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ORIGINAL RESEARCH

Acute Effects of Core Stabilization Exercise on Beta-Endorphin and Cortisol Levels among patients with Chronic Non-Specific Low Back Pain - A Randomized Cross over Design

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4 **Abstract**
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7 **Objective:** Core stabilization exercise (CSE) is widely prescribed to treat chronic non-specific
8 low back pain (CLBP). However, the neuro-endogenous mechanism behind the pain relieving
9 effect by CSE is still unknown. The main objective of the study was to measure the levels of
10 plasma beta-endorphin (PB) and plasma cortisol (PC) under CSE, placebo and control conditions
11 in CLBP patients.
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20 **Methods:** Twenty-four subjects with CLBP participated in a randomized, placebo-controlled,
21 cross-over design study. There were 3 experimental exercise conditions; control condition
22 (positioning in crook lying and rest), placebo condition (passive cycling in crook lying using
23 automatic cycler), and CSE on a Pilates device tested with 48 hours interval between sessions by
24 concealed randomization. Blood sample was collected before and after the exercise conditions.
25 PB and PC were measured through enzyme-linked immunosorbent assay and
26 electrochemiluminescence in Cobas E411 auto analyzer.
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38 **Result:** PB level showed a significant difference before and after the CSE condition ($P<0.05$),
39 while no significant differences were noticed in control and placebo exercise conditions. Also,
40 the trend of elevation of PB under the CSE was significantly different when compared to the
41 placebo and control conditions ($P<0.01$). In contrast, the PC level remains unchanged in all the
42 three conditions.
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51 **Conclusion:** CSE potentially influences PB level but not PC level among CLBP patients. The
52 mechanism of action for pain relieving effect by CSE might be possibly related to an
53 endogenous opioid mechanism as part of its effects, and might not be involved with ‘stress
54 induced analgesia mechanism.
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4 **Introduction**
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7 Lumbopelvic core stability exercise training (LPST) is a common therapeutic
8 management in day to day practice for the low back pain patients. The LPST exercises recruit
9 and train certain specific muscles such as transverses abdominis and multifidus in order to
10 provide spinal stabilization.¹ Evidence from the systematic reviews suggests that the LPST is an
11 effective treatment for low back pain patients as it reduces pain and improves function.² The
12 mechanical and neuro-physiological effects of the LPST such as improved contractility of core
13 muscle, enhanced feed forward mechanism and improved lumbopelvic stability are accounted as
14 established effects for pain reduction among patients with CLBP.³⁻⁶ While the mechanical
15 effects of LPST are well documented, the biochemical effects of the LPST behind pain relief are
16 yet to be fully understood. Exercise can cause endogenous induced analgesia through release of
17 endogenous opioids.⁷ However, it is not clear whether the LPST can release the endogenous
18 opioids that may be behind the mechanism of pain relief among CLBP patients.
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38 Endogenous opioids have been found to have analgesic effect in a variety of chronic pain
39 syndromes including CLBP.⁸ Beta-endorphin (BE) is one such peptide released with
40 adrenocorticotrophic hormone from the anterior pituitary gland as a result of exercise stress.⁹ The
41 release of BE works on the endogenous opioid receptors and acts on the descending inhibitory
42 system that modulates pain at the spinal cord level.¹⁰ An appropriate metabolic and thermal
43 stress is required in any exercise for the release of BE.¹¹ Moreover, it is suggested that exercise
44 intensity and BE release are correlated in producing the opioid induced analgesic effect in human
45 beings after exercises.¹² However, it is not sure whether the LPST has enough physical stress to
46 induce BE release and to increase PB in CLBP.
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4 Evidence suggests that functional somatic symptoms like CLBP are often associated with
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6 physical and mental stress.^{13,14} Cortisol is a primary peripheral hormone from hypothalamic-
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8 pituitary adrenocortical activity that reflects the coping mechanism of body for stress response
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10 and pain adaptation.^{13,14} The cortisol is released by the adrenal cortex by stimulation of
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12 adrenocorticotrophic hormone at the anterior pituitary and corticotrophin releasing factor from
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14 the paraventricular nucleus of the hypothalamus.¹⁵ The mental and physical stress associated
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16 with an acute pain and anxiety causes an increased hypothalamus-pituitary-adrenal axis
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18 activation (HPA).¹³ Furthermore, the altered activity of HPA axis contributes to evolution of
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20 stressful characteristics of a clinical problem into chronic pain disorder.¹³ An increased
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22 concentration of the PC has been postulated to cause attenuation of pain perception in acute
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24 stress conditions.¹⁶ Also, the cortisol regulates sympathetic and opioid mechanisms related with
25
26 central pain processing.¹⁵ While altered level of the stress hormone cortisol influences stress-
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28 somatic complaints in functional somatic syndromes, such theory may exists in conditions like
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30 CLBP.¹⁷ Therefore, in a common stressful condition like back pain or a physical stressor like
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32 exercise, it may be possible that both the BE and cortisol are released as response from body
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34 mechanism to exhibit endogenous opioid induced analgesia and stress induced analgesia
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36 respectively.
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48 While clinical studies have focused on roles of the BE and cortisol on health, the
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50 biochemical changes on the PB and PC levels after the LPST have not been studied previously.
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52 Moreover, a recent systematic review has a conclusion that there were limited evidence to
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54 conclude whether or not the exercise therapy induces pain modulating substances, and therefore
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56 warrants for an investigation to explore the effects of the LPST on the PB and PC.¹⁸ In addition,
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4 stress induced analgesia and placebo induced analgesia are two of several other clinical
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6 reasoning mechanisms proposed to effect low back pain patients.^{19,20} Thus, it is appropriate to
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8 do a deductive reasoning to differentiate and investigate the real effects of the LPST on the PB
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10 and PC in comparison to the placebo and controlled intervention. Therefore, the main aim of the
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12 current study was to investigate the effects of three groups of exercise interventions; the lumbo-
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14 pelvic core stability training (LPST), the placebo (automated passive cycling training), and the
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16 control (rest) interventions on the levels of PB and PC on CLBP patients. The current study
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18 hypothesized that the LPST might increases the PB and reduces the plasma cortisol when
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20 compared to the placebo and control interventions among the patients with CLBP. The
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22 information from the study may help clinicians to understand the potential biochemical effects of
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24 the LPST against the other exercise regimes. Such knowledge may assist the clinicians to design
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26 appropriate exercise prescription in low back pain rehabilitation.
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36 **Methods**

37 *Subjects*

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39 A total of 24 participants (7 males, 17 females; age 33.76 ± 14.51 years) with CLBP
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41 residing in a community and university area participated in the study. An advertisement about
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43 the research study was placed around the community and university locations and the
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45 participants were recruited through pre-defined inclusion and exclusion criteria. The participants
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47 aged 20-35 years with mild to moderate back pain, with a visual analog scale pain score between
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49 **2-7 cm** and presence of pain for more than 3 months with location of pain in the area between the
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51 lowest rib 12th to gluteal folds participated in the study. Any participants with referred pain or
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53 neurological involvement in lower limbs, with history of past surgery, **with history of smoking**,
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4 who had history of injury in the last 3 months, were not recruited for the study. In addition,
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6 pregnant women and women who reported menstruation three days before or three days after the
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8 study period were not included. Also, any participants who performed any forms of physical
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10 exercises regularly were not considered for the study as routine exercises may potentially
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12 influence the physiological levels of beta endorphins. None of the participants took stimulants,
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14 medications, alcohol and involved in heavy physical activities at least 12 hours prior to the test.
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16 All the participants gave written informed consent to join the study and the whole research
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18 project was conducted in an outpatient physiotherapy department of a university teaching
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20 hospital. A university ethical committee approved the human ethics for the study as per the
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22 standards of Declaration of Helsinki, Finland.
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31 Study Design

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33 The effects of the three different groups of intervention; the LPST training, the placebo
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35 (automated passive cycling training), and the control (rest) group on the levels of plasma BE and
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37 PC were investigated through a randomized, placebo-controlled, cross over trial. An envelope
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39 based concealed randomization and allocation of the participants was used in the study. Two
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41 independent physiotherapists who were not the part of the study assisted the process of
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43 randomization and allocation. All of the three interventions were numerically coded in an
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45 opaque sealed envelope and placed in a closed box by one of the independent physiotherapy staff
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47 prior to the screening of the participants. The second staff who was not aware of the coded
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49 allocation sequence for intervention assisted with the screening of the participants for the study
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51 and collected the written consent. Then, each of the participants were asked by the other staff to
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53 choose any one envelope during the first visit and allocated to the corresponding intervention
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4 group accordingly. During the second visit, the same procedure was repeated and the second
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6 allocation was carried out for the second intervention. The participants went allocated to the third
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8 intervention based on the last envelope left inside the box. Thus, the participants crossed over
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10 from one intervention to the other intervention during the course of the trial. The cross-over
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12 design was employed in this study to avoid variations in the physical, physiological and
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14 psychosocial characteristics among the study participants.
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21 *Experimental Exercise Training Conditions*

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24 The three types of exercise interventions; the LPST, the placebo (automated passive
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26 cycling training), and the control (rest) intervention for this study was implemented as per the
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28 previously established protocols.^{3,4,6} All the participants performed each type of exercises in a
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30 controlled environmental room with temperature of $24.5 \pm 0.5^{\circ}\text{C}$, relative humidity of $60 \pm 5\%$,
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32 for approximately 15 minutes randomly with 48 hours between sessions to minimize any
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34 possible carry over effect. The participants performed all the exercises under the supervision of
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36 a qualified physiotherapist who had a post graduate clinical training in musculoskeletal
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38 physiotherapy. The position of the participants for all types of exercise interventions was supine
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40 crook lying. All of the participants received one familiarization trial for each exercise on the day
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42 of the corresponding exercise intervention. The familiarization trial was strictly maintained to a
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44 single attempt in order to prevent any learning effects. The therapist instructed all the
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46 participants to report any incidence of increased intensity of pain during the course of the
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48 interventions. Any participant who reported increased intensity of pain during the intervention
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50 was stopped from further participation in the trial due to safety and ethical reasons as per the
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4 **study protocol.** The exercises were monitored and corrected by the therapist if necessary in order
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7 to adhere to the exercise protocols.

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9 A Pilates power gym transformer device (Thane Fitness, UK) and air pressure
10 biofeedback unit were used to perform the LPST. For the LPST intervention, the participants lay
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12 down on the pilates exercise training device with hip and knee flexed to 70° and 90°,
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14 respectively. The core muscle activation was facilitated by encouraging abdominal hollowing
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16 and co-contraction of trunk muscles. The core muscle contractility and the progression of the
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18 exercises were monitored by an air pressure biofeedback unit that was also placed beneath the
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20 lumbar spine from L2 to S1 and inflated to 40 mmHg. **All the participants performed core**
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22 **muscle exercise with a resistance of approximately 10% of the body weight using weight pulley**
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24 **system on the device incorporating the limb movements in the following progressive order; core**
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26 **with alternate hip abduction, core with alternate leg raise, core with both arms adduction, core**
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28 **with both arms extension, core with alternate arm lift, core with alternate leg lift, core with**
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30 **alternate leg and arm.**³ Figure 1 shows one of the exercise description of the core stability
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32 **training.** The progression of the exercises was stopped when the subject could not maintain the
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34 registered air pressure at 40 ± 10 mmHg. Every stages of exercise were repeated for 10 times.
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43 The Placebo (automated passive cycling) intervention was administered by using an automatic
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45 bicycle (ReckMotomed Viva, RECK Technik, Germany). In supine crook lying position, the
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47 participants performed alternate leg movements passively at the speed of 30 revolutions per
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49 minute (rpm) with their feet attached to the pedals of the automatic bicycle. For the control (rest)
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51 intervention, the participants were made to relax completely on the Pilates power gym in the
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53 similar supine crook lying position. In addition, both knees were supported by pillows with hip
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55 flexion of 70° and knee flexion of 90° in order to ensure complete rest and relaxation.
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4 Measurements

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6 *Procedures*

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9 A qualified biomedical person who was independent to the study protocol was employed to
10 collect the blood samples from the participants. A 10 ml of blood was drawn from each
11 participant through a venipuncture at the cubital vein. The blood samples from the participants
12 were collected at two points such as one minute prior to the exercise intervention and one minute
13 after the exercise intervention. All of the blood samples were collected during the late morning
14 period between the time frame of 09:00 hours and 11:00 hours respectively in order to
15 standardize the circadian rhythm effects on the blood markers. The samples of blood were
16 collected in vacutainer plastic tubes and stored on an iced container and moved for the
17 centrifugation process at the university hospital.
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31 *Plasma β -endorphin*

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33 The PB was collected as per an established protocol.²¹ A 500 μ l of plasma was acidified
34 with 500 μ l of 1% trifluoroacetic acid (TFA) and mixed, then centrifuged at 17,000 \times g for 20
35 min at 4 degrees Celsius. A SEP-Column containing 200 mg of C18 was equilibrated by
36 washing with 60% acetonitrile in 1% TFA (1 ml, once) followed by 1% TFA (3 ml, 3 times).
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38 Acidified plasma solution was loaded onto the pre-treated C-18 SEP-COLUMN. The column
39 was slowly washed with 1% TFA (3 ml, twice). The peptide was eluted slowly with 60%
40 acetonitrile (3 ml, once). The eluant was collected in a polypropylene tube and evaporated to dry
41 in a centrifugal concentrator. The dried sample was kept at -20°C. A 50 μ l of sample or
42 standard, 25 μ l of primary anti-serum, and 25 μ l of biotinlyated β -endorphin were added into
43 ELISA well plate and incubated for 2 hours at room temperature. The plate was washed 6 times
44 with assay buffer and dried by inverting the plate on an absorbent material. A 100 μ l of diluted
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4 SA-HRP solution was added into each well, except for the blank, and incubated for 1 hour at
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6 room temperature. The plate was washed again three times and dried. Finally, a 100 µl of TMB
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8 solution was added to each well, and incubated for 1 hour at room temperature. The reaction was
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10 stopped with 2N HCl and absorbance was read at 450 nm. The concentration of β-endorphin was
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12 calculated with the standard curve of standard β-endorphin (0.01-1,000 ng/mL).
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15 16 *Plasma Cortisol*

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18 The PC levels were detected using Elecsys® Cortisol (Roche) in Cobas E411 auto analyzer. The
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20 principle of the test was electrochemiluminescence immune assay method. The sample was
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22 incubated with a cortisol specific biotinylated antibody, a ruthenylated cortisol derivative and
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24 releasing agent. The antibody binding sites were occupied either by cortisol or the ruthenylated
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26 derivative, with the proportion of each depending on the concentration of cortisol in the sample.
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28 Streptavidin-coated micro particles were added to the reaction mixture and the immune
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30 complexes bind to the solid phase via biotin streptavidin interactions. The reaction mixture was
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32 transferred to a measuring cell and the micro particles were magnetically captured onto the
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34 surface of an electrode; unbound sample was washed away before a chemiluminescent reaction
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36 was induced by applying a voltage to the electrode. The detection limit was 0.5 nmol/L and
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38 measuring range was 0.5-1,750 nmol/L.
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45 46 *Data Analysis*

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48 The sample size for the current study was established in previous studies using G*power
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50 statistical program.^{3,6} A significant alpha level of 0.05 and power analysis of 0.80 with an
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52 estimated moderate effect size of 0.54 resulted in a sample size of approximately 24 participants
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54 for this study. The Shapiro-Wilk test was used for examination of the data normality. The
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56 percentage change (%change) of the PB and PC levels were also estimated by the difference
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4 between pre- and post-changes divided by hundred. Since the data were not normally distributed,
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6 the non-parametric statistics, Friedman's Rank Test and post-hoc analysis by Wilcoxon Signed-
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8 Rank Test were used to analyze the changes of the PB and PC levels after three types of exercise
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10 interventions. The statistical software package (SPSS) for windows version 20.0 was used to
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12 analyze the data.
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19 Results

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21 All of the participants had low back pain with reported pain intensity of 4.28 ± 1.82 cm in
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23 the pain visual analog scale and the mean duration of the low back pain was 41.54 ± 35.81
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25 months. The mean weight and height of the participants were 59.02 ± 9.38 kilograms and
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27 162.42 ± 10.89 centimeters, respectively. None of the participants reported any increase in the
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29 pain intensity during the course of the interventions.
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33 Table I shows the PB and PC levels in all the three exercise interventions. The PB and
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35 PC levels between the three groups of exercise interventions showed no significant difference
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37 ($P > 0.05$) at baseline measurements. The percentage change in the level of PB was
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39 approximately 142.30% which suggested a trend of elevation in the release of PB before and
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41 after the LPST intervention ($P < 0.05$). On the other hand, the level of PB dropped with total
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43 percentage change of -40.59% and -44.18% after the placebo intervention ($P < 0.001$) and control
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45 (rest) interventions ($P < 0.001$), respectively. The results showed that there was a significant
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47 difference between the three groups on the total percentage change of the PB after the exercise
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49 interventions ($P < 0.01$). The results from the levels of PC analysis showed that there was no
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51 significant difference in the percentage change of PC when compared between the three groups
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4 of exercise interventions ($P>0.05$); the LPST intervention (-15.83%), the placebo (automated
5 passive cycling training) (-33.70%), and the control (rest) intervention (-29.19%).
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10 11 12 13 14 **Discussion**

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16 The current study investigated the effects of LPST on the levels of PB and PC in
17 comparison to placebo (automated passive cycling) and control (rest) interventions. The PB and
18 PC are related substances as both of these substances are generated by hypothalamus-pituitary-
19 adrenal axis activation in chronic painful conditions such as CLBP or due to the presence of any
20 stressors such as physical exercise. Also, both these substances are reported to modulate pain
21 through descending inhibitory system in chronic pain conditions. Therefore, it is important for
22 clinicians to understand the regulating mechanism of these substances after LPST among low
23 back pain patients. The effects of general exercises on the endogenous opioids are well
24 established in literature⁹. However, very limited understanding exists on the effects of the LPST
25 on the biochemical substances.¹⁸ In our opinion, the current study might be the first study to
26 investigate the changes in the PB in combination with PC after administration of the LPST
27 intervention. The knowledge on the biochemical changes in PB and PC after LPST may help
28 clinicians to understand the mechanism behind the analgesic effect of LPST among low back
29 pain patients. Thus, the current study findings are important to understand the changes in the
30 levels of PB and PC after LPST prescription among CLBP. **However, the current study did not
31 focused on the correlation between the observed changes in the levels of PB and the pain
32 intensity reported among the patients. Therefore, further studies are warranted in this field
33 particularly to report on the levels of PB and PC with regard to changes in the pain intensity**
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4 among CLBP patients in LPST training as such knowledge may further strengthen the evidence
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7 for the use of LPST in clinical practice.
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11 The result showed that a trend of increase in the average PB level from 6ng/ml at the
12 baseline to 15ng/ml immediately after the LPST intervention. The result suggested that the
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14 LPST exercise could influence circulating PB levels in blood stream which might explain as one
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16 of the possible facets for pain suppression mechanisms based on the endogenous pain-relieving
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18 peptides in CLBP. Past evidences that supports an increase in the plasma concentration of
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20 serotonin after application of lumbar stabilization exercises among low back pain patients may
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22 convince to accept the rationale that the LPST increased the levels of PB among the participants
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24 in the current study.^{22,23} Exercise intensity in relation to the release of PB has been discussed in
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26 literatures.^{24,25,26} A critical intensity of exercise is pre requisite for elevation of PB levels in
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28 blood stream. An exercise intensity exceeding 60%-80% VO₂ max was reported to increase the
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30 blood BE levels in most individuals.²⁵ It was possible that the LPST may provide an adequate
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32 stimulus to trigger the release of pain relieving peptides. As none of the participants reported
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34 any increase in the pain intensity during the course of interventions, perhaps it could be
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36 suggested that the observed elevation in the levels of PB might be directly related to the LPST
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38 and not as a result of increased pain while performing the intervention. On the other hand, no
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40 elevated levels of PB was observed in the placebo (passive automated cycle) and control (rest)
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42 interventions. It could be explained that these two forms of interventions did not have adequate
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44 physical stimulants to release PB in comparison to LPST among CLBP.
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55 The result showed that there was no significant change in the levels of PC between the
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57 three types of exercise intervention groups. Cortisol is released by adrenal cortex following
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4 physiological and psychological stress.¹¹ The range of PC level in this current study was found
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6 to be within the normal range.²⁷ Excessive release of cortisol has negative effect on muscle,
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8 immune cell function and metabolism.²⁸ Also, when cortisol is released higher than the normal
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10 range, it is counterproductive to optimal tissue repair and remodeling.²⁸ Exercise is a primary
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12 stimulant for the release of PC which depends on exercise protocols, where intensive anaerobic
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14 demands increases PC while moderate aerobic exercise are reported not to elicit any responses.²⁹
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18 The result of the current study might explain that LPST intervention possibly belong to moderate
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20 intensity which was evidenced by the fact that the percentage change of PC levels were not
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22 elevated. **In our opinion, the relationship between the level of PC and PB with the intensity of**
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24 **LPST using tools such as perceived exertion scale needs to be ascertained in future trials as it**
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26 **may help clinicians to understand the dosage and intensity behind LPST for the desired**
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28 **therapeutic effects from endogenous opioids.** It could be suggested that although the LPST
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30 might contribute to endogenous induced analgesic response as supported by elevated PB levels,
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32 the participants were not stressed due to LPST or laboratory environment during the study.
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There is a huge paucity of knowledge in evidence based practice towards the effects of LPST on the PB and PC in CLBP. Therefore, further research is recommended on this field on topics such as establishing a dosage and response between LPST, PB and PC, understanding the pain modulating effects by PB and PC following LPST and alterations in the autonomic nervous system. Any spinal therapies such as spinal mobilization and spinal manipulation were reported to have an effect on the autonomic nervous system and cardio vascular system.^{30,31,32} It is

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4 suggested that stimulation of spinal joints through manipulative therapy decreases heart rate and
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6 blood pressure through the autonomic system activity.³³ Furthermore, aberrant stimulation of the
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8 spinal or paraspinal structures were reported to cause segmentally organized reflex responses of
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10 the autonomic system and visceral function.³² Also, the endogenous opioid system is activated
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12 by various stimuli such as hypoglycaemia, severe hypotension, acute myocardial ischemia and
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14 congestive cardia failure.³⁴ As LPST is believed to act on the segmental stability of the lumbar
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16 spine, it may be relevant to investigate the effects of LPST on PB and PC in the context of
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18 autonomic nervous system activity, visceral function and cardiovascular system. Therefore,
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20 future studies may investigate the mechanism of interaction between PB, PC, autonomic, visceral
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22 and cardiovascular during LPST among CLBP. The current study contributes to some important
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24 clinical implications for clinicians towards rehabilitation of CLBP. Neuroendocrine and
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26 neuroimmune effects are two important output mechanisms hypothesized in clinical reasoning of
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28 CLBP. The current study might facilitate an understanding of the levels of PB and PC among
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30 patients with low back pain as indicators of neuroendocrine and neuroimmune mechanisms.
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32 Such knowledge may help clinician to strengthen the hypothesis on the pain and tissue healing
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34 mechanisms in management of CLBP. Also, the study might provide an explanation over the
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36 analgesic mechanism reported in LPST by documenting the biochemical changes in levels of
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38 plasma BE. Additionally, the current study might help clinicians to differentiate endogenous
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40 opioid versus stress related analgesia in low back pain conditions.
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50 The study acknowledges few limitations. The study did not directly investigate the
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52 changes in the pain intensity in relationship to the changes with the levels of PB and PC during
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54 LPST. Therefore, it was not possible to comment on the direct pain inhibition effects by the
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56 levels of PB and PC observed during the study after the LPST. The smaller sample size could be
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4 considered as another limitation. Nevertheless, the current study was the first study to report on
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6 the effects of LPST on PB and PC, thereby it might be useful for other researchers to calculate an
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8 adequate sample size in further studies on this topic. Various sub stages (acute versus sub-acute)
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10 and sub populations of low back pain were not taken into account while sample recruitment.
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12 Therefore, the study findings may differ in different sub populations of low back pain and hence,
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14 it requires further investigations in future studies. The outcome of the study did not consider
15
16 long term follow up of the PB and PC levels. However, the current study design was not
17
18 appropriate to evaluate long term follow up, as several confounding factors such as neuroticism,
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20 positive and negative emotions, sleep deprivation, socio-economical and psychosocial variables
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22 might influence the PB and PC levels in a longitudinal design. Therefore, the clinicians may
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24 consider all of the above limitations before translating the study findings to the wider clinical
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26 practice.
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36 **Conclusion**

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38 The LPST intervention potentially increased the PB levels when compared to placebo and
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40 control interventions in CLBP. In addition, the percentage change of plasma cortisol levels
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42 remains unchanged after LPST intervention. Further studies are advocated to confirm and
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44 translate the study findings to a wider clinical practice.
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Table I. Data of beta-endorphin and cortisol levels, which are showed as mean \pm standard deviation (SD) and overall percentage change (%Ch) values across all three experimental conditions (i.e., core exercise, placebo, control).

Outcomes	Conditions								
	Core Exercise			Placebo			Control		
	Pre-	Post-	%Ch	Pre-	Post-	%Ch	Pre-	Post-	%Ch
Beta-endorphin	6.24 (17.10)	15.11* (32.95)	142.30% ^{a,b}	6.02 (11.30)	3.58 (9.57)	-40.59%**	10.75 (19.20)	6.00 (16.06)	-44.18%***
Plasma cortisol	8.91 (4.61)	7.50 (4.95)	-15.83%	10.48 (6.97)	6.95 (3.79)	-33.70%	8.81 (4.76)	6.24 (4.31)	-29.19%

Unit: plasma beta-endorphin (ng/ml), Plasma cortisol (ng/ml)

Note: No significant differences of the baseline data among three conditions ($P > 0.05$)

* Significant differences between pre-post under core stability exercise group ($P < 0.01$)

** Significant differences between pre-post under placebo exercise group ($P < 0.001$)

*** Significant differences between pre-post under control group ($P < 0.001$)

^a Significant differences between placebo ($P < 0.01$)

^b Significant differences between control ($P < 0.01$)

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Legends:

Table Caption:

Table I. Data of beta-endorphin and cortisol levels, which are showed as mean \pm standard deviation (SD) and overall percentage change (%Ch) values across all three experimental conditions (i.e., core exercise, placebo, and control).

Figure Caption:

Figure 1: A description of one of the core stabilization exercise with alternate leg lifting on the Pilates power gym device

Figure 1
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Section 1. Identifying Information

1. Given Name (First Name) Aadit 2. Surname (Last Name) Paungmali 3. Effective Date (07-August-2008)

4. Are you the corresponding author? Yes No

5. Manuscript Title Effects of Core Stabilization Exercise on Beta-endorphin and Cortisol levels among chronic non-specific low back pain patients

6. Manuscript Identifying Number (if you know it)

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4. Are you the corresponding author? Yes No

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11. Stock/stock options	<input checked="" type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>			X
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12. Travel/accommodations/meeting expenses unrelated to activities listed**	<input checked="" type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>			X
						ADD
13. Other (err on the side of full disclosure)	<input checked="" type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>			X
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						X
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						ADD
2. Consultancy	<input checked="" type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>			X
						ADD
3. Employment	<input checked="" type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>			X
						ADD
4. Expert testimony	<input checked="" type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>			X
						ADD
5. Grants/grants pending	<input checked="" type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>			X
						ADD
6. Payment for lectures including service on speakers bureaus	<input checked="" type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>			X
						ADD
7. Payment for manuscript preparation	<input checked="" type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>			X
						ADD

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						X
						ADD
9. Royalties	<input checked="" type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>			X
						ADD
10. Payment for development of educational presentations	<input checked="" type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>			X
						ADD
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						ADD
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4. Fees for participation in review activities such as data monitoring boards, statistical analysis, end point committees, and the like	<input checked="" type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>			X
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Author email address aatit.p@cmu.ac.th

<u>AAIT PAUNGMALI</u>	<u>X</u>	<u>Aait Paungmali</u>	<u>20/01/2016</u>
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Author email address leonardjoseph85@hotmail.com

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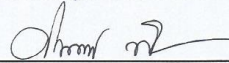
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Author email address Khanittha.Taneyhill@cmu.ac.th

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Author email address patraporn.s@umc.ac.th

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