. Basler, C. Franz, B. Kroener-Herwig, H.P. Rehfisch, & H. Seemann (Eds.). *Psychologische Schmerztherapie*. Heidelberg: Springer.

Kroener-Herwig, B. (1999). Psychosoziale Diagnostik in der Schmerztherapie [Psychosocial diagnostic criteria in pain therapy]. In H.-D. Basler, C. Franz, B. Kroener-Herwig, H.P. Rehfisch, & H. Seemann (Eds.). *Psychologische Schmerztherapie*. Heidelberg: Springer.

Kroener-Herwig, B. (2000). *Rückenschmerz* [Back Pain]. Hogrefe: Göttingen.

Kroener-Herwig, B., Jakle, C., Frettloh, J., Peters, K., Seemann, H., Franz, C., & Basler, H.-D. (1996). Predicting subjective disability in chronic pain patients. *International Journal of Behavioral Medicine*, *3*, 30-41.

Lahad, A., Malter, A.D., Berg, A.O. et al. (1994). The effectiveness of four interventions for the prevention of low back pain. *Journal of American Medical Association* (JAMA), *272(16)*, 1286-1291.

Lau, E.M., Egger, P., Coggon, D., Cooper, C., Valenti, L, & O'Connell, D. (1996). Low-back pain in Hong Kong: Prevalence and characteristics compared to Britain. *Journal of Epidemiology and Community Health*, *49(5)*, 492-494.

Lazarus, R.S., & Folkman, S. (1984). *Stress, appraisal, and coping*. New York: Springer.

Leboeuf-Yde, C., Klougart, N., & Lauritzen, T. (1996). How common is low back pain in the Nordic population?: Data from a recent study on middle-aged general Danish population an four surveys previously conducted in the Nordic countries. *Spine*, *21(13)*, 1518-1525.

Leino, P., & Magni, G. (1993). Depressive and distress symptoms as predictors of low back pain, neck-shoulder pain, and other musculoskeletal morbidity: A ten-year follow-up of metal industry employees. *Pain*, *53*, 89-94.

Lewis, J.W., Terman, S.W., Shavit, Y., Nelson, L.R., & Liebeskind, J.D. (1984). Neural, neurochemical, and hormonal bases of stress-induced analgesia. In L. Kruger & J.C. Liebeskind (Eds.), *Advances in pain research and therapy*, *6*, 277-288. New York: Raven Press.

Linton, S. (1985). The relationship between activity and chronic pack pain. *Pain*, *21*, 288-294.

Linton, S.J.(1997). A population-based study of the relationship between sexual abuse and back pain: Establishing a link. *Pain*, *73*, 147-153.

Linton, S.J. (2001). Occupational psychological factors increase the risk for back pain: A systematic review. *Journal of Occupational Rehabilitation*, *11(1)*, 53-66. Abstract from PsycINFO, Number: 17847-005.

Linton, S.J., Hellsing, A.-L., & Bryngelsson, I.-L. (2001). Psychosocial and functional risk factors in the early stages of back pain: Effect on moderate pain. International *Journal of Behavioral Medicine*, *7(4)*. Special Issue: Behavioral factors and interventions in pain and musculoskeletal disorders, 291-304. Abstract from PsycINFO, Number: 17145-001.

Loranger, A.W., Lehmann-Susman, V., Oldham, J., & Russakof, L.M. (1985). *Personality Disorder Examination (PDE): A structured interview for DSM-III-R personality disorders*. White Plain, NY: New York Hospital-Cornell Medical Center, Westchester Division.

Love, A.W., & Peck, C.L. (1987). The MMPI and psychological factors in chronic low back pain: A review. *Pain*, *28*, 1-12.

Maes, S., Leventhal, H., & DeRidder, D.T.D. (1996). Coping with chronic disease. In M. Zeidner & N.S. Endler (Eds.), *Handbuch of coping: Theory, research, and applications*. New York: Wiley.

Magni, G. Moreschi, C., Rigatti-Luchini, S., & Merskey, H. (1994). Prospective study on the relationship between depressive symptoms and chronic musculoskeletal pain. *Pain*, *56*. 289-297.

Malenfant, A., Forget, R., Papillon, R., Amsel, R., Frigon, J.Y., & Choinière, M. (1996). Prevalence and characteristics of chronic sensory problems in burn patients. *Pain*, *67*, 493-500.

Mannion A.F., Dolan, P., Adams M.A. (1996). Do "abnormal" scores precede or follow first-time low back pain?. *Spine*, *21*, 2603-2611.

Mayer, T.G. (1991). Rationale for modern spinal care. In T.G. Mayer, V. Mooney, & R.J. Gatchel (Eds.), *Contemporary conservative care for painful spinal disorders*. Philadelphia: Lea & Febiger.

Mayer, T.G., & Gatchel, R.J. (1988). *Functional restoration for spinal disorders: The sports medicine approach*. Philadelphia: Lea & Febiger.

McCarron, R.F. & Laros, G.S. (1987). What is the cause of your patient's sciatica? *Journal of Musculoskeletal Medicine*; June, 59-77.

McCracken, L.M., Gross, R.T., Sorg, P.J., & Edmands, T.A. (1993). Prediction of pain in patients with chronic low back pain: Effects of inaccurate prediction and pain-related anxiety. *Behaviour Research and Therapy*, *31*, 647-652.

McCrae, R.R. & Costa, R.T. (1986). Personality, coping, and coping effectiveness in an adult sample. *Journal of Personality*, *54*, 385-405.

Melzack, R. (1975). The McGill Pain Questionnaire: Major properties and scoring methods. *Pain*, *1*, 277-299.

Melzack, R. (1999). Pain and Stress: A New Perspective. In R.J. Gatchel & D.C. Turk (Eds.) *Psychosocial Factors in Pain*: *Critical Perspectives*. New York: Guilford Press.

Melzack, R., & Wall, P.D. (1965). Pain mechanisms: A new theory. *Science*, *150*, 971-979.

Melzack, R., & Wall, P.D. (1982). *The challenge of pain*. New York: Basic Books.

Melzack, R., & Wall, P.D. (1996). *The challenge of pain*. (2nd ed.). London: Penguin Books.

Mercado, A.C., Carroll, L.J., Cassidy, J.D., & Cöté, P. (2000). Coping with neck and low back pain in the general population. *Health Psychology*, *19(4)*, 333-338.

Merskey, H. (Ed.).(1986). Classification of chronic pain: Descriptions of chronic pain syndromes and definitions of pain terms. *Pain*, *3*, 1-225.

Miaskowski, C. (1999). The Role of Sex and Gender in Pain Perception and Response to Treatment. In R.J. Gatchel & D.C. Turk (Eds.) *Psychosocial Factors in Pain*: *Critical Perspectives*. New York: Guilford Press.

Miedema, H.S., Chorus, A.M.J., Wevers, C.W.J., & van der Linden; S. (1998). Chronicity of back problems during working life. *Spine*, *23(18)*, 2021-2028.

Mobbs, R.J., Newcombe, R.L., Nadana, & Chandran, K.N. (2000). Lumbar discectomy and the diabetic patient: Incidence and outcome, *Journal of Clinical Neuroscience*, *10*, 1-4. Abstract from Idealibrary, Number: 0967-5868.

Neubauer, E. (2002). Kurzfragebogen zur Einschätzung des Chronifizierungsrisikos bei Rückenschmerzen [Short Questionnaire for classifying the risk of chronification in back pain patients.] *Orthopädie, 4*, 234-236.

Nicassio, P.M., Radojevic, V., Schoenfeld-Smith, K., & Dwyer, K. (1995). The contribution of family cohesion and the pain-coping process to depressive symptoms in fibromyalgia. *Annals of Behavioral Medicine*, *17*, 349-356.

Novy, D.M., Nelson, D.V., Frances, D.J., & Turk, D.C. (1995). Perspectives of chronic pain: An evaluative comparison of restrictive and comprehensive models. *Psychological Bulletin*, *118*, 238-247.

Olmarker, K., Hasue, M. (1995). Classification and pathophysiology of spinal pain syndromes. In J.N. Weinstein, A.B.L. Rydevik, K.H. Sonntag (Eds.) *Essentials of the Spine*. New York: Raven.

Pennebaker, J.W. (1982). *The psychology of physical symptoms*. New York: Springer.

Perrot, S. (2000). [Factors in the chronic progression of common low back pain.] [Article in French]. *Rev Prat*, *50(16)*, 1770-1773. Abstract from PubMed; Number:11103127.

Pert, C.B., & Snyder, S. (1973). Opiate receptor: Demonstration in nervous tissue. *Science*, *179*, 1011-1014.

Pfingsten, M., Kaluza, G., Hildebrant, J. (1999). Rückenschmerzen [Back Pain]. In H.-D. Basler, C. Franz, B. Kröner-Herwig, H.-P. Rehfisch & H. Seemann (Eds.) *Psychologische Schmerztherapie*. Heidelberg: Springer.

Pfohl, B., Blum, N., & Zimmerman, M. (1995). *Structured Interview for DSM-IV Personality Disorders*. Iowa City: University of Iowa.

Pincus, T., Burton, A.K., Vogel, S., & Field, A.P. (2002). A Systematic Review of Psychological Factors as Predictors of Chronicity/Disability in Prospective

Cohort of Low Back Pain. *Spine*, *27(5)*, 109-120. Abstract from Lippincott Williams & Wilkins, Number: Spine 2002:27:E109-E120.

Polatin, P.B., Kinney, R.K., Gatchel, R.J., Lillo, E., & Mayer, T.G. (1993). Psychiatric illness and chronic low-back pain. *Spine*, *18(1)*, 66-71.

Rachman, S., & Arntz, A. (1991). The overprediction and underprediction of pain. *Clinical Psychology Review*, *11*, 339-356.

Reesor, K.A., & Craig, K. (1988). Medically incongruent chronic pain: Physical limitations, suffering and ineffective coping. *Pain*, *32*, 35-45.

Reynolds, D.V. (1969). Surgery in the rat during electrical analgesia induced by focal brain stimulation. *Science*, *164*, 444-445.

Richard, K. (1988). The occurrence of maladaptive health-related behaviors and teacher-related conduct problems in children of low back pain patients. *Journal of Behavioral Medicine*, *11*, 107-116.

Riley, J.L., & Robinson, M.E. (1997). The Coping Strategies Questionnaire: Five factors or fiction? *Clinical Journal of Pain*, *13*, 156-162.

Robinson, M.E., Riley, J.L., III (1999). The Role of Emotion in Pain. In R.J. Gatchel and D.C. Turk (Eds.), *Psychosocial Factors in Pain: Critical Perspectives*, 18-34. New York: Guilford Press.

Salovey, P., & Birnbaum, D. (1989). Influence of mood on health-relevant cognitions. *Journal of Personality and Social Psychology*, *57*, 539-551.

Sanders, S.H. (2000). Risk Factors for Chronic, Disabling Low-Back Pain: An Update for 2000. *American Pain Society Bulletin*, *10(2)*, 1-6.

Sapolsky, R.M. (1992). Neuroendocrinology of the stress-response. In J.B. Becker, S.M. Breedlove, & D. Crews (Eds.). *Behavioral endocrinology*. Cambridge, MA: MIT Press.

SAS Institute Inc. (1990). *SAS Procedures Guide*, *6(3)*. Cary, NC: SAS Institute Inc.

SAS Institute Inc. (1990). *SAS User's Guide*, *6(4)*, Volumes 1, 2. Cary, NC: SAS Institute Inc.

Schmidt-Rathjens, C. (1998). *Persönlichkeit und Krebs: Studien zur subjektiven und objektiven Relevanz von psychologischen Faktoren bei der Krebsentstehung*. [Personality and cancer: A study of the subjective and objective relevancy of psychological factors for the onset of cancer.] (Doctoral thesis) Düsseldorf: Pabst.

Schmidt-Rathjens, C., Benz, D., Van Damme, D., Feldt, K., & Amelang, M. (1997). Über Zwiespältige Erfahrungen mit Fragebögen zum Kohärenzsinn sensu Antonovsky [Conflicting results from questionnaires measuring Antonovsky's Sense of Coherence.] *Diagnostica*, *43(4)*, 327-346.

Scholz, O.B. (1999). Schmerzmessung [Pain measurement]. In H.-D. Basler, C. Franz, B. Kroener-Herwig, H.P. Rehfisch, & H. Seemann (Eds.). *Psychologische Schmerztherapie*. Heidelberg: Springer.

Scholz, O.B., & Gerber, W.-D. (1999). Klassifikation chronischer Schmerzen [Classification of chronic pain]. In H.-D. Basler, C. Franz, B. Kroener-Herwig, H.P. Rehfisch, & H. Seemann (Eds.). *Psychologische Schmerztherapie*. Heidelberg: Springer.

Schwarzer, R. (1996). *Psychologie des Gesundheitsverhaltens* [The Psychology of Health Behavior]. Göttingen: Hogrefe-Verlag.

Schwenkmezger, P. (1994). Gesundheitspsychologie: Die persönlichkeits-psychologische Perspektiv. [Health psychology: The personality perspective.] In P. Schwenkmezger & L.R. Schmidt (1994) *Lehrbuch der Gesundheitspsychologie*. Stuttgart: Enke.

Schwenkmezger, P. & Schmidt, L.R. (1994). *Lehrbuch der Gesundheitspsychologie*. [Health Psychology.] Stuttgart: Enke.

Seyle, H. (1950). *Stress*. Montreal: Acta Medical Publisher.

Smedley, J., Egger, P., Cooper, C., & Coggon D. (1995). Manual handling activities and risk of low back pain in nurses. *Occupational and Environmental Medicine*, *52*, 160-163.

Spielberger, C.D. (1985). *State-Trait Anger Expression Inventory Professional Manual*. Odessa, FL: Psychological Assessment Resources.

Spitzer, R.L., Williams, J.B., Gibbon, M., & First, M.B. (1988). *Structured Clinical Interview for DSM-III-R*. New York: Academic Press.

Sternbach, R.A. (1974). *Pain patients: Traits and treatment*. New York: Academic Press.

Swenson, R. (1999). Differential Diagnosis: A Reasonable Clinical Approach. *Lower Back Pain*, *1*, 43-63.

Taylor, S.E. (1999). *Health Psychology*. 4th Edition. Boston: McGraw Hill.

Taylor, S.E., Heigeson, V.S., Reed, G.M., Skokan, L.A. (1991). Self-generated feelings of control and adjustment to physical illness. *Journal of Social Issues*, *47*, 91-109.

Thompson, S. C., Sobolew-Shubin, A., Galbraith, M.E., Schwnakovsky, L., & Cruzen, D. (1993). Maintaining perceptions of control: Finding perceived control in low-control circumstances. *Journal of Personality and Social Psychology*, *64*, 293-304.

Toshima, M.T., Kaplan, R.M., Ries, A.L. (1992). Self-efficacy expectations in chronic obstructive pulmonary disease rehabilitation. In R. Schwarzer (Ed.), *Self-efficacy: Thought control of action*. London: Hemisphere.

Trief, P.M., Carnike, C.L.M., & Drudge, O. (1995). Chronic pain and depression: Is social support relevant? *Psychological Reports*, *76*, 227-236.

Turk, D.C. (1996). Biopsychosocial perspective on chronic pain. In R.J. Gatchel and D.C. Turk (Eds.), *Psychological approaches to pain management: A practitioner's handbook*, 3-32. New York: Guilford Press.

Turk, D.C. & Flor, H. (1999). Chronic Pain: A Biobehavioral Perspective. In R.J. Gatchel and D.C. Turk (Eds.), *Psychosocial Factors in Pain: Critical Perspectives*, 18-34. New York: Guilford Press.

Turk, D.C., Kerns, R.D:, & Rosenberg, R. (1992). Effects of marital interaction on chronic pain and disability: Examining the downside of social support. *Rehabilitation Psychology*, *37*, 259-274.

Turk, D.C., & Okifuji, A. (1996). Perception of traumatic onset, compensation status, and physical findings: Impact on pain severity, emotional distress, and disability of chronic pain patients. *Journal of Behavioral Medicine*, *19*, 435-454.

Turk, D.C., & Rudy, T.E. (1990). The robustness of an empirically derived taxonomy of chronic pain patients. *Pain*, *43*, 27-35.

Turk, D.C., & Rudy, T.E. (1992). Cognitive factors and persistent pain: A glimpse into Pandora's box. *Cognitive Therapy and Research*, *16*, 99-112.

Turnquist, D.C., Harvey, J.H. & Andersen, B.L. (1988). Attributions and adjustment to life-threatening illness. *British Journal of Clinical Psychology*, *27*, 55-65.

Unruh, A.M. (1996). Gender variations in clinical pain experience. *Pain*, *65*, 123-167.

Verres, R. (1989). Zur Kontextabhängigkeit subjektiver Krankheitstheorien [Subjective Theories about illness depending upon context]. In C. Bischoff & H. Zenz (Eds.), *Patientenkonzepte von Körper und Krankheit*. Bern: Huber.

Von Korff, M. (1994). Studying the natural history of back pain. *Spine*. *19(18S)*:2041S-2046S.

Waddell, G. (1987). Volvo award in clinical sciences. A new clinical model for the treatment of low back pain, *Spine*, *12*, 632-644.

Waddell, G., Newton, M., Henderson, I., Somerville, D., & Main, C. (1993). A Fear-Avoidance Beliefs Questionnaire (FABQ) and the role of fear-avoidance beliefs in chronic low back pain and disability. *Pain*, *52*, 157-168.

Wade, J.B., Dougherty, L.M., Hart, R.P., & Cook, D.B. (1992). Patterns of normal personality structure among chronic pain patients. *Pain*, *48*, 37-43.

Waring, E.M., Weisz, G.M., & Bailey, S.I. (1976). Predictive factors in the treatment of low back pain by surgical intervention. In J.J. Bonica & D. Albefessard (Eds.) *Advances in pain research and therapy* (Vol. 1). New York: Raven Press.

Weickgenant, A.L., Slater, M.A., Patterson, T.L., Atkinson, J., Hampton, et al. (1993). Coping activities in chronic low back pain: Relationship with depression. *Pain*, *53(1)*, 95-103.

Weisberg, J.N., & Keefe, F.J. (1999). Personality, Individual Differences, and Psychopathology in Chronic Pain. In R.J. Gatchel and D.C. Turk (Eds.), *Psychosocial Factors in Pain: Critical Perspectives*, 18-34. New York: Guilford Press.

Weisberg, J.N., & Keefe, F.J. (1997). Personality disorders in the chronic pain population: Basic concepts, empirical findings and clinical implications. *Pain Forum*, *6(1)*, 1-9.

Weisel, S., Tsourmas, N., Feffer, N., et al. (1984). A study of computer-assisted tomography: the incidence of positive CT scans in an asymptomatic group of patients. *Spine, 9(6)*, 549-551.

Willis, W.D., Jr. (1992). *Hyperalgesia and allodynia*. New York: Raven Press.

Willis, W.D., Jr., & Westlund, K.N. (1997). Neuroanatomy of the pain system and of the pathways that modulate pain. *Journal of Clinical Neurophysiology*, *14*, 2-31.

Wiltse, L.L., & Rocchio, P.D. (1975). Preoperative psychological tests as predictors of success of chemonucleolysis in the treatment of low back syndrome. *Journal of Bone and Joint Surgery*, *57(A)*, 478-483.

Xuan, J. Kirchdoerfer, L.J., Boyer, J.G., & Norwood, G.J. (1999). Effects of comorbidity on health-related quality of life scores: an analysis of clinical trial data. *Clinical Therapy*, *21(2)*, 383-403. Abstract from PubMed, Number: 10211540.

Zatzick, D.F., & Dimsdale, J.E. (1990). Cultural variations in response to painful stimuli. *Psychosomatic Medicine*, *52*, 544-557.

Zerssen, D. von (1976). *Die Paranoid-Depressivitäts-Skala*. [The paranoid-depression-scale.] Weinheim: Beltz.

Zillman, D., de Wied, M., King-Jablonski, C., & Jenzowsky, S. (1996). Drama-induced affect and pain sensitivity. *Psychosomatic Medicine*, *58*, 333-341.

8. APPENDIX

Causal Attributions About Disease Onset

Item	Text
1	Dissatisfaction with the work situation.
2	Constantly overtaxed/constant stress.
3	Disappointment over unattainable (life) goals.
4	Constantly recurring family problems.
5	Recurring anxieties about the future.
6	Hopelessness and resignation.
7	Time pressure and long-term stress.
8	Loss of a loved one through death or divorce.
9	Frequent repression of rage and anger.
10	Loneliness and isolation.
11	Constant denial of one's own needs.
12	Frequent repression of sadness and despondency.
13	High self-imposed expectations for achievement.
14	Continuous emotional instability and sensitivity.
15	Constant depressed mood.
16	Traumatic life event(s).

Sense of Coherence (SOC)

Item	Text
1*	"I often don't understand how I got myself into these circumstances."
2*	"I often ask myself: Why is this happening to me?"
3	"I love life."
4	"I believe that I can accomplish any task set before me."
5*	"My life is chaotic, every day inexplicable things happen or I find myself in unexpected predicaments."
6	"With respect to my future, I'm very optimistic."
7*	"I often don't understand why things turn out as they do."
8	"I am an optimistic person."
9	"In general, I have confidence in the skills and the goals of our politicians."

* indicates negatively poled items.

Item Test Correlations for the Sense of Coherence Scale (Schmidt-Rathjens, 1998). *N* = 100 male and *n* = 100 female participants. An asterisk indicates negatively poled items.

Item Number	r_{it}, males	r_{it}, females
SOC 1*	.59	.27
SOC 2*	.41	.13
SOC 3	.47	.23
SOC 4	.51	.31
SOC 5*	.55	.24
SOC 6	.62	.52
SOC 7*	.44	.31
SOC 8	.57	.42
SOC 9	.18	.17

Depression Scale

Item	Text
1	″I've been anxious and jumpy lately.″
2	″I feel "down" and out of energy.″
3	"I understand a lot less written text than I used to."
4	″I would consider killing myself.″
5	"I no longer have any deep relationships."
6	"I feel like I'm about to fall apart."
7	"I'm constantly anxious that I'll say or do something wrong."
8	"I am less interested in my love relationship(s) than I used to be."
9	"I often feel simply miserable."
10	″Even when I try, I can't think straight.″
11	″I don't have any emotions anymore.″

Item Test Correlations for the Sense of Coherence Scale (Schmidt-Rathjens, 1998). *N* = 100 male and *n* = 100 female participants. An asterisk indicates negatively poled items.

Item Number	r_{it}, males	r_{it}, females
1	.75	.54
2	.65	.67
3	.70	.54
4	.63	.67
5	.54	.62
6	.68	.57
7	.65	.67
8	.57	.54
9	.67	.63
10	.59	.60
11	.56	.62

Job Satisfaction

Item	Text
1	"I'm excited to get back to work after vacations."
2	"I'm content with my work situation and I hope that it stays that way."
3*	"I don't have much control over my work situation."
4*	"I'm not ecstatic about my job, but it could be worse."
5*	"I almost ready to quit. My work situation is no longer tolerable."
6	"In general, how content are you with your job?"

* indicates negatively poled items.

Exaggerated Pain Reaction

Item	Text
SKIN	"Is the skin sensitive in the lumbar spine area, or does the patient report pain when pressing upon the hip bone, Sacrum, or upper back?"
PRESS	"Does the patient report pain when applying light pressure to the top of the head when standing up?"
LIFT	"Does the patient report more pain when lifting the leg with an extended knee while sitting, than while lying down?"
NERVE	"Does the patient show motoric and sensory deficits that are hard to attribute to one or more nerve roots?"
OVER	„Does the patient tend to exaggerate the pain reaction or overreact?"

List of Diagnoses
Genetic Malformation
Herniated Disc without neural damage
Herniated Disc with neural damage
Degeneration
Psychosomatic Origin

Var. Names	Variable definitions	
Group	1	back pain
	0	no back pain
Chronic	0	pain experience lasting less than 6 months.
	1	pain experience lasting 6 months and more.
Intensity	0	no pain (0), little pain (1), minimal pain (2).
	1	intense pain (3), unbearable pain (4).
Intensity		0 no pain – 4 unbearable pain
Age	1	less than 35
	2	35 – 48
	3	49 and older
Age		Range 15-82
Sex	1	female
	2	male
Education	1	completed „abitur" (high)
	2	no „abitur" (low)
Comorb	0	presently, no comorbidity, but back pain.
	1	presently, at least one disease and back pain.
Comorp	0	no comorbidity in the past, but back pain.
	1	at least one comorbidity in the past and back pain.
Combined	0	low-low
	1	high-high
SOC	1	low
	2	high
Depression	1	low
	2	high
Job	1	low
Satisfaction	2	high
Smoking	1	ex-smoker
	2	smoker
	3	non-smoker
Physical	1	< 2 hours weekly
Exercise	2	>= 2 hours weekly

Significant Chi-Square Tests of Independence

(Back Pain Group and No Back Pain Group, Together)

Age and Education: Age and education showed a significant association (Chi-Square = 13.27; $p < 0.005$). In the high education group, 51% of the participants are under 35 years of age, 28% are between 35 and 48 years of age, and 21% are over 48 years. In the low education group, 25% are under 35 years of age, 30% are between 35 and 48, and 45% are over 48 years. Statement: Higher educated participants tend to be younger (under 35 years of age), the majority of the low educated participants are over 48 years of age.

Table A1: Chi-Square Frequency Table: Age and Education
(Back Pain Group and No Back Pain Group, together)

Frequency (Col Pct)	Low Education	High Education	Total
< 35	**57** (25%)	**20** (51%)	**77**
35-48	**69** (30%)	**11** (28%)	**80**
>48	**105** (45%)	**8** (21%)	**113**
Total	**231**	**39**	**270**

Age and Smoking: A significant association was found between age and smoking (Chi-Square = 25.32; $p < 0.0001$). 43% of the smokers are under 35 years of age, 34% are between 35 and 48 years, and 23% are over 48. For ex-smokers, 16% are under 35 years, 27% are between the ages of 35 and 48, and the majority of ex-smokers tend to be over 48 years of age (57%). A similar pattern was found for non-smokers, 24% are under 35 years of age, 27% are between the ages of 35 and 48 years, and 49% are over 48 years. Statement: Smokers tend to be younger (under 35 years of age), ex-smokers and non-smokers tend to be over 48 years of age.

Table A2: Chi-Square Frequency Table: Age and Smoking
(Back Pain Group and No Back Pain Group, together)

Frequency (Col Pct)	Ex-Smoker	Smoker	Non-Smoker	Total
< 35	**10** (16%)	**40** (43%)	**26** (24%)	**76**
35-48	**17** (27%)	**31** (34%)	**30** (27%)	**78**
>48	**36** (57%)	**21** (23%)	**54** (49%)	**111**
Total	**63**	**92**	**110**	**265**

Gender and Smoking: Chi-Square analysis revealed a significant association between gender and smoking (Chi-Square = 9.37; $p < 0.01$). 59% of the females are smokers, compared to 41% of the male participants. 44% of the women are ex-smokers while 56% of the men have quit smoking. 68% of the women report never smoked relative to 32% of the men. Statement: Women tend to be smokers or non-smokers, men tend to have quit smoking.

Table A3: Chi-Square Frequency Table: Gender and Smoking (Back Pain Group and No Back Pain Group, together)

Frequency (Col Pct)	Ex-Smoker	Smoker	Non-Smoker	Total
Females	**28** (44%)	**54** (59%)	**75** (68%)	**157**
Males	**35** (56%)	**38** (41%)	**35** (32%)	**108**
Total	**63**	**92**	**110**	**265**

Education and Smoking: Education and smoking are significantly related (Chi-Square = 7.86; $p < 0.05$). Fifty-four percent of the participants with a higher education smoke. In the high education group, 13% are ex-smokers, 33% are non-smokers. For participants with a lower education, 31% smoke, 26% are ex-smokers, and 43% are non-smokers. Statement: A larger percentage of participants with a higher education smoke.

Table A4: Chi-Square Frequency Table: Education and Smoking (Back Pain Group and No Back Pain Group, together)

Frequency (Col Pct)	Ex-Smoker	Smoker	Non-Smoker	Total
Low Education	**58** (26%)	**71** (31%)	**97** (43%)	**226**
High Education	**5** (13%)	**21** (54%)	**13** (33%)	**39**
Total	**63**	**92**	**110**	**265**

Significant Chi-Square Tests of Independence

(Back Pain Group and No Back Pain Group, Separate)

Age and Education (Back Pain Group): Analysis revealed a significant relationship between age and education in the back pain group (Chi-Square = 17.16; $p < 0.0005$). Fifty-four percent of those participants with a higher education are under 35 years of age, 27% are between 35 and 48 years of age, and 19% are over 48 years. In the low education group, 18% are under 35 years of age, 27% are between 35 and 48 years, and 55% are over 48 years. Statement: Participants with a higher education tend to be younger (under 35 years of age), participants from the low education group tend to be older (over 48 years of age). In the no back pain group, age and education showed no significant association.

Table A5: Chi-Square Frequency Table: Age and Education (Back Pain Group)

Frequency (Col Pct)	Low Education	High Education	Total
< 35	**23** (18%)	**14** (54%)	**37**
35-48	**35** (27%)	**7** (27%)	**42**
>48	**70** (55%)	**5** (19%)	**75**
Total	**128**	**26**	**154**

Age and Smoking (Back Pain Group): Analysis revealed a significant relationship between age and smoking in the back pain group (Chi-Square = 15.78; $p < 0.005$). 41% of the smokers are under 35 years of age, 31% are between 35 and 48 years, and 29% are over 48 years of age. For ex-smokers, 11 % are under 35 years, 25% are between 35 and 48 years, and 64% are over 48 years of age. For non-smokers, 18% are under 35, 26% are between 35 and 48, and 55% are over 48 years of age. Statement: Back pain patients who smoke tend to be younger than ex-smokers or non-smokers.

Table A6: Chi-Square Frequency Table: Age and Smoking (Back Pain Group)

Frequency (Col Pct)	Ex-Smoker	Smoker	Non-Smoker	Total
< 35	**4** (11%)	**20** (41%)	**12** (18%)	**36**
35-48	**9** (25%)	**15** (31%)	**17** (26%)	**41**
>48	**23** (64%)	**14** (29%)	**36** (55%)	**73**
Total	**36**	**49**	**65**	**150**

Age and Smoking (No Back Pain Group): Significant associations were also found for the no back pain group (Chi-Square = 9.82; $p < 0.05$). The same trends can be found as described above, i.e., smokers tend to be younger (under 35 years of age) while ex-smokers and non-smokers tend to be slightly older (over 48 years of age). Please refer to the percentages and frequencies in the table.

Table A7: Chi-Square Frequency Table: Age and Smoking (No Back Pain Group)

Frequency (Col Pct)	Ex-Smoker	Smoker	Non-Smoker	Total
< 35	**6** (22%)	**20** (47%)	**14** (31%)	**40**
35-48	**8** (30%)	**16** (37%)	**13** (29%)	**37**
>48	**13** (48%)	**7** (16%)	**18** (40%)	**38**
Total	**27**	**43**	**45**	**115**

Gender and Smoking (Back Pain Group): A Chi-Square test revealed a significant relationship between gender and smoking in the back pain group (Chi-Square = 7.19; $p < 0.05$). 55% of the smokers are female compared to 45% of the males. In the ex-smoker group, 44% are female while 56% are male. A strong trend was found in the non-smoker group, 71% are women relative to 29% men. No significant associations were found between gender and smoking in the no back pain group.

Table A8: Chi-Square Frequency Table: Gender and Smoking (Back Pain Group)

Frequency (Col Pct)	Ex-Smoker	Smoker	Non-Smoker	Total
Females	**16** (44%)	**27** (55%)	**46** (71%)	**89**
Males	**20** (56%)	**22** (45%)	**19** (29%)	**61**
Total	**36**	**49**	**65**	**150**

Education and Smoking (No Back Pain Group): A significant relationship between education and smoking was found in the no back pain group (Chi-Square = 9.79; $p < 0.01$). In the high education group, 77% are smokers, 8% are ex-smokers, and 15% are non-smokers. For those participants receiving a lower education, 32% are smokers, 25% are ex-smokers, and 42% are non-smokers. Statement: Smokers tended to receive a higher education in the no back pain group. Chi-Square tests revealed no significant associations between education and smoking in the back pain group.

Table A9: Chi-Square Frequency Table: Education and Smoking (No Back Pain Group)

Frequency (Col Pct)	Ex-Smoker	Smoker	Non-Smoker	Total
Low Education	**26** (25%)	**33** (32%)	**43** (42%)	**102**
High Education	**1** (8%)	**10** (77%)	**2** (15%)	**13**
Total	**27**	**43**	**45**	**115**

Table: Comorbidity Scale: Assessed Diseases and Frequencies (Back Pain and No Back Pain Groups)

Disease	Present	Past
Cancer	4	6
Heart disease	10	12
Diseases of the blood	2	2
Diabetes	4	5
Thymus dysfunction	16	17
Diseases of the liver	2	4
Allergies	46	39
Asthma	9	7
Arthritis	34	23
Depression	14	16
Heartburn	36	27
Ulcers	1	6
High blood pressure	27	16
Digestive complications	33	31
Other	22	23

Note: $N = 270$

Causal Attributions About Disease Onset
One-way repeated-measures ANOVA (Proc GLM, repeated measures):

Class Level Information

Class	Levels	Values
Group 2	1	2

Repeated Measures Level Information

Dependent Variable		Back	Cancer	Heart Disease
Level of Attribution (Questions 1-16)		1	2	3
Level of Group		1		2
Variable	N	Mean Std.Dev.	N	Mean Std.Dev.
Att1-back	115	3.24 1.20	152	3.47 1.24
Att1-cancer	115	3.17 1.06	152	3.29 1.14
Att1-heart	115	3.73 1.12	152	3.96 0.88
Att2-back	110	4.23 1.01	150	4.20 1.11
Att2-cancer	110	3.62 1.04	150	3.61 1.12
Att2-heart	110	4.44 0.91	150	4.45 0.89
Att3-back	113	2.94 1.06	151	3.21 1.06
Att3-cancer	113	3.21 1.06	151	3.45 1.06
Att3-heart	113	3.63 0.97	151	3.53 0.91
Att4-back	114	3.36 1.23	150	3.41 1.31
Att4-cancer	114	3.48 1.17	150	3.73 1.15
Att4-heart	114	3.92 1.03	150	4.03 0.94
Att5-back	111	3.17 1.12	149	3.25 1.24
Att5-cancer	111	3.47 1.11	149	3.45 1.11
Att5-heart	111	3.86 1.04	149	3.87 0.88
Att6-back	112	3.23 1.07	145	3.30 1.19
Att6-cancer	112	3.57 1.06	145	3.66 1.15
Att6-heart	112	3.70 0.97	145	3.63 0.96
Att7-back	113	3.93 1.09	149	3.92 1.14
Att7-cancer	113	3.73 1.01	149	3.73 1.11
Att7-heart	113	4.56 0.81	149	4.66 0.71
Att8-back	113	3.19 1.15	153	3.08 1.29
Att8-cancer	113	3.57 1.16	153	3.54 1.25
Att8-heart	113	3.97 1.06	153	3.96 0.97

Att9-back	113	3.42	1.08	153	3.44	1.28
Level of Group			1			2
Variable	N	Mean	Std.Dev.	N	Mean	Std.Dev.
Att9-cancer	113	3.61	1.05	153	3.69	1.13
Att9-heart	113	4.00	0.94	153	4.15	0.83
Att10-back	113	2.89	1.15	151	3.01	1.19
Att10-cancer	113	3.28	1.04	151	3.23	1.15
Att10-heart	113	3.39	1.04	151	3.32	1.06
Att11-back	116	3.11	1.17	153	3.27	1.21
Att11-cancer	116	3.25	1.05	153	3.35	1.23
Att11-heart	116	3.52	1.02	153	3.43	1.02
Att12-back	110	3.15	1.0	148	3.26	1.23
Att12-cancer	110	3.42	0.95	148	3.57	1.06
Att12-heart	110	3.63	0.99	148	3.78	0.84
Att13-back	113	3.77	1.15	149	3.64	1.20
Att13-cancer	113	3.27	1.00	149	3.19	1.09
Att13-heart	113	3.97	1.08	149	4.05	1.01
Att14-back	112	3.29	1.00	148	3.32	1.19
Att14-cancer	112	3.42	0.98	148	3.42	1.07
Att14-heart	112	3.62	1.02	148	3.70	0.95
Att15-back	111	3.48	1.06	150	3.48	1.18
Att15-cancer	111	3.64	1.04	150	3.63	1.13
Att15-heart	111	3.80	1.00	150	3.69	0.96
Att16-back	111	3.32	1.13	149	3.30	1.23
Att16-cancer	111	3.37	1.05	149	3.59	1.12
Att16-heart	111	3.60	1.01	149	3.75	1.03

Psychologisches Institut der Universität Heidelberg
Forschungsgruppe Gesundheit

RÜCKENSCHMERZEN

Liebe Patientin, lieber Patient,

heute suchen Sie Ihren Orthopäden wegen Rückenschmerzen auf. Viele Menschen leiden unter Rückenschmerzen. Allerdings geht jeder Mensch unterschiedlich mit Schmerzen um. Daher bitten wir Sie, jetzt in der Wartezeit einen Fragebogen auszufüllen. Dieser Fragebogen soll uns helfen, Ihre Beschwerden besser zu verstehen. So können wir für Sie und andere Menschen, die unter ähnlichen Beschwerden leiden, langfristig eine erfolgreiche Therapie entwickeln.

Hinweise zur Bearbeitung des Fragebogens:
1. Es gibt keine richtigen oder falschen Antworten, sondern nur Ihre persönliche Meinung.
2. Wenn keine Antwort vollständig für Sie zutrifft, antworten Sie dann bitte so, wie es am ehesten für Sie paßt.
3. Bitte lassen Sie keine Frage aus.

Ihre Antworten haben keinen Einfluß auf Ihre Behandlung und Ihre Angaben werden vertraulich behandelt.

Vielen Dank für Ihre Mithilfe!

A. Krankheitsgeschichte: Rückenschmerzen

1. Haben Sie akute Rückenschmerzen zur Zeit? Wenn ja, wie lange dauert diese aktuelle akute Phase schon?	❑1 weniger als eine Woche	❑2 2-11 Wochen	❑3 3-6 Monate	❑4 ein halbes Jahr oder mehr

2. Wann sind Ihre Rückenschmerzen zum allererstenmal aufgetreten?	❑1 in der letzten Woche	❑2 in dem letzten halbes Jahr	❑3 in diesem Jahr	❑4 in den letzten 5 Jahren

❑5 in den letzten 10 Jahren	❑6 vor mehr als 10 Jahren

3. Wie stark waren Ihre Rückenschmerzen durchschnittlich in der letzten Woche? *(Zutreffendes bitte ankreuzen)*

0	1	2	3	4
keine Schmerzen				unerträgliche Schmerzen

4. Haben sich Ihre Rückenschmerzen im Laufe der Zeit verändert? (*Mehrere Anworten möglich*)

❑1 Nein.	❑2 Ja, sie sind stärker geworden.	❑3 Ja, sie sind häufiger geworden.	❑4 Ja, sie sind schwächer geworden.	❑5 Ja, sie sind seltener geworden.

5. Waren Sie wegen der aktuellen Rückenschmerzen bereits in Behandlung?

❑1 Nein.	❑2 Ja, bei einer Hausärztin / einem Hausarzt.	❑3 Ja, bei einer Fachärztin / einem Facharzt (Orthopäden).	❑4 Ja, bei einer/einem Kranken-gymnastIn.	❑5 Ja, bei einer/einem MasseurIn.

6. Wie oft haben Sie wegen der Schmerzen bereits eine Ärztin / einen Arzt aufgesucht?

❑1 Ein Mal.	❑2 2-3 Mal.	❑3 4-5 Mal.	❑4 6 Mal und mehr.

7. Strahlen Ihre aktuellen Rückenschmerzen in das Bein aus?

❑1 Nein.	❑2 Ja, in den Oberschenkel oder bis zum Knie.	❑3 Ja, bis in den Unterschenkel oder bis zum Fuß.

8. Sind Sie durch Ihre aktuellen Rückenschmerzen in Ihrem täglichen Leben eingeschränkt?

❑1 Nein.	❑2 Ja, aber nur wenig.	❑3 Ja, ich bin stark eingeschränkt.	❑4 Ja, ich bin sehr stark ein-geschränkt.

9. In welcher Aktivität des täglichen Lebens sind Sie am meisten eingeschränkt?

_____

10. Nehmen Sie wegen Ihrer Rückenschmerzen Schmerzmittel?

❑1 Nein.	❑2 Ja, täglich.	❑3 Ja, mehrmals in der Woche.	❑4 Ja, mehrmals im Monat.

11. Hatten Sie früher schon Rückenschmerzen? Wenn nein, beantworten Sie weiter Frage 12. Wenn ja, wie sind Sie damals behandelt worden? *(Mehrere Antworten möglich)*

❑1 Medikamente.	❑2 Kranken-gymnastik.	❑3 Massage.	**❑4** Sport/ Bewegung.	
❑6 Entspannung/ Bettruhe.	❑7 Kur.	❑8 Krank-schreibung.	❑9 Operation.	❑10 andere Behandlungen: _____

12. Was hilft Ihnen, die Rückenschmerzen zu lindern? *(Mehrere Antworten möglich)*

❑1 Medikamente.	❑2 Kranken-gymnastik.	❑3 Massage.	**❑4** Sport/ Bewegung.	❑5 Spritzen.
❑6 Entspannung.	❑7 Einreiben.	❑8 Wärme- oder Kälte-behandlung.	❑9 Keine Linderung möglich.	❑10 anderes: _____

13. Worauf sind Ihre Rückenschmerzen Ihrer Meinung nach zurückzuführen? (*Mehrere Antworten möglich*)

❑1 Körperliche Erkrankung (z.B. der Wirbelsäule oder der Bandscheibe).	❑2 Körperliche Überlastung.	❑3 Unfallfolgen.	**❑4** Operations-folge.	❑5 Einseitige Belastung (z.B. langes Sitzen, Stehen, etc.).

❑6 Streß am Arbeitsplatz.	❑7 Private Sorgen.	❑8 Sportfolgen.	❑9 anderes: _____

14. Wie stark ist – Ihrer Meinung nach – Ihr Rücken durch Ihre berufliche Tätigkeit belastet?
(*Bitte machen Sie entsprechend Ihrer beruflichen Rückenbelastung ein Kreuz auf der Skala*)

0	1	2	3	4	6	7	8	9	10
überhaupt nicht				mittel					extrem stark

15. Arbeiten Sie am Computer? Wenn ja, wieviel Stunden am Tag?

❑1 Nein.	❑2 Ja, und zwar _____ Stunden am Tag.

16. Wie stark belasten Sie – Ihrer Meinung nach – Ihren Rücken im Alltagsleben (*z.B. durch Gartenarbeit, Hausarbeit, Kleinkindpflege, usw.*)?

0	1	2	3	4	6	7	8	9	10
überhaupt nicht				mittel					extrem stark

17. Leiden Sie aufgrund Ihrer Rückenschmerzen unter einer der folgenden Beschwerden? (*Mehrere Anworten möglich*)

❑1 Appetitmangel.	❑2 Abnahme oder Verlust sexueller Bedürfnisse.	❑3 Allgemeine Reizbarkeit.	**❑4** Schlafprobleme.

18. Waren Sie jemals wegen Ihrer Rückenschmerzen krank geschrieben?

❑1 Nein.	❑2 Ja.

19. Sind Sie zur Zeit wegen Ihrer Rückenschmerzen krank geschrieben?

❑1 Nein.	❑2 Ja.

20. Wenn nein, beantworten Sie weiter Frage 21.
Wenn ja, wie lange waren / sind Sie insgesamt im letzten Jahr wegen Rückenschmerzen krank geschrieben?

❑1 eine Woche	❑2 2-11 Wochen	❑3 3-6 Monate	❑4 mehr als ein halbes Jahr	❑5 Ich werde / Ich bin berenntet.

21. Was hat Ihre Ärztin / Ihr Arzt Ihnen über die Diagnose mitgeteilt?

_____.

B. Begleiterkrankungen

1. Haben Sie – außer Rückenschmerzen – auch andere Schmerzen? Wenn nein, beantworten Sie weiter Frage 2.

❑1 Kopfschmerzen.	❑2 Schmerzen infolge einer Krebs-erkrankung.	❑3 Magen-schmerzen.	❑4 Gelenk-schmerzen.	❑5 andere Schmerzen. Welche? _____

2. Haben Sie – außer Rückenbeschwerden – auch andere Beschwerden oder Krankheiten? Wenn nein, beantworten Sie weiter Frage 3.

❑1 Krebs-erkrankung.	❑2 Herzerkrankung	❑3 Bluterkrankung.	❑4 Diabetes.	❑5 Schildrüsen-erkrankung.
❑6 Leber-erkrankung.	❑7 Allergien.	❑8 Asthma.	❑9 Arthritis / Arthrose.	❑10 Depression.
❑11 Sodbrennen.	❑12 Magen-geschwür.	❑13 Bluthochdruck.	❑14 Magen- / Darm-beschwerden.	❑15 Andere: _____

3. Hatten Sie – außer Rückenbeschwerden – auch andere Beschwerden oder Krankheiten? Wenn nein, beantworten Sie weiter Frage C1.

❑1 Krebs-erkrankung.	❑2 Herzerkrankung	❑3 Bluterkrankung.	❑4 Diabetes.	❑5 Schildrüsen-erkrankung.
❑6 Leber-erkrankung.	❑7 Allergien.	❑8 Asthma.	❑9 Arthritis / Arthrose.	❑10 Depression.
❑11 Sodbrennen.	❑12 Magen-geschwür.	❑13 Bluthochdruck.	❑14 Magen- / Darm-beschwerden.	❑15 Andere: _____

C. Allgemeine Gesundheit

1. Wie alt sind Sie? _____Jahre.

2. Welches Geschlecht haben Sie?

❑1 Weiblich.	❑2 Männlich.

3. Wieviel wiegen Sie?_____ kg.

4. Wie groß sind Sie?_____ cm.

5. Treiben Sie regelmäßig Sport? Wenn nein, beantworten Sie weiter Frage 8.

❑1 Nein.	❑2 Ja.

6. Wieviel Stunden pro Woche treiben Sie Sport?

_____ Stunden / Woche.

7. Wenn ja, welche Sportart(en)?

---

8. Wie ernähren Sie sich? (*Mehrere Anworten möglich*)

❑1 Keine besondere Diät.	❑2 Vegetarisch.	❑3 Vollwertig.	❑4 Fettarm.	❑5 Wenig Milchprodukte.
❑6 Schlankheitskur	❑7 Trennkost.	❑8 Anderes: _____		

9. Ergänzen Sie Ihre Ernährung mit der Einnahme von Vitaminen- und/oder Mineralientabletten?

❑1 Nein.	❑2 **Ja.**

10. Wie würden Sie Ihren Gesundheitszustand insgesamt einschätzen?
(*Bitte machen sie entsprechend Ihres Gesundheitszustandes ein Kreuz auf der Skala*)

0	1	2	3	4	6	7	8	9	10
sehr schlecht				mittel					sehr gut

11. Haben Sie früher geraucht oder rauchen Sie zur Zeit?

❑1 Habe früher geraucht, rauche jetzt nicht mehr.	❑2 Rauche zur Zeit.	❑3 Habe noch nie geraucht.

12. Wenn ja, wieviel rauchen Sie durchschnittlich?

❑1 1-10 Zigaretten am Tag.	❑2 11-20 Zigaretten am Tag.	❑3 mehr als 20 Zigaretten am Tag.	❑4 ich rauche Zigarren, Zigarillos, oder Pfeifen. Anzahl pro Tag: _____

13. Wieviel Bier, Wein oder andere alkoholischen Getränken nehmen Sie durchschnittlich in einer Woche zu sich?

❑1 6x ein halbes Liter Bier oder weniger.	❑2 7x ein halbes Liter Bier, oder mehr.	❑3 6x ein Glas Wein oder Sekt (0,2L), oder weniger.	❑4 7x ein Glas Wein oder Sekt (0,2L), oder mehr.	❑5 6x ein Glas Spirituosen (0,2dl), oder mehr.
❑6 7x ein Glas Spirituosen (0,2dl), oder mehr.	❑7 Ich trinke gelegentlich Alkohol (1-2 Mal im Monat).	❑8 Ich trinke keinen Alkohol.		

D. Es gibt unterschiedliche Meinungen über mögliche Zusammenhänge zwischen psychischen Faktoren und der Entstehung von bestimmten Krankheiten. Im folgenden haben wir einige solcher Faktoren aufgelistet. Bitte kreuzen Sie zu jedem Punkt an, ob bzw. in welchem Ausmaß er Ihrer Meinung nach zur Entstehung von Rückenschmerzen, Krebs- und/oder Herzkrankheiten beiträgt. Die Zahlen entsprechen folgenden Antwortmöglichkeiten:

-2	-1	0	+1	+2
völlig unzutreffend		weder zutreffend noch unzutreffend		völlig zutreffend

Bei der Entstehung dieser Krankheiten spielen folgende Ursachen eine wichtige Rolle:

	Rückenschmerzen	Krebserkrankung	Herzerkrankung
1. Unzufriedenheit am Arbeitsplatz.	-2 –1 0 +1 +2	-2 -1 0 +1 +2	-2 -1 0 +1 +2
2. Dauerhafte Überforderung.	-2 –1 0 +1 +2	-2 -1 0 +1 +2	-2 -1 0 +1 +2
3. Enttäuschungen über bestimmte nicht erreichte (Lebens-)Ziele.	-2 –1 0 +1 +2	-2 -1 0 +1 +2	-2 -1 0 +1 +2
4. Andauernde familiäre Probleme.	-2 –1 0 +1 +2	-2 -1 0 +1 +2	-2 -1 0 +1 +2
5. Immer wiederkehrende (Zukunfts-) Ängste.	-2 –1 0 +1 +2	-2 -1 0 +1 +2	-2 -1 0 +1 +2
6. Hoffnungslosigkeit und Resignation.	-2 –1 0 +1 +2	-2 -1 0 +1 +2	-2 -1 0 +1 +2
7. Ständiger Streß und Zeitdruck.	-2 –1 0 +1 +2	-2 -1 0 +1 +2	-2 -1 0 +1 +2
8. Verlust geliebter Personen durch Tod oder Trennung.	-2 –1 0 +1 +2	-2 -1 0 +1 +2	-2 -1 0 +1 +2
9. Häufige Unterdrückung von Wut und Ärger.	-2 –1 0 +1 +2	-2 -1 0 +1 +2	-2 -1 0 +1 +2
10. Einsamkeit und Kontaktarmut.	-2 –1 0 +1 +2	-2 -1 0 +1 +2	-2 -1 0 +1 +2
11. Ständiges Zurückstellen eigener Bedürfnisse.	-2 –1 0 +1 +2	-2 -1 0 +1 +2	-2 -1 0 +1 +2
	Rückenschmerzen	Krebserkrankung	Herzerkrankung

	Rückenschmerzen	Krebserkrankung	Herzerkrankung
12. Häufige Unterdrückung von Gefühlen von Trauer und Niedergeschlagenheit.	-2 –1 0 +1 +2	-2 -1 0 +1 +2	-2 -1 0 +1 +2
13. Hohe Leistungsanforderungen an sich selbst.	-2 –1 0 +1 +2	-2 -1 0 +1 +2	-2 -1 0 +1 +2
14. Anhaltende emotionale Unausgeglichenheit und Empfindlichkeit.	-2 –1 0 +1 +2	-2 -1 0 +1 +2	-2 -1 0 +1 +2
15. Anhaltende depressive Verstimmung.	-2 –1 0 +1 +2	-2 -1 0 +1 +2	-2 -1 0 +1 +2
16. Traumatische Erlebnisse.	-2 –1 0 +1 +2	-2 -1 0 +1 +2	-2 -1 0 +1 +2

E. Beantworten Sie jede Frage spontan nach dem Lesen. Dazu kreuzen Sie bitte gemäß dem Grad ihrer Zustimmung oder Ablehnung jeweils eine der folgenden Antwortmöglichkeiten an:

0	1	2	3	4
völlig unzutreffend		weder zutreffend noch unzutreffend		völlig zutreffend

1. Oft stehe ich fassungslos den Ereignissen in meinem Leben gegenüber.	0	1	2	3	4
2. Ich frage mich häufig: „Warum muß mir das gerade passieren?“.	0	1	2	3	4
3. Ich liebe das Leben.	0	1	2	3	4
4. Ich glaube, daß ich fast jeder Lebensaufgabe gewachsen bin.	0	1	2	3	4
5. Mein Leben ist ein einziges Chaos, da sich jeden Tag Dinge oder Situationen ereignen, die nicht vorhersehbar sind.	0	1	2	3	4
6. Was mein zukünftiges Leben angeht, bin ich sehr optimistisch.	0	1	2	3	4
7. Ich kann oft nicht verstehen, warum die Dinge sich so entwickeln und nicht anders.	0	1	2	3	4
8. Ich bin ein Optimist.	0	1	2	3	4
9.Im Großen und Ganzen habe ich großes Vertrauen in die Fähigkeiten und Absichten unserer Politiker.	0	1	2	3	4

F. Lesen Sie bitte die folgenden Sätze. Entscheiden Sie bei jeder Fragestellung, ob sie für Sie zutrifft oder nicht. Kreuzen Sie eine der vier Ziffern entsprechend der Stärke Ihrer Zustimmung bzw. Ablehnung an. Lassen Sie bitte keinen Satz aus.

0	1	2	3
trifft gar nicht zu	trifft etwas zu	trifft über-wiegend zu	trifft ausge-sprochen zu

1. In letzter Zeit bin ich sehr ängstlich und schreckhaft.	0	1	2	3
2. Ich fühle mich niedergeschlagen und schwermütig.	0	1	2	3
3. Ich kann das, was ich lese, nicht mehr so gut verstehen wie früher.	0	1	2	3
4. Am liebsten würde ich mir das Leben nehmen.	0	1	2	3
5. Ich habe zu anderen Menschen keine innere Beziehung mehr.	0	1	2	3
6. Ich fühle, daß ich nahe daran bin, zusammenzubrechen.	0	1	2	3
7. Ich habe ständig Angst, daß ich etwas Falsches sagen oder tun könnte.	0	1	2	3
8. Ich bin jetzt viel weniger am Liebesleben interessiert.	0	1	2	3
9. Oft fühle ich mich einfach miserabel.	0	1	2	3
10. Ich komme beim besten Willen nicht mit den kleinsten Gedankenschritten voran.	0	1	2	3
11. Ich habe keine Gefühle mehr.	0	1	2	3

G. Arbeit / Beschäftigung

Wie betrachten Sie ihre Arbeitsstelle? Kreuzen Sie eine der fünf Ziffern entsprechend der Häufigkeit der folgenden Gedanken an. Lassen Sie bitte keinen Satz aus.

0	1	2	3	4
sehr selten	selten	hin und wieder	oft	sehr oft

1. Ich freue mich nach einem Urlaub, wieder arbeiten gehen zu können.	0	1	2	3	4
2. Ich bin mit meiner Arbeitssituation zufrieden und ich hoffe, daß sie so bleibt.	0	1	2	3	4
3. Als ArbeitnehmerIn habe ich wenig Einfluß auf meine Arbeitssituation.	0	1	2	3	4
4. Ich bin von meiner Arbeitsstelle nicht begeistert, aber sie könnte schlimmer sein.	0	1	2	3	4
5. Ich bin nahe dran, zu kündigen. Die Arbeitssituation ist nicht mehr erträglich.	0	1	2	3	4

6. Wie zufrieden Sie sind insgesamt mit Ihrer Arbeitsstelle?

0	1	2	3	4
sehr unzufrieden		weder / noch		sehr zufrieden

H. Ausbildung

1. Welchen Schulabschluß haben Sie?
(Falls Sie mehrere Abschlüsse haben, nennen Sie bitte nur den höchsten.)

❑1 Volksschul-/Hauptschul-abschluß.	❑2 Mittlere Reife, Realschul-abschluß.	❑3 Fachhochschul-reife.	❑4 Abitur.

❑5 Ich habe keinen Schulabschluß.	❑6 Anderer Schulabschluß, und zwar: _____

2. Haben Sie eine abgeschlossene Berufsausbildung oder Hochschulausbildung? Wenn ja, welche?
(Falls Sie mehrere Ausbildungen abgeschlossen haben, geben Sie bitte die letzte an.)

❑1 Gewerbliche oder landwirt-schaftliche Lehre.	❑2 Kaufmännische oder sonstige Lehre.	❑3 Berufsschule, oder Handelsschule.	❑4 Schule des Gesundheits-wesens.	❑5 Fachschule, z.B. Technikerschule

❑6 Beamten-ausbildung.	❑7 Fachhochschule oder Ingenieurschule	❑8 Universität oder Hochschule.	❑9 Ich habe keinen Ausbildungs-abschluß.	❑10 Sonstiger Ausbildungs-abschluß, und zwar: _____

Fragebogen für die Ärztin / den Arzt

1. Wird die oberflächliche Haut im Lumbalbereich als schmerzhaft empfunden oder gibt der Patient/die Patientin Schmerzen bei Druck auf die Beckenkämme, das Sakrum oder die BWS an?

❑1 Nein.	❑2 Ja.

2. Ist sanfter Druck auf den Kopf bei stehendem/stehender PatientIn schmerzhaft?

❑1 Nein.	❑2 Ja.

3. Ist die Schmerzangabe beim Anheben des gestreckten Beines im Sitzen heftiger als im Liegen?

❑1 Nein.	❑2 Ja.

4. Gibt es motorische oder sensorische Defizite, die sich nicht einer oder mehreren Wurzeln zuordnen lassen?

❑1 Nein.	❑2 Ja.

5. Zeigt die Patientin / der Patient eine übertriebene Reaktion auf den Schmerz (z.B. durch Reiben der schmerzhaften Regionen, durch schmerzgeplagte Mimik, Stöhnen, Ächzen oder Abstützen im Gehen)?

❑1 Nein.	❑2 Ja.

6. Wie würden Sie die Ursachen des Schmerzes klassifizieren?

❑1 Angeborene/ erworbene Formfehler der Wirbelsäule.	❑2 Bandscheiben-vorfall ohne radikuläre Reizung.	❑3 Bandscheiben-vorfall mit radikulärer Reizung.	❑4 Spezifische Erkrankung der Wirbelsäule.	❑5 Unspezifischer Rückenschmerz bei Störung von: _____
❑6 Posttraumati-scher Schmerz.	❑7 Infolge eines benigner Tumors.	❑8 Infolge eines Malignom.	❑9 Rückenschmerz sonstiger Genese.	❑10 Postoperativer Schmerz.
❑11 Degenerativer Zustand.	❑12 Psycho-somatische Genese.			

Psychologisches Institut der Universität Heidelberg

FRAGEBOGEN GESUNDHEIT

Liebe Patientin, lieber Patient,
heute suchen Sie Ihren Arzt auf. Unsere Forschungsgruppe beschäftigt sich mit den Bedingungen von Krankheit und Gesundheit. Dieser Fragebogen soll helfen, bestimmte Krankheiten besser zu verstehen. So können wir für Sie und andere Menschen, die unter ähnlichen Beschwerden leiden, langfristig eine erfolgreiche Therapie entwickeln.

Hinweise zur Bearbeitung des Fragebogens:

1. Es gibt keine richtigen oder falschen Antworten, sondern nur Ihre persönliche Meinung.
2. Wenn keine Antwort vollständig für Sie zutrifft, antworten Sie dann bitte so, wie es am ehesten für Sie paßt.
3. Bitte lassen Sie keine Frage aus.

Ihre Antworten haben keinen Einfluß auf Ihre Behandlung und Ihre Angaben werden vertraulich behandelt.

Vielen Dank für Ihre Mithilfe!

Krankheitsgeschichte

1. Was ist der Grund Ihres heutigen Arztbesuches?

_____

2. Haben Sie Schmerzen? Wenn nein, beantworten Sie weiter Frage 5.

❑1 Kopfschmerzen.	❑2 Schmerzen infolge einer Krebs-erkrankung.	❑3 Magen-schmerzen.	**❑4** Rücken-schmerzen.	❑5 andere Schmerzen. Welche? _____

3. Haben Sie zur Zeit akute Schmerzen? Wenn ja, wie lange dauert diese aktuelle Phase schon?	❑1 weniger als eine Woche	❑2 2-11 Wochen	❑3 3-6 Monate	❑4 ein halbes Jahr oder mehr

4. Wie stark waren Ihre Schmerzen durchschnittlich in der letzten Woche?
(Zutreffendes bitte ankreuzen)

0	1	2	3	4
keine Schmerzen				unerträgliche Schmerzen

5. Haben Sie (in der Gegenwart) eine oder mehrere der folgenden Beschwerden oder Krankheiten? Wenn nein, beantworten Sie weiter Frage 1 „Allgemeine Gesundheit“.

❑1 Krebs-erkrankung.	❑2 Herzerkrankung	❑3 Bluterkrankung.	❑4 Diabetes.	❑5 Schildrüsen-erkrankung.

❑6 Leber-erkrankung.	❑7 Allergien.	❑8 Asthma.	❑9 Arthritis / Arthrose.	❑10 Depression.

❑11 Sodbrennen.	❑12 Magen-geschwür.	❑13 Bluthochdruck.	❑14 Magen- / Darm-beschwerden.	❑15 Andere: _____

6. Hatten Sie (in der Vergangenheit) eine oder mehrere der folgenden Beschwerden oder Krankheiten? Wenn nein, beantworten Sie weiter Frage 1 „Allgemeine Gesundheit".

❑1 Krebs-erkrankung.	❑2 Herzerkrankung	❑3 Bluterkrankung.	❑4 Diabetes.	❑5 Schildrüsen-erkrankung.
❑6 Leber-erkrankung.	❑7 Allergien.	❑8 Asthma.	❑9 Arthritis / Arthrose.	❑10 Depression.
❑11 Sodbrennen.	❑12 Magen-geschwür.	❑13 Bluthochdruck.	❑14 Magen- / Darm-beschwerden.	❑15 Andere: _____

Allgemeine Gesundheit

1. Wie alt sind Sie? _____Jahre.

2. Welches Geschlecht haben Sie?

❑1 Weiblich.	❑2 Männlich.

3. Wieviel wiegen Sie?_____ kg.

4. Wie groß sind Sie?_____ cm.

5. Treiben Sie regelmäßig Sport? Wenn nein, beantworten Sie weiter Frage 8.

❑1 Nein.	❑2 Ja.

6. Wieviel Stunden pro Woche treiben Sie Sport?

_____ Stunden / Woche.

7. Wenn ja, welche Sportart(en)?

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8. Wie ernähren Sie sich? (*Mehrere Anworten möglich*)

❑1 Keine besondere Diät.	❑2 Vegetarisch.	❑3 Vollwertig.	❑4 Fettarm.	❑5 Wenig Milchprodukte.
❑6 Schlankheitskur	❑7 Trennkost.	❑8 Anderes: _____		

9. Ergänzen Sie Ihre Ernährung mit der Einnahme von Vitaminen- und/oder Mineralientabletten?

❑1 Nein.	❑2 Ja.

10. Wie würden Sie Ihren Gesundheitszustand insgesamt einschätzen? (*Bitte machen sie entsprechend Ihres Gesundheitszustandes ein Kreuz auf der Skala*)

0	1	2	3	4	6	7	8	9	10
sehr schlecht				mittel					sehr gut

11. Haben Sie früher geraucht oder rauchen Sie zur Zeit?

❑1 Habe früher geraucht, rauche jetzt nicht mehr.	❑2 Rauche zur Zeit.	❑3 Habe noch nie geraucht.

12. Wenn ja, wieviel rauchen Sie durchschnittlich?

❑1 1-10 Zigaretten am Tag.	❑2 11-20 Zigaretten am Tag.	❑3 mehr als 20 Zigaretten am Tag.	❑4 ich rauche Zigarren, Zigarillos, oder Pfeifen. Anzahl pro Tag: _____

13. Wieviel Bier, Wein oder andere alkoholischen Getränken nehmen Sie durchschnittlich in einer Woche zu sich?

❑1 6x ein halbes Liter Bier oder weniger.	❑2 7x ein halbes Liter Bier, oder mehr.	❑3 6x ein Glas Wein oder Sekt (0,2L), oder weniger.	❑4 7x ein Glas Wein oder Sekt (0,2L), oder mehr.	❑5 6x ein Glas Spirituosen (0,2dl), oder mehr.
❑6 7x ein Glas Spirituosen (0,2dl), oder mehr.	❑7 Ich trinke gelegentlich Alkohol (1-2 Mal im Monat).	❑8 Ich trinke keinen Alkohol.		

Es gibt unterschiedliche Meinungen über mögliche Zusammenhänge zwischen psychischen Faktoren und der Entstehung von bestimmten Krankheiten. Im folgenden haben wir einige solcher Faktoren aufgelistet. Bitte kreuzen Sie zu jedem Punkt an, ob bzw. in welchem Ausmaß er Ihrer Meinung nach zur Entstehung von Rückenschmerzen, Krebs- und/oder Herzkrankheiten beiträgt. Die Zahlen entsprechen folgenden Antwortmöglichkeiten:

-2	-1	0	+1	+2
völlig unzutreffend		weder zutreffend noch unzutreffend		völlig zutreffend

Bei der Entstehung dieser Krankheiten spielen folgende Ursachen eine wichtige Rolle:

	Rückenschmerzen	Krebserkrankung	Herzerkrankung
1. Unzufriedenheit am Arbeitsplatz.	-2 –1 0 +1 +2	-2 -1 0 +1 +2	-2 -1 0 +1 +2
2. Dauerhafte Überforderung.	-2 –1 0 +1 +2	-2 -1 0 +1 +2	-2 -1 0 +1 +2
3. Enttäuschungen über bestimmte nicht erreichte (Lebens-)Ziele.	-2 –1 0 +1 +2	-2 -1 0 +1 +2	-2 -1 0 +1 +2
4. Andauernde familiäre Probleme.	-2 –1 0 +1 +2	-2 -1 0 +1 +2	-2 -1 0 +1 +2
5. Immer wiederkehrende (Zukunfts-) Ängste.	-2 –1 0 +1 +2	-2 -1 0 +1 +2	-2 -1 0 +1 +2
6. Hoffnungslosigkeit und Resignation.	-2 –1 0 +1 +2	-2 -1 0 +1 +2	-2 -1 0 +1 +2
7. Ständiger Streß und Zeitdruck.	-2 –1 0 +1 +2	-2 -1 0 +1 +2	-2 -1 0 +1 +2
8. Verlust geliebter Personen durch Tod oder Trennung.	-2 –1 0 +1 +2	-2 -1 0 +1 +2	-2 -1 0 +1 +2
9. Häufige Unterdrückung von Wut und Ärger.	-2 –1 0 +1 +2	-2 -1 0 +1 +2	-2 -1 0 +1 +2
10. Einsamkeit und Kontaktarmut.	-2 –1 0 +1 +2	-2 -1 0 +1 +2	-2 -1 0 +1 +2
11. Ständiges Zurückstellen eigener Bedürfnisse.	-2 –1 0 +1 +2	-2 -1 0 +1 +2	-2 -1 0 +1 +2

	Rückenschmerzen	Krebserkrankung	Herzerkrankung
12. Häufige Unterdrückung von Gefühlen von Trauer und Niedergeschlagenheit.	-2 –1 0 +1 +2	-2 -1 0 +1 +2	-2 -1 0 +1 +2
13. Hohe Leistungsanforderungen an sich selbst.	-2 –1 0 +1 +2	-2 -1 0 +1 +2	-2 -1 0 +1 +2
14. Anhaltende emotionale Unausgeglichenheit und Empfindlichkeit.	-2 –1 0 +1 +2	-2 -1 0 +1 +2	-2 -1 0 +1 +2
15. Anhaltende depressive Verstimmung.	-2 –1 0 +1 +2	-2 -1 0 +1 +2	-2 -1 0 +1 +2
16. Traumatische Erlebnisse.	-2 –1 0 +1 +2	-2 -1 0 +1 +2	-2 -1 0 +1 +2

Beantworten Sie jede Frage spontan nach dem Lesen. Dazu kreuzen Sie bitte gemäß dem Grad ihrer Zustimmung oder Ablehnung jeweils eine der folgenden Antwortmöglichkeiten an:

0	1	2	3	4
völlig unzutreffend		weder zutreffend noch unzutreffend		völlig zutreffend

1. Oft stehe ich fassungslos den Ereignissen in meinem Leben gegenüber.	0	1	2	3	4
2. Ich frage mich häufig: „Warum muß mir das gerade passieren?“.	0	1	2	3	4
3. Ich liebe das Leben.	0	1	2	3	4
4. Ich glaube, daß ich fast jeder Lebensaufgabe gewachsen bin.	0	1	2	3	4
5. Mein Leben ist ein einziges Chaos, da sich jeden Tag Dinge oder Situationen ereignen, die nicht vorhersehbar sind.	0	1	2	3	4
6. Was mein zukünftiges Leben angeht, bin ich sehr optimistisch.	0	1	2	3	4
7. Ich kann oft nicht verstehen, warum die Dinge sich so entwickeln und nicht anders.	0	1	2	3	4
8. Ich bin ein Optimist.	0	1	2	3	4
9.Im Großen und Ganzen habe ich großes Vertrauen in die Fähigkeiten und Absichten unserer Politiker.	0	1	2	3	4

Lesen Sie bitte die folgenden Sätze. Entscheiden Sie bei jeder Fragestellung, ob sie für Sie zutrifft oder nicht. Kreuzen Sie eine der vier Ziffern entsprechend der Stärke Ihrer Zustimmung bzw. Ablehnung an. Lassen Sie bitte keinen Satz aus.

0	1	2	3
trifft gar nicht zu	trifft etwas zu	trifft überwiegend zu	trifft ausgesprochen zu

1. In letzter Zeit bin ich sehr ängstlich und schreckhaft.	0	1	2	3
2. Ich fühle mich niedergeschlagen und schwermütig.	0	1	2	3
3. Ich kann das, was ich lese, nicht mehr so gut verstehen wie früher.	0	1	2	3
4. Am liebsten würde ich mir das Leben nehmen.	0	1	2	3
5. Ich habe zu anderen Menschen keine innere Beziehung mehr.	0	1	2	3
6. Ich fühle, daß ich nahe daran bin, zusammenzubrechen.	0	1	2	3
7. Ich habe ständig Angst, daß ich etwas Falsches sagen oder tun könnte.	0	1	2	3
8. Ich bin jetzt viel weniger am Liebesleben interessiert.	0	1	2	3
9. Oft fühle ich mich einfach miserabel.	0	1	2	3
10. Ich komme beim besten Willen nicht mit den kleinsten Gedankenschritten voran.	0	1	2	3
11. Ich habe keine Gefühle mehr.	0	1	2	3

Arbeit / Beschäftigung

Wie betrachten Sie ihre Arbeitsstelle? Kreuzen Sie eine der fünf Ziffern entsprechend der Häufigkeit der folgenden Gedanken an. Lassen Sie bitte keinen Satz aus.

0	1	2	3	4
sehr selten	selten	hin und wieder	oft	sehr oft

1. Ich freue mich nach einem Urlaub, wieder arbeiten gehen zu können.	0	1	2	3	4
2. Ich bin mit meiner Arbeitssituation zufrieden und ich hoffe, daß sie so bleibt.	0	1	2	3	4
3. Als ArbeitnehmerIn habe ich wenig Einfluß auf meine Arbeitssituation.	0	1	2	3	4
4. Ich bin von meiner Arbeitsstelle nicht begeistert, aber sie könnte schlimmer sein.	0	1	2	3	4
5. Ich bin nahe dran, zu kündigen. Die Arbeitssituation ist nicht mehr erträglich.	0	1	2	3	4

6. Wie zufrieden Sie sind insgesamt mit Ihrer Arbeitsstelle?

0	1	2	3	4
sehr unzufrieden		weder / noch		sehr zufrieden

Ausbildung

1. Welchen Schulabschluß haben Sie?
(Falls Sie mehrere Abschlüsse haben, nennen Sie bitte nur den höchsten.)

❑1 Volksschul-/Hauptschul-abschluß.	❑2 Mittlere Reife, Realschul-abschluß.	❑3 Fachhochschul-reife.	❑4 Abitur.

❑5 Ich habe keinen Schulabschluß.	❑6 Anderer Schulabschluß, und zwar: _____

2. Haben Sie eine abgeschlossene Berufsausbildung oder Hochschulausbildung? Wenn ja, welche?
(Falls Sie mehrere Ausbildungen abgeschlossen haben, geben Sie bitte die letzte an.)

❑1 Gewerbliche oder landwirt-schaftliche Lehre.	❑2 Kaufmännische oder sonstige Lehre.	❑3 Berufsschule, oder Handelsschule.	❑4 Schule des Gesundheits-wesens.	❑5 Fachschule, z.B. Technikerschule

❑6 Beamten-ausbildung.	❑7 Fachhochschule oder Ingenieurschule	❑8 Universität oder Hochschule.	❑9 Ich habe keinen Ausbildungs-abschluß.	❑10 Sonstiger Ausbildungs-abschluß, und zwar: _____