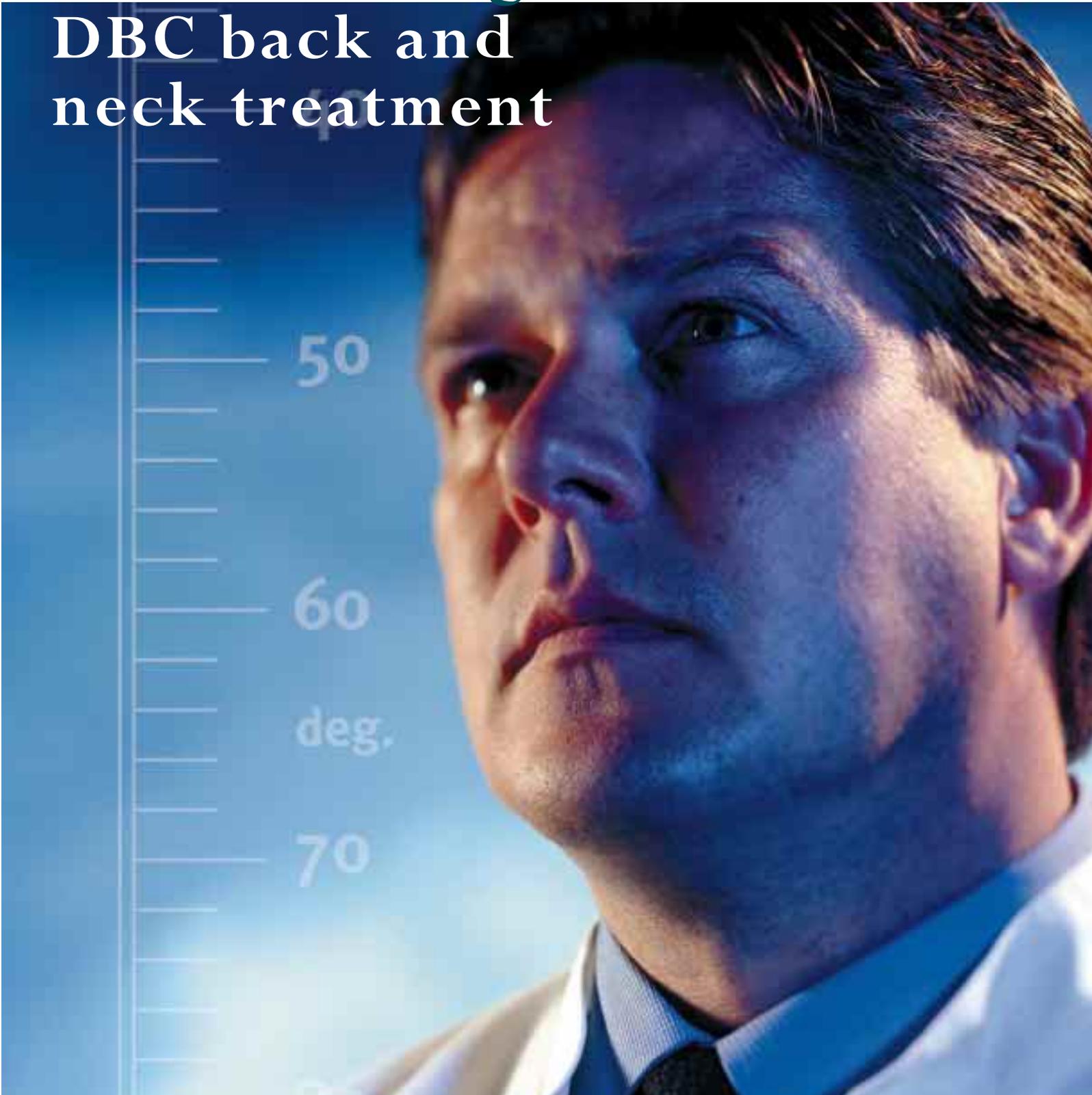


medical background

**DBC back and
neck treatment**



contents

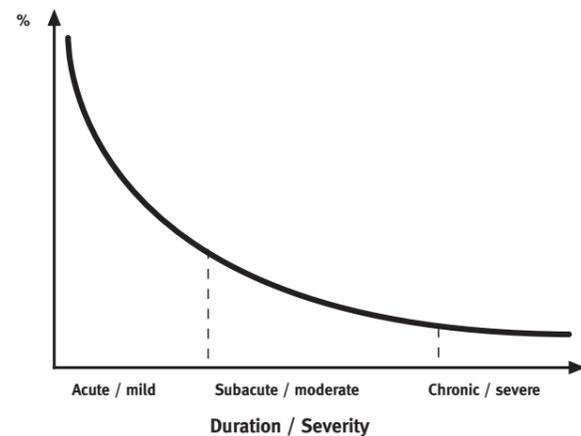
1. Introduction	4	6. DBC Active Spine Care	20
2. LBP Treatment Approaches	5	Basic principles	20
Outcome criteria	6	Selection criteria for DBC Active Spine	
Treatment efficacy	7	Care	20
The evidence	7	Evaluation protocols and follow-up	
3. The Background of Back Problems	8	of the treatment	21
Pain control and its changes with		Active treatment	22
chronic pain	9	<i>The DBC Devices</i>	23
Contribution of brain anatomy to		<i>The therapist's role</i>	23
information processing and pain	10	<i>Additional treatments</i>	23
Pain and deconditioning syndrome	10	Outcome criteria	24
Avoidance behaviour	11	7. DBC Results	24
Failures of trunk movement control	12	Pain reduction and psychological well	
<i>Protective guarding and reflex inhibition</i>	13	being	24
<i>Missing flexion relaxation</i>	13	Mobility and strength gains	24
<i>Delayed responses to sudden loading</i>	14	Associations between pain, mobility	
<i>Ineffective anticipatory trunk</i>		and strength changes	25
<i>stabilization</i>	14	Pain reduction and lumbar endurance	
<i>Abnormal postural control</i>	15	improvement in a randomized setting	25
<i>Delayed reaction times</i>	15	Active treatment in chronic neck pain	
<i>Abnormal lumbar fatigue</i>	16	-A prospective randomized study	26
<i>Abnormal gluteal fatigability</i>	17	Absenteeism from work after DBC	27
Summary: A model	17	8. DBC Quality Assurance	28
4. The Aim in Active Rehabilitation	17	9. References	30
5. Exercise Physiology	18		
Dosage and target	18		
Efficacy of exercises in LBP	19		
<i>Acute LBP</i>	19		
<i>Subacute LBP</i>	19		
<i>Chronic LBP</i>	19		
<i>Chronic Disabling LBP</i>	19		
<i>Recurrent LBP</i>	19		
<i>Postoperative / Post-traumatic LBP</i>	19		
Back specificity of exercises	20		



Simo Taimela, MD, DrMedSc
Research Associate Professor
Medical Director, DBC International

1. introduction

Low back pain (LBP) affects nearly half of the adult population in one given year and up to 80% of all adults will have at least one episode of back pain in their lifetime^{11, 21, 36, 95, 117, 151}. Most of these episodes are transient in their character and will not cause the patients to see a doctor. About 70% of all patients claim to be well and back at work within 14 days and about 90% within two months. The remaining 10% will tend to become chronic and have pain and disability beyond three months. However, many studies indicate that the natural course of low back trouble in reality is fluctuating, i.e. varies over time^{37, 48}. Each recurrence increases the likelihood of a new one, previous LBP being a strong predictor of the next LBP^{18, 58, 170}. Recent studies indicate that the first acute pain episode may cause recurrent symptoms longer than previously believed. Half of the adult population suffers from LBP yearly^{21, 95, 117}. The high prevalence of low back trouble (LBT) and its recurrence is mirrored in the number of lost working days resulting from LBT and the number of individuals seeking medical care. Many cases of backache are never reported and may not cause much worry¹¹⁷. There are, however, other cases where the individual will, for various reasons, take prolonged duration of absence from work. Eventually some will progress to a chronic state, representing a major socio-economic problem. Effective management of the disabling consequences of LBT is a major challenge for industrialized society^{123, 125}.



The relative proportion of population by duration/severity of low back trouble.

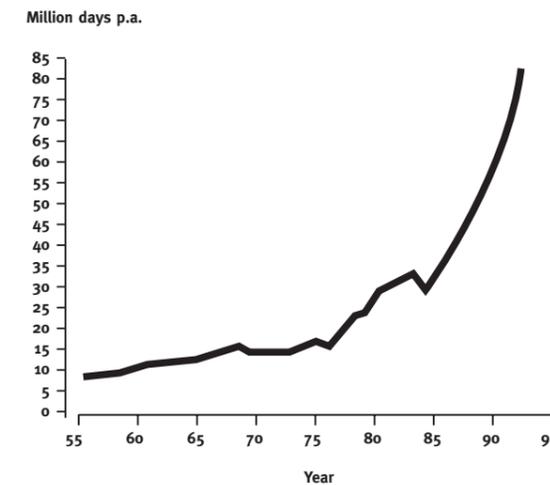
The costs of back and neck trouble are enormous. For example, the estimate of total costs for back and neck-shoulder pain in Sweden (population some 8 million people) is 22 billion SEK (\$2.8 billion) per year, the main part (>90%) being costs for work absenteeism and early retirement. Despite the fact that there has been no increase in the incidence of back pain over the years, there has been a steady increase in disability and costs because of back pain during the last decades. In the USA, for example, the rate of disability claims has increased 14 times faster than the population growth. Despite the magnitude of the problem the cause of back pain is still not known in up to 85% of the cases³⁷.

A wide range of different treatment modalities has been introduced over the years with a large variation in their use in different countries and within different specialties. For example, there is a five-fold difference in the rates of back surgery in the developed countries²⁷. The introduction of modern diagnostic tools such as computerized tomography (CT) and magnetic resonance imaging (MRI) has not only refined the possibility of obtaining a specific diagnosis, but has also lead to an increase in the number of evaluations done and to an unacceptable rate of false positive pathologic findings. This has lead to an increase in back surgery and other invasive procedures based on imaging modalities. In recent studies from 26 % up to 67% of pathologic spinal findings have been diagnosed in MRI-examinations of healthy subjects in lumbar^{15, 19, 72, 171}, thoracic and cervical spine¹⁶. All this has lead to an increase in the costs for treating back and neck patients with improved imaging methods, although there has been no evidence of a significant decrease in the long-term disability of patients with the disorders.

As there are only certain specific back and neck problems such as disc herniations, spinal stenosis and spondylolisthesis that may benefit from surgery, other treatment modalities for non-specific mechanical back and neck pain such as pain relief, training programs and manipulative therapy need to be used. In recent years, several studies have shown the efficacy of exercises in the subacute and

2. LBP treatment approaches

chronic phase of back pain, i.e. from four weeks and onwards^{25, 56, 91, 97, 98, 106-108, 114, 122, 134}. Recent government-based consensus statements in e.g. United States, Great Britain, the Netherlands, Norway and Finland, and systematic literature reviews¹⁶⁵ have subsequently acknowledged the role of exercises in the treatment of subchronic and chronic back pain. No clear evidence for training in the acute phase of back pain exists^{44, 45, 105}. The U.S. Agency for Health Policy and Research² stated in their Management Guidelines for Acute Low-Back Pain that bedrest for acute LBP should be as short as possible, low-stress aerobic exercise can prevent debilitation due to inactivity, and endurance exercise programs can be started during the first two weeks. Gradually increased conditioning exercises for trunk muscles are helpful according to the statement also in the treatment of acute back pain, especially if symptoms persist².



The number of days lost due to low back trouble during the last few decades: note the changes at the time of the introduction of CT (~75) and MRI (~85).

The DBC Active Spine Care method was introduced as a structured evaluation and treatment concept for patients with low back and neck trouble in 1992.

The active rehabilitation program has been shown to increase strength, mobility and endurance and to decrease pain in lumbar spine^{69, 82, 153}. Decrease of pain and improved function after active rehabilitation are long-term^{83, 111},

especially if the patient remains active after the treatment¹⁵². At least short-term changes in psychological well being have also been documented in these patients⁶⁹. After DBC active rehabilitation program as much as 80 percent of chronic low back pain patients are shown to remain working without absenteeism in a two-year follow-up, if they stay active after the treatment¹⁵². For each patient, a standardized set of evaluations and an individual active rehabilitation program based on them are performed; specially designed training equipment for extension, flexion, rotation and lateral flexion of the lumbar spine are used under the strict supervision of a specially trained therapist. During the treatment, the therapists encourage positive attitudes and beliefs to alter avoidance behaviors by encouraging an active approach. The treatment program will later be described in detail. Also, the concept includes formal education and quality assurance of the involved clinics, and a central data collection of results, which are reported by the clinics into special computer software. The software contains questionnaires with pertinent questions regarding different aspects of the medical history and status of the patients in a structured manner. The database information is continuously updated and available for statistical analyses. Ongoing research and development is done for the improvement of the method.

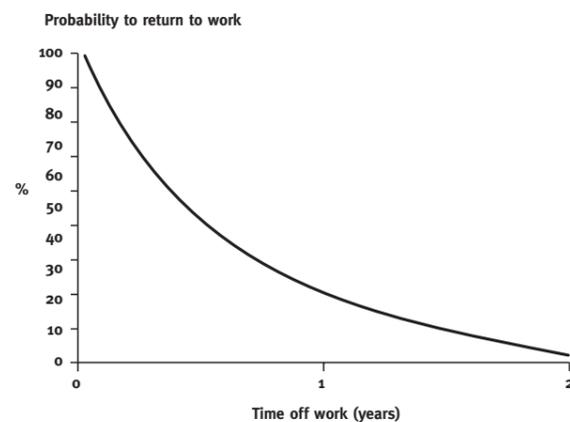
In the following chapters the DBC-concept will be described in more detail.

2. LBP Treatment Approaches

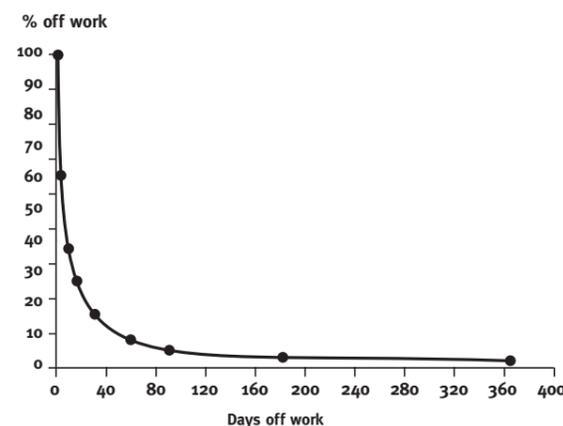
Back pain has a high recurrence rate and one of the best predictors for future back pain is a previous experience of the disorder. With highly prevalent disorders it is difficult to identify risk factors and determinants. Very few risk factors for low back pain are known and they predict only a minor part of the disorders. Furthermore, most of the risk factors identified to date such as age, sex, previous back pain history, initial pain severity or the presence of sciatica appear to be either by definition invariable or, at least, very difficult to change with current treatments. It may be that

back pain itself is so common that we should not expect to prevent a large proportion of it. Instead, several experts recommend that the focus should be on reducing the socially and economically important consequences of impairment and disability of back pain, and especially on preventing chronic low back trouble. It has been widely recognized that a small percentage of LBP patients (some 10%) produce the majority (over 75%) of LBT-related costs mainly due to prolonged absenteeism and early retirement from work⁵.

The probability to return-to-work reduces drastically after a person has been out of work for a few weeks. After six months of absenteeism, the probability to return to work is only some 50% and after one year some 15%,



The probability to return to work diminishes by the time out of work. Therefore, early active rehabilitation is preferred to waiting for pain chronicity.



A small proportion of LBP patients are responsible for the majority of costs due to prolonged absenteeism; the majority of chronic LBP patients have not been out of work or have been out of work just a few occasional days.

regardless of treatment. Moreover, the majority of chronic back sufferers have not (yet) been out of work, but are facing a situation where each relapse increases the probability to drop out of work. These facts emphasize the importance of early rehabilitation to maintain normal function and ability to work.

Outcome criteria

It is important to distinguish between the different types of outcome that follow prolonged LBP. One way to present the outcome criteria is division into pain, function, degeneration and costs. Pain by definition (by the International Association for the Study of Pain) is “a subjective emotional experience that is described as a tissue injury, or as the threat of tissue injury”. Being entirely subjective and next to impossible to measure in an objective way, it has to be regarded separately from the level of physical function that can also be measured objectively for example strength, mobility, endurance and co-ordination, or assessed as experienced impairment of daily functioning with questionnaires. Pain and pain behavior may limit physical function per se, but another possible reason for physical impairment may be physical deconditioning due to disuse even without pain. Tissue degeneration is another feature associated with LBT. However, although previously considered as an important factor in low back pain, disc pathology and degeneration is now considered to be only moderately associated with low back pain^{42, 72, 127, 132, 133}. Moreover, disc degeneration is strongly determined by genetic factors and only with a relatively small proportion by physical loading or other external factors^{9, 166}. However, one feature in the degenerative process, paraspinal muscle atrophy, has been well documented with prolonged LBT^{30, 47, 55, 94, 142}. Still direct and indirect costs are the key factors that make LBT such an important issue from the society’s and insurance industry’s point of view. LBT-related costs are enormous, the disorder being one of the most expensive health problems in western societies^{5, 36, 123, 125}.

Treatment efficacy

Three major categories can be defined in the approaches for rehabilitation of chronic back and neck trouble. In pain management, the main emphasis is on pain eradication. This can be achieved by various techniques such as medication, manipulation, physical modalities, acupuncture etc. In work hardening, the critical physical demands of the previous job are simulated in a clinical setting. This approach has gained popularity in the United States being less frequently practiced in European countries. An active rehabilitation program uses exercises in the treatment, and its main emphasis is on restoring full physical function. This approach has gained popularity especially in the treatment of LBT since very good results were presented in the mid 80’s^{107, 113, 114}. Different modes of exercises have had their role in treatments following the active rehabilitation approach^{57, 115}. Functional restoration programs are a specific form of active rehabilitation. They generally use an aggressive program of physical exercises and psychosocial support with the focus on improving function despite the pain.

The evidence

Evidence-based medicine is the devoted and judicious use of the best current evidence in making decisions about the care of individual patients. It means integrating individual clinical expertise with the best available external clinical

evidence from systematic research. Individual clinical expertise means the talent and judgment that an individual clinician acquires through clinical experience and clinical practice. Best available external clinical evidence means clinically relevant research, which sometimes includes basic sciences, but especially patient-centered clinical research into the accuracy and precision of diagnostic tests, the power of prognostic factors, and the efficacy and safety of therapeutic procedures. External clinical evidence both invalidates previously accepted diagnostic tests and treatments and replaces them with new ones that are more accurate, more efficacious and safer. Good doctors use both individual clinical expertise and the best available external evidence. Neither one alone is enough. Without clinical expertise, practice becomes dominated by evidence, which may be inapplicable for an individual patient. Without the current best evidence, practice becomes outdated, to the disadvantage of patients¹⁴¹.

The selection of the treatment modality should be based on the strength of the scientific evidence. Randomized controlled trial (RCT) is largely regarded to be the strongest scientific proof of the efficacy of an intervention. However, not only the type of the study but also the methodological quality of the study is important when valuing the level of scientific evidence. An increasingly popular way to tackle the question of scientific evidence is the publication of

Strong Evidence Multiple relevant, high quality RCTs
<ul style="list-style-type: none"> • NSAIDs are more effective than placebo in the treatment of uncomplicated LBP, but not in acute sciatica. Various types of NSAIDs are equally effective. • Muscle relaxants are more effective than placebo for acute LBP. Different types of muscle relaxants are equally effective. • Bed rest is not effective. • Exercise therapy is not more effective than other conservative treatments, including no intervention.
Moderate evidence One relevant, high quality RCT and ≥ 1 relevant, low quality RCT
<ul style="list-style-type: none"> • Analgesics are not more effective than NSAIDs
Limited evidence One relevant, high quality RCT or multiple relevant, low quality RCTs
<ul style="list-style-type: none"> • Manipulation is better than placebo • Traction • Epidural steroid injections for acute LBP with nerve root pain and radicular neurologic deficit
No evidence ≤ 1 relevant, low quality RCT or multiple relevant, low quality RCTs
<ul style="list-style-type: none"> • Manipulation versus other physiotherapeutic applications or drug therapy • TENS • Back schools • Behaviour therapy

The level of scientific evidence of therapeutic interventions for acute LBP¹⁶⁵ based on systematic review of randomized controlled studies (RCTs).

3. the background of back problems

Strong Evidence Multiple relevant, high quality RCTs
<ul style="list-style-type: none"> • Exercise therapy is effective. • Manipulation is more effective than placebo. • Intensive back school in occupational setting is more effective than no actual treatment.
Moderate evidence One relevant, high quality RCT and ≥1 relevant, low quality RCT
<ul style="list-style-type: none"> • Manipulation is more effective than usual care by general practitioner, bed rest, analgesics and massage. • Epidural steroid injections are more effective than placebo. • NSAIDs • Antidepressants are not effective.
Limited evidence One relevant, high quality RCT or multiple relevant, low quality RCTs
<ul style="list-style-type: none"> • Behavioural therapy is effective • Muscle relaxants are effective • Traction is not effective • Biofeed is not effective
No evidence ≤1 relevant, low quality RCT or multiple relevant, low quality RCTs
<ul style="list-style-type: none"> • Orthoses • TENS • Acupuncture

The level of scientific evidence of therapeutic interventions for chronic LBP¹⁶⁵ based on systematic review of randomized controlled studies (RCTs).

systematic literature reviews. In this approach, outcomes of RCTs and their methodological quality are assessed in a systematic way. The evidence is typically rated as 1) strong evidence 2) moderate evidence 3) limited evidence 4) no evidence^{2, 88, 164, 165}. Several systematic reviews have been published of RCTs on the efficacy of various therapeutic interventions available for the treatment of LBP^{8, 87, 89, 90, 162, 163}. Moreover, a recent publication¹⁶⁵ evaluated the scientific evidence for acute and chronic LBP separately. The tables regarding the level of scientific evidence on various treatment modalities have been adopted from van Tulder et al¹⁶⁵.

Based on the evidence so far, the management of acute LBP should include the following: first there should be a triage to exclude simple backache from sciatica and spinal pathology requiring orthopedic or other specific interventions. For a non-specific backache, simple analgesia is recommended and physical therapy may be applied if symptoms persist longer than a few days. Bed rest is recom-

mended only if essential and then as short as possible; instead, early activity is recommended. Psychosocial management includes enhancement of positive attitudes towards activity and return to work, and absence from work is applicable only if unavoidable^{2, 29, 165}.

For chronic LBP, there is strong evidence for efficacy regarding exercise therapy, intensive back school (including exercises) in an occupational setting, and manipulation^{2, 29, 165}. Thus, the treatment options in the acute stage target on pain control, and in the chronic phase aim at restoring function.

3. The Background of Back Problems

Back pain by definition is a symptom, not a diagnosis, and a precise diagnosis usually is missing. Specific causes of back pain are rare. Thus, perhaps 95% of patients have what is sometimes called "mechanical" low back pain, although local inflammation and muscle tension may have important

	Acute	Subacute	Recurrent	Chronic
Primary aims	1) Pain relief	1) Pain relief 2) Prevention of chronicity	1) Pain relief 2) Prevention of chronicity	1) Minimizing disability 2) Treatment of physical, psychological and social consequences of LBT
Means	Medication; Physical therapy	Medication; Active rehabilitation	Medication; Active rehabilitation	Multidisciplinary rehabilitation

The primary aims of the treatment of back disorders.

etiologic roles. A relevant question is why does a simple, acute backache in some individuals turn into recurrent/chronic disabling pain. Traditionally, doctors and scientists have focused on causes of pain, assuming a physical basis for pain that, once identified, could be eliminated or blocked. Assessments are often focused on identifying the physical basis. When no clear physical basis is found, a psychological cause is assumed, hence the term "psychogenic pain." The traditional view of persistent pain includes a simple dichotomy - the pain is physical or psychological. However, this concept of a simple dichotomy has proven to be inadequate. How do the sensory apparatus of the body and system of signal transmission and modulation relate to the experience of pain of peripheral origin? What kind of (reflex) motor responses and physical consequences does a painful peripheral stimulus initiate? Are these mechanisms different in acute and chronic pain? These are the topics that need to be covered to understand the mechanisms of pain control and consequences of pain.

Regardless of the type of injury, the injured tissue should heal within some six weeks. Thus, pain reaching beyond this time limit can be considered to have other components involved than activation of nerve endings sensitive for pain in the peripheral tissue. In this model, chronic back trouble is considered as a series of physical, psychological and social consequences due to prolongation of the original problem.

Pain control and its changes with chronic pain

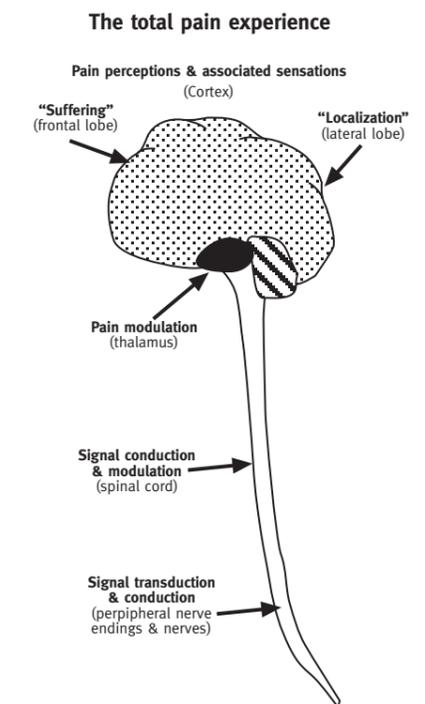
The total pain experience consists of:

1. signal transduction at the peripheral receptor site such as spinal structures
2. signal conduction along the peripheral nerve
3. pain modulation at the level of the spinal cord
4. pain perception at the supraspinal sites
5. associated sensations, emotional reactions, and affective state

Pain perception begins with activation of peripheral nociceptors and conduction through myelinated A delta and

unmyelinated C fibres to the dorsal root ganglion. Nociceptors are found in e.g. facets, annulus fibrosus, ligamentum longitudinale anterior, interspinal ligaments, and ligamentum supraspinale^{41, 73-75, 118, 136, 172}. From here, signals travel via the spinothalamic tract to the thalamus and the somatosensory cortex. Modulation of sensory input (i.e. pain) occurs at many levels. Nociceptors are also neuroeffectors, and transmission can be modulated by their cell bodies, which secrete inflammatory mediators, neuropeptides, or other pain-producing substances. Descending pathways from the hypothalamus, which has opioid-sensitive receptors and is stimulated by arousal and emotional stress, can transmit signals to the dorsal horn that modulate ascending nociceptive transmissions. Modulation to alter the perception of pain can also occur at higher centres (e.g. frontal cortex, midbrain, and medulla) by opioids, anti-inflammatory agents, as well as antagonists and agonists of neurotransmitters^{39, 112}.

Chronic pain differs from acute pain in that it serves no useful function and causes suffering, while acute pain can be considered as an useful warning signal to avoid



The total pain experience changes with pain chronicity at all levels.

harmful situations. The transmission and control of acute pain is not steady, but subject to plasticity so that the differentiation between acute and chronic pain is difficult to make. Very brief acute pain is transmitted in a simple way and rarely produces difficulties in pharmacological treatment, for example, with paracetamol or NSAIDs. The situation changes if the stimulus continues. Modulation of prolonged pain occurs in different levels. Soon after, genes are induced in central neurons and increases and decreases in diverse pharmacological systems involved in pain transmission and modulation occur over periods of only a few hours. Also, recent findings emphasize the importance of certain cortical areas (retroinsular and the anterior cingulate cortices) in the conscious appreciation of pain³⁵. Both the level of pain transmission and pain modulation will alter over time^{39, 112}.

Contribution of brain anatomy to information processing and pain

A certain area in the frontal lobe of brains, anterior cingulate cortex (ACC), appears to play a crucial role in initiation, motivation, and goal-directed human behaviors^{35, 169}. The ACC is considered as a part of a matrix of cortical areas involved in attention and ACC is activated in tasks that are attention-demanding^{33, 35}. Besides attention, ACC may also have contribution to learning and memory networks³⁵ and error processing⁶⁵.

Brain research with modern imaging techniques such as positron emission tomography (PET) has revealed interesting avenues to understand features related to pain perception. PET studies report metabolic activation of ACC by painful stimuli, suggesting that ACC receives direct nociceptive inputs^{31, 77, 169}. It appears unlikely that the ACC has anything to do with localization of pain^{6, 169}, but PET studies confirm activity in the ACC during the emotional, suffering component of pain^{31, 33, 130, 169}. Davis et al³³ found that the ACC was not activated during pain rated as mildly painful, but was activated during pain rated as moderate or intense. This result suggests that ACC is involved

particularly in severe pain.

ACC seems to be involved in chronic pain syndromes. It has been recently shown that patients with chronic inflammatory pain develop adaptive cortical responses to noxious stimulation characterized by reduced anterior cingulate response⁷⁶. Even an illusion of pain may produce activation in the anterior cingulate cortex, without noxious stimulus³¹. Thus, chronic pain patients may experience pain without peripheral noxious stimulation. This mechanism may partly explain chronic pain without tissue origin. On the other hand, both the attention-demanding premotor response selection and the emotional component of severe pain are processed in the same highly specialized area of the cortex, ACC. Although the exact areas for these functions are probably not overlapping on an individual level^{33, 34}, they most probably affect the functioning of one another.

Pain and deconditioning syndrome

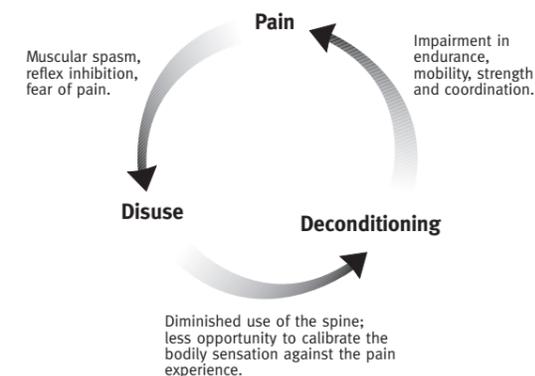
It is widely believed that many chronic low back and neck pain patients suffer from a physical "deconditioning syndrome". According to this model, some patients develop pain and illness (avoidance) behaviors in the early phase of pain^{86, 96, 116}. The pain avoider is fearful of the pain and its consequences. Behavioral avoidance may cause different physical and behavioral problems. A decrease in daily physical activity can result in reduced mobility and loss of muscle strength, endurance and co-ordination because of e.g. paraspinal muscle wasting^{30, 47, 55, 66, 94}. Paraspinal muscles in CLBP patients are smaller, contain more fat and present selective muscle fibre atrophy^{30, 47, 55, 66, 94}. Deconditioning will eventually lead to more pain and will reinforce the avoidance cycle. Patients land in a vicious circle with an ever decreasing physical condition of the back muscles and other structures. This model strongly suggests an interrelationship between impairment in physical condition and illness behavior in chronic pain. Different physical outcomes may result from deconditioning. Reduced peak strength^{1, 13, 66, 116, 119, 124, 157} and endurance capacity^{13, 66, 149} of paraspinal muscles have been related to chronic LBT in

many studies.

Several researchers have assumed that strong back is a synonym for healthy back. This is partly based on the finding that, on the average, back pain patients are weaker than healthy controls. However, a few prospective studies have tackled the question whether back strength does protect from injury and pain, or whether the lack of strength is just one consequence in deconditioning. Based on the existing evidence, trunk or spinal strength is not a protection from back injury or pain^{93, 121, 131}. Thus, the strengthening approach in rehabilitation may be questioned. Moreover, a good outcome in rehabilitation is not related to strength per se¹⁵³, but to functional improvement¹⁵³, reduction of symptoms and overall satisfaction^{49, 62} and work satisfaction⁶⁷.

Avoidance behavior

There can be little doubt that psychological factors such as fear of pain are involved in the course of chronic LBT. Scores obtained from psychological questionnaires correlate highly with work absence, claims for financial compensation, and response to treatment. They can predict which subjects with acute back pain will progress to chronic pain and disability^{23, 50, 104, 160}. Related factors such as job satisfaction also influence reports of back pain and work absence^{14, 67, 150}.



The chronic pain/deconditioning cycle. Some back pain patients land in a never-ending vicious circle.

Associations between psychometric scores and various aspects of behavior related to chronic back pain do not prove, however, that "abnormal" psychosocial characteristics precede or cause back problems⁵⁹. On the contrary, it could be argued that depression, antisocial attitudes and litigation merely reflect normal human reactions to vague diagnosis, ineffective treatment, and inconsiderate employers. Those questions in the widely used depression questionnaires, for example, that are most closely associated with severe back pain refer to symptoms such as sleep disturbance, which appear to be more of a consequence than a cause of pain.

The fundamental problem of cause and effect has been tackled in prospective studies that link psychometric scores with future back pain. However, an interpretation of the findings is not straightforward because abnormal scores recorded at the outcome may depend themselves on previous experiences of pain and because abnormal scores may either predict or follow chronic pain. Moreover, a statistically significant association does still not indicate clinical significance, i.e. the magnitude of the association has been omitted in most of the studies.

A recent study of Mannion et al¹¹⁰ addressed the issue of quantity of the association between psychometric scores and development of LBP. The study included 403 volunteers with no history of "serious" low back pain, which was defined as pain requiring medical attention or absence from work. At the time of initial assessment and at six-month intervals thereafter, the volunteers completed the following questionnaires: the Health Locus of Control, which was subdivided into three sections labeled "Internal," "Powerful others," and "Chance"; the Modified Somatic Perception Questionnaire (MSPQ), and the Zung depression scale. Scores from the MSPQ and from the Zung scale were added to form a combined measure of psychological distress. Additional questionnaires inquired about any back pain experienced in the previous six-months. At 18-month follow-up 162 participants had reported "any" low back pain, of which 79 were "serious." None of the psychometric

scores were affected by "any" low back pain. The MSPQ scores only changed after "serious" back pain was reported. In a multivariate analysis, the most significant predictor of first time "serious" or "any" back pain was a history of non-"serious" back pain. Of the psychological factors, the sum of MSPQ and Zung questionnaire scores was the best predictor of "serious" back pain, and the MSPQ score was the best predictor of "any" back pain. However, after accounting for the effects of a history of non-"serious" back pain, psychometric scores predicted less than an additional 4% of reported back pain. Thus, although a statistically significant association was found, the association was very weak. It can be with good reason questioned whether the development of severe LBP is due to "avoidance behavior".

Two other recent studies agree with the findings of Mannion et al that the role of avoidance behavior and distress in the development of LBP is minor. Psychological distress, depression, self-efficacy beliefs, subjective work prognosis, disability, and work characteristics were assessed at baseline in a prospective, two-year follow-up study of a working population⁴³. The best predictor of future pain was disability. The psychometric measures did not predict changes in pain at all⁴³. In a large prospective cohort study in the general population by Croft et al³², symptoms of psychological distress in individuals without back pain predicted new episodes of LBP. However, the proportion of new episodes of low back pain that might be attributable to such psychological factors in the general population was 16% only.

Thus, a minor part of the onset of severe LBP can be explained with avoidance behavior or distress only. Other explanations for the prolongation and recurrence of the disorder need to be found. Failures of reflexive trunk movement control have raised interest among back researchers recently. It may rather be that avoidance behavior, distress and depression are consequences of long-lasting pain and disability, and that the process of pain chronicity is initiated by other factors, such as failures of reflexive movement

control⁶¹.

Failures of trunk movement control

Abnormal loading to the spine can take many forms and may occur both at workplace and during leisure-time. Protection against excess loading and injury requires anticipation of events and reasonable muscular responses. Both the perception of trunk position and motion, and appropriate responses are essential for the correct placement of the trunk. In addition to their mechanically restraining function, ligaments and muscles provide neurologic feedback that directly mediates joint (or vertebral segment) position sensibility and muscular reflex stabilization about the respective area¹³⁸. Other sense organs producing information for co-ordination and postural control are vision, vestibular end organs and receptors of the skin that are sensitive to pressure^{3, 138, 143}. In case an event of abnormal loading occurs faster or at a higher loading level than the control system can respond, one is at risk of mechanical damage or injury.

The human motor (movement) control is based on interactions of several cortical, subcortical and somatosensory levels. Using a simplified schematic model, these can be presented as follows. The lowest level is the most automatic and consists of spinal neurons and motor units. The highest level consists of a number of adjacent cerebral areas that stand for abstract global planning. The intermediate level consists of primary motor cortex and the pyramidal, extrapyramidal and cerebellar connections, which bridge the gap between the lowest and the highest levels. Motor behavior varies from reflexive responses at the lowest level to voluntary movements at the highest level. The voluntary movements include many non-stereotypical motor acts that are typically guided by sensory information from the external world, and by motor strategies based on previous experience and training^{80, 138, 143}.

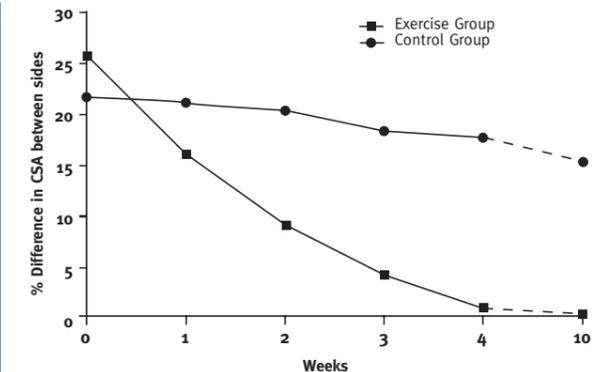
Several types of failures in movement control of the spine have been found in back pain patients. Protective

guarding refers to a situation where the patient favors the injured area, i.e. both reflexive and conscious mechanisms are utilized not to move the painful site. It may be questioned whether the patients actively choose to disuse the spine as assumed in the concept of deconditioning due to avoidance behavior, or whether the protective disuse is rather on a reflexive basis.

Protective guarding and reflex inhibition

Hides et al⁶⁰ conducted a clinical study on 39 patients with acute, first-episode, unilateral low-back pain and unilateral, segmental inhibition of the multifidus muscle. Patients were allocated randomly to a control or treatment group. The controls received medical treatment only, while the treatment group received also specific, localized, exercise therapy. Outcome measures included four weekly assessments of pain, disability, range of motion, and size of the multifidus cross-sectional area and the patients were reassessed at a 10-week follow-up examination. Multifidus muscle recovery was not spontaneous on remission of painful symptoms in controls. Muscle recovery was more rapid and more complete among patients who received exercise therapy. Other outcome measurements were similar for the two groups at the 4-week examination. Although they resumed normal levels of activity, controls still had decreased multifidus muscle size at the 10-week follow-up examination. Thus, multifidus muscle recovery is not spontaneous on remission of painful symptoms. Keeping in mind that previous experience of back pain is the strongest predictor for a new one, lack of localized, muscle support may be one reason for the high recurrence rate of low back pain following the initial episode⁶⁰. The previous findings of the same study group that multifidus wasting is unilateral and isolated to one level suggest that the mechanism of atrophy is not generalized disuse atrophy or spinal reflex inhibition⁶¹. Inhibition due to perceived pain, via a long loop reflex, which targets the vertebral level of pathology to protect the damaged tissues, is the likely mechanism of wasting⁶¹.

Indahl et al^{70, 71} have conducted a series of studies

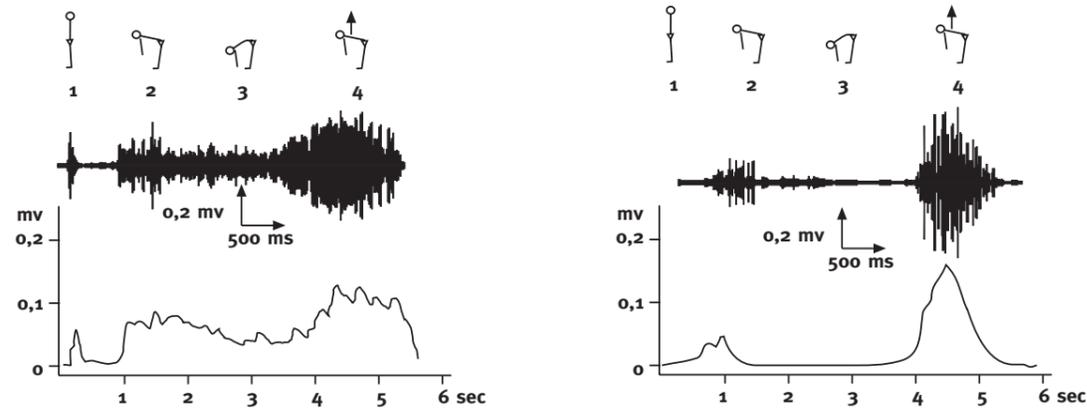


Acute LBP patients were randomized into two separate groups. The other received segment specific exercises and the other received no therapy. The exercise group had their side asymmetry removed in the cross-sectional area (CSA) of Multifidus muscle, while the CSA side difference stayed in the LBP patients in control group up to 10 weeks in spite of the pain relief earlier⁶⁰.

evaluating the control mechanisms between discs, facets and paraspinal muscles. In the studies, porcine intervertebral discs were stimulated with electricity, which produced maximal paraspinal muscle activity on the same segment level. Injection of anesthetic⁷⁰ or isotonic saline⁷¹ under the facet capsule, however, diminished the multifidus activity. Thus, there is an interaction between discs, facets and paraspinal muscles in such a way that paraspinal muscles protect the motion segment from excess motion in a painful situation. However, facet capsule stretch is capable of reducing the (abnormally) high paraspinal muscle activity. Perhaps the efficacy of manipulation and specific exercises is based just on this explanation.

Missing flexion relaxation

Reduction in lumbar muscular activity at full body flexion, known as flexion relaxation, has been noticed in relation to overall trunk, lumbar spine and hip flexion in healthy subjects⁴⁶. The hip extensors (i.e., hamstring muscles) also relax during forward flexion but with different timing¹⁴⁵. Flexion relaxation is often missing in LBP patients^{146, 147}, which has recently been confirmed with motion on the segmental level⁷⁹. Flexion relaxation occurs only in subjects in whom intervertebral rotation has reached a stage of completion considerably before full trunk flexion is achieved⁷⁹. Most

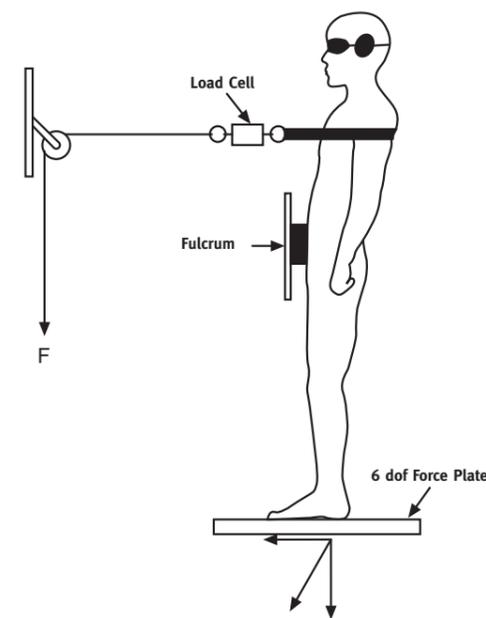


Flexion relaxation is often missing in LBP patients (the left hand) compared to healthy individuals (the right hand picture). Most likely, persistent muscle activation (protective guarding), that restricts intervertebral motion, is a means by which the neuromuscular system provides stability to help protect injured passive spinal structures from movements that may cause pain¹⁴⁷.

likely, persistent muscle activation (protective guarding), that restricts intervertebral motion, is a means by which the neuromuscular system provides stability to help protect injured passive spinal structures from movements that may cause pain.

Delayed responses to sudden loading

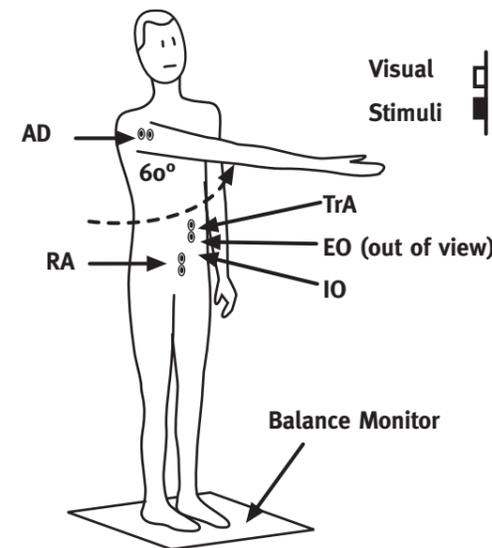
Unexpected loads, which people often experience both at workplace and in leisure activities, can lead to high forces in the spine and may be a cause of low back injury. In a series of studies^{103, 167}, subjects were exposed to fatiguing and restorative interventions to assess their response to sudden loads. Studies comparing patients with LBP and healthy control subjects indicate that back patients have a delayed response and a lower EMG amplitude, which imply an unwillingness or inability to use their muscles as effectively. They also have greater ground reaction amplitudes in the same task indicating higher mechanical loading to the body since the back muscles do not absorb the unexpected loading, as they should. The higher EMG baseline that was found in the patients indicates that they have a continuous tension of their muscles to protect their back from sudden or uncontrolled events. This increased "base-tension," however, most likely fatigues the muscles, thereby preventing a rapid response to unexpected load^{103, 167}.



The setting to study responses of trunk muscles in sudden loading; the EMG electrodes are attached to the low back muscles¹⁶⁷.

Ineffective anticipatory trunk stabilization

Hodges and Richardson have performed a series of studies assessing the trunk stabilization mechanisms among patients and controls. The subjects performed rapid shoulder flexion, abduction, and extension in response to a visual stimulus⁶³. Electromyographic activity of the abdominal muscles, lumbar multifidus, and the deltoid was evaluated using fine-wire and surface electrodes. Movement in each direction



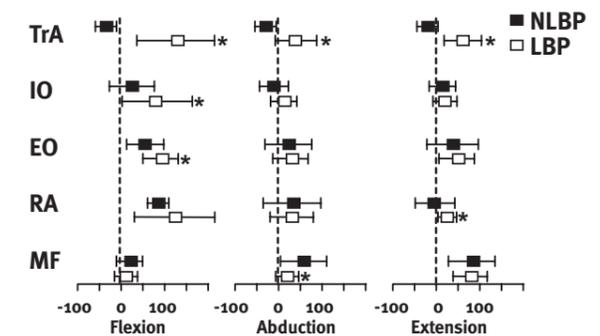
The settings for studying trunk stabilization as the function of upper arm movement. In healthy subjects the first muscle to react before arm movement is the transversus abdominis to stabilize the trunk. This activation is delayed in CLBP patients⁶³.

resulted in contraction of trunk muscles before or shortly after the deltoid in healthy controls. The transversus abdominis was invariably the first muscle active and was not influenced by movement direction, indicating a role of this muscle in spinal stiffness generation. Contraction of transversus abdominis muscle was significantly delayed among patients with low back pain with all movements. Isolated differences were noted in the other muscles⁶³. Hodges and Richardson have studied the effect of lower limb movement to the trunk responses in a similar setting also⁶⁴. The transversus abdominis muscle responses were delayed in LBP patients in lower limb movements also⁶⁴.

The delayed onset of contraction of transversus abdominis in limb movements indicates a deficit of motor control and apparently results in inefficient muscular stabilization of the spine. All this results as increased physical loading to the spine and vulnerability in physical loading.

Abnormal postural control

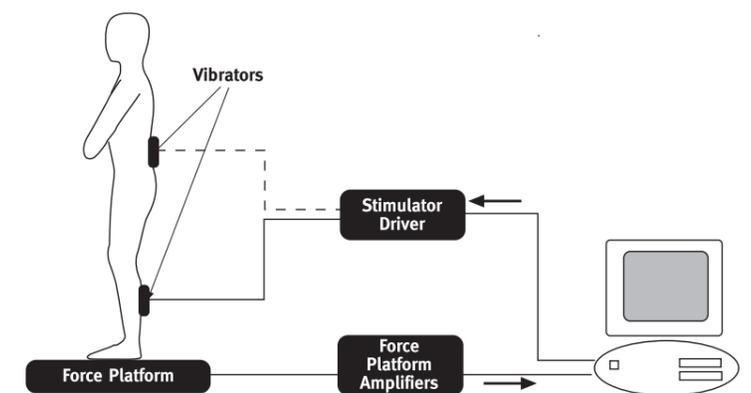
Luoto et al¹⁰¹ studied two-footed postural control with a vertical force platform among healthy control volunteers and patients with chronic LBP. Women with severe low back pain had poorer postural control



than women with moderate low back pain and women in the control group. In one-footed stance, there was a significant difference between patients and controls in both males and females⁹⁹. Byl & Sinnot have published similar findings earlier²⁴. The results indicate that patients with chronic low back pain have impaired postural control.

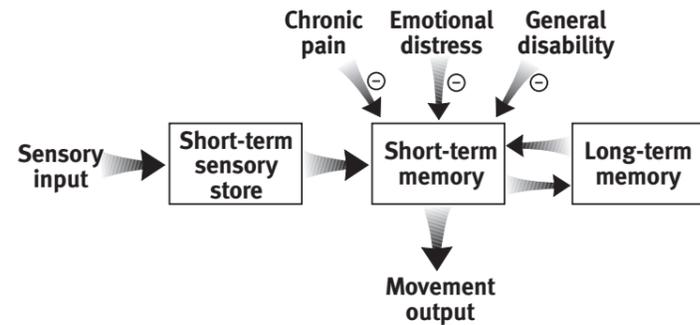
Delayed reaction times

Luoto et al¹⁰¹ studied psychomotor speed (reaction time) among healthy control volunteers and patients with chronic LBP at the beginning of an active, functional, restoration back rehabilitation program and six months after the program. Reaction times for upper and lower limbs were tested



The settings for studying postural control in CLBP patients¹⁰¹.

4. the aim in active rehabilitation



Chronic pain, emotional distress and disability are factors that hamper the function of short-term memory. This can be measured as prolonged reaction times.

with a system based on a microcomputer. A consistent trend was found in which patients with low back pain had reaction times slower than volunteer controls. Men with severe low back pain had significantly longer hand reaction times than men in the control group ($P = 0.03$). Functional restoration had an effect on reaction times. The restoration was considered successful if the condition of a patient with a disability that had resulted from low back pain improved during the follow-up examination and unsuccessful if the disability worsened. Patients who experienced these results were identified into groups called "good" and "poor," respectively. Among men, the reaction times improved in the control group and "good" groups, but they became slower in the "poor" group. The difference between "good" and "poor" groups was significant ($P = 0.008$). Women in the "good" group achieved the most improved reaction times, and the difference between these women and the control women almost reached significance ($P = 0.076$). These results indicate that patients with chronic low back pain have impaired psychomotor speed in concordance with earlier results¹⁵⁶, and that psychomotor speed can improve during an active functional restoration back rehabilitation program.

The relationship between LBT and slow reaction times is interesting, since it suggests that patients with LBP experience central impairment besides the known peripheral problems. The mechanism explaining the association between LBP and information processing seems to be impairment in the function of short-term memory due to pain and

impairment¹⁰², i.e., the "workspace" of the central nervous system¹⁴³.

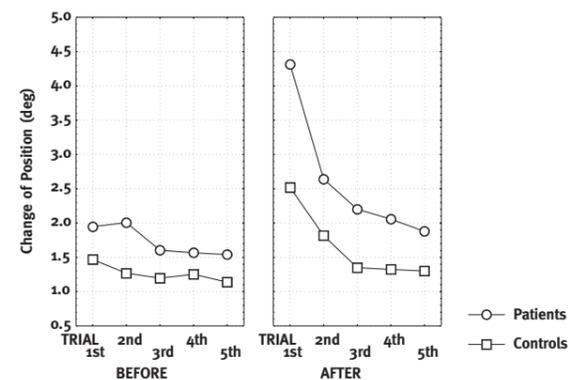
Abnormal Lumbar Fatigue

Muscle fatigue is defined as inability to maintain required muscular contraction against external resistance or repeated task. Lumbar paraspinal muscle fatigue results as abnormal trunk movements and loss of precise movement control^{103, 129, 167}. This may lead to micro injury of spinal structures, and cause LBP¹⁷³. Excessive

fatigability of back extensor muscles is common among chronic LBT patients^{12, 66, 109, 139, 144, 149}. Excess lumbar fatigability may also be a risk factor for future low back pain^{13, 100, 109}.

It has been shown that isoinertial fatiguing loading of the paraspinal muscles in sagittal plane changes the triaxial coupled lumbar movement patterns among healthy male subjects¹²⁹. The subjects became slower in their sagittal plane movements, while both the movement velocity and amplitude increased in the coronal plane, and the movement amplitude increased in transverse plane due to the lumbar fatigue¹²⁹.

Impairment of the ability to sense lumbar position and its changes may partly explain the abnormalities of the



CLBP patients and healthy controls had their lower body rotated 1 degree/second to either direction. Test subjects pressed a button when they sensed that their body was rotating. The ability to sense a change in lumbar position was hampered by fatiguing the muscles before the test¹⁵⁴.

coupling of lumbar movements during fatigue. The ability to sense a change in lumbar position was assessed in 57 chronic LBT patients and 49 healthy-back controls before and after a maximal endurance task¹⁵⁴. Lumbar fatigue induced significant impairment in the ability to sense a change in lumbar position, but the LBT patients had significantly poorer values than controls already when they were not fatigued¹⁵⁴. Thus, there is a period immediately after the fatiguing task during which the information of lumbar position and its changes available from the lower back is inaccurate. It is probable that the risk of spine injury is increased in such a situation of abnormal loading.

Abnormal gluteal fatigability

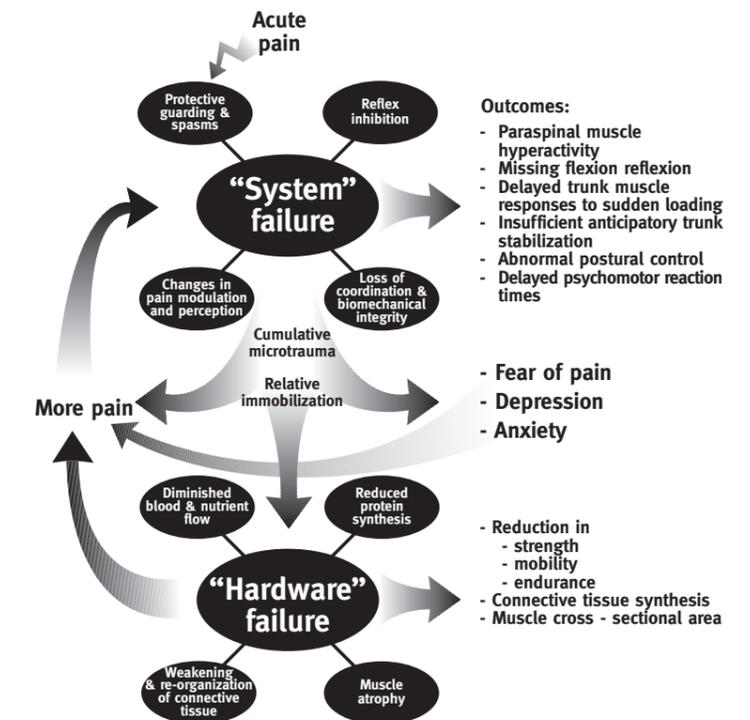
Gluteal activation and pelvic stability often are decreased in chronic low-back pain sufferers^{22, 81, 148, 168}. Kankaanpää et al⁸¹ studied LBP patients and controls in a seated static endurance task. The chronic LBP patients were weaker and fatigued faster than the healthy controls. The EMG fatigue analysis results revealed that the gluteus maximus muscles were more fatigable in chronic patients than

in healthy control subjects, but no difference was found in the fatigue of lumbar paraspinal muscles between the groups in the seated static endurance task⁸¹. It is likely that in the seated task with relatively high loading level in forward bending, gluteal and hamstring muscles are the primary actors and the paraspinal muscles are working in a supporting role. Thus the fatigue is concentrating mostly in the gluteal area.

Summary: A model

The following model attempts to summarize the recent findings on the functional ("system") and structural ("hardware") abnormalities in low back trouble. During

recurrent and chronic back pain, the relative importance of physical consequences of the back trouble increase in importance, while in the early phases of LBP functional disturbance is dominant.

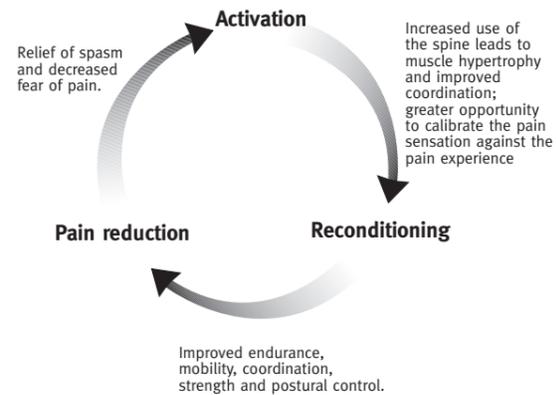


The relative importance of the control system failure is large at the beginning of the process of LBP. With prolongation of LBP and psychological disturbance, the relative importance of tissue deconditioning (hardware failure) will increase.

4. The Aim in Active Rehabilitation

The aim in active rehabilitation utilizing exercises is to restore lumbar function and movement control to minimize the above-mentioned features of disability. In this context, it does not matter whether the loss of function has been due to reflexive abnormality or avoidance behavior and deconditioning. Another aim is to influence the behavioral pattern of the LBP patient in a way that he or she would be willing and capable of taking care of himself or herself on an individual basis after the treatment. Thirdly, pain is shown to decrease in active rehabilitation aiming at restoring full function.

5. exercise physiology



Consequences of active rehabilitation.

5. Exercise Physiology

Exercise is a form of leisure-time physical activity that is performed on a repeated basis over time (exercise training) with a special objective such as the improvement of physical performance or health²⁰. When prescribed by a physician, the regimen should cover the recommended mode, intensity, frequency, duration and progression of such activity^{7, 20, 51}. The mode of exercise covers the type of activity and the temporal pattern of it that is recommended, with a detailed specification of the duration of each exercise and rest period in the case of intermittent exercise bouts. The intensity can be expressed in either absolute or relative terms. The frequency of exercise is normally reported as the number of sessions undertaken in a typical week. The duration of an individual exercise session is usually reported in minutes or hours^{7, 20, 51}.

Dosage and Target

Some basic principles are important when considering the most effective modality and dosage of exercises in chronic

	Aerobic exercises	Strengthening exercises	Trunk muscle training	Isolated, controlled back-specific exercises
Method	Walking, jogging, aerobics etc.	Resistance training w/wo external loads	Resistance training w/wo external loads	Back specific loading, slow progression
Outcome	General fitness; VO ₂ max	General fitness	Trunk strength	Trunk co-ordination and control

The different aims / targets on various types of exercises

or recurrent LBT. One of them is the task-specificity of training. This means that training is specific for its target tissues in a dose-related manner. Training effect can be obtained in the trained tissues only. With increased amount of training there is an increased training effect unless overloading leads to a repetitive strain (stress) injury. Also, training effects are specific to the type of training. Peak strength training does not produce major endurance improvements and vice versa. Also, other techniques than strength and endurance training are needed to train coordination and skills. Thus the mode of exercise used and the theoretically possible outcomes need to be kept in mind when assessing the efficacy of exercise regimen on the back and neck pain outcome. It is, for example, unrealistic to expect aerobics-type exercises to have a major effect on back muscle hypertrophy, peak strength, co-ordination and endurance since that type of exercise stresses primarily the cardiovascular system rather than trunk muscles^{84, 92, 143}.

Some studies have been done on training and detraining specificity and frequency for lumbar strengthening. A training frequency as low as once a week provided an effective training stimulus for the development of lumbar extension strength with back-specific training devices⁵³. However, it is obvious that higher training frequency provides benefits at least regarding endurance and co-ordination. Isometric lumbar extension strength can be maintained after a training program with a reduced frequency of training; even training once every four weeks was capable of maintaining extension strength in one study¹⁵⁹. The key issue is, however, that in case of detraining after functional restoration, it is likely that the results obtained regarding for example peak strength will be lost within months. Thus, long-term changes in exercise habits and lifestyle are required

to maintain optimal physical functioning.

Efficacy of exercises in LBP

Back and neck pain definitions vary in different countries and areas. The following definitions are, however, the most widely used ones.

Acute back pain	0-7 weeks' duration (upper limit 4-7 weeks)
Recurrent (acute on chronic) LBP	recurrence of new or exacerbation of pre-existing pain
Subacute LBP	7 weeks to 3 months' duration (6 weeks to 6 months)
Chronic LBP	Duration > 3 months (> 6 months)

Some definitions of back pain.

Acute LBP

There is strong evidence that exercises are not more effective than ordinary activities in the treatment of acute LBP^{44, 45, 105}. However, bed rest should be as short as possible and early return to normal activities is recommended^{38, 105}. In case the symptoms persist, low-stress aerobic exercise can prevent debilitation due to inactivity, and endurance exercise programs can be started during the first two weeks. Gradually increased conditioning exercises for trunk muscles are also helpful if the symptoms persist².

Subacute LBP

There is evidence that in this patient group a progressive activity program, with a behavioral therapy approach, is superior to traditional LBP care such as conservative physical therapy^{97, 98}. It is especially important to prevent pain chronicity in this stage of the problem.

Chronic LBP

In this patient group, significant improvements in both experienced (subjective) and objective function has been recorded consistently with exercises in subchronic and chronic LBP¹⁶⁵. Exercises increase muscular strength and endurance along with the reduction in experienced pain and disability^{107, 116, 135, 140}. Also, psychomotor control

improves with a functional restoration approach rehabilitation¹⁰¹. Regarding degeneration, muscular hypertrophy with exercises^{128, 135} has been documented. Good results concerning return-to-work with treatment modalities including exercises have been obtained in some studies also^{10, 114}.

Chronic Disabling LBP

If chronic LBP leads to early retirement or recurrent prolonged absenteeism from work, it can be defined as chronic disabling LBP. The results concerning return-to-work rates have been partly controversial in this patient group between United States¹¹⁴ and Scandinavia^{4, 78, 120}. Return-to-work in chronic LBP appears to be a tremendous task in countries that have a highly developed social security system and high unemployment rates. It seems that early rehabilitation activities - preferable in the recurrent or subacute phase of LBT - are needed to prevent the recurrent or subacute LBP turning into permanently disabling, chronic LBT.

Recurrent LBP

Many studies indicate that the natural course of low back trouble in reality is fluctuating, i.e. varies over time^{37, 48}. Each recurrence increases the likelihood of a new one, previous LBP being a strong predictor of the next LBP^{18, 58, 170}. Roughly half of the population in industrialized countries suffer from LBP at least once a year. Many of them would benefit from early intervention to prevent pain chronicity. The latest time point for intervention should be recurrence of work absenteeism due to LBP.

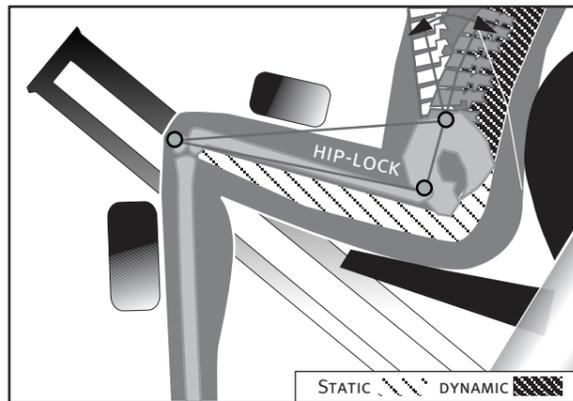
Postoperative / Post-traumatic LBP

Postoperative or post-traumatic immobilization may lead to deconditioning per se in addition to the initial injury⁸⁴. This should be prevented with the shortest possible immobilization and early rehabilitation. Preliminary results show that DBC treatment is applicable after both disc or fusion operations producing pain reduction and increases in both strength and mobility¹⁷.

6. DBC Active Spine Care

Back-specificity of exercises

One issue to be considered in assessing the possible efficacy of exercises for the treatment of LBT is whether the training effects are back specific. This is achieved with pelvic stabilization techniques using devices employing a “hip lock mechanism”. A large variation in strength production is a result of lumbar posture and the involvement of pelvic sagittal rotation^{26, 28, 46, 52}. Pelvic stabilization excluding strong gluteus and hamstring muscles is required to specifically test and train the lumbar extensor function^{52, 54}.

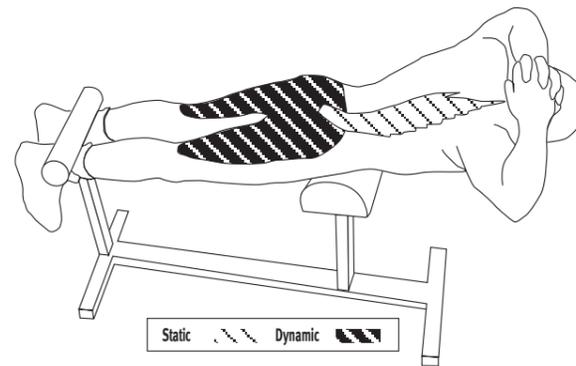


A hip lock mechanism. Cushions support the thighs and pelvis in such a way that pelvic rotation is prevented and the dynamic movement is targeted to the lumbar spine.

Trunk extension involves strong gluteal and hamstring muscles and especially in erected position back extensors are only little involved, mostly in a static way^{28, 46, 126}. In isolated spinal extension, the aim is to exclude the function of the gluteus and hamstring muscles with a specific “hip lock”. The lock system aims at preventing pelvic sagittal rotation and, subsequently, the dynamic movement of the muscles involved. Specific devices are required for this function. The difference is emphasized in Graves et al.⁵⁴. Recent electromyographic findings confirm, for example, that static loading of upper body extension primarily targets the gluteal and hamstring muscles, rather than back extensor muscles⁸¹.

The key difference between back-specific and non-specific exercises is that the loading and, subsequently, the effect can be targeted in an isolated and safe way to the

lumbar spine in the former.



Trunk extension is often considered as a function of back extensors. However, in this task back extensors work mostly in a static way and the loading is mainly targeted to the gluteal and the hamstring muscles.

6. DBC Active Spine Care

Basic principles

Recent consensus statements in different countries^{2, 29} as well as systematic literature reviews of randomized controlled trials¹⁶⁵ indicate strong evidence that exercise is effective in the treatment of prolonged and chronic LBP. More specifically, both internal analyses by DBC¹⁵³ and independent efficacy studies indicate that the DBC protocol is effective in the treatment of these disorders⁸³. These two facts provide the basis for the documentation-based care at DBC.

The British expert panel review²⁹ requests that “There should be a fundamental change in management strategy directed towards early active rehabilitation and return to work. It should be based on physical, psychological and social needs of the individual patient.” The DBC approach has been designed to fulfill these demands.

Selection criteria for DBC Active Spine Care

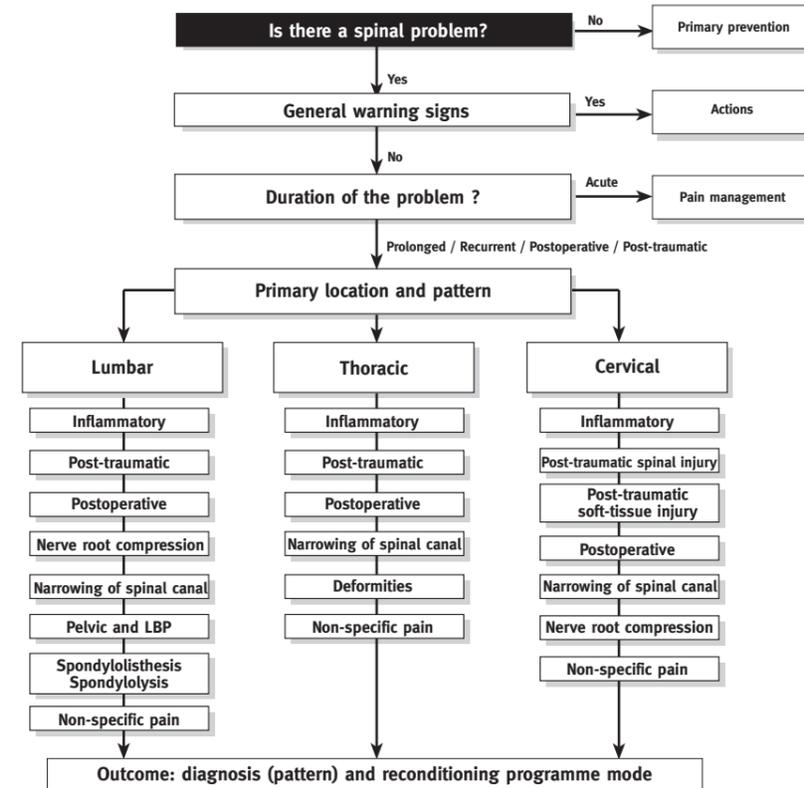
If there are signs or symptoms indicating a severe disease such as malignancy, infection, potential neurologic catastrophe or systemic disease, these patients deserve immediate treatment given by a corresponding special unit. Certain specific spine problems such as disc herniations, spinal

stenosis and spondylolisthesis may benefit from surgery. The majority of patients, however, has non-specific mechanical back or neck pain that will benefit from treatment modalities such as training programs.

Evaluation protocols and follow-up of the treatment

The evaluation protocols consist of subjective questionnaire techniques on pain and impairment, and objective measurements of lumbar function.

The participants answer a structured questionnaire and their trunk endurance and mobility are measured at the beginning and at the end of the treatment period. Progress can also be monitored during the treatment. Specially trained physiotherapists are responsible for the interviews, measurements and treatments.



Selection criteria for DBC.

- | |
|--|
| <p>Neural Tissue involvement</p> <ul style="list-style-type: none"> • Current nerve root entrapment with intolerable pain • Cauda Equina • Spinal cord compression • Tumors • Other corresponding disorders <p>Disorders of the spine</p> <ul style="list-style-type: none"> • Severe instability indicative for stabilization surgery • Severe osteoporosis • Recent fracture • Other corresponding disorders preventing active rehabilitation <p>Other (systemic) diseases</p> <ul style="list-style-type: none"> • Severe cardiovascular diseases • Severe metabolic diseases • Other corresponding disorders preventing active rehabilitation <p>Recent major operation</p> <p>Acute infection</p> <p>Lack of co-operation</p> <ul style="list-style-type: none"> • Severe psychological disturbance/ Psychiatric disease |
|--|

Some warning signs (exclusion criteria from DBC)

Questionnaires

The questionnaires include, in addition to sociodemographic variables, the duration in years of the low back pain, its regularity (no pain, intermittent, regular, continuing pain) and intensity (visual analogue scales).

Self-experienced impairment and disability, depression⁸⁵, fear avoidance^{137, 160} and recovery beliefs^{68, 158, 161} are also sought.

Mobility

Mobility is measured with the treatment devices. The movement amplitude and fixation mechanisms of the devices are adjusted to focus the movement on the specific part of spine. The results are given as deviations from the neutral position (degrees).

Endurance

In the dynamic endurance test the subjects are seated on a special testing unit (DBC LTE) where a hip lock mechanism targets the loading on the lumbar paraspinal muscles. Subjects

perform dynamic upper trunk extensions (30 repetitions/min. with a movement range 25 degrees flexion to 5 degrees extension) up to 90 sec against the movement bar with a load that is calculated on the basis of upper body weight. Continuous surface EMG recordings are made bilaterally over the paraspinal muscles at L5-S1 spinal level during the endurance test and the spectral zero crossing rate compression (% change/min) is calculated of the raw EMG signal as an objective index of muscle fatigue.

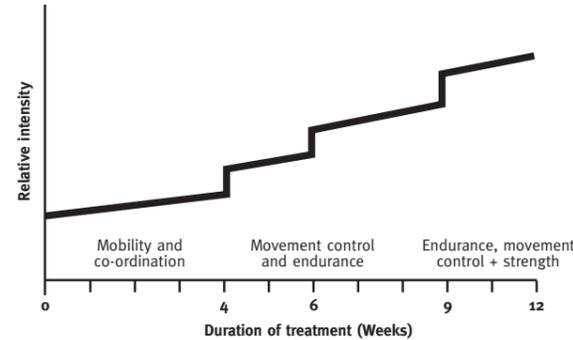
Relative changes (percentage) during active rehabilitation for measured values are calculated [(value post - value pre)/ value pre].

Active treatment

The duration of the active rehabilitation program is defined on the basis of the severity of pain and deconditioning. The program is at minimum 6 weeks with 12 treatment visits but the more common approach is to provide a treatment program of 12 weeks and 24 visits.

The treatment includes co-ordination, mobility and muscle endurance exercises with specific equipment. In addition, stretching and relaxation exercises, and functional muscle and co-ordination exercises (sit-ups, etc.) are included. A specially trained therapist guides the active treatment program.

The treatment is primarily based on equipment-exercises; correct loading and range limiters ensure that exercises are performed in a painless range of motion and

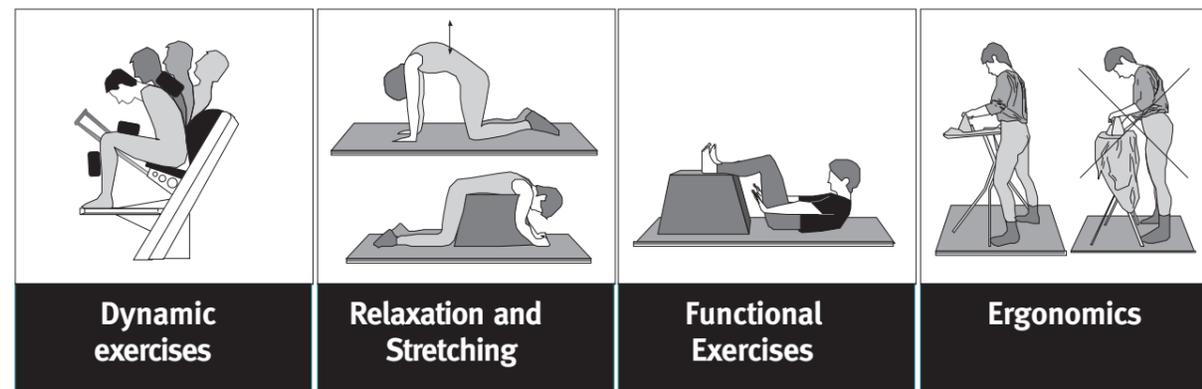


The relative progression during 12-week DBC functional restoration program.

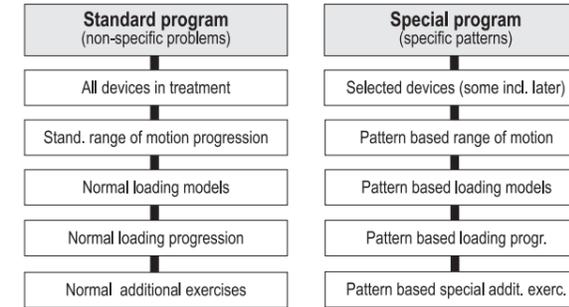
that they find their right target in the lumbar spine. Treatment includes controlled movements in lumbar/thoracic flexion, extension, rotation and lateral flexion.

Treatment is planned on the basis of initial endurance and mobility measurements and interviews, and records are kept of the progress. The treatment begins on low loads for the first weeks with the object of improving mobility and especially teaching proper co-ordination and control of the spine. The load is gradually increased so that only at the sixth to eighth week subjectively strenuous applied for the first time, but within the pain tolerance of the individual patient. The load is further increased in a gradual and controlled manner until, at the end of the program, the patients are instructed to continue an individual secondary prevention program once or twice a week with or without guidance depending on their individual needs.

The inclusions of exercises, rate of progression in



The DBC Active Spine Care program



The DBC Active Spine Care program is individual based on the diagnosis and severity of the back problem.

loading and ranges of motion are individual based on the type (diagnosis) and severity of the back problem.

The DBC devices

The measurement and treatment devices have been specifically designed for both testing and treatment according to the latest know-how in biomechanics and functional disorders of the lumbar and cervical spine.

Lumbar Thoracic Extension

Lumbar Thoracic Rotation

Lumbar Thoracic Flexion

Lumbar Thoracic Lateral Flexion

Cervicothoracic Elliptic Extension

Cervical 3D Rotation

Shoulder Blade Adduction

Multipurpose Low-Friction Unit

Horizontal Leg Press

Abdominal Crunch

DBC software is a Microsoft Windows®-based computer program for documentation and management of patient information. It features functions to design treatment programs, evaluate test results, print reports and manage the data on a patient, group or clinic level.

The therapist's role

Hands on

The skill of the therapists is to target the loading accurately

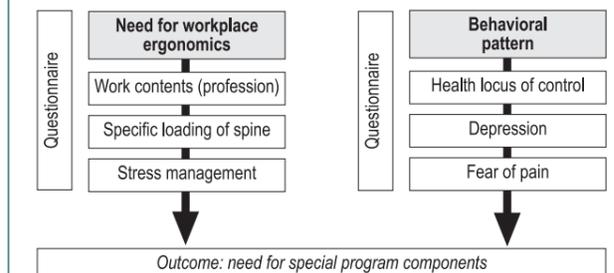
in the right place(s) especially at the early phase of the active treatment as it plays a crucial role in the success of the treatment program. The aim is to achieve segmental motion of the lumbar or cervical spine in a controlled manner. Very few individuals are able to produce the motion without the hip locking system or the three-dimensional guidance in the devices and external guidance from the therapist. Later on after the correct movements have been learned, the role of the therapist concerning the active treatment is primarily in guiding the progress in loading and movement ranges.

Cognitive and behavioral support

An elementary part of the treatment program is behavioral and cognitive support and motivation given by the therapists. This is given using discussions concerning the "benign nature and good prognosis" of low back pain during treatment sessions. In addition, the evaluation results, especially concerning the objective measurements and their changes, are used as a tool to convince the patient about progress. All this results in diminished fear of pain and increased self-efficacy beliefs. Also, individualized ergonomics guidance and psychological support can be included in the program according to the needs.

Additional treatments

Additional treatments such as psychological counseling and workplace interventions can be added as external treatments to the active rehabilitation of the back. Their needs are evaluated with the DBC questionnaire scores concerning avoidance behavior, depression and recovery beliefs.



Depending on the need, additional program components can be added for psychological and work-related subjects.

7. DBC results

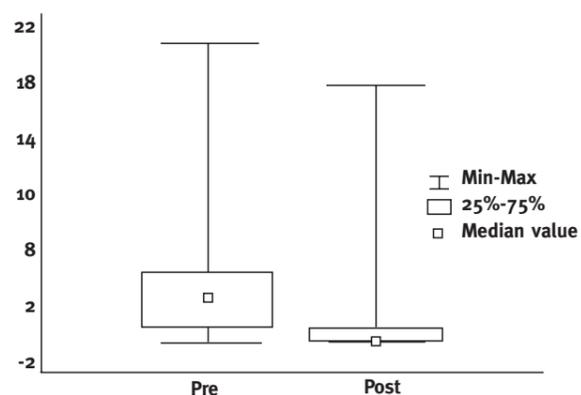
Outcome Criteria

Outcome criteria used include changes in pain, mobility, lumbar endurance, and self-experienced impairment.

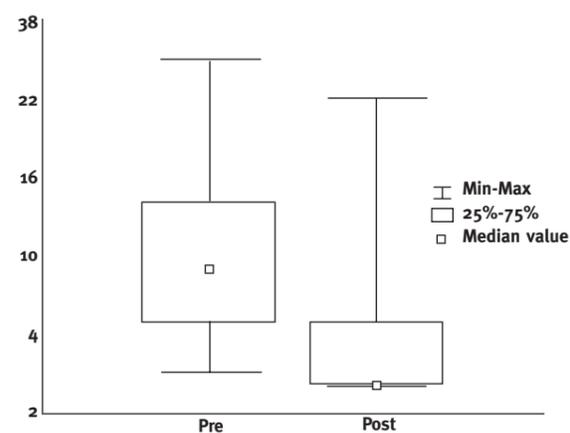
7. DBC Results

Pain reduction and psychological well-being

Pain reduction and changes in psychological well-being with DBC 12-week intervention was studied in 125 chronic/recurrent LBT patients (71 M, 54 F) whose mean age 43 years (SD 10 yr.) and the average recurrent or chronic LBP duration was 9.0 years (SD 9.5 yr.). The majority of them was leading a working life. The data was collected from various DBC outpatient clinics in Finland in early 1995. Significant reduction in pain severity (intensity VAS 62 mm at the beginning versus 31 mm at the end of the program $p < 0.0001$; frequency reduction $p < 0.0001$) on the average was found⁶⁹. Significant reduction in depression scale and improvements in perceived competence (39.0 vs. 41.4, $p < 0.0001$) and self-efficacy towards physical activities (58.7 vs. 62.1, $p < 0.0001$) were also registered⁶⁹. All these changes were significant in both sexes although depression was more often present (higher score) in females at the beginning; however, similar scores were found between sexes at the end of the program on average⁶⁹. Thus, DBC 12-week outpatient program that focuses on restoration of physical function in low back trouble can lead to significant



Changes in depression scale during the 12-week DBC Active Spine Care program.



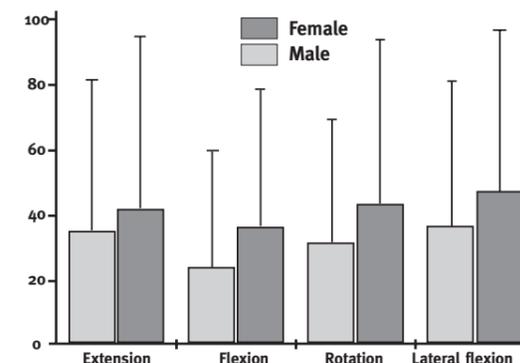
Changes in impairment index during the 12-week DBC Active Spine Care program.

pain reduction and improvements in psychological factors.

Mobility and strength gains

Significant gains in lumbar mobility in all directions have been recorded with DBC functional restoration¹⁵³. Also, trainability of back muscles was studied in 744 LBT patients (373 M, 371 F) whose mean age was 43 yr. (SD 10 yr.; range 15-74 yr.) and who were referred to DBC functional restoration⁴⁰. Their average recurrent or chronic LBP duration was 8.7 yr. (SD 8.6 yr.) and the data was collected from various DBC outpatient clinics in Sweden and Finland in 1994-1995. DBC strength measurements were utilized and correlation coefficients between strength gains and age were calculated, the effect of sex on strength trainability was analyzed with multivariate (different measurements) analysis of variance (sex) and covariance (age) (MANCOVA). The correlations between age and changes in muscle strength expressed either as absolute or relative values were very low (r 's below 0.15) in both sexes which means that age does not affect trainability of back muscles. MANCOVA revealed a significant sex difference both in relative ($R=6.8$, $p=0.00002$) and absolute ($R=6.9$, $p=0.00002$) strength gains. The relative strength gains were higher in females (37-47%) than in males (24-43%) on the average, but the absolute strength gains were higher in males (0.33-0.64

Nm/kg) than in females (0.27-0.43 Nm/kg). In conclusion, both sexes can gain strength significantly, but the absolute strength gains are bigger in males. Since females are weaker than males at the beginning, even small changes in strength can produce high relative changes among them⁴⁰.



Relative strength gains during 12-week DBC functional restoration.

Associations between pain, mobility and strength changes

The association between subjective experience in pain reduction and objective measurements in improvement of physical functioning was analyzed with 143 recurrent/chronic LBT patients¹⁵³. The data was collected from various DBC outpatient clinics in Sweden and Finland in 1994. The associations between DBC strength and mobility measurements and pain inquiries and their changes were calculated. The results showed that 79 % of the subjects reported subjective decrease in LBP during the 12-week restoration program and simultaneous increases in isometric strength and mobility were measured in some 80 per cent of the subjects also. Concordance of these findings was high, i.e., the reduction of pain and improvement of function occurred mostly in the same subjects. However, the correlations between physical functioning parameters and pain reduction were low (r 's below 0.22). Baseline strength and mobility values did not differ between those who benefited from the treatment regarding pain, and those who did not. Thus, absolute levels at the baseline or magnitude of changes in the measurements of maximum isometric strength or

mobility were not associated with pain reduction¹⁵³.

These results indicate that subjective pain reduction is significantly associated with improvement *per se* in trunk muscle function and spinal mobility during active functional restoration, but not with the absolute or relative magnitude of the improvements. Thus self-experienced pain reduction seems to be independent, although concordant, with strength gains. This is taken in to account in the DBC treatment program putting a special emphasis on cognitive and behavioral support of the patients.

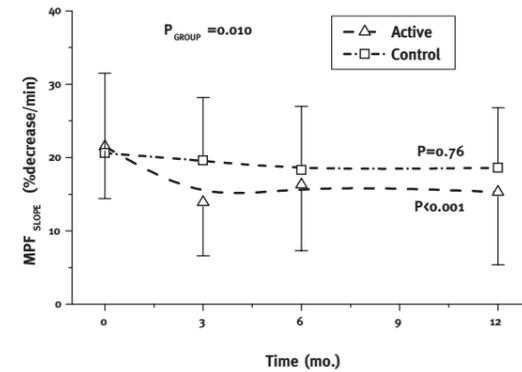
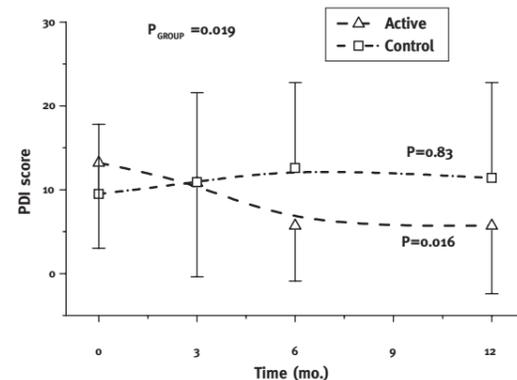
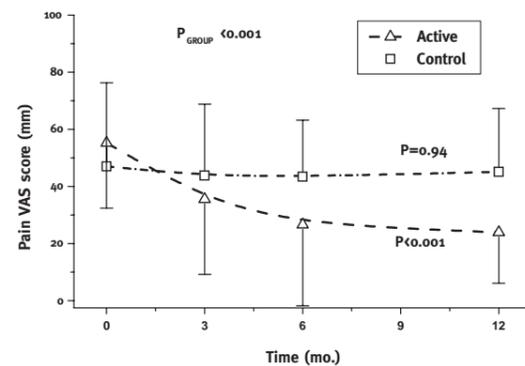
Pain reduction and lumbar endurance improvement in a randomized setting

The efficacy of the DBC protocol in improving lumbar endurance was studied in a randomized setting⁸³. A total of 57 middle-aged patients with non-specific, chronic LBP (35 men and 22 women) were randomly assigned to either a 12 week DBC treatment program, or to a four week passive control treatment program, which was focusing on pain relief with the means of physical and thermal therapy. 19 men and 11 women completed the active program, and 16 men and 8 women completed the passive treatment program. After the intervention patients were followed-up and re-measured at six months and one year.

Pain and disability index (PDI), low back pain intensity (100 mm visual analogue scale, VAS), and paraspinal muscle fatigability (spectral EMG) in the DBC 90 sec submaximal isoinertial back endurance test were recorded before and after the interventions as well as at a six-months and one-year of follow-up. The changes in back pain intensity (VAS scale), disability (PDI score) and lumbar fatigability (MPF_{SLOPE}) were significantly larger ($P < 0.05$) in the active DBC than in the passive control treatment program. The changes were not significantly different between men and women ($P > 0.05$). Pain intensity, disability and lumbar fatigability all decreased significantly ($p < 0.05$) during the active program (VAS_{PRE} 55.2±22.8 mm vs. VAS_{POST} 35.5±26.3 mm; PDI_{PRE} 13.2±10.2 vs. PDI_{POST} 10.8±11.6; MPF_{SLOPE} PRE -21.5±7.1%/min vs. MPF_{SLOPE} POST -

13.9±7.3 %/min). No significant changes were observed during the passive treatment program in any of these outcome variables (VAS_{PRE} 47.0±29.3 mm vs. VAS_{POST} 43.8±23.0 mm; PDI_{PRE} 9.5±8.4 vs. PDI_{POST} 10.9±10.7; MPF_{SLOPE} PRE -20.6±10.9%/min vs. MPF_{SLOPE} POST -19.6±8.6 %/min). The change in fatigability (MPF_{SLOPE}) did not correlate with changes in either pain intensity (VAS) or disability (PDI) (P>0.05). The difference between groups in all outcome measures remained significant during the one year of follow up.

Thus, the DBC treatment was successful in reducing pain, self-experienced disability and lumbar fatigability compared to the passive treatment program, which was focusing on pain relief⁸³. The study also reveals that the benefits regarding reduction in pain and physical impairment and the improvement in lumbar endurance remain over one-year follow-up period.



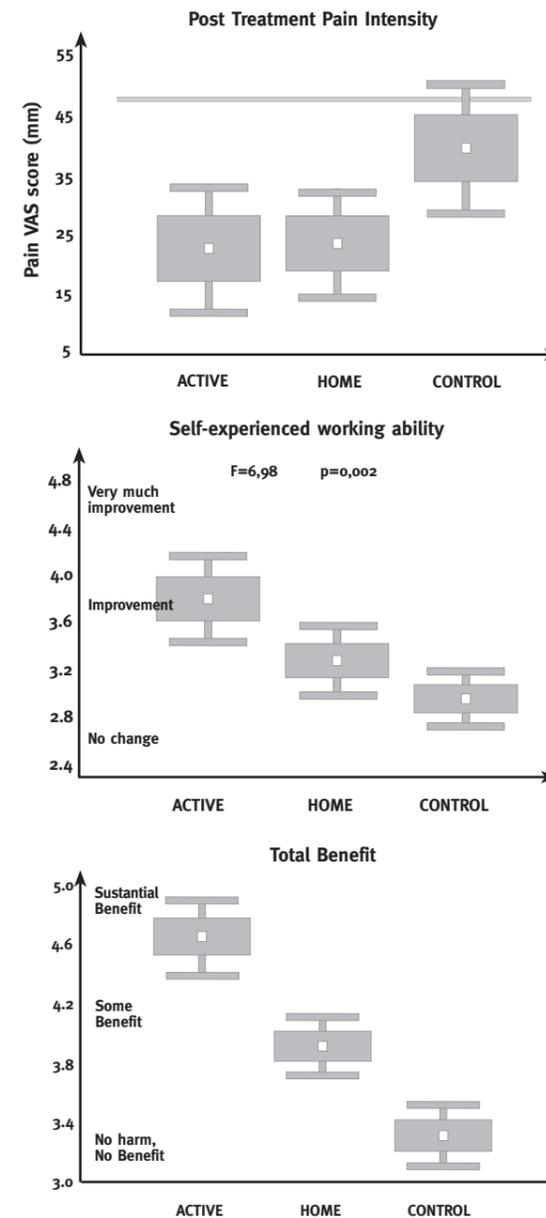
A randomized controlled study compared DBC Active Spine Care and passive control treatment and the results showed that pain intensity (VAS), physical impairment (PDI) and lumbar fatigability (MPF) were reduced during the active treatment. The difference between active and passive treatment results became even more distinct during the one year follow-up after the treatment period⁸³.

Active treatment in chronic neck pain – A prospective randomized study

A randomized comparative study with single-blind outcome assessments compared the efficacy of the multimodal DBC treatment emphasizing proprioceptive training (DBC) with activated home exercises (HOME) and recommendation of exercise (CONTROL) in patients with non-specific chronic neck pain¹⁵⁵. The study group consisted of seventy-six patients (22 men, 54 women) with chronic, non-specific neck pain. Sixty-two participated in the one-year follow-up. Subjective pain and disability, cervical ranges of motion, and pressure pain threshold in the shoulder region were measured at baseline, at three months, and at 12 months. The DBC treatment consisted of 24 sessions of proprioceptive exercises, relaxation, and behavioral support. The HOME regimen included a neck lecture and two sessions of practical training for home exercises and instructions for maintaining a diary of progress. The CONTROL treatment included a lecture regarding care of the neck with a recommendation to exercise. According to the exercise diaries the actual amount of exercise was largest in HOME group and smallest in CONTROL group.

The average self-experienced total benefit was highest in the DBC group, and the HOME group rated over

the CONTROL group (P < 0.001). Differences between the groups in favor of the DBC treatment were recorded in reduction of neck symptoms and improvements in general health and self-experienced working ability (P < 0.01–0.03). Changes in measures of mobility and pressure pain threshold were minor. Since no major differences were noted in



The randomized study revealed that active exercise significantly reduced the pain symptoms. Active exercise program with cognitive behavioral approach completed in out-patient setting gave, however, significantly greater improvement in patients' working ability than home exercise program¹⁵⁵.

objective measurements of cervical function between the groups, it can be assumed that neck pain and, especially, its chronicity comprises a condition where motivation and perceiving the problem plays a significant role. These findings support the idea that multimodal treatment integrating both proprioceptive and endurance exercises as well as behavioral support is more efficacious in treating chronic neck pain patients than solitary training¹⁵⁵.

Absenteeism from work after DBC

A follow-up study investigating the long-term results of DBC treatment was conducted in Luxembourg¹⁵². Consecutive 125 chronic or recurrent low back pain patients (76 women, 49 men) participated in a 12-week active low-back rehabilitation program at an outpatient DBC unit, were followed up on the average 14 months before reassessing their back symptoms and function. The outcomes of the study were defined as a recurrence of persistent pain and work absenteeism and a survival/failure analysis was performed between those who had continued exercising and who had been physically inactive.

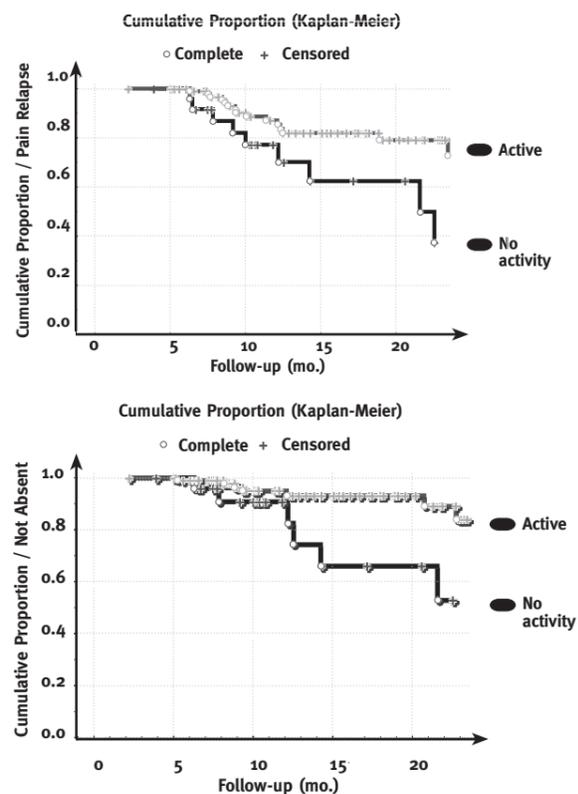
Twenty-five subjects out of the 125 followed (20%) had been physically inactive during the follow-up, 36 subjects (29%) had practiced individual home exercises, 21 (17%) had participated in fitness training, and 43 (34%) had participated in ongoing training once a week in a DBC unit with back specific devices. Kaplan-Meier survival function was used to assess the occurrence of outcome variables (pain relapse or absenteeism) during the follow up. Recurrences of persistent pain during the follow-up period were fewer (p=0.03) among those who had maintained regular exercise habits after the treatment than among those who had been physically inactive. Similarly, work absenteeism was fewer (p<0.01) among physically active than among physically inactive.

After two years of follow-up over eighty percent of subjects remaining active after the DBC treatment continued to work without absenteeism. In the group of physically inactive, roughly 50 percent continued working without

8. DBC quality assurance

absenteeism. In multiple regression analysis it became evident, that patients with good outcome regarding pain reduction in the LBP rehabilitation were more likely to participate in physical exercise.

This study reveals that DBC treatment, when completed successfully, predicts low rates of absenteeism due to back pain after the treatment. An essential part of DBC treatment is to modify patient's behavior towards physical activity and self-responsibility, and when the treatment succeeds in terms of reducing the pain level, patients are likely to remain physically active after the DBC treatment. To achieve a sufficient level of pain reduction during the active treatment program necessitates thorough assessment of a patient's symptoms and function as well as individual planning of contents and length of the treatment program.



After two years of follow-up over eighty percent of subjects remaining active after the DBC treatment continued to work without absenteeism. In the group of physically inactive both pain relapses and absenteeism were more common, and the poor prognosis was strongly related to too short treatment or unsatisfactory outcome initially 152.

Thus, significantly low absenteeism rates can be achieved after the DBC functional restoration 152.

8. DBC Quality Assurance

There are two levels, which need to be addressed when assessing the outcome of a treatment modality. First, the efficacy of a therapeutic procedure, e.g. active rehabilitation, is proven in tightly controlled studies where there are well-described diagnostic and inclusion criteria, and carefully standardized rehabilitation methods provided by well-trained professionals. Preferably a blinded, independent observer assesses the outcome. Then there is a need to assess the effectiveness at the community level, i.e. in real-life conditions. At this point the health-care providers and patients are much more variable than in controlled trials, and the outcome criteria include also applicability and practicality of the treatment regimen.

The basis for good treatment results throughout the whole chain of DBC units includes two main levels. First, the method is evidence-based including controlled efficacy studies. Second, a quality assurance system is applied to ensure real-life effectiveness.

Assurance of quality in DBC to ensure effectiveness contains the following elements: unique technology; basic education; ongoing training; control visits; ongoing analysis of treatment results; and customer satisfaction survey. The rehabilitation equipment used at DBC is available for the DBC units only. A central element of the quality assurance is the basic education, which is given centrally by DBC International in Finland to everyone working in the chain. In addition, ongoing training is provided at minimum on a yearly basis. The ongoing assurance of quality in DBC in practical level is done through quality assurance visits and by analyzing the medical results and customer satisfaction results of the clinics. The persons in charge of the local operations on national level visit each clinic twice a year. Every other of these visits is primarily concentrated on the details in evaluation techniques and rehabilitation method-

ology the clinic is applying, and the treatment results the clinic has achieved with the patients. There is a central data collection of treatment results, which are reported by the clinics into the special computer software. The results are analyzed twice a year and major deviations in outcome in the specific DBC unit demand corrections in the local rehabilitation application. Another tool, which is used to analyze and develop the quality in the DBC units, is the customer satisfaction survey. Each patient fills an anonymous survey at the end of the treatment and the answers are analyzed and the feedback is given to the clinics.

With structured, ongoing quality assurance there is a possibility to ensure effectiveness of the DBC method also in real-life conditions throughout the DBC clinic chain since major deviations from required quality are noticed early and corrections can be made.

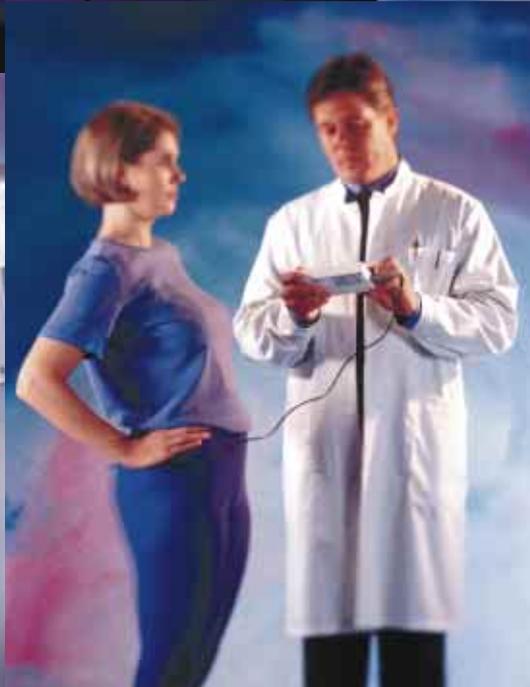
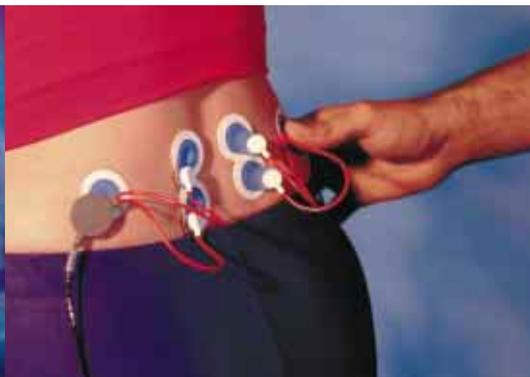
9. references

1. Addison R, Schultz A. Trunk strengths in patients seeking hospitalization for chronic low-back disorders. *Spine* 1980;5:539-544.
2. Agency for Health Policy and Research UDoHHS. Management Guidelines for Acute Low-Back Pain. Washington, DC: US Government Printing Office, 1994.
3. Alaranta H, Moffroid M, Elmqvist L, Held J, Pope M, Renström P. Postural control of adults with musculoskeletal impairment. *Critical Reviews in Physical & Rehabilitation Medicine* 1994;6:337-370.
4. Alaranta H, Rytokoski U, Rissanen A, et al. Intensive physical and psychosocial training program for patients with chronic low back pain. A controlled clinical trial. *Spine* 1994;19(12):1339-1349.
5. Andersson GBJ. Epidemiological aspects on low-back pain in industry. *Spine*. 1981;6:53-60.
6. Andersson JL, Lilja A, Hartvig P, et al. Somatotopic organization along the central sulcus, for pain localization in humans, as revealed by positron emission tomography. *Exp Brain Res* 1997;117(2):192-9.
7. American College of Sports Medicine position stand. The recommended quantity and quality of exercise for developing and maintaining cardiorespiratory and muscular fitness in healthy adults. *Medicine and Science in Sports and Exercise* 1990;22(2):265-274.
8. Assendelft WJ, Koes BW, van der Heijden GJ, Bouter LM. The effectiveness of chiropractic for treatment of low back pain: an update and attempt at statistical pooling. *J Manipulative Physiol Ther* 1996;19(8):499-507.
9. Battie M, Videman T, Gibbons L, Fisher L, Manninen H, Gill K. 1995 Volvo Award in Clinical Sciences: Determinants of Lumbar Disc Degeneration: A Study Relating Lifetime Exposures and Magnetic Resonance Imaging Findings in Identical Twins. *Spine* 1995;20:2601-2612.
10. Bendix A, Bendix T, Vaegter K, Lund C, Frolund L, Holm L. Multidisciplinary intensive treatment for chronic low back pain: A randomized, prospective study. *Cleve Clinics Journal of Medicine* 1996;63:62-69.
11. Biering-Sorensen F. A prospective study of low back pain in a general population. I. Occurrence, recurrence and aetiology. *Scandinavian Journal of Rehabilitation Medicine* 1983;15(2):71-79.
12. Biering-Sorensen F, Thomsen CE, Hilden J. Risk indicators for low back trouble. *Scandinavian Journal of Rehabilitation Medicine* 1989;21(3):151-157.
13. Biering-Sorensen F. Physical measurements as risk indicators for low back trouble over one-year period. *Spine*. 1984;9:106-119.
14. Bigos SJ, Battie MC, Spengler DM, et al. A prospective study of work perceptions and psychological factors affecting the report of back injury. *Spine*. 1991;16:1-6.
15. Boden SD, Davis DO, Dina TS, Patronas NJ, Wiesel SW. Abnormal magnetic-resonance scans of the lumbar spine in asymptomatic subjects. A prospective investigation. *J Bone Joint Surg [Am]* 1990;72(3):403-8.
16. Boden SD, McCowin PR, Davis DO, Dina TS, Mark AS, Wiesel S. Abnormal magnetic-resonance scans of the cervical spine in asymptomatic subjects. A prospective investigation. *J Bone Joint Surg [Am]* 1990;72(8):1178-84.
17. Bogdanffy M, Taimela S, Rashbaum R, Hochschuler S. Back-specific physical reconditioning after laminectomy or fusion spine surgery: A controlled intervention. 8th Annual Meeting of the European Spine Society. Kos, Greece, 1997.
18. Bombardier C, Kerr MS, Shannon HS, Frank JW. A Guide to Interpreting Epidemiologic Studies on the Etiology of Back Pain. *Spine* 1994;19:2047S-2056S.
19. Boos N, Rieder R, Schade V, Spratt KF, Semmer N, Aebi M. 1995 Volvo Award in Clinical Sciences: The Diagnostic Accuracy of Magnetic Resonance Imaging, Work Perception, and Psychosocial Factors in Identifying Symptomatic Disc Herniations. *Spine* 1995;20:2613-2625.
20. Bouchard C, Shephard RJ. Physical activity, fitness and health: the model and key concepts. In: Bouchard C, Shephard RJ, Stephens T, eds. *Physical activity, fitness, and health. Consensus statement*. Champaign, Ill.: Human Kinetics Publishers, 1993:11-23.
21. Brown JJ, Wells GA, Trottier AJ, Bonneau J, Ferris B. Back pain in a large Canadian police force. *Spine* 1998;23(7):821-7.
22. Bullock-Saxton JE, Janda V, Bullock MI. Reflex activation of gluteal muscles in walking. An approach to restoration of muscle function for patients with low-back pain. *Spine*. 1993;18(6):704-708.
23. Burton AK, Tillotson KM, Main CJ, Hollis S. Psychosocial Predictors of Outcome in Acute and Subchronic Low Back Trouble. *Spine* 1995;20(6):722-728.
24. Byl N, Sinnott P. Variations in balance and body sway in middle-aged adults: subjects with healthy backs compared with subjects with low-back dysfunction. *Spine* 1991;16:325-330.
25. Campello M, Nordin M, Weiser S. Review article. Physical exercise and low back pain. *Scandinavian Journal of Medicine and Science in Sports* 1996;6:63-72.
26. Cartas O, Nordin M, Frankel VH, Malgady R, Sheikhzadeh A. Quantification of trunk muscle performance in standing, semistanding and sitting postures in healthy men. *Spine* 1993;18:603-609.
27. Cherkin DC, Deyo RA, Loeser JA, Bush T, Waddell G. An international comparison of back surgery rates. *Spine*. 1994;19:1201-1206.
28. Cholewicki J, Panjabi MM, Khachatryan A. Stabilizing function of trunk flexor-extensor muscles around a neutral spine posture. *Spine* 1997;22(19):2207-12.
29. Clinical Standards Advisory Group. Report on Back Pain. London: Her Majesty's Stationery Office, 1994.
30. Cooper RG, StClair Forbes W, Jayson MIV. Radiographic demonstration of paraspinal muscle wasting in patients with chronic low back pain. *British Journal of Rheumatology* 1992;31:389-394.
31. Craig AD, Reiman EM, Evans A, Bushnell MC. Functional imaging of an illusion of pain. *Nature* 1996;384(6606):258-60.
32. Croft PR, Papageorgiou AC, Ferry S, Thomas E, Jayson MI, Silman AJ. Psychologic distress and low back pain. Evidence from a prospective study in the general population. *Spine* 1995;20(24):2731-7.
33. Davis KD, Taylor SJ, Crawley AP, Wood ML, Mikulis DJ. Functional MRI of pain- and attention-related activations in the human cingulate cortex. *J Neurophysiol* 1997;77(6):3370-80.
34. Derbyshire SW, Vogt BA, Jones AK. Pain and Stroop interference tasks activate separate processing modules in anterior cingulate cortex. *Exp Brain Res* 1998;118(1):52-60.
35. Devinsky O, Morrell MJ, Vogt BA. Contributions of anterior cingulate cortex to behavior. *Brain* 1995;118(Pt 1):279-306.
36. Deyo R, Tsui-Wu Y-J. Descriptive Epidemiology of Low-back Pain and Its Related Medical Care in the United States. *Spine* 1987;12:264-268.
37. Deyo RA. Fads in the treatment of low back pain. *New England Journal of Medicine* 1991;325:1039-1040.
38. Deyo RA, Diehl AK, Rosenthal M. How many days of bed rest for acute low back pain? A randomized clinical trial. *New England Journal of Medicine* 1986;315:1064-1070.
39. Dickenson AH. Central acute pain mechanisms. *Ann Med* 1995;27(2):223-7.
40. Dvorak V, Taimela S. Trainability of back muscles in chronic low back pain patients: the effect of sex and age. *EuroSpine* 96, October 16-19. Zurich, Switzerland, 1996.
41. Eisenstein SM, Ashton IK, Roberts S, et al. Innervation of the spondylolysis "ligament". *Spine* 1994;19(8):912-6.
42. Erkintalo MO, Salminen JJ, Alanen A, Paaanen HEK, Korhano M. Development of degenerative changes in the lumbar intervertebral disk: results of prospective MR imaging study of subjects with and without low back pain. *Radiology*. 1995;196:529-533.
43. Estlander AM, Takala EP, Viikari-Juntura E. Do psychological factors predict changes in musculoskeletal pain? A prospective, two-year follow-up study of a working population. *J Occup Environ Med* 1998;40(5):445-53.
44. Faas A, Chavannes AW, van Eijk JM, Gubbels JW. A Randomized, Placebo-Controlled Trial of Exercise Therapy in Patients With Acute Low Back P. *Spine* 1993;18:1388-1395.
45. Faas A, van Eijk J, Chavannes AW, Gubbels JW. A Randomized Trial of Exercise Therapy in Patients With Acute Low Back Pain: Efficacy on Sickness Absence. *Spine* 1995;20:941-947.
46. Farfan H. Form and Function of the Musculoskeletal System As Revealed by Mathematical Analysis of the Lumbar Spine: An Essay. *Spine* 1995;20:1462-1474.
47. Fidler MW, Jowett RL, Troup JDG. Myosin ATPase activity in multifidus muscle from cases of lumbar spinal derangement. *Journal of Bone and Joint Surgery* 1975;57B:220-227.
48. Frymoyer JW. Back pain and sciatica. *New England Journal of Medicine* 1988;318(5):291-300.
49. Gallagher RM, Rauh V, Haugh LD, et al. Determinants of return-to-work among low back pain patients. *Pain*. 1989;39(1):55-67.
50. Gatchel RJ, Polatin PB, Mayer TG. The Dominant Role of Psychosocial Risk Factors in the Development of Chronic Low Back Pain Disability. *Spine* 1995;20(24):2702-2709.
51. Goldberg L, Elliot D. Exercise for prevention and treatment of illness. Philadelphia, PA: F.A. Davis, 1994.
52. Graves JE, Pollock ML, Carpenter DM, et al. Quantitative assessment of full range-of-motion isometric lumbar extension strength. *Spine*. 1990;15(4):289-294.
53. Graves JE, Pollock ML, Foster D, et al. Effect of training frequency and specificity on isometric lumbar extension strength. *Spine*. 1990;15(6):504-509.
54. Graves JE, Webb DC, Pollock ML, et al. Pelvic stabilization during resistance training: its effect on the development of lumbar extension strength. *Archives of Physical Medicine and Rehabilitation* 1994;75(2):210-215.
55. Hadar H, Gadoth N, Heifetz M. Fatty replacement of lower paraspinal muscles: normal and neuromuscular disorders. *American Journal of Roentgenology* 1983;141(5):895-898.
56. Hansen FR, Bendix T, Skov P, et al. Intensive, dynamic back-muscle exercises, conventional physiotherapy, or placebo-control treatment of low-back pain. A randomized, observer-blind trial. *Spine*. 1993;18(1):98-108.
57. Hazard RG. Spine Update: Functional Restoration. *Spine* 1995;20:2345-2348.
58. Heliövaara M. Risk factors for low back pain and sciatica. *Annals of Medicine* 1989;21(4):257-264.
59. Heliövaara M. Risk factors of low back pain - A review. In: Ernst J, Jayson MIV, Pope MH, Porter RW, eds. *Advances in idiopathic low back pain*. Wien: Blackwell-MZV, 1993:41-51.
60. Hides JA, Richardson CA, Jull GA. Multifidus muscle recovery is not automatic after resolution of acute, first-episode low back pain. *Spine* 1996;21(23):2763-9.
61. Hides JA, Stokes MJ, Saide M, Jull GA, Cooper DH. Evidence

- of lumbar multifidus muscle wasting ipsilateral to symptoms in patients with acute/subacute low back pain. *Spine* 1994;19(2):165-72.
62. Hildebrandt J, Thees C, Pechstein U, Scheufler KM, Wurker J, Nadstawek J. Prediction of success from a multidisciplinary treatment program for chronic low back pain. *Spine* 1997;22:990-1001.
 63. Hodges PW, Richardson CA. Inefficient muscular stabilization of the lumbar spine associated with low back pain. A motor control evaluation of transversus abdominis. *Spine* 1996;21(22):2640-50.
 64. Hodges PW, Richardson CA. Delayed postural contraction of transversus abdominis in low back pain associated with movement of the lower limb. *J Spinal Disord* 1998;11(1):46-56.
 65. Holroyd CB, Dien J, Coles MG. Error-related scalp potentials elicited by hand and foot movements: evidence for an output-independent error-processing system in humans. *Neurosci Lett* 1998;242(2):65-8.
 66. Hultman G, Nordin M, Saraste H, Ohlson H. Body composition, endurance, strength, cross-sectional area and density of Mm. erector spinae in men with and without low back pain. *Journal of Spinal Disorders* 1993;6:114-123.
 67. Hurri H. The Swedish back school in chronic low back pain. Part II. Factors predicting the outcome. *Scandinavian Journal of Rehabilitation Medicine* 1989;21(1):41-44
 68. Härkäpää K. Relationships of psychological distress and health locus of control beliefs with the use of cognitive and behavioral coping strategies in low back pain patients. *Clinical Journal of Pain* 1991;7(4):275-282.
 69. Härkäpää K, Taimela S. Pain reduction and changes in depression and perceived competence during active functional restoration for chronic low-back pain: a 12-week follow-up. *EuroSpine* 96, October 16-19. Zurich, Switzerland: Schulthess Clinic, 1996:76.
 70. Indahl A, Kaigle A, Reikeras O, Holm S. Electromyographic response of the porcine multifidus musculature after nerve stimulation. *Spine* 1995;20(24):2652-8.
 71. Indahl A, Kaigle AM, Reikeras O, Holm SH. Interaction between the porcine lumbar intervertebral disc, zygapophysial joints, and paraspinal muscles. *Spine* 1997;22(24):2834-40.
 72. Jensen MC, Brant-Zawadzki MN, Obuchowski N, Modic MT, Malkasian D, Ross JF. Magnetic resonance imaging of the lumbar spine in people without back pain. *New England Journal of Medicine* 1994;331:69-73.
 73. Jiang H, Moreau M, Raso J, Russell G, Bagnall K. Identification of the location, extent, and pathway of sensory neurologic feedback after mechanical stimulation of a lateral spinal ligament in chickens. *Spine* 1997;22(1):17-25.
 74. Jiang H, Raso JV, Moreau MJ, Russell G, Hill DL, Bagnall KM. Quantitative morphology of the lateral ligaments of the spine. Assessment of their importance in maintaining lateral stability. *Spine* 1994;19(23):2676-82.
 75. Jiang H, Russell G, Raso VJ, Moreau MJ, Hill DL, Bagnall KM. The nature and distribution of the innervation of human supraspinal and interspinal ligaments. *Spine* 1995;20(8):869-76.
 76. Jones AK, Derbyshire SW. Reduced cortical responses to noxious heat in patients with rheumatoid arthritis. *Ann Rheum Dis* 1997;56(10):601-7.
 77. Jones AK, Friston K, Dolan R. Positron emission tomography as a research tool in the investigation of psychiatric and psychological disorders. *Baillieres Clin Endocrinol Metab* 1991;5(1):187-203.
 78. Järvikoski A, Mellin G, Estlander AM, et al. Outcome of two multimodal back treatment programs with and without intensive physical training. *Journal of Spinal Disorders* 1993;6(2):93-98.
 79. Kaigle AM, Wessberg P, Hansson TH. Muscular and kinematic behavior of the lumbar spine during flexion-extension. *J Spinal Disord* 1998;11(2):163-74.
 80. Kalaska J, Drew T. Motor cortex and visuomotor behavior. In: Holloszy J, ed. *Exercise and Sport Sciences Review*. Baltimore: Williams & Wilkins, 1993:397-436.
 81. Kankaanpää M, Taimela S, Laaksonen D, Hanninen O, Airaksinen O. Back and hip extensor fatigability in chronic low back pain patients and controls. *Arch Phys Med Rehabil* 1998;79(4):412-7.
 82. Kankaanpää M, Taimela S, Airaksinen O, Hanninen O. The efficacy of active rehabilitation in chronic low back pain - Effect on pain intensity, self-experienced disability and lumbar fatigability. *Spine* 1999;24:1034-1042.
 83. Kannus P, Josza L, Renström P, et al. e. The effects of training, immobilization and remobilization on musculoskeletal tissues. I. Training and immobilization. *Scandinavian Journal of Medicine and Science in Sports* 1992;2:100-108.
 84. Keltikangas-Järvinen L, Rimon R. Rimon's brief depression scale, a rapid method for screening depression. *Psychol. Rep.* 1987;60:111-119.
 85. Klenerman L, Slade PD, Stanley IM, et al. The Prediction of Chronicity in Patients With an Acute Attack of Low Back Pain in a General Practice Setting. *Spine* 1995;20:478-484.
 86. Koes BW, Assendelft WJ, van der Heijden GJ, Bouter LM. Spinal manipulation for low back pain. An updated systematic review of randomized clinical trials. *Spine* 1996;21(24):2860-71; discussion 2872-3.
 87. Koes BW, Bouter LM, van-der-Heijden GJ. Methodological quality of randomized clinical trials on treatment efficacy in low back pain. *Spine*. 1995;20(2):228-235.
 88. Koes BW, Scholten RJ, Mens JM, Bouter LM. Efficacy of epidural steroid injections for low-back pain and sciatica: a systematic review of randomized clinical trials. *Pain* 1995;63(3):279-88.
 89. Koes BW, Scholten RJ, Mens JM, Bouter LM. Efficacy of non-steroidal anti-inflammatory drugs for low back pain: a systematic review of randomised clinical trials. *Ann Rheum Dis* 1997;56(4):214-23.
 90. Kohles S, Barnes D, Gatchel RJ, Mayer TG. Improved physical performance outcomes after functional restoration treatment in patients with chronic low-back pain. Early versus recent training results. *Spine*. 1990;15(12):1321-1324.
 91. Komi P. *Strength and power in sport*. London: Blackwell, 1992.
 92. Kujala UM, Taimela S, Videman T, Battie MC, Viljanen T. Physical loading and performance as predictors of back pain in healthy adults. A 5-year prospective study. *European Journal of Applied Physiology and Occupational Physiology* 1996;73:452-458.
 93. Laasonen EM. Atrophy of sacrospinal muscle groups in patients with chronic, diffusely radiating lumbar back pain. *Neuroradiology*. 1984;26:9-13.
 94. Leboeuf-Yde C, Klougart N, Lauritzen T. How common is low back pain in the Nordic population? Data from a recent study on a middle-aged general Danish population and four surveys previously conducted in the Nordic countries. *Spine* 1996;21(13):1518-25; discussion 1525-6.
 95. Letham J, Slade PD, Troup JDG, Bentley G. Outline of a fear-avoidance model of exaggerated pain perception. Part 1. *Behavioral Research and Therapy* 1983;21:401-408.
 96. Lindstrom I, Ohlund C, Eek C, Wallin L, Peterson LE, Nachemson A. Mobility, strength, and fitness after a graded activity program for patients with subacute low back pain. A randomized prospective clinical study with a behavioral therapy approach. *Spine*. 1992;17(6):641-652.
 97. Lindström I, Öhlund C, Eek C, et al. e. The Effect of Graded Activity on Patients with Subacute Low Back Pain: A Randomized Prospective Clinical Study with an Operant-Conditioning Behavioral Approach. *Physiotherapy* 1992;72:279-293.
 98. Luoto S, Aalto H, Taimela S, Hurri H, Pyykkö I, Alaranta H. One-footed and externally disturbed two-footed postural control in chronic low-back pain patients and healthy controls: A controlled study with follow-up. *Spine* 1998;23:2081-2090
 99. Luoto S, Heliövaara M, Hurri H, Alaranta H. Static back endurance and the risk of low back pain. *Clinical Biomechanics* 1995;10:323-324.
 100. Luoto S, Taimela S, Hurri H, Aalto H, Pyykkö I, Alaranta H. Psychomotor speed and postural control in chronic low-back pain patients: A controlled follow-up study. *Spine* 1996;21:2621-2627.
 101. Luoto S, Taimela S, Hurri H, Alaranta H. Mechanisms explaining the association between low-back trouble and deficits in information processing: A controlled study with follow-up. *Spine* 1999; 24(3):255-62
 102. Magnusson ML, Aleksiev A, Wilder DG, et al. European Spine Society--the AcroMed Prize for Spinal Research 1995. Unexpected load and asymmetric posture as etiologic factors in low back pain. *Eur Spine J* 1996;5(1):23-35.
 103. Main CJ, Wood PL, Hollis S, Spanswick CC, Waddell G. The Distress and Risk Assessment Method. A simple patient classification to identify distress and evaluate the risk of poor outcome. *Spine* 1992;17(1):42-52.
 104. Malmivaara A, Hakkinen U, Aro T, et al. The treatment of acute low back pain--bed rest, exercises, or ordinary activity? *New England Journal of Medicine* 1995;332(6):351-355.
 105. Manniche C, Asmussen K, Lauritsen B, et al. Intensive dynamic back exercises with or without hyperextension in chronic back pain after surgery for lumbar disc protrusion. A clinical trial. *Spine*. 1993;18(5):560-567.
 106. Manniche C, Hesselsoe G, Bentzen L, Christensen I, Lundberg E. Clinical trial of intensive muscle training for chronic low back pain. *Lancet*. 1988;2(8626-8627):1473-1476.
 107. Manniche C, Lundberg E, Christensen I, Bentzen L, Hesselsoe G. Intensive dynamic back exercises for chronic low back pain: a clinical trial. *Pain* 1991;47:53-63.
 108. Mannion AF, Connolly B, Wood K, Dolan P. The use of surface EMG power spectral analysis in the evaluation of back muscle function. *J Rehabil Res Dev* 1997;34(4):427-39.
 109. Mannion AF, Dolan P, Adams MA. Psychological questionnaires: do "abnormal" scores precede or follow first-time low back pain? *Spine* 1996;21(22):2603-11.
 110. Mannion AF, Muntener M, Taimela S, Dvorak J. A randomized clinical trial of three active therapies for chronic low back pain. *Spine* 1999;24(23):2435-48.
 111. Markenson JA. Mechanisms of chronic pain. *Am J Med* 1996;101(1A):6S-18S.
 112. Mayer TG, Gatchel RJ, Kishino N, et al. Objective assessment of spine function following industrial injury. A prospective study with comparison group and one-year follow-up. *Spine*. 1985;10(6):482-493.
 113. Mayer TG, Polatin P, Smith B, et al. Contemporary Concepts in Spine Care: Spine Rehabilitation: Secondary and Tertiary Nonoperative Care. *Spine* 1995;20(18):2060-2066.
 114. Mayer TG, Smith SS, Keeley J, Mooney V. Quantification of

- lumbar function. Part 2: Sagittal plane strength in low back pain patients. *Spine*. 1985;10:765-772.
117. McKinnon ME, Vickers MR, Ruddock VM, Townsend J, Meade TW. Community studies of the health service implications of low back pain. *Spine* 1997;22(18):2161-6.
118. McLain RF. Mechanoreceptor endings in human cervical facet joints. *Spine* 1994;19(5):495-501.
119. McNeill T, Warwick D, Andersson C, Schultz A. Trunk strength in attempted flexion, extension, and lateral bending in healthy subjects and patients with low back disorders. *Spine* 1980;5:529-538.
120. Mellin G, Harkapaa K, Vanharanta H, Hupli M, Heinonen R, Jarvikoski A. Outcome Of A Multimodal Treatment Including Intensive Physical Training Of Patients With Chronic Low Back Pain. *Spine* 1993;18:825-829.
121. Mooney V, Kenney K, Leggett S, Holmes B. Relationship of lumbar strength in shipyard workers to workplace injury claims. *Spine* 1996;21(17):2001-5.
122. Mälkiä E, Ljunggren A. Exercise Programs for Subjects with Low Back Disorders. *Scandinavian Journal of Medicine and Science in Sports* 1996;6:73-81.
123. Nachemson A. Chronic pain--the end of the welfare state? *Quality of Life Research* 1994;3:11-17.
124. Nachemson A, Lindh M. Measurement of abdominal and back extension strength with and without low-back pain. *Scandinavian Journal of Rehabilitation Medicine* 1969;1:60-65.
125. Nachemson AL. The lumbar spine: an orthopedic challenge. *Spine*. 1976;1:59-71.
126. Nelson JM, Walmsley RP, Stevenson JM. Relative lumbar and pelvic motion during loaded spinal flexion/extension. *Spine*. 1995;20(2):199-204.
127. Paajanen H, Erkintalo M, Kuusela T, Dahlström S, Kormanen M. Magnetic resonance study of disc degeneration in young low-back pain patients. *Spine*. 1989;14:982-985.
128. Parkkola R, Kujala UM, Rytökoski U. Response of the muscles to training assessed by MRI and muscle strength. *European Journal of Applied Physiology and Work Physiology* 1992;65:383-387.
129. Parnianpour M, Nordin M, Kahanovitz N, Frankel V. The triaxial coupling of torque generation of trunk muscles during isometric exertions and the effect of fatiguing isoinertial movements on the motor output and movement pattern. *Spine* 1988;13:982-992.
130. Rainville P, Duncan GH, Price DD, Carrier B, Bushnell MC. Pain affect encoded in human anterior cingulate but not somatosensory cortex. *Science* 1997;277(5328):968-71.
131. Ready AE, Boreskie SL, Law SA, Russell R. Fitness and lifestyle parameters fail to predict back injuries in nurses. *Can J Appl Physiol* 1993;18(1):80-90.
132. Riihimäki H, Mattson T, Zitting A, Wickstrom G, Hanninen K, Waris P. Radiographic changes of the lumbar spine among concrete reinforcement workers and house painters. *Spine* 1990;15:114-119.
133. Riihimäki H, Luoma K, Raininko R, Viikari-Juntura E, Lamminen A, Luukkonen R. Low-back pain and disc degeneration of the lumbar spine - A MRI study. ISSLS 23rd Annual Meeting, Burlington, Vermont, June 25-29, 1996:170.
134. Risch SV, Norvell NK, Pollock ML, et al. Lumbar strengthening in chronic low back pain patients. Physiologic and psychological benefits. *Spine*. 1993;18(2):232-238.
135. Rissanen A, Kalimo H, Alaranta H. Effect of intensive training on the isokinetic strength and structure of lumbar muscles in patients with chronic low back pain. *Spine*. 1995;20:333-340.
136. Roberts S, Eisenstein SM, Menage J, Evans EH, Ashton IK. Mechanoreceptors in intervertebral discs. Morphology, distribution, and neuropeptides. *Spine* 1995;20(24):2645-51.
137. Rose MJ, Klenerman L, Atchison L, Slade PD. An application of the fear avoidance model to three chronic pain problems. *Behav Res Ther* 1992;30(4):359-65.
138. Rothwell J. Control of human voluntary movement. (2 ed.) Cambridge: University Press, 1994.
139. Roy SH, De Luca CJ, Casavant DA. Lumbar muscle fatigue and chronic lower back pain. *Spine*. 1989;14:992-1001.
140. Roy SH, De-Luca CJ, Emley M, Buijs RJ. Spectral electromyographic assessment of back muscles in patients with low back pain undergoing rehabilitation. *Spine*. 1995;20(1):38-48.
141. Sackett DL. Evidence-based medicine. *Spine* 1998;23(10):1085-6.
142. Salminen JJ, Erkintalo-Tertti MO, Paajanen HE. Magnetic resonance imaging findings of lumbar spine in the young: correlation with leisure time physical activity, spinal mobility, and trunk muscle strength in 15-year-old pupils with or without low-back pain. *Journal of Spinal Disorders* 1993;6(5):386-391.
143. Schmidt RA. Motor control and learning. A behavioral emphasis. (2nd ed.) Champaign, Illinois: Human Kinetics Publishers, 1988.
144. Seidel H, Beyer H, Brauer D. Electromyographic evaluation of back muscle fatigue with repeated sustained contractions of different strengths. *Eur J Appl Physiol* 1987;56(5):592-602.
145. Sihvonen T. Flexion relaxation of the hamstring muscles during lumbar-pelvic rhythm. *Arch Phys Med Rehabil* 1997;78(5):486-90.
146. Sihvonen T, Lindgren KA, Airaksinen O, Manninen H. Movement disturbances of the lumbar spine and abnormal back muscle electromyographic findings in recurrent low back pain. *Spine* 1997;22(3):289-95.
147. Sihvonen T, Partanen J, Hanninen O, Soimakallio S. Electric behavior of low back muscles during lumbar pelvic rhythm in low back pain patients and healthy controls. *Arch Phys Med Rehabil* 1991;72(13):1080-7.
148. Snijders CJ, Slagter AHE, van Strik R, Vleeming A, Stoeckart R, Stam HJ. Why Leg Crossing? The Influence of Common Postures on Abdominal Muscle Activity. *Spine* 1995;20:1989-1993.
149. Suzuki N, Endo S. A quantitative study of trunk muscle strength and fatigability in the low back pain syndrome. *Spine*. 1983;8:69-74.
150. Svensson HO, Andersson GB. The relationship of low-back pain, work history, work environment, and stress. A retrospective cross-sectional study of 38- to 64-year-old women. *Spine*. 1989;14(5):517-522.
151. Svensson HO, Andersson GBJ. Low-back pain in 40- to 47-year-old men. *Spine*. 1983;8(3):272-276.
152. Taimela S, Didrich C, Hubsch M, Heinrich M. The role of physical exercise and inactivity on low back pain recurrence and absenteeism from work after active outpatient rehabilitation for recurrent-chronic LBP. *Spine* 2000:in press.
153. Taimela S, Härkäpää K. Strength, mobility, their changes and pain reduction during active functional restoration for chronic low back disorders. *Journal of Spinal Disorders* 1996;9:306-312.
154. Taimela S, Kankaanpää M, Luoto S. The effect of lumbar fatigue on the ability to sense a change in lumbar position - a controlled study. *Spine* 1999;24(13):1322-7.
155. Taimela S, Takala EP, Asklof T, Seppala K, Parviainen S. Active Treatment of Chronic Neck Pain: A Prospective Randomized Intervention. *Spine* 2000;25(8):1021-1027.
156. Taimela S, Österman K, Alaranta H, Soukka A, Kujala UM. Long psychomotor reaction time in patients with chronic low-back pain - preliminary report. *Archives of Physical Medicine and Rehabilitation* 1993;74:1161-1164.
157. Thorstensson A, Arvidsson A. Trunk muscle strength and low back pain. *Scandinavian Journal of Rehabilitation Medicine* 1982;14:69-75.
158. Triano JJ, McGregor M, Cramer GD, Emde DL. A comparison of outcome measures for use with back pain patients: results of a feasibility study [see comments]. *J Manipulative Physiol Ther* 1993;16(2):67-73.
159. Tucci JT, Carpenter DM, Pollock ML, Graves JE, Leggett SH. Effect of reduced frequency of training and detraining on lumbar extension strength. *Spine*. 1992;17(12):1497-1501.
160. Waddell G, Newton M, Henderson I, Somerville D, Main CJ. A Fear-Avoidance Beliefs Questionnaire (FABQ) and the role of fear-avoidance beliefs in chronic low back pain and disability. *Pain* 1993;52(2):157-68.
161. Wallston KA, Wallston BS, DeVellis R. Development of the Multidimensional Health Locus of Control (MHLC) Scales. *Health Educ Monogr* 1978;6(2):160-70.
162. van der Windt DA, van der Heijden GJ, Scholten RJ, Koes BW, Bouter LM. The efficacy of non-steroidal anti-inflammatory drugs (NSAIDs) for shoulder complaints. A systematic review. *J Clin Epidemiol* 1995;48(5):691-704.
163. van Poppel MN, Koes BW, Smid T, Bouter LM. A systematic review of controlled clinical trials on the prevention of back pain in industry. *Occup Environ Med* 1997;54(12):841-7.
164. van Tulder MW, Assendelft WJ, Koes BW, Bouter LM. Method guidelines for systematic reviews in the Cochrane Collaboration Back Review Group for Spinal Disorders. *Spine* 1997;22(20):2323-30.
165. van Tulder MW, Koes BW, Bouter LM. Conservative treatment of acute and chronic nonspecific low back pain. A systematic review of randomized controlled trials of the most common interventions. *Spine* 1997;22(18):2128-56.
166. Videman T, Sarna S, Battie MC, et al. The long-term effects of physical loading and exercise lifestyles on back related symptoms, disability, and spinal pathology among men. *Spine*. 1995;20(6):699-709.
167. Wilder D, Aleksiev A, Magnusson M, Pope M, Spratt K, Goel V. Muscular response to sudden load - A tool to evaluate fatigue and rehabilitation. *Spine* 1996;21(22):2628-2639.
168. Vleeming A, Pool-Goudzwaard AL, Hammudoghlu D, Stoeckart R, Snijders CJ, Mens JMA. The Function of the Long Dorsal Sacroiliac Ligament: Its Implication for Understanding Low Back Pain. *Spine* 1996;21:556-562.
169. Vogt BA, Derbyshire S, Jones AK. Pain processing in four regions of human cingulate cortex localized with co-registered PET and MR imaging. *Eur J Neurosci* 1996;8(7):1461-73.
170. Von-Korff M. Studying the natural history of back pain. *Spine*. 1994;19(18):2041S-2046S.
171. Wood K, Garvey T, Gundry C, Heithoff K. Magnetic resonance imaging of the thoracic spine. Evaluation of asymptomatic individuals. *Journal of Bone and Joint Surgery* 1995;77-A:1631-1638.
172. Yamashita T, Cavanaugh JM, el-Bohy AA, Getchell TV, King AI. Mechanosensitive afferent units in the lumbar facet joint. *J Bone Joint Surg [Am]* 1990;72(6):865-70.
173. Yoganandan N, Maiman DJ, Pintar F, Ray G, Myklebust JB, Sances A, jr. Microtrauma in the lumbar spine: a cause of low-back pain. *Neurosurgery*. 1988;23:162-168.

DBC International Ltd.
PO Box 125, 01511 Vantaa, Finland
Tel. +358 9 870 0640
Fax + 358 9 870 06450
Email: dbc@dbc.fi



DBC America

3725 Cockrell
Forth Worth, TX 76110
USA
Tel. +1 817 921 9981
Fax +1 817 921 1407

DBC Belgium

Ortho-Spine bvba
Kortrijksesteenweg 1065
B-9051 Gent
Belgium
Tel. +32 9 221 46 22
Fax +32 9 220 29 68

DBC Finland

Suomen Aktiivikuntoutus Ky
Marttilantie 53
60100 Seinäjoki, Finland
Tel. +358 6 414 1723
Fax +358 6 414 1673
Email:
ali.laitasaari@aktiivikuntoutus.nic.fi

DBC Luxembourg

**Mondorf le domaine
Thermal**
BP 52
L-5601 Mondorf-les-Bains
Luxembourg
Tel. +352 66 12 12 603
Fax +352 66 12 12 620
Email: m.heinricy@mondorf.lu

DBC Netherlands

Buuserstraat 216
7544 RG Enschede
The Netherlands
Tel. +31 53 478 2871
Fax +31 53 478 2873
Email: info@dbc.nl

DBC Norway

Aktive Rehab Norge AS
Johan Berentsensvei 63
N-5161 Laksevåg
Norway
Tel. +47 55 349 032
Fax +47 55 349 031

DBC Sweden

Håkan Kihlström AB
Hattmakargatan 5
80311 Gävle
Sweden
Tel. +46 26 106 541
Fax +46 26 649 464
Email: info@dbesweden.com

DBC Singapore

**TeBex Medica Singapore
Pte Ltd.**
Camden Medical Centre
1 Orchard Boulevard
#09-05
Singapore 249615
Tel. +65 835 2896
Fax +65 835 2897
Email: tebexmedica@pacific.net.sg

DBC Switzerland

Medaktiv AG
Schulweg 9
8610 Uster
Switzerland
Tel. +41 1 940 6354
Fax +41 1 940 8827

DBC UK

P.O. Box 183
Chertsey
Surrey KT16 OYS
United Kingdom
Tel +44 781 803 1685
Fax. +44 171 656 2703
Email: fiona.long@dbc.fi