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Investigation of Beta Endorphin Changes after Bruce Test in Active and Sedentary Individuals

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Opioid peptides have been implicated in many biological processes acting as hormones, neurohormones or neurotransmitters. The aim of this research was to investigate effect of endurance training on plasma beta endorphin (BA) changes in active and sedentary girls. 12 healthy physical education students as active group (n = 12, active group with, 21.6 ± 0.6 year age, 162.25 ± 3.68 cm in height, and 54.13 ± 6.35 kg, weight) and 12 healthy non physical education as sedentary group (n = 12, sedentary group, age 20.55 ± 0.69 year, high, 161.40 ± 3.14 cm, weight, 59.25 ± 4.45 kg) requited. Subjects entered the study on their usual diet and after familiarization with training protocol, Bruce test (treadmill test) as aerobic test applied. The blood sample was collected before and after training in both groups. Beta-endorphin concentration was determined by radioimmunoassay kits. The analyses and data processing showed that there is no significant difference within active (0.77 ± 0.1 pmol/l) and sedentary group (0.82 ± 0.01 pmol/l) in basal BE at rest. The BE levels after endurance training in active group and sedentary group increase respectively (0.83 ± 0.09 pmol/l), (compared with basal) however, significant difference was found between pre and post test group in active group ($P > 0.05$). In this research, BE secretion increased in response to endurance exercise both in active and sedentary girls.

Keywords: Beta Endorphin; Treadmill Test; Endurance Training; Active; Sedentary

Introduction

Endogenous opioid peptides constitute a flexible and widespread regulatory modulatory system. Opioid peptides have been implicated in many biological processes acting as hormones, neurohormones or neurotransmitters. Their influence is mediated by specific opioid receptors. Three classes of endogenous opioid peptides can be discriminated between: Enkephalins, Endorphins and Dynorphines (Hackney, 2006). Enkephalines are found mainly in brain. In this tissue methione and leucine enkephalins (met- and leu-enkephalins) are purified and characterized. None is very potent because of their rapid enzymatic degradation. Met-enkephalin is also found in the blood plasma and in a relatively high concentration in comparison with other opioid peptides (Angelo et al., 2001). The circulating met-enkephalin is in a several fold molar excess over the circulating endorphin. An extremely high concentration of enkephalins is co-localized and co-released with catecholamines in adrenal medulla. In this connection the met-enkephalin concentrations in the adrenal vein is higher than in other parts of the circulation. However the majority of the circulation met-enkephalin originnates in the sympathetic nervous system (Bender et al., 2007; Pierce et al., 1993).

Dynorphins are extended forms of leu-enkephalins. They are among highly potent opioid peptides. The dynorphin family includes dynorphin A, dynorphin B, and neo-endorphin. A common precursor for endorphins and pituitary corticotropin, as well as for β-lipotropin, β-melanotropin and probably also for met-enkephalin is pro-opiomelanocortin (Farrel, 1998; Kraemer et al., 1993).

Opioid peptides activate three different types of receptors; mu, kappa and delta receptors, all of which act through a second messenger (Andrea, 2006; Tamas et al., 2007).

Endorphins (endogenous morphines) are produced in various brain structures, with the highest rate in the arculate nucleus of the hypothalamus. The endorphin is synthesized in the hypophysis and adrenals. It is not excluded that there are other sites. However, the main source of blood endorphins is the hypophysis. Endorphins primarily operate via the mu receptor. This receptor is known to mediate analgesic effects as well as play a role in the reward system within the brain. Evidence showing that endorphins can interfere with the release of other neurotransmitters, including norepinephrine, dopamine, and acetylcholine, have led to a belief that they work by modulating the presynaptic membranes of synapses other than own (Tamas et al., 2007; Goldfarb et al., 1997) The main opioid peptide is beta endorphin. Products of its metabolic degradation are alpha and gama endorphins.

The production of endorphins is stimulated by a hypothalamic neuropeptide, corticoliberine. It acts via corticoliberine receptors located in the hypophysis as well as in various nervous structures. In the hypophysis the result of the activity of corticoliberine receptors is the formation from pro-opiomelanocortin of corticotropin, β-endorphin and β-lipotropin, as well as the secretion of these substances into the circulation. The resulting increase in the corticotrophin and β-endorphin blood levels is rather parallel after endogenous administration of corticoliberine or under the influence of various stressors.

Blood-borne endorphins do not gain entry into the central

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nervous system. They actualize their regulatory/modulators function through peripheral receptor sites. Only a limited amount of blood endorphins can reach neural receptors in sites not protected by the blood-brain barrier. Anyway, there are two discrete parts of the endogenous opioid system: central nervous and peripheral parts. The activities of these two parts may be in good correlation, but the produced opioids are not transferred from one compartment into the other. However, met-enkephalin and dynorphin may cross the blood-brain barrier using an allosterically modulated saturable transport mechanism (Gorostiaga et al., 2004; Taylor et al., 2000).

In order to establish the physiological function of endogenous opioids, an opioid receptor antagonist, naloxane, is widely used. In this way their role has been indicated in decreased pain perception, suppression of affective disorders, promotion of learning and memory, hunger and thirst influence, glucose homeostasis, regulation of cardiovascular functions, respiration, endocrine activities, renal function, gastrointestinal activity, lymphatic function/immunity, and sexual behavior as well as thermal regulation.

The euphoric feelings generated by endorphins which may result from strenuous exercise, are believed to play an important role in addiction (Goldfarb et al., 1997; Farrel, 1998; Gorostiaga et al., 2004; Anthony et al., 2006; Hegadon et al., 2009; Courteny et al., 2004). While it is generally accepted that endorphins induce euphoria, it is unclear whether exercise causes an increase in endorphin levels (Armstrong, 2006). Additionally, if exercise increases endorphin levels, few studies have been performed to measure whether this increase plays a role in exercise dependence. Because findings of endorphin elevations are so inconsistent, researchers continually alter experimental strategies. Unfortunately, this makes it difficult to determine what, if any, strategies effectively measure endorphin and the physiological response to exercise. Based on different research, it appears that endorphin activity may be highly variable from one individual to the next, making this analysis even more complex (Taylor et al., 2000). Many researches have not found significantly elevated endorphin levels after exercise (Heitkamp et al., 1998; Harbach et al., 2000).

Farrell (1985) assessed the threshold by looking at the effects of intensity and distance of running on endorphin release in male subjects by compiling results from multiple studies with varied time and distance of running. Analyses of these data indicated a trend of elevated endorphin levels after exercise in all studies, although not all were significant. Pierce et al. (1993) performed a study measuring plasma endorphin levels before and after endurance exercise, 45 minutes of high intensity aerobics. Results indicate a significant increase in endorphin levels after the exercise while compared to levels before. These findings support the idea that opioid peptides may be released as a result of exercising vigorously for a specific amount of time. A study by Goldfarb et al., (1997) agrees with the conclusion, but claims that a critical intensity of exercise must be attained to induce elevation in plasma endorphin levels. However, contrary to the data by Goldfarb et al., (1997), Farrell's (1985) evidence shows that the increase in endorphin release did not appear to be dependent on the intensity of running.

While much data has been published about the relationship between endorphins and intensity of the exercise, other researches tested different forms of exercise, mostly the different between running and bicycling. In fact Pierce et al. (1994) asserted that a 70% VO is required to significantly elevate endorphin levels in the blood plasma.

All these studies demonstrate a variety of measurements on human participants examining endorphin activity. Non indicate strong evidence of increase of endorphin level release at a result of vigorous exercise, although many suggest trend such a response. This trend is enough to compel researchers to continually question if such a response exists.

So the aim of this research was to study changes of plasma beta endorphin in response to endurance training in active and sedentary girls.

Methodology

Subject

Sixty volunteer female students in Alzahra University, Tehran Iran, were taken into the study. All subjects were asked to complete a physical activity, medical examination and a medical questionnaire to ensure that they were not taking any medication and free of endocrine or metabolic diseases. Menstrual cycle was controlled and all the girls were in luteal phase to reduce any side effect.

Calculated body mass index (BMI) (kg/m^2) and waist hip ratio (WHR) were measured to analyses anthropometry factors in two groups. Waist circumference was measured in a horizontal plane at the level of natural waist that was the narrowest part of the torso. Hip circumference was measured at the largest protrusion of the buttock without compressing the skin (Latifah & Hanachi, 2008).

Finally 12 healthy physical education students as active group (21.6 ± 0.6 year, 162.25 ± 3.68 cm, 54.13 ± 6.35 kg) and 12 healthy non physical education as sedentary group (20.55 ± 0.69 year, 161.40 ± 5.14 cm, 59.25 ± 6.45 kg) were studied during a medical check up in order to measure their exercise induced plasma beta endorphin responses. All students in active group were following their regular university sport programs, but were not involve vigorous and high intensity physical training. Non were receiving medication at the time of the study. The training protocols acquainted to participants and inform consent was obtained from all subjects. The anthropometrical characteristics of active and sedentary groups were shown in **Table 1**.

Exercise Test

Bruce test (treadmill test) was exerted as endurance training which had 10 steps with duration of 3 minutes in each steps. The protocol of this test mentioned below in **Table 2**. Then subjects warm up for 5 minutes and start doing (treadmill test) protocol (Bruce et al., 1994). The test finished when subjects could not continue the training sections. Prior to experimental

Table 1.
Mean values of anthropometric characteristics of active and sedentary groups.

	Sedentary group (n = 12)	Active group (n = 12)
Age (year)	20.55 ± 0.69	21.67 ± 0.65
Height (cm)	161.40 ± 5.14	162.25 ± 3.68
Weight (kg)	59.25 ± 6.45	54.13 ± 6.35
BMI (kg/m^2)	21.89 ± 2.40	20.52 ± 2.23
WHR	0.83 ± 0.04	0.80 ± 0.02

trial, a flexible catheter was placed into an antecubital right hand vein and two blood samples were collected in resting period and after the training. The serum beta endorphin levels measured in both active and sedentary groups (**Figure 1**).

Serum Beta Endorphin Levels

Venous blood samples from the right hand arm of the subjects were taken to measure levels of beta-endorphin before and immediately after specialized training protocol under stress-free condition, as possible.

Serum blood samples immediately were centrifuged at 5000 rpm for 10 min at 4˚C and serum were stored at –70˚C until beta endorphin assayed. serum level of Beta-endorphin was determined with standard radioimmunoassay kit (Phonix Pharmaceuticals, Inc., California, USA) with high affinity for beta endorphin and cross reactivity of less than 6.2% for B-lipotropin levels and less than 0.01% for other peptides.

Statistical Analysis

The statistical analyses were performed with the SPSS 17 for Windows program. The differences between groups and between values before and after each group were calculated as mean ± standard deviation. Independent samples t-test were used in the statistical analyses. $P < 0.05$ was considered to be statistically significant.

Table 2.
The experimental protocol of Bruce test.

Step	Time (min)	Speed (km/hr)	Gradient
1	0	2.74	10%
2	3	4.02	12%
3	6	5.47	14%
4	9	6.76	16%
5	12	8.05	18%
6	15	8.85	20%
7	18	9.65	22%
8	21	10.42	24%
9	24	11.26	26%
10	27	12.07	28%

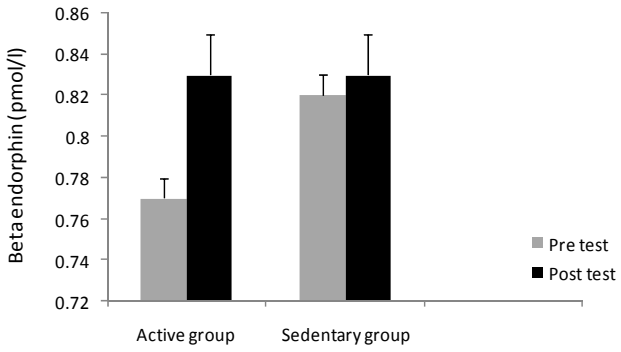

Figure 1.
Beta endorphin levels (pmol/l) before and after Bruce test as an endurance training protocol in active and sedentary group. Data were mean ± S.E.M. *Statistical significance was accepted at $P \leq 0.05$.

Results

The analyses and data processing showed that there is no significant difference within active (0.77 ± 0.1 pmol/l) and sedentary group (0.82 ± 0.01 pmol/l) in basal BE at rest. BE levels after endurance training in active group and sedentary group increase respectively (0.83 ± 0.09 pmol/l), (compared with basal) however, significant difference was found between pre and post test group in active group ($P > 0.05$). The increase in beta endorphin in active group after endurance training was higher than sedentary group (**Figure 1**).

Discussion of Findings and Recommendations

The hormonal changes can have important effect on physical activity functions. In particular, exercise leads to the release of certain neurotransmitters in the brain that alleviate pain, both physical and mental. Much of the research done in this area has focused on running, but all types of aerobic exercise provide benefits. Although the exact nature of these benefits is still being determined, enough research has been done to provide even skeptics with a motivation to take up exercise. Exercise exerts its effects on the brain through several mechanisms, including neurogenesis, mood enhancement, and endorphin release (Andrea, 2006).

Exercise-induced increases in the peripheral beta-endorphin concentration and mainly associated both with changes in pain perception and mood state. A more precise understanding of opioid function during exercise can be achieved by investigating the changes in beta-endorphin concentrations dependent upon intensity and duration of physical exercise and in comparison to other stress hormones. Published studies reveal that incremental graded and short term anaerobic exercise leads to an increase in beta-endorphin levels, the extent correlating with the lactate concentration (Goldfarb et al., 1997; Armstrong, 2006).

Many studies have examined the relationship between exercise and endorphin release, studying the role of these peptides in exercise induce euphoria as well as the reduction of pain (Farrel, 1998; Bouix et al., 1993; Fry et al., 1997).

All these studies demonstrate a variety of measurements on human participants examining endorphin activity. Non indicate strong evidence of increase of endorphin level release at a result of vigorous exercise, although many suggest trend such a response.

In this study the result showed that the levels of plasma beta endorphin increases after exercises both in active and sedentary groups. It could be related to physical stress produced by long distance running. Other possible functions of beta endorphin increase could be related to the endocrine response to exercise, because a role in glucorticoid release and energy balance (Anthony et al., 2006). It is possible that the release of beta endorphin is linked to other functions that must be controlled during the practice of exercise, as are antihypertensive mechanisms, respiratory response and immuno response. However, further studies at biochemical and physiological levels are necessary in order to elucidate the role played by B-end release during physiccal exercise. The main finding of this study was that experimental inconsistence makes it nearly impossible to draw a distinct relationship between exercise and elevation in endorphin blood plasma levels. Therefore further investigations are necessary to determine the role of beta-endorphin in exercise-mediated physiological and psychological events.

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REFERENCES

Andrea, L. (2006). Endorphins, exercise, and addictions: A review of exercise dependence. *Journal for Undergraduate Publications in the Neurosciences, 55,* 291-297.

Angelo Tirelli, A., Guastafierro, S., & Annunziata, S. (2001). Effects of β-endorphin and met-enkephalin on platelet activity. *American Journal of Hematology, 68,* 1-3. doi:10.1002/ajh.1140

Anthony, C., & Hackney, B. (2006). Exercise as a stressor to the human neuroendocrine system. *Medicina (Kaunas), 42,* 122-134.

Armstrong, D. W. (2006). Hormonal responses to opioid receptor blockade during rest and exercise in cold and hot environments. *European Journal of Applied Physiology, 97,* 43-51. doi:10.1007/s00421-006-0135-7

Bender, T., Nagy, G., Barna, I., Tefner, I., Kádas, E., & Géher, P. (2007). The effect of physical therapy on beta-endorphin levels. *European Journal of Applied Physiology, 100,* 371-382. doi:10.1007/s00421-007-0469-9

Bouix, O., Brun, F., & Raynaud, E. (1993). Plasma β-endorphin, corticotrophin and growth hormone responses to exercise in pubertal and prepubertal children. *European Journal of Applied Physiology, 26,* 195-199.

Bruce, R. A., Lovejoy, F. W. Jr., Pearson, R. et al. (1994). Normal respiratory and circulatory pathways of adaptation in exercise. *The Journal of Clinical Investigation, 28,* 1423-1430. |doi:10.1172/JCI102207

Courteny, A., Rocheleau, M., & Gregory, D. (2004). Moderates of the relationship between exercise and mood changes: Gender, exertion level, workout duration. *European Journal of Applied Physiology, 19,* 491-506.

Farrell, P. A. (1998). Exercise and endorphins-male responses. *Medicine and Science in Sports and Exercise, 17,* 89-93.

Fry, A. C., & Kraemer, W. J. (1997). Resistance exercise overtraining and overreaching: Neuroendocrine responses. *Resistance Exercise Overtraining and Overreaching, 23,* 106-129. doi:10.2165/00007256-199723020-00004

Goldfarb, A. H., & Jamurtas, A. Z. (1997). Beta-endorphin response to exercise. *Sports Medicine, 24,* 8-16. doi:10.2165/00007256-199724010-00002

Gorostiaga, E. M., Izquierdo, M., Ruesta, M., Iribarren, J., Gonzalez-Badillo, J. J., & Ibanez, J. (2004). Strength training effects on physical performance and serum hormones in young soccer players. *European Journal of Applied Physiology, 91,* 698-707. doi:10.1007/s00421-003-1032-y

Latiffah, L., & Hanachi, P. (2008). To investigate the relation of hypertension and anthropometric measurement. *Journal Applied Science, 8,* 3963-3968. doi:10.3923/jas.2008.3963.3968

Hegadoren, K. M., O'Donnell T., Lanius, R., Coupland, N. J., & Lacaze-Masmonteil, N. (2009). The role of β-endorphin in the pathophysiology of major depression. *Neuropeptides, 43,* 341-353. doi:10.1016/j.npep.2009.06.004

Heitkamp, H. C., Schulz, H., Rocker, K., & Dickhuth, H. H. (1998). Endurance training in females: Changes in beta-endorphin and ACTH. *International Journal of Sports Medicine, 19,* 260-264. doi:10.1055/s-2007-971915

Harbach, H., Hell, K., Gramsch, C., Katz, N., Hempelmann, G., & Teschemacher, H. (2000). β-endorphin (1-31) in the plasma of male volunteers undergoing physical exercise. *Psychoneuroendocrinology, 25,* 551-562. doi:10.1016/S0306-4530(00)00009-3

Kraemer, W. J., Dziados, J. E., Marchitelli, L. J., Gordon, S. E., Harman, E. A., Mello, R., Fleck, S. J., Frykman, P. N., & Triplett, N. T. (1993). Effects of different heavy-resistance exercise protocols on plasma beta-endorphin concentrations. *Journal of Applied Physiology, 74,* 450-459.

Pierce, E. F., Eastman, N. W., Tripathi, H. T., Olson, K. G., & Dewey, W. L. (1993). Plasma beta-endorphin immunoreactivity: Response to resistance exercise. *Journal of Sports Science and Medicine, 11,* 499-452. doi:10.1080/02640419308730019

Pierce, E. F., Eastman, N. W., McGowan, R. W., Tripathi, H., Dewey, W. L., & Olson, K. G. (1994). Resistance exercise decreases beta-endorphin immune reactivity. *British Journal of Sports Medicine, 28,* 164-166. doi:10.1136/bjsm.28.3.164

Taylor, D. V., Boyajian, J. G., James, N., Woods, D., Chicz-Demet, A., Wilson, A. F., & Sandman C. A. (2000). Acidosis stimulates beta-endorphin release during exercise. *Journal of Applied Physiology, 77,* 1913-1918.

Tamas, B., & Gyorgy, N. (2007). The effect of physical therapy on beta-endorphin levels. *European Journal of Applied Physiology, 100,* 371-382. doi:10.1007/s00421-007-0469-9