THE NATURAL COURSE OF LOW BACK PAIN AND EARLY INTERVENTION OF HIGH RISK POPULATION

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ABSTRACT

STUDY OBJECTIVES: (1) To apply the afore-mentioned methodological guidelines to the literature on community prevalence of LBP and assign an overall methodological score, (2) To compare studies determined to be methodologically acceptable in an attempt to draw conclusions about the prevalence of LBP in the world population, (3) To estimate of the point prevalence of LBP in North America, and (4) To make suggestions for improving the methodological quality of these types of studies. **DESIGN:**RCT**. SETTING:**Patients were recruited from the orthopedic clinic of a local hospital and several general practitioners' practices. **METHODS:**An RCT was performed with patients randomly assigned to 1 of 2 treatment groups: (1) a group that received general exercise combined with specific trunk muscle stabilization exercise techniques or (2) a group that received general exercise only. The research physical therapist (GAK) who was in charge of the study and who performed the outcome assessments of subjects and data analyses was unaware of group allocation throughout the study. However, the clinical physical therapist (FR) who administered the exercise programs could not bemasked to group allocation. Patients were not aware of the theoretical bases of each of the exercise regimensbecause the study's objective was described to them in the following way: "to identify any differential effect between 2 exercise regimens for the trunk muscles,which have a role in protecting the spine from further injury **OUTCOME MEASURE:** Short-Form McGill Pain Questionnaire (SF-MPQ), visual analog scale (VAS), Roland-Morris Disability Questionnaire (RMDQ). **CONCLUSION:** LBP is a common and usually benign disorder, which is characterized by exacerbations and remissions. However, about 10% of the adult Danish population develops chronic or frequently recurring LBP over several years. Therefore, an effort must be made to identify a high-risk population early in order to implement selective primary preventive measures. LBP is associated with the presence of other disorders in both adults and adolescents, thus, these associations cannot be explained merely by external factors in adult life, but probably have, at least partly, a common origin based on an inherent frailty. We could not predict future LBP by birth factors, although birth weight often is associated with other diseases, but the liability to develop LBP seems to have a genetic component. It is possible that it is the individuals with a strong genetic disposition for LBP, who develop chronic/recurrent LBP in adulthood. If such an underlying genetic predisposition exists, the recurring pattern of LBP is a logical consequence. This predisposition might also indicate a general frailty making individuals more prone to other disorders as well, which would explain the associations between LBP and other disorders.

INTRODUCTION

The term *low back pain* (LBP) is very broad and covers a large heterogeneous group of disorders. These cannot only be characterized by pain, but also by discomfort and/or stiffness. "LBP" probably covers several sub-groups with differing etiologies and prognoses, but since current knowledge does not allow us to determine the exact medical cause of LBP in most patients, ´nonspecific ´ LBP is determined by exclusion. In fact, it has been estimated by some that a somatic cause is found in 10-20% of cases with LBP (1), whereas others find that as much as 97% of LBP is called "non-specific" or "sprain/strain" (2). Thus, LBP refers to a set of symptoms or a syndrome rather than a diagnosis (3). Although low back trouble would be a more precise term, LBP (or nonspecific LBP) is the most commonly used term for non-specific trouble relating to the lower back. As it is also commonly used for indexing purposes, it will be used throughout this thesis.

Although 60-65% of the Nordic population experience LBP during their lifetime (4), only a small subset of these become chronic/recurrent LBP-sufferers (5-7). It has been concluded in American economic analyses that 60-70% of the total costs associated with LBP are related to this subset of individuals (8). According to Petersen et al., musculo-skeletal problems are the most common reason for long-term sick-leave in Denmark (24%) with permanent or recurring back trouble

occurring in 10% of the Danish workforce (9). Despite the difference in social security coverage and reimbursement systems, similar figures have been reported from the United States (10). Obviously, there is a lot to gain for society as well as for the individual, if it were possible to identify, at an early stage, those with a high risk of chronicity/recurrence. To gain insight into disease development, it is necessary to understand the natural course of the disease. In a disorder with a highly variable course, such as LBP, this is difficult, and requires long-term follow-up as well as careful considerations of outcome measures. Available data on the natural history of LBP were found to be incomplete and confusing by Von Korff in 1994 (11) and unfortunately this has not improved much since. It still needs to be established what the chances are, that the pain will run a transient, recurrent or chronic course.

In order to prevent long-lasting LBP it is not sufficient to identify risk factors. During the past decades, numerous factors, such as physical characteristics (17), psychological characteristics (18), lifestyle factors (19), employment (20-23), social factors (24,25) andgenetic components (26), have been considered risk factors for developing LBP. Despite considerable research efforts, no clear picture has emerged. As proposed by Hartvigsen (27), this might be due to poor design of the studies (cross-sectional, lack of clear definitions of both LBP and the exposure), but it might also be due to more complicated relationships between outcome and exposure. Even thoughdifferent factors are found to be dominant risk factors in different studies (24,28,29), they may all at the same time be complicating factors or confounders of varying importance.Some factors might enhance each other while some might suppress the effect of others. Furthermore, the same factor may have various influences on different body types, personalities, genetic make-up or subgroups of LBP. It is necessary to recognize the fact th t potential risk factors affect people at an individual level, i.e. different people may react differently to a specific stimulus. Etiologic research has so far mainly focused on risk factors. To make prevention efficient, risk factors must be known, but risk groups must also be identified to target the prevention at the most needing group.

To date, only few attempts have been made to describe the group which is most susceptible to LBP and which will respondwith morbidity to external stressors. Epidemiological studies have mainly focused on adult populations and several attempts have been made to predict recovery versus nonrecovery of LBP (24,28-30). Since these studies obviously focus on people who alreadyare affected by LBP, this limits the scope to secondary prevention. Ideally, epidemiological studies of the development of LBP should be performed on large, young populations in order to assess the impact of a general inherent frailty on the development of LBP. Obviously, the older a population is, the more difficult it will be to detect the impact of possible inherent risk factors of LBP, as other causative or aggravating factors gradually will confuse the picture.

A group with a high risk of low back pain, might have a high a risk of other disorders as well. Due to some underlying disposition, physiological, genetic, social or otherwise, they may be generally susceptible to disease, i.e. they are more 'frail' than the average population The concept of frailty was introduced in demographic research at the end of the 1970s (31) as a non-observable, hidden susceptibility to death, and is mainly used in relation to aging populations. A full understanding of the concept of frailty may result from a description of how multiple systems intersect to produce frailty (32). The musculoskeletal system is considered to be an essential part of the development of such a frailty, and loss of leg strength has been noted to be the strongest single predictor for subsequent institutionalization, stronger than other physiological markers and disease diagnosis (33). In this way, a cycle of frailty is introduced in old people, where deficits in other systems can impair functions of the musculoskeletal system. Loss of movement capacity, in turn, frequently accelerates declines in other systems, which in turn feed back on the ability to move (34). Bortz even consider the musculoskeletal system to be the entrance pathway for frailty in the elderly (34).

To explore the natural course of LBP, a systematic and critical review of the literature was done, based on articles retrieved from Medline and EMBASE using the comprehensive search strategy recommended by the Back Review Group of the Cochrane Collaboration (44). Articles had to be published from 1992 to 1999 and include at least one-year follow-up of LBP. Furthermore, only original articles with a sample size of 50 or more, written in English or the Scandinavian languages, were included. Methodological quality of the articles was evaluated independently by two reviewers, and the results were analyzed in relation to outcome, nationality, age, gender, and previous history of LBP.

In order to estimate the natural course of LBP in a Danish population, data from the Ebeltoft project, considered to be representative for the Danish middle-aged population, were analyzed for change in LBPstatus over time. The outcome variable used was number of days with LBP during the past year. On this basis, the cohort was divided into three subgroups: no LBP, short-lasting LBP and long-lasting/recurrent LBP. The transition between these groups over a five-year period was investigated and prevalences, stability within groups, and transition probabilities between groups were reported.

METHODS

As a first step in identifying a frail sub-population with a high risk of LBP, a review of the literature relating to comorbidity with LBP was performed. Medline was searched for articles relating LBP to frailty, comorbidity or a number of individual disorders. A manual search was done through the authors' personal archives relating to LBP, and reference lists were screened. Articles were included if they related LBP to at least one other physical disorder, but excluded if the prevalence of such disorders could not be compared to that of a control group or the general population. The retrieved articles were evaluated for quality and information relating to strength of associations and temporality was extracted.

LBP and birth-factors: The results of the comorbidity study indicated the existence of a generally frail subgroup among teenagers, while others are more robust. The next step was to investigate if a possible susceptibility could be detected already at birth. For this purpose, data about LBP from the Twin Registry were combined with information regarding birth weight, birth length, gestational age and Apgar scores from The Danish Medical Birth Register. Possible associations between LBP in adolescence and birth characteristics were investigated by means of multiple logistic regression analysis and again a twin control study was conducted to control for various confounders.

Heredity of LBP: To investigate whether LBPsusceptibility was present even before birth, the relative contribution of genes and environment on the development of LBP in adolescent twins was explored. Information about LBP, gender, age and zygosity was extracted from The Danish Twin Registry. Casewise concordance rates were estimated to test the null hypothesis that genetic factors do not influence the variance of a trait (48). Common odds ratios for MZ- and DZ-twins

(comparing the odds for having LBP in twins with affected co-twins to those with unaffected co-twins) were calculated to determine if there was familial aggregation, and MZ and DZ odds ratios were compared in search of an underlying genetic influence (49). Finally, tetrachoric correlation coefficients as well as path analysis were performed to estimate the relative contribution of additive effects of genes, dominant effects of genes, common (shared) family environment and unique (within-family) environment (50). The differences between MZ and DZ tetrachoric correlation coefficients were used to supply evidence of absence/presence of genetic influence on the liability of LBP, and could also indicate the relative distribution of the variance components. In the path analysis, seven different liability models were fitted todata and the Akaike Information Criterion was used to determine the best-fitting model (51). All results were stratified for age to illustrate the variability between age groups.

Design: An RCT was performed with patients randomly assigned to 1 of 2 treatment groups: (1) a group that received general exercise combined with specific trunk muscle stabilization exercise techniques or (2) a group that received general exercise only. The research physical therapist (GAK) who was in charge of the study and who performed the outcome assessments of subjects and data analyses was unaware of group allocation throughout the study. However, the clinical physical therapist (FR) who administered the exercise programs could not bemasked to group allocation. Patients were not aware of the theoretical bases of each of the exercise regimensbecause the study's objective was described to them in the following way: "to identify any differential effect between 2 exercise regimens for the trunk muscles,which have a role in protecting the spine from further injury."

Subjects: Patients were recruited from the orthopedic clinic of a local hospital and several general practitioners' practices. Patients took part in the study after informed consent had been obtained. The rights of human subjects were protected at all times.Patients were eligible for the study if they had a history of recurrent LBP (repeated episodes of pain in past year collectively lasting for less than 6 months)31 of a nonspecific nature, defined as back pain complaints occurring without identifiable specific anatomical or neurophysiological causative factors.2 To establish this, all patients included in the trial had a prior clinical examination by their physician, including a radiograph or a magnetic resonance imaging scan. Patients with previous spinal surgery, "red flags" (ie, serious spinal pathology or nerve root pain signs) as outlined in the Clinical Standards Advisory Group (CSAG) report for back pain, or signs and symptoms of instability (radiological diagnosis of spondylolysis or spondylolisthesis corresponding to a symptomatic spinal level; "catching," "locking," "giving way," or "a feeling of instability" in one direction or multiple directions of spinal movements) were excluded. Patients were recruited for the trial at the subacute or chronic stage (onset of their current episode of pain 6 weeks) if their symptoms persisted. The anthropometric and LBP history data of patients who took part in the RCT are presented in Table 1. Patients had to be medically fit (no heart problems, pregnancy, or inflammatory arthritis) and willing to participate in the exercise program and be able to travel independently to the hospital. All subjects were employed at the time of study and were not involved in any current workers' compensation or litigation procedures. The subjects' progress throughout the trial is outlined in the Figure.

	Stabilization–Enhanced General Exercise Group (n=29)		General Exercise–Only Group (n=26)		
	$\bar{X}$	SD	$\bar{X}$	SD	P
Anthropometry					
Age (y)[b]	39.2	11.4	35.2	9.7	.16
Height (cm)[b]	170.1	7.5	174.4	9.1	.06
Body mass (kg)[b]	75.9	12.8	80.5	12.0	.18
BMI (kg/m^2)[b]	26.2	4.2	26.4	3.2	.87
History of LBP					
Time since first onset (mo)[b]	57.1	48.1	44.2	51.6	.34
Current duration (wk)[c]	12.0	7.3–22.0	12.0	8.0–12.0	.78

DATA ANALYSIS

Normality of distribution for all data collected was analyzed with the Kolmogorov-Smirnov test. Summary statistics for anthropometric and outcome variables were compared at baseline for the 2 exercise groups (independent-samples t test or Mann-Whitney U test) to establish whether the applied randomization procedure was successful.

A 2 x 3 (exercise group x time) analysis of variance for repeated measures on the second factor was used to analyze each outcome measure separately. The sphericity assumption was checked with the Mauchly test. In addition to examining statistical significance, calculation of mean differences and 95% CIs between each follow-up point and pretreatment data were performed (independent-samples t tests). The level of significance was set at P.05 for all comparisons.

All analyses were performed primarily according to the "intention-to-treat" (ITT) principle, with all subjects randomly assigned for intervention analyzed in their assigned groups.53,54 Friedman et al,54 however, also suggest that, when withdrawals are inevitable, both a per-protocol analysis and an ITT analysis should be performed; if both types of analysis concur, the result can be accepted with more confidence. A per-protocol analysis was performed alongside the ITT, using only data from subjects who provided follow-ups on both occasions (n38). Missing data for ITT analyses were handled with a relatively conservative approach by inserting group means in the place of missing values. Statistical analyses were performed using SPSS software, version 9.0.

RESULT

Thirty-six articles fulfilled our inclusion criteria.. Studies are listed in alphabetical order according to the name of the first author.

Quality of data:

The overall quality was generally good, but the following concerns are noteworthy: 1) In 42% (13/31) of the relevant articles, comparison of responders and nonresponders was missing. 2) The exact anatomical demarcation of LBP was not defined in 33% (12/36) of the studies. 3) In 8% (3/36) of the studies, data had not been collected in the preferred manner, i.e. sickleave data from administrative sources and symptom data from interviews or questionnaires. All other criteria were fulfilled,and no studies scored below 67%. It was therefore decided not to exclude any of the studies on basis of the quality assessment.

Number and type of studies:

The 36 included studies were published from 1981 to 1999 (October). Only 4 studies were published in the 1980s [1,2,27,37]. Six studies were RCTs [6,16,17,25,34,35], five were retrospective observational studies [2,19,20,21,46], and the remaining 25 were prospective observational studies. No difference in outcome was noted between these three types of design.

Study populations:

The majority of studies had a population size between 100 and 500 with a range of 62 [32] to 89,190 [20].

Study populations were drawn from several sources: the army [10], schools[4,21,29,32], the general population [28,30], workers receiving compensation [1,2,20,25,27,35,37] and clinical populations [5-9,12,13,16,17,19,22-24,31,33,34,36,39,40,44-46].

Description of LBP:

The gluteal folds were commonly defined as the lower border in the definition ofLBP [16,17,22-24,28,31,36,45] whereas the upper border varied from the scapula [45] to the first lumbar vertebra [28]. In several studies the only description provided was ”back pain” or ”low back pain”. Patients withradiating pain were specifically excluded in only one study [17]. In 14 studies [2,5,6,16,20- 23,27,34,35,37,39,46] both patients with and without leg pain were included and in the remaining 21 studies there was no mention of radiating pain at all.

table are reference numbers

Definition of LBP \ Length of follow-up	1 week	2 wks	1 mths	2 mths	3 mths	4 mths	6 mths	8 mths	1 year	18 mths	22 mths	2 years	3 years	4 years	≥5 years
Back pain			12,13, 40				7				7	12,13			
Low back pain	8		8,33		8		33		4,8,29,33			4,8	4,34	4	4,25, 30
Pain bt. scapulae and gluteal folds					45				45						
Pain bt. T12 and gluteal folds		17	17						17						
Pain bt. T12 and gluteal folds or radiating there from		16	16,22, 23,24	22,23, 24	22,23, 24		22,23, 24		16,22, 23,24						
Pain bt. L1 and gluteal folds							28		28	28					
LBP with/without radiation		6	20,34	20	6,20, 34	39	20,35	39	6,20, 34,35,39			46			
Back and/or leg/sciatic pain			5		5				5,27, 37			37			
Any back related problems															10
Pain bt. 12th rib and gluteal folds	36				36				31,36						
Generalized pain including the lower back	9				9				9						
Pain below T6							44		44						
Pain or discomfort in the lower part of the spine															21
MS complaints in the lumbar or lumbosacral region									1			1	1		
Pain, ache, stiffness or fatigue in the lower back w/wo radiation		2	2				2								
Back, hip and/or leg pain									19						

Changes With Exercise

For all self-report measures used (pain, disability, and all pain belief scales), the interaction of time with exercise class participation were not significant(P .05), thus indicating that both groups had achieved similar change over time (Tab. 2). The RMDQ data just failed to reach statistical significance when all 3 time points were analyzed together with an analysis of covariance (ANCOVA) (P.05, Tab. 2). When the 2 follow-up time points were analyzed separately and for the RMDQ only,

there was a statistically significant between-group difference immediately following exercise (mean difference2.55, P.027) in favor of the general exercise– only group, but this difference was no longer present at the 3-month follow-up. Both groups improved immediately following intervention (P.001), and these improvements were maintained 3 months later for all outcome measures apart from the PLC pain control subscale, which remained unchanged (Tab. 3). For all outcome measures, results were the same with both types of a lyses (ITT and per protocol). Only the results of the ITT analyses, therefore, are presented (Tabs. 2 and 3). The VAS B data were adjusted for the differences in baseline using an ANCOVA.

DISCUSSION

One of the strengths of our study is the age of the cohorts. Participants in the Ebeltoft study belong to the age group, where chronicity could be expected to be initiated, and is therefore an ideal age to study the natural course. Our participants from The Danish Twin Register are young and therefore relatively unaffected by the influences of adult life. The young twin cohort is also large enough to study comorbidity. Further, it requires large data bases to do the agestratified biometric modeling, and The Danish Twin Register is one of the few twin data bases world-wide where this is possible (39).

The use of questionnaires designed to investigate several aspects of life in the general population (this was the case for both The Ebeltoft Project and The Twin Register) has the advantage, that the population is not selfselected in terms of LBP. Previous work has shown that respondents to LBP surveys tend todiffer from non-responders in their experience of LBP (56,57). However, participants in health surveys generally may be self-selected by their interest in health-related questions and characterized by a lower threshold for the recognition of health problems, including LBP (7). This might have been the case in the Ebeltoft study, where women were somewhat over-represented in the study population compared to the target population and the prevalence of LBP was higher among women than among men. Fortunately, such a possible over-reporting is unlikely to affect the analysis of transition patterns between LBP-groups, but should be considered, when interpreting prevalence data. In the twin study, the bias resulting from such a self-selection process is probably negligible due to the high response rate.

In the literature many types of morbidity are linked to low birth weight (54), however this was not the case for LBP in our population. No previous studies about the relationship between LBP and birth weight or other birth factors were found. Since we did not find any associations with other birth factors either, a possible predisposition for LBP cannot be detected at birth in our data.

There seems to be a significant genetic component in the liability to develop LBP, which is most pronounced in the younger years, and with a non-additive genetic component in the adult population. Such a genetic component has previously been demonstrated for degenerative disc disease (67-69), for sciatica (70), for LBP of functional importance for work (26), and for self-reported non-specific LBP in the older cohort of The Danish Twin Register (71). The increasing influence of non-shared environment with age was also indicated in other studies (52), but to our knowledge, biometric modeling of the liability to LBP has not been attempted previously and thus the existence of nonadditive components in the liability to LBP has so far been undetected. Path analysis has been done for the liability of degenerative disc disease. An AE model, including additive genetic effects and individual environment,while ignoring dominant genetic effects and shared environment, was found to have the best fit. However, the analyses were not stratified for age, so a possible dominant effect in certain age groups might have beenover looked.

According to some authors, all patients with LBP may benefit from spinal stabilization exercise retraining on the premise that deconditioning of trunk muscles leads to instability symptoms,16–19 without any definitive proof from a relevant RCT yet. To test for this, we recruited subjects with nonspecific LBP. However, our findings tend to suggest that general trunk muscle exercises alone, without the addition of stabilization exercises, reduce patient self-reported disability more effectively immediately after the end of a 2-month exercise period. A statistically significant difference was observed between the 2 groups for the reduction in RMDQ scores (mean difference2.55, P.027) in favor of the general exercise– only group for the RMDQ data acquired immediately posttreatment. Both groups made a clinically significant improvement based on a 4-point withingroup change56; however, the improvement in the stabilization–enhanced exercise group was suboptimal compared with the general exercise– only group for the mmediatepostexercise comparison. According to previous research, a 2.5-point between-group difference in RMDQ scores can be considered as minimally important51; therefore, the null hypothesis for our study can be rejected based on this result. However, for all of the remaining outcome measures, no between-group differences could be detected either immediately postexercise or 3 months later. The difference in the RMDQ scores also was no longer present at the 3-month follow-up.

Scores by Group Over Time and *P* Values for the Interaction Effect[a]

	Pretreatment[b]				8 Weeks				20 Weeks				
	Stabilization–Enhanced General Exercise Group (n=29)		General Exercise–Only Group (n=26)		Stabilization–Enhanced General Exercise Group (n=29)		General Exercise–Only Group (n=26)		Stabilization–Enhanced General Exercise Group (n=29)		General Exercise–Only Group (n=26)		
	$\bar{X}$	SD	$\bar{X}$	SD	$\bar{X}$	SD	$\bar{X}$	SD	$\bar{X}$	SD	$\bar{X}$	SD	*P*
Pain scale													
SF-MPQ, sensory descriptors	12.2	4.0	12.9	5.2	7.9	4.1	7.7	5.2	6.4	4.8	8.3	5.2	.29[c]
SF-MPQ, affective descriptors	3.5	2.9	3.5	2.8	1.7	1.6	1.1	1.3	1.3	1.9	1.9	2.0	.18[c]
SF-MPQ, total score	15.7	5.4	16.3	6.4	9.6	5.2	8.8	5.9	7.7	6.4	10.2	6.3	.15[c]
VAS B (pain in past week)	26.9	20.6	40.2	24.6	12.3	13.7	21.3	17.3	15.8	15.3	17.8	14.2	.30[d]
VAS C (pain in past month)	49.9	26.4	55.9	25.5	22.3	18.3	27.8	15.6	23.1	18.8	28.8	16.9	.98[c]
Disability													
RMDQ	9.2	4.6	11.3	5.2	5.1	4.0	4.7	3.5	4.5	3.8	5.2	3.5	.05[c]
Pain beliefs													
Fear of movement (TSK)	37.6	6.3	40.5	8.9	33.7	6.5	35.1	7.1	31.5	6.1	32.9	5.3	.57[c]
PSEQ	42.0	12.3	37.3	11.1	49.2	8.6	48.1	7.7	51.2	8.3	48.9	9.4	.38[c]
PLC, pain control	12.4	4.5	11.2	6.0	12.4	4.3	11.3	5.0	10.9	3.6	9.9	4.1	.99[c]
PLC, pain responsibility	8.4	1.9	8.0	2.4	9.4	1.9	9.3	2.2	9.7	1.9	10.2	1.9	.23[c]

[a] SF-MPQ=Short-Form McGill Pain Questionnaire, VAS=visual analog scale, RMDQ=Roland-Morris Disability Questionnaire, TSK=Tampa Scale of Kinesiophobia, PSEQ=Pain Self-Efficacy Questionnaire, PLC=Pain Locus of Control Scale.

[b] Independent-samples *t* test showed no differences at baseline between the 2 groups for all outcome measures ($P>.05$) apart from VAS B ($P=.034$).

[c] 2 × 3 (exercise group × time) analysis of variance.

[d] Adjusted for baseline, 2 × 3 analysis of covariance.

	Stabilization–Enhanced General Exercise Group (n=29)		General Exercise–Only Group (n=26)		Between-Group Mean Difference	95% CI
	$\bar{X}$	SD	$\bar{X}$	SD		
Pain scale						
MPQ, sensory descriptors[b]						
8 wk–pretreatment	−4.25	4.63	−5.21	5.48	0.95[c]	−1.78 to 3.68
20 wk–pretreatment	−5.79	5.05	−4.63	6.00	−1.16[c]	−4.15 to 1.82
MPQ, affective descriptors[b]						
8 wk–pretreatment	−1.81	2.87	−2.32	2.34	0.51[c]	−0.91 to 1.94
20 wk–pretreatment	−2.23	3.30	−1.52	2.65	−0.71[c]	−2.34 to 0.92
MPQ, total score[b]						
8 wk–pretreatment	−6.06	6.44	−7.49	6.43	1.42[c]	−2.06 to 4.91
20 wk–pretreatment	−8.02	7.39	−6.11	7.30	−1.91[c]	−5.89 to 2.07
VAS B (pain in past week)[b,d]						
8 wk–pretreatment	−18.18	18.80	−14.92	16.52	−3.26[c]	−10.15 to 3.63
20 wk–pretreatment	−15.16	19.10	−17.78	19.70	2.62[c]	−4.58 to 9.82
VAS C (pain in past month)[b]						
8 wk–pretreatment	−27.57	29.96	−28.16	26.64	0.58[c]	−14.82 to 15.99
20 wk–pretreatment	−26.82	27.23	−27.10	27.14	0.28[c]	−14.45 to 15.00
Disability						
RMDQ[b]						
8 wk–pretreatment	−4.05	3.26	−6.60	4.97	2.55[e]	0.30 to 4.81
20 wk–pretreatment	−4.65	3.26	−6.03	4.98	1.38[c]	−0.87 to 3.64
Pain beliefs						
Fear of movement (TSK)[b]						
8 wk–pretreatment	−3.95	5.11	−5.40	6.51	1.46[c]	−1.69 to 4.61
20 wk–pretreatment	−6.13	6.57	−7.62	7.09	1.49[c]	−2.21 to 5.18
PSEQ[b]						
8 wk–pretreatment	7.17	11.41	10.75	11.22	−3.58[c]	−9.71 to 2.55
20 wk–pretreatment	9.19	11.06	11.53	10.97	−2.34[c]	−8.31 to 3.62
PLC, pain control[f]						
8 wk–pretreatment	0.04	5.05	0.09	5.96	−0.05[c]	−3.05 to 2.96
20 wk–pretreatment	−1.43	5.24	−1.26	5.76	−0.17[c]	−3.17 to 2.84
PLC, pain responsibility[b]						
8 wk–pretreatment	0.97	2.06	1.33	2.09	−0.36[c]	−1.50 to 0.77
20 wk–pretreatment	1.26	2.26	2.24	2.13	−0.97[c]	−2.18 to 0.23

The greater improvement in the general exercise– only group may signify that perhaps specific muscle stabilization retraining is more relevant to patients with either gross spinal instability symptoms 12 or pronounced side-to-side differences in the size of the multifidus muscle 11 than to our subjects, who did not present any signs and symptoms of clinical instability as described in the literature. 32,57 The patients in the study by O'Sullivan et al12 had radiological confirmation of an unstable segment related to the pain distribution, and also the patients in the study by Hides et al11 showed a good correlation between the level of side-to-side multifidus muscle CSA imbalance and the level of their pain.

The mode of action of stabilization retraining still remains unclear, because it has not been shown to be capable of mechanically containing an unstable segment, even upon improvement of muscle activation. No direct long-term effect of stabilization exercises on the status of the local stabilizing muscles has been demonstrated. Hides et al21 demonstrated less LBP symptom recurrence 3 years after treatment but did not verify the role of CSA, which was measured only in the initial study11 and not the follow-up.21 Similarly, no long-term improvement in the activation of the local stabilizing muscles has been presented. Thus, these studies suggest only a possible role for "stabilization" and illustrate the need for more comprehensive long-term assessments.

From a methodological point of view, the frequency and duration of the studied interventions (2–5 times per week for 8 weeks) were deemed appropriate to produce demonstrable benefits, based on previous studies of similar or less exercise duration.9,48,59,60 Because increasing doses of low back active exercises have been associated with an increase in reported benefits,61 we attempted to avoid confounding our results due to this factor by balancing the exercise dosage between the groups, based on prior literature on the loading imposed on the trunk muscles with each type of exercise. Exercises were administered in a progressive manner for both groups, and classes were supplemented with exercise leaflets to maintain motivation. The relatively high level of adherence both during classes and at home confirms patient motivation to complete the exercise program. The treating physical therapist had extensive expertise in stabilization exercise intervention delivery through attendance of specialized seminars on the t ic and its subsequent application. However, correct contraction of the stabilizing muscles could not be achieved in all subjects in the stabilization–enhanced exercise group until 2 to 3 sessions had passed, and subjects had to be constantly corrected by the treating physical therapist each time new exercises were introduced, similar to the study by O'Sullivan et al.12 However, the

subjects in the general exercise– only group could perform the exercises correctly by following the leaflets provided, with minimal instruction required from the physical therapist.

A limitation to our study was that, apart from the clinical physical therapist palpating the transverses abdominis and multifidus muscle contraction in the subjects in the stabilization–enhanced exercise group, there was no other means of verifying whether these muscles were recruited appropriately. However, due to our intention to monitor the effect of stabilization exercises delivered under pragmatic, clinical conditions used in everyday practice, the use of sophisticated devices such as electromyographic biofeedback units or real-time ultrasound scanners, as advised by some authors,[11,62] was avoided. Positive effects of stabilization exercises also have been reported by O'Sullivan et al,[12] who used less sophisticated feedback techniques such as the facilitation techniques used in our study.

Two subjects dropped out from the stabilization– enhanced exercise group due to complaints of pain. Their increase in pain, however, could not be attributed with certainty to the exercises, because pain did not begin during exercise performance time. The percentage of subjects from this group who developed pain(6.9%) was not alarmingly high enough to suspect that the increase in pain was due to the exercises administered, nor has such an incident been reported in any similar previous study.

An important finding of our study was that, although exercise was prescribed under a biomechanical framework (to train the muscles surrounding the spine in order to protect it) and we did not adopt strict psychological principles of exercise delivery, within-group improvement in 3 of the 4 psychological outcome measures was documented for both groups. Namely, participants' ideas about fear of movement/injury or reinjury, self-confidence in the performance of activities despite the pain, and the PLC pain responsibility subscale (patients' degree of responsibility in controlling their pain levels) registered improvements on both posttreatment follow-ups. However, no appreciable change was noted in one other outcome measure (PLC pain control subscale). Similar multidimensional changes have been reported by several researchers who adopted primarily a "physiological type" of approach to intervention63 as well as those who used psychological approaches in conjunction with exercise.

The information provided in The Back Book may have resulted in a positive shift in patient beliefs regarding LBP, as previously demonstrated.37 In our opinion, however, the shift in beliefs also was reinforced by patient problem-solving interactions with the treating physical therapist on how to perform the exercises and by the fact that some pain during exercise was to be considered normal may have led to increased patient adherence, allowing the subjects to participate in a number of exercise routines. Patients' exposure to potentially back-straining movements, such as spinal flexion, has been shown to decrease the avoidance of such activities and perhaps patient levels of disability in general. Exercises were delivered in a progressive method, from easier to more difficult for both programs, to progressively introduce patients to more demanding exercises, according to graded exposure principles. Due to the design of our study, it was not clear whether all of these factors resulted in the improvement of patient beliefs regarding LBP.

Several studies have shown that patients who are less fearful and more optimistic about their abilities to function despite LBP report less pain behavior and disability and demonstrate fewer functional limitations compared with patients who have increased fear and decreased pain self-efficacy beliefs. The reduction noted in some of the psychological factors measured also may have been related to decreased pain and disability report. However, due to the nature of our trial and the very few time points when the data were collected, a clear order of the change in the variables measured (pain, disability, and patient beliefs) could not be established. This can be a future avenue for exploration.

CONCLUSION

LBP is a common and usually benign disorder, which is characterized by exacerbations and remissions. However, about 10% of the adult Danish population develops chronic or frequently recurring LBP over several years. Therefore, an effort must be made to identify a high-risk population early in order to implement selective primary preventive measures.

LBP is associated with the presence of other disorders in both adults and adolescents, thus, these associations cannot be explained merely by external factors in adult life, but probably have, at least partly, a common origin based on an inherent frailty. We could not predict future LBP by birth factors, although birth weight often is associated with other diseases, but the liability to develop LBP seems to have a genetic component. It is possible that it is the individuals with a strong genetic disposition for LBP, who develop chronic/recurrent LBP in adulthood. If such an underlying genetic predisposition exists, the recurring pattern of LBP is a logical consequence. This predisposition might also indicate a general frailty making individuals more prone to other disorders as well, which would explain the associations between LBP and other disorders.

Follow-up studies of young populations are needed to confirm the existence of a high-risk group.

ACKNOWLEDGMENTS

We would like to thank all our well wishers who help us in this study and give us support and courage do this.

FUNDING: There was no funding source for this study.

CONFLICT OF INTEREST: Nil

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