Available online at www.scholarsresearchlibrary.com

Scholars Research Library

Scholars Research Library

Annals of Biological Research, 2012, 3 (3):1255-1263
(http://scholarsresearchlibrary.com/archive.html)

Annals of Biological Research
Scholars Research Library
ISSN 0976-1233
CODEN (USA): ABRNBW

The effect of sleep deprivation due to land travel on the concentration of immunoglobulins A, G, and M and serum cortisol in young soccer players

Abolhasan Hedayati[1], Maghsoud Peeri[2], Mohammad Ali Azarbayjani[2], Ahmad Mazidi[3]

[1]Department of Physical Education, Aliabad Katoul Branch, Islamic Azad University, Aliabad Katoul, Iran
[2]Department of Physical Education and Sport Sciences, Central Tehran Branch, Islamic Azad University, Tehran, Iran
[3]Department of Physical Education, Sari Branch, Islamic Azad University, Sari, Iran

---

ABSTRACT

The purpose of the present research was to determine the effect of sleep deprivation due to land travel on the concentration of Immunoglobulins A, G, and Mand serum cortisol in young soccer players. Thus, IgM, IgG, IgA, and serum cortisol concentration of 15 soccer players (mean and standard deviation of 18.40 ± 1.24 years of age, 167.66 ± 6.22 cm height, 66.6 ± 7.28 kg weight, and 23.60 ± 2.47 kg/m^2 BMI) was measured after two trips from Gorgan to Tehran and Khorramabad. The subjects were selected using purposive and convenience sampling. Repeated measures analysis of variance was applied for data analysis. The results of Bonferroni correction revealed a significant increase in IgG, IgA, and serum cortisol concentration in Tehran ($P = 0.020$) and Khorramabad ($P = 0.045$), a significant increase in serum cortisol concentration between the two cities of Gorgan and Tehran ($P = 0.003$), a significant decrease in serum cortisol concentration between Tehran and Khorramabad ($P = 0.045$), and no significant difference in IgM concentration ($P = 0.539$). The results of the present research showed that sleep deprivationmay play an important role in immune and endocrine-related physiological function and suggest that quality and depth of sleep can be a more significant determinant for immune and endocrine function.

Keywords: sleep deprivation, travelling across time zones, immunoglobulin, cortisol.

INTRODUCTION

Sleep deprivation is a stressor that affects the brain and many body systems [17]. Travel across multiple time zones is a common feature of the lifestyle of contemporary international sport competitors. This entails a disruption of the body's circadian timing mechanisms [21]. The circadian rhythms may take seven days to normalize when more than five time zones have been crossed [28]. Long-distance flights can cause a number of clinical problems in both the

passengers and flight attendants [7]. Laboratory studies have shown that relative sleep deprivation following immunization against hepatitis reduces the production of antibody titers approximately four weeks after immunization. In addition, partially sleep-deprived individuals also exhibit impairment of antibody production against Influenza virus. Sleep disorders in which sleep deprivation is a major feature such as insomnia result in major alterations in the immune system, which may be mediated by the augmented activity of the HPA axis and/or the sympathetic nervous system [20]. It has been reported that sleep loss affects the immune system and results in reduced serum IgG, IgA, and IgM [2], while Ozturk (1999) showed that there is no significant difference in IgM and IgG levels following insomnia. Accordingly, there is evidence that sleep deprivation affects the immune system [12]. While short-term sleep deprivation can enhance host defenses, long-term sleep deprivation is devastating. This may be associated with alterations in immune parameters with different lengths of sleep alteration [17].

Soccer players always travel to other cities and countries for participating in national and international tournaments which can affect their humoral immune system. Travelling to different geographical locations is one of the common characteristics of the lifestyle of international athletes in the contemporary age and this entailsa disruption of body's circadian timing mechanisms [23]. Most studies measuring the effect of jet lag on athletic performance have methodological problems; yet, it is prudent to provide suggestions for minimization of jet lags [27]. In land travels, sleep disorder is an inevitable outcome that needs special attention. Many studies have examined the effect of insomnia of hormonal and immune indices, yet not much research has been carried out on the effect of sleep deprivation on the performance in a sport like soccer. Despite great effort, it was impossible to find a study done in this regard.

MATERIALS AND METHODS

Participants

The participants of the present research are 15 soccer players with 18.40 ± 1.24 years of age, 167.66 ± 6.22 cm height, and 66.6 ± 7.28 kg weight. They had the record of playing in national league tournaments and were selected as sample after filling out the consent form using purposive and convenience sampling. It must be noted that all the participants were completely healthy and took no medications. Before selecting the participants, all the purposes and dimensions of the research, the testing procedures, and the risks were fully explicated to the subjects in an introduction session.

Test Procedures

To examine the effect of sleep deprivationdue to land travel on the concentration of immunoglobulins G, A, and M and serum cortisol in young soccer players, a group of the subjects were examined in a pretest-posttest design. The first blood sample was taken at 10 A.M. in Gorgan City at 54-56° latitude and 37-38° longitude (as the reference city). A week later and immediately after finishing a bus travel to Tehran, the second blood sample was taken at 10 A.M. in Tehran at 50-53° latitude and 35-36° longitude. The final blood sampling was done in Khorramabad at 47-50° latitude and 33-34° longitude and at a similar time to that of the other cities. The samples were immediately placed in an ice-pack and were transported to a physiopathology laboratory. The samples were frozen and kept at -20°. Then the samples were arranged in the laboratory according to the work list. Due to the formation of two phases during the freezing of samples, the samples were mixed using a shaker. Blood samples were centrifuged at 4000 rpm for 10 minutes so that serum would be separated from the cells and then the samples were examined in Gama Counter System made by GENESYS Inc. for counting cortisol, Hitachi System for measuring the levels of IgG and IgA, and COBAS Mira System for measuring the

level of IGM. It must be noted that these systems were all computerized and human factor played the slightest role in measurements unlike common traditional methods.

Data Collection

Cortisol: The level of serum cortisol concentration of the participants was measured using RIA (radioimmunoassay) method and Immunotech Kit (made in France) with a precision of 0.02 Micg/dl (CV).

Immunoglobulin M (IgM): IgM concentration was measured using turbidimetry and Pars Azmoon kit (made in Iran). This kit is designed to measure IgM at the range of 3 to 780 mg/dl. The sensitivity of this kit is 3 mg/dl. The between-subject and within-subject accuracy were 2.35 and 2.04 respectively.

Immunoglobulin A (IgA): IgA concentration was measured using turbidimetry and Pars Azmoon kit. This kit is designed to measure IgM at the range of 70 to 400 mg/dl. The sensitivity of this kit is 3.25 mg/dlin 400 mg/dl.

Immunoglobulin G (IgG): IgG concentration was measured using turbidimetry and ParsAzmoon kit. This kit is designed to measure IgM at the range of 700 to 1600 mg/dl. The sensitivity of this kit is 0.580 mg/dl in 1600 mg/dl.

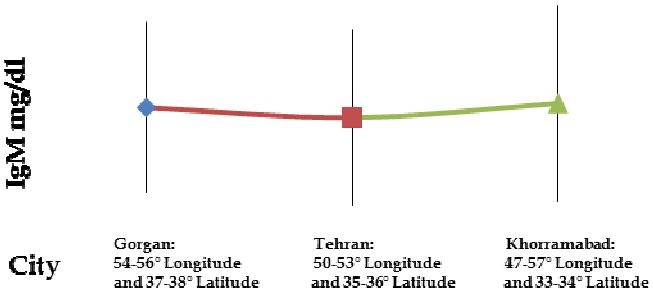
Data Analysis

Descriptive and inferential statistics have been used for data analysis. All the data obtained in the present research has been reported based on mean and standard deviation. First, normal distribution of the data was examined using Kolmogorov-Smirnov test and due to the normal distribution, repeated measures analysis of variance (ANOVA) was applied. Further, Bonferroni test was applied in case of significant difference in order to determine the source of variance. All the statistical operations were done using SPSS 16.

RESULTS

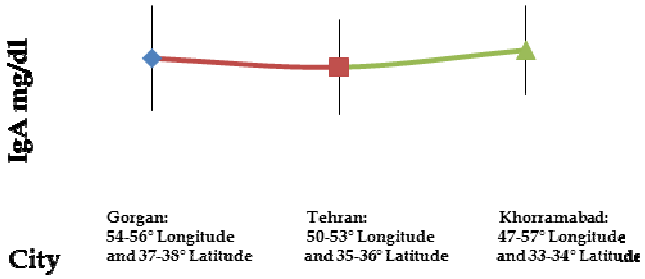
The results showed that there is a significant difference in the concentration of IgG, IgA, and serum cortisol of the participants following sleep deprivation due to land travel, but no significant difference was observed in the level of IgM concentration. Moreover, within-group comparisons revealed that there is a significant increase in IgG and IgA concentration between Tehran and Khorramabad ($P = 0.020$ *and* $P = 0.045$), where the level of IgG and IgA increases in Khorramabad which was farther from city of origin, but decreases in Tehran which has a less distance from the city of origin. Regarding cortisol, the results showed the level of serum cortisol concentration increases significantly from Gorgan to Tehran ($P = 0.003$). Further, serum cortisol concentration decreased significantly between Tehran and Khorramabad ($P = 0.045$), but no significant difference was observed in IgM concentration ($P = 0.539$).

F(2,13) = 0.649, P= 0.539

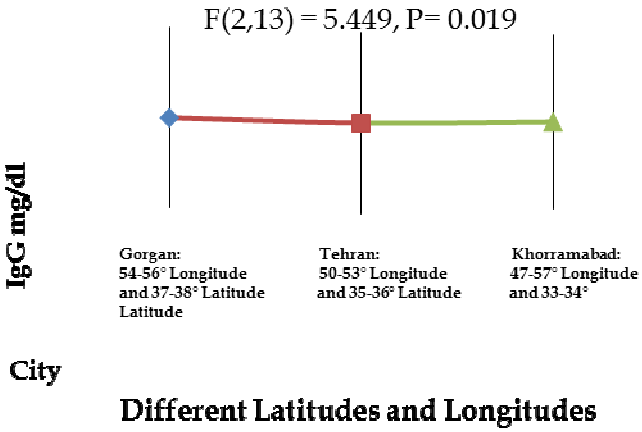


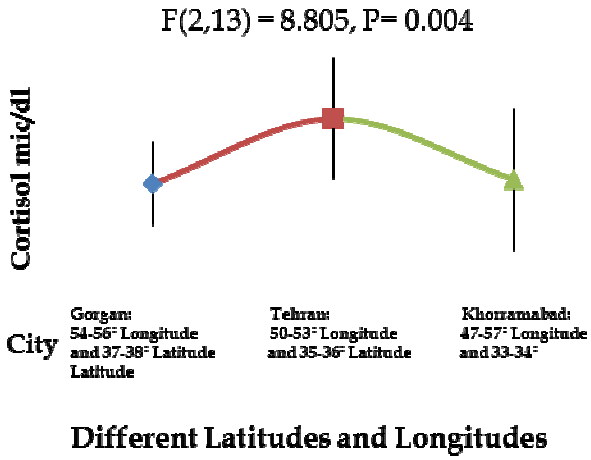
Different Latitudes and Longitudes

F(2,13) = 5.250, P= 0.021



Different Latitudes and Longitudes





DISCUSSION

The purpose of the present research was to determine the effect of sleep deprivation due to land travel on the level of IgG, IgA, IgM, and serum cortisol concentration in young soccer players. Interestingly, the results showed that sleep deprivation due to land travel leads to significant difference in the level of IgG, IgA, and serum cortisol concentration, while no significant difference was observed in the level of IgM concentration. Further, within-group comparisons revealed that there is a significant difference between travelling to Tehran and Khorramabad in IgG and IgA concentration, where the level of IgG and IgA increases in Khorramabad which was farther from city of origin, but decreases in Tehran which has a less distance from the city of origin. By traveling across several time zones, the circadian rhythm of the body retains its initial symptoms. New environmental forces affect these circadian rhythms, mainly at sunrise and sunset, and they act as a time period that adjusts body's clock [21]. An impairment of immunity is reported after long-haul flights, and the mild hypobaric hypoxia caused by pressurization in the passenger airline cabin may contribute to it [6]. This information is helpful for athletes and coaches in planning their itineraries. There is scant evidence regarding the changes in the function of human's immune system during insomnia due to land travelling across various latitudes and longitudes. Thus, the results of the present research support the other findings that examined the effects of sleep deprivation on humans and animals. Hui and colleagues studied the

effect of 24 hours of sleep deprivation on parameters of humoral immunityand observed that the level of serum IgG and IgA increased in the sleep-deprived group and this finding is consistent with our results in the travel to Khorramabad [12]. Costa and colleagues studied the effect of two nights of sleep deprivation with or without energy restriction on immune indices at rest and in response to cold exposure. The results indicated that one night on sleep-deprived (SLED) increased bacterially stimulated neutrophil degranulation and two nights on SLED and SLED+ER (energy-restricted) increased saliva secretory IgA concentration. The decrease in saliva during experiments is probably due to the level of sympathetic balance as a result of anxiety related to prolonged sleep deprivation [5]. 48 hours of energy restriction alone reduces the level of lymphocyte diffusion, while adding sleep deprivation to energy restriction prevents the reduction and diffusion of lymphocytes. These possibilities include the effect of sleep on alternative regulation of local blood flow and possibly discontinuous effects on lymphocyte distribution. Increased distribution of melatonin during sleep deprivation can regulate local blood flow and thus account for the change in redistribution of lymphocytes, but this has not been proven [5]. Zager and colleagues studied the effects of chronic and acute sleep loss on immune modulation of rats. TThe procedure consisted of placing 10 rats in a tiled water tank for 24 to 96 hours. They found that the production of IgM increased which is not consistent with our findings [28]. In another research consistent with our findings, Gümüştekín and colleagues studied the effects of sleep deprivation on wound healing in rats and measured the level of IgG in wound area. They observed that the level of IgG was higher in the sleep-deprived group in comparison with the control group [9]. Everson engaged in the clinical assessment of blood leukocytes, serum cytokines, and serum immunoglobulins as responses to sleep deprivation in laboratory rats. Serum IgM, IgG, and IgA concentration increased dramatically from the fifth to the tenth day of prolonged sleep deprivation. Generally, sleep patterns can provide contradictory findings that can be due to differences in sampling (every 30 minutes of every 24 hours) and the type of sleep deprivation in individuals [8]. Renegar and colleagues studied the effect of sleep deprivation on serum influenza-specific IgG in old mice that were passively immunized intravenously with IgG anti-influenza monoclonalantibodies. Then, the mice were sleep-deprived for either 9 hours (one episode) or for 9 hours followed by 6 hours on the consecutive day (two episodes). They found that sleep deprivation did not increase the level of influenza-specific IgG in serum, but increased it in comparison with mice with regular sleep patterns. They concluded that short-term sleep deprivation has insignificant effect on the existing mucous membrane and secretory immunization in young and old mice [22]. Increase in IgG and IgA concentration somehow may be attributed to reduced level of cortisol and HPA-axis hypoactivity at night-time. It has been reported that there is a close link between sleep deprivation and activity of HPA-axis as well as the level of cortisol [26]. Meanwhile, inhibition of the activity of HPA-axis could be associated with enhanced activity of the growth hormone axis, for HPA-axis is opposite to the growth hormone axis and prolactin affects the immune system [17]. It may well be stated that this was more evident in the participants in the present research who were in the growth age. One of the major effects of glucocorticoids on the immune system is suppressing cell-mediated immunity and increasing lymphatic immunity, a process that takes place through changing T_H1 into T_H2 in production of cytokines [20]. Further, activation of immunoglobulins at the level of B-cells is affected by released cytokines (such as IL2-IL4 and IL-6) from T helper cells. The mechanism is due to increased secretory components including production and release of cytokines such as IL-6 and IL-2 during sleep loss [13]. During normal sleep the level of distribution of lymphocyte subgroups starts again and increases some cell-mediated immunity factors [12]. Thus, increase or decrease in the level of cortisol can at least explain the changes in the reactions of T_H1 into T_H2 during sleep deprivation. In our research, the total level of immunoglobulins increased except for IgM that decreased insignificantly. This shows that activity and wakefulness play an important role humoral-mediated immunity, although

interventions that target sleep might constitute new strategies to constrain inflammation with effects on inflammatory disease risk [13]. Immune functioning decreases with stress and social and physiological stressors are a part of daily life and the source of life changing events [10]. Our results indicated the there is a significant difference in the level of serum cortisol concentration following sleep deprivation due to land travel. Based on Bonferroni test, a significant difference was observed in cortisol concentration between Tehran and Gorgan and as well between Tehran and Khorramabad. Sleep deprivation is an inherently stressful procedure and it may not be possible to completely extricate the effects of SD from general non-specific stress effects. There is evidence regarding the interaction between immune functioning and increased activity of hypothalamus-pituitary-adrenal (HPA) axis in response to stress (Andersen et al., 2005). For example, the levels of plasma cortisol decreases a few hours after sleeping, it increases during deep sleep, and reaches its peak amount early in the morning. The increase and decrease of the level of cortisol in Tehran and Khorramabad respectively may be associated with greater awareness and better sleep during the trip which affects the activity of the HPA-axis. It has been suggested that sleep loss at different times of the night affects the hypothalamic-pituitary-adrenal axis (HPA-axis) differentially [26]. On the other hand, the change in the level of cortisol can also be attributed to the stressors during trips (such as sudden honks, brakes, etc.). It has been reported that the activity of HPA-axis increases in preparation for facing stressors and in response to real stressful events [20]. Hui and colleagues studied the effects of partial sleep deprivation (SD) serum cortisol concentrations in two protocols in healthy adult men. The results indicated that the level of serum cortisol decrease in both protocols after sleep restriction and this is consistent with our results [26]. Lac and Chamoux studied the increasing levels of salivary cortisol as a result of sleep deprivation in shift-workers. The results showed that similar levels of cortisol were measured for evening and night shifts, but an approximately 6-fold increase was observed for the morning shift. This cortisol rise was deduced to be caused by sleep deprivation as a result of rapidly rotating shift patterns and this finding is consistent with our results [15]. Heiser and colleagues examined the level of white blood cells and cortisol after sleep deprivation and recovery sleep in humans. This study involved a night of total sleep deprivation between the first and second days and recovery sleep between the second and the third. The results suggested that cortisol rhythm was affected neither by sleep deprivation nor recovery sleep [11]. Meerlo and colleagues studied the effect of sleep restriction on the hypothalamic-pituitary-adrenal response to stress. The results showed mild increases in ACTH and more robust increases in corticosterone levels. Sleep loss not only had acute and short-lasting effects on the resting levels of the hormones, but it also affected the subsequent HPA response to a novel stressor [18]. Mougin and colleagues studied the hormonal responses to exercise after partial sleep deprivation and after a hypnotic drug-induced sleep. There were 5 sleep deprivation conditions in this research. Plasma growth hormone, prolactin, cortisol, catecholamine and lactate concentrations were measured at rest, during exercise and after recovery. The results showed that the concentration of plasma growth hormone and catecholamine were not affected by partial sleep deprivation, whereas that of plasma prolactin was higher [19]. Sgoifo and colleagues studied the effects of sleep deprivation on cardiac autonomic and pituitary-adrenocortical stress reactivity in rats. The results indicated that sleep deprivation produced a tonic increase of heart rate and HPA axis activity. The data show that sleep deprivation not only affects the baseline activity of the stress system, but it also alters its response to a subsequent stressor. These results were reported for 3 sleep deprivation conditions that were more distinct in comparison with the control group [24]. The direct mechanisms for the interaction between stress and immune function have also been postulated. Stress is associated with alterations in sympathetic nervous system and hypothalamic-pituitary-adrenal axis activation. Functional relationships between these neuroendocrine pathways and the immune system have been acknowledged for some time. Therefore, it is feasible that changes in basal activity in these systems, or their repeated

activation in response to stress, could impact upon antibody development and maintenance. Finally, individual differences in the extent to which these systems respond to standardized stressors have been shown to be predictive of reactivity to real-life stressors [3]. The association between psychological stress and humoral immune (antibody) response to immunization is convincing in the case of secondary immune response but weak for primary response. The lack of consistent evidence for a relation with primary response maybe attributed to a failure to consider the critical points when stress needs to be elevated in the course of the production of antibody. Only in studies of secretory immunoglobulin A antibody the psychological and antibody measures were linked very closely in time. Health practices did not mediate relations between stress and antibody responses; however, there were indications that elevated cortisol levels among stressed patients could play a role [4]. It appears that sleep and athletic performance are related. Many studies have focused on the relationship between sport and sleep and have studied the effects of exercise on the quality and quantity of sleeping [16]. Sleep deprivation may affect the peak power and performance of athletes [25]. These findings show the athletes to be concerned about the effects of inadequate sleep on their performance though the effect of sleep deprivation on physiological performance has not yet been fully understood.

CONCLUSION

Finally, considering the findings of the present research we can come to the conclusion that sleep deprivation due to land travel has a significant effect on serum IgG, IgA, and cortisol concentrations of the participants and increases the concentrations, but it has no significant effect of IgM concentration. It seems that increased cortisol concentration decreases IgG, IgA, and IgM concentrations and vice versa. Considering the results it can be concluded that sleep deprivation plays an important role in various physiological effects on the immune and endocrineparameters and the quality and depth of sleep can be an important determinant of immune function.

REFERENCES

[1] M.L. Andersen, P.J. Martins, V., D'Almeida, M. Bienotto, S. Tufik, *J Sleep Res*, **2005**, 14: 83-90.
[2] A. Bøyum, P. Wiik, E. Gustavsson, O.P. Veiby, J. Reseland, A.H. Haugen, P.K. Opstad, *Scand J Immunol*, **1996**, 43(2): 228-35.
[3] V.E. Burns, D. Carroll, C. Ring, M. Drayson, *Vaccine*, **2003**, 21: 2523-2534.
[4] S. Cohen, G.E. Miller, B.S. Rabin, *Psychosomatic Med*, **200**1, 63: 7–18.
[5] R.J. Costa, A.H. Smith, S.J. Oliver, R. Walters, N. Maassen, J.L. Bilzon, N.P. Walsh, *Eur J Appl Physiol*, **2010**, 109: 417–428.
[6] O. Coste, P. Van Beers, A. Bogdan, Y. Touitou, *Chronobiol Inter*, **2007**, 24(1): 877-98.
[7] C.A. Eriksen, T. Akerstedt, *Chronobiol Int*, **2006**, 23: 843-858.
[8] C.A. Everson, *Am J Physiol Regul Integr Comp Physiol*, **2005.**
[9] K. Gümüştekín, *Int J Neuroscience,* **2004**, 114: 1433–1442.
[10] L. Hawkley, J.T. Cacioppo, *Brain, Beh Immun*, **2004**, 18: 114–119.
[11] P. Heiser, B. Dickhaus, W. Schreiber, H.W. Clement, C. Hasse, J. Hennig, H. Remschmidt, J.C. Krieg, W. Wesemann, C. Opper, *Eur Arch Psychiatry Clin Neurosci*, **2000**, 250: 16-23.
[12] L. Hui, F. Hua, H. Diandong, Y. Hong, *Brain, Beh Immun*, **2007**, 21:308–310.
[13] M. Irwin, J. Thompson, C. Miller, J.C. Gillin, M. Ziegler, *J Clin Endocrinol Metab*, **1999**, 84(6): 1979–1985.
[14] M.D. Irwin, M. Wang, C.O. Campomayor, A. Collado-Hidalgo, S. Cole, *Arch Intern Med*, **2006,** 166: 1756-1762.
[15] G. Lac, A. Chamoux, *Occupational Med*, **2003**, 53: 143-145.

[16] B.J. Martin, *Eur J Appl Physiol*, **1981**, 47: 345-354.
[17] E. Maurovich-Horvat, T. Pollmächer, K. Sonka, *Prague Medical Report*, **2008**, 109(4): 275-285.
[18] P. Meerlo, M. Koehl, K. Van Der Borght, F.W. Turek, *J Neuroendocrinology*, **2002,** 14: 397-402.
[19] F. Mougin, H. Bourdin, M.L. Simon-Rigaud, N.U. Nguyen, J.P. Kantelip, D. Davenne, *J Sports Sci*, **2001**, 19: 89-97.
[20] B.D. Palma, P.A. Tiba, R.B. Machado, S. Tufik, D. Suchecki, *Rev Bras Psiquiatr*, **2007**, 29: S33-8.
[21] T. Reilly, G. Atkinson, J. Waterhouse, *J Sports Sci*, **1997**, 15(3): 365-369.
[22] K.B. Renegar, R.A. Floyd, J.M. Krueger, *Sleep*, **1998**, 21(1): 19-24.
[23] R.V.T. Santos, S. Tufik, M.T. De Mello, *Sleep Med Rev*, **2007**, 11: 231-239.
[24] A. Sgoifo, B. Buwalda, M. Roos, T. Costoli, G. Merati, P. Meerlo, *Psycho neuro endocrinology*, **2006**, 31: 197-208.
[25] N. Souissi, B. Sesboüé, A. Gauthier, J. Larue, D. Davenne, **2003.**
[26] H. Wu, Z. Zhao, W.S. Stone, L. Huang, Z. Zhuang, B. He, P. Zhang, Y. Li, *Brain res Bulletin*, **2008**, 77: 241-245.
[27] M. Young, P. Fricker, R. Maughan, D. MacAuley, *Br Sports Med*, **1998**, 32: 77-81.
[28] A. Zager, M.L. Anderson, F.S. Ruiz, I.B. Antunes, S. Tufik, *Am J Physiol Regul Integr Comp Physiol*, **2007**, 293:504-509.