Research Report

A Pilot Study of the Effects of High-Intensity Aerobic Exercise Versus Passive Interventions on Pain, Disability, Psychological Strain, and Serum Cortisol Concentrations in People With Chronic Low Back Pain

Dimitris Chatzitheodorou, Chris Kabitsis, Paraskevi Malliou, Vassilis Mougios

Background and Purpose

Given the complex nature of chronic pain, the effects of high-intensity aerobic exercise on pain, disability, psychological strain, and serum cortisol concentrations in people with chronic low back pain were investigated.

Subjects

Twenty subjects receiving primary health care were randomly allocated into exercise and control groups.

Methods

Subjects in the exercise group received a 12-week, high-intensity aerobic exercise program. Subjects in the control group received 12 weeks of passive modalities without any form of physical activity.

Results

Data analysis identified reductions in pain (41%, $t_{10}=8.51$, $P<.001$), disability (31%, $t_{10}=7.32$, $P<.001$), and psychological strain (35%, $t_{10}=7.09$, $P<.001$) in subjects in the exercise group and no changes in subjects in the control group. High-intensity exercise failed to influence serum cortisol concentrations.

Discussion and Conclusion

Regular high-intensity aerobic exercise alleviated pain, disability, and psychological strain in subjects with chronic low back pain but did not improve serum cortisol concentrations.

D Chatzitheodorou, PT, MSc, is Laboratory Instructor, Department of Physical Therapy, Technological Educational Institute of Thessaloniki, PO Box 141, 57400 Sindos, Greece. Address all correspondence to Mr Chatzitheodorou at: dim_chatzi@yahoo.com.

C Kabitsis, PhD, is Professor, Department of Physical Education and Sport Science at Serres, Aristotle University of Thessaloniki, Thessaloniki, Greece.

P Malliou, PhD, is Assistant Professor, Department of Physical Education and Sport Science, Demokretus University of Thrace, Komotini, Greece.

V Mougios, PhD, is Associate Professor, Department of Physical Education and Sport Science, Aristotle University of Thessaloniki.

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For The Bottom Line:
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Musculoskeletal pain is extremely common in the general population. A considerable number of patients develop chronic symptoms that do not respond well to conventional therapeutic approaches.[1,2] Chronic pain, defined by Merksey and Bogduk[3] as pain that has passed the span of 3 months, has a multifactorial etiology.[2,4] As far as the somatic aspect of pathology is concerned, the processing of the noxious stimuli does not relate proportionately to the sensory perception and the physical findings.[4]

The nervous and endocrine systems collaborate in integrating the perception of pain. This process modifies the physiology and effectiveness of the 2 systems.[5-7] Almost all endocrine actions are controlled by higher brain centers and, in fact, most hormones affect brain function.[5,7] Pain as a reflex sensory response is accompanied by an altered neuroendocrine response mediated by the hypothalamic-pituitary-adrenal (HPA) axis. The emotional aspect of the pain response is encoded by corticolimbic systems (including the HPA axis) to integrate the relationship among pain, memory, and mood. The starting point of the HPA axis, the hypothalamus, is controlled by unknown excitation mechanisms of first-order brain nuclei, which receive input through the final effectors of the axis, glucocorticoids.[5,7] Glucocorticoids play a key regulatory role in the basal activity of the HPA axis.[5,7]

Both clinical and preclinical neuroendocrine studies have strongly suggested that dysregulation of the HPA axis and cortisol play a causal role in the development and course of chronic pain.[5,7-12] In addition to its effects on the capacity of the body to heal wounds, cortisol acts on human behavior: mood, appetite, sleep, cognitive level, and sensory input perception.[13] Therefore, it may influence the dimensions of pain perception and the mechanisms of its integration,[14] thus constituting an index of endocrine system activation for pain integration and modulation.[9,11]

Irregularities in HPA axis function have been associated with pain syndromes that show little or no evidence of pathology in the painful tissue, such as fibromyalgia and chronic low back pain.[8,12,15] Abnormalities in the HPA axis are expressed in 2 forms. One is characterized by chronic hyperactivation of the stress system; cortisol secretion is increased, and the plasma corticotropin response to exogenous corticotropin-releasing hormone is decreased.[11,16,17] The other form is characterized by hypoactivation of the stress system; in this form, chronically reduced hormonal secretion may result in pathological hypoarousal.[9,11,18]

Both forms share a hyporeactive HPA axis, which develops after prolonged periods of stress or exposure to painful stimuli, together with hyperactive HPA axis and excessive glucocorticoid release.[11] These alterations are determined by the reduced biosynthesis or release of cortisol from the adrenal glands; this process is accompanied by subsequent stimulation of the respective target receptors, hypersecretion of a secretagogue (with a subsequent down-regulation of the respective target receptors), enhanced sensitivity to the negative feedback of glucocorticoids, decreased availability of free cortisol, and reduced effects of cortisol on the target tissue.[9,11,19] As a result, different patient subgroups with a single disorder are characterized by different patterns of HPA axis dysregulation.[9,11]

In people who are healthy, prolonged physical exercise of moderate to high intensity leads to transient increases in serum cortisol concentrations and then a return to baseline levels.[20] The increases may be attributable to either augmented secretion or decreased metabolic removal. Serum cortisol concentrations rise progressively, and a workload of about 60% maximal aerobic capacity seems to be the critical level above which a rise in cortisol concentrations occurs,[20] although the threshold may depend on exercise duration and interindividual differences.[21]

The majority of the relevant studies underline the importance of psychological factors in glucocorticoid secretion as related to exercise.[21,22] In people who are healthy and in the absence of emotional stress, exercise does not induce substantial short-term changes in cortisol concentrations, although the data are not uniform across studies.[20] On the other hand, pain states with chronicity or relapse are correlated with persistent HPA axis abnormalities, although these abnormalities are not fully explained by the accompanying psychological stress.[23] Additionally, not all patients with chronic pain have abnormal cortisol concentrations. Findings have been inconsistent, and issues of measurement validity and unknown underlying mechanisms have been raised.[24]

The increased or decreased corticosteroid concentrations in chronic pain conditions may be an important contributor to the increased psychological strain and may account for the variability in the clinical presentation and in the therapeutic response to exercise. When cortisol was measured in relation to self-expressed mood, it was correlated positively with the reported level of "alertness."[25] Infusion of pharmacological doses of cortisol, however, did not cause any significant changes in the measured affective states of people who are healthy.[25] Nevertheless, it should be noted that the effects of cortisol on mood are probably more long-lasting and dose-

dependent than the effects that are sometimes seen in patients receiving prolonged treatment with systemic steroids; short-term infusions may not be able to mimic the action of endogenous cortisol in the brain.[25]

The negative correlation between physical activity and morbidity is well documented.[26] However, there is no sufficient evidence for the effectiveness of exercise in alleviating chronic pain.[2,27,28] The implementation of exercise in studies of chronic low back pain has produced controversial results. Until 2005, there had been 4 systematic reviews on the topic. The authors of the first review evaluated 7 studies, lamented the poor quality of research on the topic, and concluded that exercise therapy has not been more efficacious than other treatment modalities.[29] The authors of the second review evaluated 6 studies and concluded that those with high methodological scores reported no effects of exercise on chronic pain, whereas studies with low scores showed positive effects.[30]

In 2000, van Tulder et al[27] published a Cochrane review of the literature assessing the effectiveness of exercise therapy in relation to pain intensity, functional status, overall improvement, and return to work. They concluded that exercise therapy may be helpful in alleviating chronic low back pain. Hayden et al[28] published a meta-analysis of randomized controlled trials evaluating exercise therapy for adult nonspecific low back pain and measuring pain, function, return to work or absenteeism, and global improvement outcomes. They concluded that exercise therapy seems to be slightly effective at decreasing pain and improving function.

In a clinical update on the management of chronic low back pain, Bogduk[31] concluded that exercise therapy is more effective than usual care by a general practitioner and better than back schools, but evidence is conflicting on whether exercise therapy is more effective than an inactive, sham treatment.

In clinical trials that evaluated the effectiveness of exercise, the high-intensity component of exercise referred to strengthening exercises,[32,33] whereas aerobic forms of exercise were either of moderate intensity or general fitness programs.[34–41] The role of high-intensity aerobic exercise in confronting chronic musculoskeletal conditions has not been studied. Therefore, the purpose of the present study was to investigate the effects of this type of exercise on pain, disability, anxiety or depression, and serum cortisol concentrations in people with chronic musculoskeletal lumbar pain. We hypothesized that exercise would reduce all of these parameters.

Method

Subjects

After the consecutive referral of 25 patients with chronic low back pain, we assessed the eligibility of the patients for the trial. The inclusion criterion was low back pain lasting over 6 months; the symptoms had to be present for over half of this period. Exclusion criteria were medical history of serious injury, spinal surgery or malignant pathology, and obesity (a body mass index of over 30 kg/m^2) because of its association with hypercortisolism[13] and for safety reasons (possible cardiovascular complications and injury during high-intensity exercise).

One patient was excluded because of obesity. After discussion of general aspects of the trial and time requirements but no information concerning group assignment, interventions, and prognostic comments, 4 patients declined to participate because of personal restrictions (difficulties in transportation from their homes to the exercise establishment). These patients did not differ in age, sex, or duration of symptoms from the patients who agreed to participate. The remaining 20 subjects were recruited for the study over a period of 8 months. They had chronic low back pain, presenting disk disruption (n=15), lumbar spondylosis (n=3), or zygapophyseal joint pain (n=2), diagnosed through clinical and radiographic examinations by an orthopedic surgeon or a neurosurgeon at primary health care or 2 orthopedic outpatient hospital departments in Thessaloniki, Greece. The subjects were not exercising regularly.

After signing an informed consent form, eligible subjects were recruited for the study and were randomly assigned to an exercise group or to a control group by block in order to ensure equal group sizes. To assign by block, we generated a random-numbers table with 70 columns and 200 lines, and we randomly selected 2 columns. Looking at the last 2 digits of the numbers in each column and proceeding from left to right and from top to bottom, we located numbers 01 to 20. The numbers in the first column corresponded to subjects in the exercise group, and the numbers in the second column corresponded to subjects in the control group. As eligible subjects were enrolled in the trial, they were assigned to the group that contained their number of recruitment. Eligibility assessment and administration of interventions were implemented by the first author, and preparation of the allocation system and assignment to groups were performed by the third author. The characteristics of the 2 groups are shown in Table 1.

Procedure

Meetings were arranged for all subjects to complete the McGill Pain Questionnaire (MPQ) for pain evaluation,[42,43] the Roland-Morris Disability Questionnaire (RMDQ) for dis-

ability evaluation,[44,45] and the Hospital Anxiety and Depression Scale (HADS) for psychological strain evaluation.[46,47] These questionnaires are described in detail below. The meetings took place in a private physical therapy clinic in the absence of other patients or personnel. Next, a meeting was set for blood sample collection for the measurement of cortisol. Five milliliters of blood was collected from an upper arm vein in the morning (8 AM–12 noon) in the fasting state, without any morning exercise and at least 3 hours after waking. The blood sample was allowed to coagulate and was centrifuged at 1,500*g* for 10 minutes. Serum was removed and stored at −20°C until it was analyzed. After this step, the subjects started following the protocol of the group to which they belonged.

The subjects in the exercise group followed a 12-week exercise training program supervised by a physical therapist (the first author) in the presence of a physician. The resting heart rate of the subjects was measured for 5 consecutive days, and the mean value was calculated. On the basis of this value and on the age-predicted maximal heart rate (220−age), we calculated individualized heart rate reserves and heart rates during exercise through the use of the Karvonen formula.[48]

A 15-minute warm-up, consisting of calisthenic exercises of the arms and legs followed by mild stretching exercises of the legs, preceded each aerobic exercise session. Aerobic exercise consisted of running on a horizontal treadmill at 60% to 85% the heart rate reserve for 30 to 50 minutes 3 times per week for 12 weeks. Taking into account the reduced adaptive ability that subjects might have because of their symptoms, a 3-week period of adjustment was given; during this period, exercise intensity was increased from 60% to 85% and exercise duration was increased from 30 to 50 minutes, both in a steady, almost linear, fashion. Thereafter, intensity remained at 85% the heart rate reserve and duration remained at 50 minutes. That intensity was achieved by running at a velocity of 7 to 8 km/h. Each exercise session ended with a 3-minute cool-down consisting of walking on the treadmill at a decelerating pace. All subjects completed the exercise program successfully. Adherence to the exercise program (calculated as the ratio of sessions attended to sessions scheduled) was 98%.

Table 1.
Characteristics of Subjects

Variable	Exercise Group (n=10)	Control Group (n=10)
Age, y		
$\bar{X}$	42.4	41.5
SD	12.7	12.9
Range	25-65	25-64
Sex, no. of subjects		
Male	5	6
Female	5	4
Diagnosis, no. of subjects		
Disk disruption	8	7
Lumbar spondylosis	1	2
Facet joint pain	1	1
Out of work	2	1

The subjects in the control group received 12 weeks of passive modalities without any form of physical activity. The specific modalities that were administered included shortwave diathermy (10 minutes, continuous pulse at moderate to high intensity according to each subject's tolerance), ultrasound (5 minutes, continuous pulse, 45 W/cm^2), laser (10 minutes, He-Ne, λ=632.8 nm), and DF (difase fixe), CP (module en courtes periodes), and LP (module en longues periodes) current forms of electrotherapy (4 minutes each at moderate to high intensity according to each subject's tolerance) applied to the area of the lumbar spine. Each session lasted approximately 45 minutes. The subjects remained prone on the physical therapy couch, and the intervention was administered by the physical therapist. Adherence was 96%.

After the end of each subject's program, he or she again completed the questionnaires mentioned above and under the same circumstances. Similarly, a second blood sample was collected in the same way as the first sample, within the hour during which the first blood sample was collected, 3 days after the completion of the program. Because we were interested in the long-term responses of cortisol to the interventions, measurements were aimed at determining the basal levels of the hormone before and after the interventions. Thus, single blood samplings at these time points were selected. The questionnaires were scored, and the serum samples were analyzed for cortisol concentrations.

Cortisol concentrations were determined with an established enzyme immunoassay and a kit from DRG.*

* DRG, Frauenbergstr. 18, Marburg, Germany.

Table 2.
Outcome Measures for Subjects in Exercise and Control Groups at Baseline and 12 Weeks

Variable	Score			
	Exercise Group (n=10)		Control Group (n=10)	
	$\bar{X}$	SD	$\bar{X}$	SD
Pain (McGill Pain Questionnaire)				
Baseline	53.9	10.4	53.0	11.7
12 wk	32.3[a]	7.9	53.3	10.0
Disability (Roland-Morris Disability Questionnaire)				
Baseline	13.8	2.4	14.4	2.8
12 wk	9.6[a]	2.6	14.3	3.6
Anxiety or depression (Hospital Anxiety and Depression Scale)				
Baseline	24.8	5.0	22.6	4.1
12 wk	16.2[a]	3.4	21.9	4.5
Cortisol concentrations (ng/mL)				
Baseline	184.1	56.8	164.4	51.4
12 wk	188.5	77.6	147.5	52.3

[a] Significantly different from baseline value ($P<.001$).

Measurements were obtained with an Anthos 2001 photometer.[†]

Questionnaires

The MPQ was designed to provide quantitative measures of clinical pain that could be treated statistically.[42] It was intended to provide a means of detecting differences among different methods of relieving pain, but it has also been used for diagnostic differentiation. It describes pain not solely in terms of intensity but additionally in terms of sensory, affective, and evaluative qualities. Each of the 78 description words is ranked within 1 of 20 groups in order of increasing intensity. The values of the words chosen, multiplied by the weight of the corresponding group, are added to produce a score ranging from 0 to 78.[42] With good test-retest reliability, construct validity, and responsiveness to change, the MPQ is a simple and self-completed questionnaire that provides a measure of the complexity of the pain experience.[43,47]

The RMDQ was chosen because it is reported frequently in the literature and its measurement properties are better than or equal to those of competing measures.[49] It is a self-administered questionnaire consisting of 24 items abstracted from the Sickness Impact Profile. The items are intended to represent a variety of activities with which people with low back pain may have difficulty. Test-retest reliabilities of .91 and .83 have been achieved.[44,50] Concurrent validity has been demonstrated to the extent that the RMDQ has been shown to correlate with Sickness Impact Profile scores ($r=.85$).[50] The ability of the RMDQ to detect valid change over time has been expressed with receiver operating characteristic curves.[51]

The HADS is designed to detect the presence and severity of a relatively mild mood disorder likely to be found in nonpsychiatric hospital outpatients. It is intended both as a screening device and to chart progress over time.[47] The scale consists of 14 items, 7 of which relate to depression and 7 of which relate to anxiety. Each item provides 4 response categories in terms of frequency or severity. The wording of questions and response categories is positive (ie, 0–3) for 6 items and negative for the remaining 8 items (ie, 3–0). Item scores for each subscale are summed and indicate noncases (7 or less), doubtful cases (8–10), and definite cases (11 or more).[46] The scale is self-completed. There is less evidence of the reliability of HADS than of their validity. Satisfactory internal consistency was reported.[47] Subsequent evidence confirmed the validity of the HADS when it was tested against other measures of anxiety and depression.[46,47] There seems to be no evidence concerning the responsiveness of the scale to treatment effects, although it was recommended for use as a means of monitoring progress over time.[47]

Data Analysis

All data were entered into a computer, and statistical analysis was performed with the use of the Statistical Package for the Social Sciences, version 10.[‡] Descriptive statistics (means and standard deviations) were determined for all dependent variables for each group. For all statistical tests conducted, the alpha level was set at .05.

A chi-square test produced no significant differences between groups with respect to sex, and independent Student t tests produced no

[†] Anthos, Lagerhausstr. 507, Salzburg, Austria.

[‡] SPSS Inc, 233 S Wacker Dr, Chicago, IL 60606.

significant differences between groups with respect to age and duration of symptoms. Application of the Shapiro-Wilk and Levene tests revealed no violation of the assumptions of normal distribution and of homogeneity of variance, respectively.

Next, we examined whether the assumptions of homogeneity of covariance between groups and of correlation of the dependent variables for a multivariate analysis of variance (MANOVA) were not violated by using, respectively, the Box M test of equality of covariance matrices ($F_{20}=1.50$, $P=.028$) and the Bartlett test of sphericity (between subjects, $\chi^2_{20}=159.1$, $P<.001$; within subjects, $\chi^2_{20}=205.7$, $P<.001$). All of the assumptions for the parametric analysis and the MANOVA were not violated, except for the homogeneity of covariance; it seemed reasonable that this problem could be overcome because of expected multiple dependent variables.

Thus, a 2×2 MANOVA with a between-subjects factor (intervention) and a within-subjects factor (time) was used for the analysis of differences between groups in the dependent variables. Moreover, for the analysis of changes in each dependent variable within each group, paired *t* tests were conducted because of the identification of a statistically significant interaction between factors in the MANOVA.

Results

Descriptive statistics for all dependent variables are shown in Table 2. Baseline values for the outcome measures for subjects in both groups were as follows: the mean pain score was 53 or 54 on a 78-point scale, the mean disability score was 14 on a 24-point scale, the mean psychological strain score was 23 or 24 on a 42-point scale, and the mean cortisol concentrations were 164 to 184 ng/mL (normal: 30–230 ng/mL).[52]

Table 3.
Multivariate Analysis of Variance Results (Overall F Values)

Main Effect	Wilks Lambda F Value	*P*	Effect Size	Power
Intervention	1.48	.25	0.28	0.35
Time	25.25	<.001	0.87	1
Interaction	24.40	<.001	0.86	1

For the main effect of intervention, the MANOVA overall F value was not statistically significant; for the main effect of time, the F value was significant; and for the interaction of factors, the F value was significant (Tab. 3). Because of the identification of a significant interaction, the main effects on the individual variables could not be interpreted. Paired *t* test values for subjects in the exercise group were statistically significant for pain ($t_{10}=8.51$, $P<.001$), disability ($t_{10}=7.32$, $P<.001$), and anxiety or depression ($t_{10}=7.09$, $P<.001$) but not for cortisol concentrations. Correlation coefficients ranged from .63 to .74 for these tests.

Effect size values (calculated as the difference between means divided by the pooled standard deviations) for subjects in the exercise group were 2.34 for pain, 1.68 for disability, 2.01 for psychological strain, and 0.06 for cortisol concentrations. Effect size values for subjects in the control group were 0.03, 0.03, 0.16, and 0.32, respectively. The probability of a type II error for cortisol concentrations was .49. All subjects exhibited decreases in pain, disability, and anxiety or depression with exercise. No *t* test for subjects in the control group was statistically significant.

Discussion

The results confirmed the hypothesis that high-intensity aerobic exercise would reduce pain, disability, and psychological strain in subjects with chronic musculoskeletal spinal pain, although they did not confirm the hypothesis regarding cortisol concentrations. Data analysis indicated that the implementation of high-intensity aerobic exercise for 12 weeks, unlike passive modalities, reduced pain, disability, and anxiety or depression in subjects with chronic pain, although the reduction in disability did not reach clinical significance, as described below. In contrast, the exercise program failed to influence serum cortisol concentrations in the subjects. Considering the complicated nature of chronic pain, the reduction of pain by 41%, the recovery of a large part of lost functioning (31%), and the reduction of psychological strain by 35% within a period of 12 weeks may be clinically significant. It remains to be established whether the improved state of the subjects remained after the intervention.

There are no standards of minimal clinically significant changes against which the changes observed in the present study can be compared, except for the case of the RMDQ, for which a change of 5 points is considered important,[51,53,54] although for patients having chronic pain symptoms for well over 6 months smaller changes could be considered clinically significant. The decreases in subjects in the exercise group in the present study were 4.2 on average and 5 or higher for 4 of the 10 subjects. The average decrease in subjects in the control group was 0.1, with a maximum value of 1 for 5 of the 10 subjects.

The relative normalization of the clinical scenario with exercise was not accompanied by a favorable change in cortisol concentrations. This finding could be interpreted as a lack of influence of exercise on the regulatory mechanisms of the endocrine system in our subjects, although this conclusion is based on a single hormone measurement. The level of excitation of the HPA axis and consequently the cortisol concentrations in peripheral blood are not regulated by pain only. Exercise could induce changes in endocrine mechanisms and could have a strong influence on symptom presentation, but these effects might not be reflected in the blood cortisol concentrations because complex and multiple factors interact to induce many changes in HPA axis function and clinical presentation. In addition, the duration of the exercise program might have been too short to allow changes in cortisol concentrations to be recorded, given that symptoms were present for well over 6 months.

Exercise and the immediate postexercise recovery period are associated with an increased pain threshold and analgesia.[55] Glucocorticoids have been linked to increased thresholds to dental and other types of pain.[56–58] Some exercise-associated analgesia is a result of direct activation of pain-inhibiting brain substrates by type III and IV afferent nerves.[59] Thus, although there is evidence that increased secretion of β-endorphin by the pituitary gland and by the leukocytes and macrophages in injured tissues contributes to exercise-induced analgesia, it does not account for all observed increases in the pain threshold.[55]

The magnitude of the neurohormonal response to any given physical task is augmented by associated emotional stress.[60] Aerobically fit people who continue to exercise and maintain their aerobic fitness might release more endogenous opiates than their nonexercising, less fit counterparts during exercise. This scenario, in turn, would explain why they experience a sustained elevation of mood. It also seems reasonable to postulate that exercise modifies psychological stress reactivity. According to a proposed model, exercise reduces negative affective states by increasing the release of endogenous opiates, reducing HPA axis activation, and enhancing immunity.[60] In the present study, the finding of pain and psychological strain reduction in subjects in the exercise group compared with no change in subjects in the control group, in light of unaltered cortisol levels in the subjects in both groups, could favor the hypothesis of improvement attributable to endogenous opiate action triggered by exercise; however, this hypothesis needs to be properly tested in future studies.

Improvement in the clinical scenario on the basis of the parameters measured was uniform in all subjects in the exercise group. This finding might reflect an effect of high-intensity aerobic exercise on mechanisms of pain. Both the nervous and the endocrine systems are candidate mediators of this beneficial effect. The uniformity of the responses also might reflect the potential of subjects with chronic low back pain to respond to a particular type of exercise in a uniform pattern. This notion implies that the favorable response to exercise might be determined by a powerful central mechanism that diminishes the differences in pathology at the local level through combined effects of the nervous and endocrine systems on the musculoskeletal system. Aerobic exercise constitutes a distinct mode of intervention, because its physiological effects are not confined to musculoskeletal function. Aerobic exercise, particularly in its high-intensity form, acts on most body systems; therefore, it might be effective in changing clinical symptoms.

In subjects with chronic low back pain, the potential cause of changes from hypercortisolism to hypocortisolism could be related partially to a failure of the body's self-adjusting abilities through the down-regulation of specific receptors at different levels of the HPA axis, reduced biosynthesis or depletion of hormones at several levels of the axis, and increased sensitivity to glucocorticoids through negative feedback. Thus, different subject subgroups with a single disorder are characterized by different patterns of HPA axis dysregulation. Because cortisol alterations are not a specific correlate of chronic low back pain and because the mechanisms underlying the development of cortisol alterations are complex and heterogeneous between and within subjects, we introduced unselected chronic low back pain populations to produce higher external validity (potential generalizability). It is reasonable to remain skeptical about linking cortisol, pain, and exercise with a cause-and-effect relationship. The HPA axis function alone is very complex, and there are multiple mechanisms and determinants that still remain a matter of speculation. The basic limitation of the study (small sample size) and the preference of the design for external validity warrant some skepticism.

Drawing general conclusions about the effectiveness of exercise programs in chronic pain states by reviewing the literature is difficult. In studies that used general fitness programs containing moderate-intensity exercise, the outcomes for pain (evaluated on a pain scale) and disability (measured with the RMDQ, a global assessment questionnaire, the Oswestry Disability Questionnaire, the Sickness Impact Factor, and return to work) were better when exercise was included in 5 comparisons with alternative interventions

and were the same in 6 other comparisons.[34-41,61] In studies with high-intensity strengthening programs, exercise produced better results in general but not uniformly, with subgroups of subjects not responding in the same manner.[31-33] These studies used different exercises, durations, intensities, implementations, methods, and analyses. Unlike any other study of which we are aware, our study implemented high-intensity aerobic exercise which, in addition, was adapted to each subject's abilities and was supervised individually.

Conclusion

High-intensity aerobic exercise alleviated pain, disability, and psychological strain in subjects with chronic low back pain, although the decrease in disability is not considered clinically significant, whereas a program of conservative modalities was not effective. In contrast, both interventions failed to influence serum cortisol concentrations in the subjects. These results suggest a possibly important role of high-intensity aerobic physical activity in reducing the symptoms of chronic musculoskeletal conditions and regulating the disturbed equilibrium of physiological systems. Future studies need to establish the effectiveness of a particular type of exercise with larger sample sizes. There is also a need to evaluate more accurately the contribution of the endocrine system to improvements in people with chronic low back pain.

All authors provided concept/idea/research design. Mr Chatzitheodorou, Dr Kabitsis, and Dr Malliou provided writing and data analysis. Mr Chatzitheodorou and Dr Malliou provided data collection and facilities/equipment. Dr Kabitsis and Dr Malliou provided consultation (including review of manuscript before submission). The authors thank Professor Ronald Melzack for permission to use the McGill Pain Questionnaire and for providing all of the documentation for its use. They thank Savvas Mavromoustakos for constructive criticism, Aleka Michalaki for linguistic assistance, Anatoli Petridou for technical assistance, and Konstantinos Manavis and Dimitris Efthimiadis for providing access to subjects.

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