The effects of intensive exercise after one night's sleep deprivation on muscle damage indexes in the male runners

Sajad Arshadi\textsuperscript{1*} and Mehdi Noora\textsuperscript{2}

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(1) Assistant professor in exercise physiology; Department of exercise physiology, South Tehran Branch, Islamic Azad University, Tehran, Iran., E-mail: arshadi.sajad@yahoo.com

(2) Assistant professor in exercise physiology; Department of exercise physiology, Shiraz Branch, Islamic Azad University, Shiraz, Iran

Abstract

Introduction: Sleep is generally considered to be a restorative process, having beneficial effects on physiological functions. Evidence suggests athletes worry about the effects of inadequate sleep on performance. The aim of present study was to examine the effects of intensive exercise after one night's sleep deprivation on muscle damage indexes in the male runners.

Material & Methods: Twenty four collegiate male runners in Tehran volunteered to participate in this study as the subject. All the subjects, 72h days before sleep deprivation and after about 8 hours of complete sleep were performed intensive exercise. Three days later, after a 30 hours' sleep deprivation, intensive exercise were performed again. Blood samples were taken before the complete sleep or sleep deprivation and before and after the intensive exercise to measure serum creatine kinase (CK), lactate dehydrogenase (LDH) and Aspartate transaminase (AST).
Results: The results indicated that serum CK, LDH and AST were higher in response to intensive exercise after the sleep deprivation in compare to the intensive exercise after the complete sleep.

Conclusions: In summary, our results suggest that the sleep deprivation may lead to high levels of muscle damage enzymes and is may be impairs athletic performances.

Keywords: Sleep deprivation, Muscle damage, Lactate dehydrogenase, Intensive exercise

1. Introduction

Sleep is a basic human need and a healthy adult will spend about one-third of his/her life sleeping (1). Although all the functions of sleep are still unknown (2), it has been reported through the years that sleep is closely related to physical and mental health, cognitive processes and metabolic function (3). Complete rest or sleep is still seen as the main means of restoring physical working capacity, as well as mental restoration (4,5). Although many regard sleep as the most significant aspect of recovery, Walters (2002) maintains that athletes often neglect this fact. Research findings indicate that athletes generally sleep less than non-athletes and often have difficulty sleeping (6). It is important for athletes to understand how sleep affects performance and recovery, know which factors could affect sleep quality and be able to develop optimal sleeping habits.

Sleep deprivation consists either in a complete lack of sleep during a certain period of time or a shorter-than optimal sleep time. The most common causes of sleep deprivation are those related to contemporary lifestyle and work-related factors; thus the condition affects a considerable number of people. A chronic reduction in the sleep time or the fragmentation of sleep, leading to the disruption of the sleep cycle (7), may have consequences comparable to those of severe acute sleep deprivation; this referring particularly to the cognitive functions, attention and operant memory (8-10). The changes in sleep time across the circadian pattern (11), such as during shift work (12-15) or air travel (jet-lag syndrome resulting from changing time zones) (16), prove to be unfavorable as well. Many people also experience mild discomfort while
Sleep deprivation and exercise induced-muscle damage

adjusting to the daylight saving time. Sleep deprivation lasting as long as several days usually takes place in extreme situations or under experimental conditions.

Although athletes and coaches believe that adequate sleep is essential for peak performance, there are many situations in which sleep is disturbed prior to an athletic event. An athlete may lose sleep owing to jetlag or anxiety (17). Evidence suggests athletes worry about the effects of inadequate sleep on performance (18), but there are a limited number of studies which have examined the effects of sleep deprivation on athletic performance and they have produced conflicting results. For example, Rodgers et al. (1995) reported that 48 hours period of sleep deprivation significantly decreased the physical work tasks requiring 30-45% VO2max without affecting anaerobic power (19). Further, Souissi et al. (2003) demonstrated that duration of sleepless period may be important as peak power was not affected after 24 hours sleep deprivation but significantly decreased after 36 hours of wakefulness (20). Blumert and colleagues (2007) examined the effects of 24 h of sleep deprivation in nine U.S. college-level weightlifters in a randomised counter-balanced design. There were no differences in any of the performance tasks (snatch, clean and jerk, front squat and total volume load and training intensity) following 24 h of sleep deprivation when compared to no sleep deprivation (21). The mechanism behind the reduced performance following prolonged sustained sleep deprivation is not clear; however, it has been suggested that an increased perception of effort is one potential cause. While the above studies provide some insight into the relationship between sleep deprivation and performance, most athletes are more likely to experience acute bouts of partial sleep deprivation where sleep is reduced for several hours on consecutive nights.

Regular physical exercise has many health benefits including a lowered threat of all-cause mortality along with a reduced risk of cardiovascular disease, cancer, and diabetes (22). Paradoxically, it is also clear that contracting skeletal muscles generate free radicals and that prolonged and intense exercise can result in oxidative damage to cellular constituents (23). Exercise-induced muscle damage is associated with muscle soreness or discomfort and a marked decline of muscle strength during the first 12–72 hours post-exercise (24). Furthermore, exercise-
induced muscle damage leads to the onset of an inflammatory response that is associated with the activation of leukocytes, muscle edema, deterioration of muscle function, delayed-onset of muscle soreness, and several intracellular events that aim to restore the integrity and function of the affected muscle (25). The effects of intensive exercise on muscle damage after sleep deprivation are not well known, thus the present study was done to examine the effects of intensive exercise after one night's sleep deprivation on muscle damage indexes in the male runners.

2. Materials and Methods

Subjects
Twenty four collegiate male runners volunteered to participate in this study. The subjects were given both verbal and written instructions outlining the experimental procedure, and written informed consent was obtained. Anthropometric characteristics of the subject are presented in the Table 1.

<table>
<thead>
<tr>
<th>Age (Year)</th>
<th>24.8 ± 3.1</th>
</tr>
</thead>
<tbody>
<tr>
<td>Body weight (Kg)</td>
<td>71.3 ± 4.6</td>
</tr>
<tr>
<td>Height (cm)</td>
<td>175.0 ± 4.7</td>
</tr>
<tr>
<td>BMI (Kg/m²)</td>
<td>23.2 ± 4.6</td>
</tr>
</tbody>
</table>

Study protocol
The experimental protocol was in two parts, A and B. In Part A, each subject slept at home (retiring at any time between 22:30 and 23:00 hours, as decided individually), rising at 07:00 hours to come to the laboratory. All the subjects after about 8 hours of complete sleep were performed intensive exercise. Intensive exercise consists of 100m sprint running and 4 × 9m shuttle run test. Three days later, Part B was done. Part B was identical except that no sleep was allowed during the night. Subjects arrived at the laboratory at about 22:00 hours and were not allowed to go to sleep at their usual bedtime. During this time, subjects did such things as watch television, read books, play music and chess or work at a computer. They were strictly supervised by two experimenters so as to ensure that no-one dozed off or consumed beverages containing
caffeine. After a 30 hours' sleep deprivation, intensive exercise were performed again. 3 days before the beginning of the study, each subject was supervised to continue his normal sport nutrition program. On the testing day the subjects were supervised not to use any sport or dietary supplements.

**Biochemical analysis**

Blood samples were taken from an antecubital vein in the sitting position. 5 milliliters blood from a vein was taken at 23:00 (before the complete sleep or sleep deprivation) and at 7:00 (before the intensive exercise) and at 10:00 (after the intensive exercise) to measure serum creatine kinase (CK), lactate dehydrogenase (LDH) and Aspartate transaminase (AST). CK and AST levels were measured using colorimetric analysis. The intra and inter-assay coefficients of variation for CK and AST were <1.6% and <1.9% respectively and a sensitivity of 1 U/l and 5 µg/l respectively. LDH concentrations were measured via enzymatic colorimetric method. The intra and inter-assay coefficients of variation for LDH were <2.1% and a sensitivity of 5 U/l.

**Statistical analysis**

Results were expressed as the mean ± SD and distributions of all variables were assessed for normality. 2 × 3 repeated measures ANOVA was used to evaluate time-course change in variables. Post hoc analyses (Bonferroni) were then performed when warranted and independent t-test was used to compute differences in the variables. The significant level of this study was set at P<0.05 and the data were analyzed using SPSS software for Windows (version 22, SPSS, Inc., Chicago, IL).

**3. Results**

Changes on serum CK, LDH and AST concentrations are presented in Table 2. The results indicated that there were no significant differences in serum CK concentrations in adequate sleep and sleep deprivation conditions between blood sampling at 23:00 and at 7:00. Our results revealed that although serum CK concentrations increases after the intensive exercise in the both condition, the increase of serum CK was higher in sleep deprivation than adequate sleep condition (P<0.001). For
serum LHD and AST concentration, the results also showed that there were no significant differences in adequate sleep and sleep deprivation conditions between blood sampling at 23.00 and at 7:00. By according to the Table 2, the results revealed that although serum LHD and AST concentrations increases after the intensive exercise in both condition, the increase of serum LHD and AST levels were higher in sleep deprivation than the adequate sleep condition (P<0.001).

<table>
<thead>
<tr>
<th>Parameter</th>
<th>23:00 Mean±SD</th>
<th>7:00 Mean±SD</th>
<th>10:00 Mean±SD</th>
<th>F</th>
<th>Sig</th>
</tr>
</thead>
<tbody>
<tr>
<td>CK (U/l)</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Adequate sleep</td>
<td>151.2±3.1</td>
<td>148.7±3.1</td>
<td>155.6±3.3*</td>
<td>562.5</td>
<td>0.001</td>
</tr>
<tr>
<td>Sleep deprivation</td>
<td>148.2±3.2</td>
<td>152.0±3.9</td>
<td>199.8±4.1*</td>
<td></td>
<td></td>
</tr>
<tr>
<td>LDH (U/l)</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Adequate sleep</td>
<td>309.3±3.5</td>
<td>306.0±4.5</td>
<td>313.7±3.5*</td>
<td>278.2</td>
<td>0.001</td>
</tr>
<tr>
<td>Sleep deprivation</td>
<td>311.2±4.6</td>
<td>310.2±5.2</td>
<td>336.1±6.1*</td>
<td></td>
<td></td>
</tr>
<tr>
<td>AST (µg/l)</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Adequate sleep</td>
<td>15.9±0.4</td>
<td>15.3±0.5</td>
<td>16.4±0.6*</td>
<td>57</td>
<td>0.001</td>
</tr>
<tr>
<td>Sleep deprivation</td>
<td>15.0±0.5</td>
<td>15.3±0.6</td>
<td>17.2±0.6*</td>
<td></td>
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</tr>
</tbody>
</table>

* Significant differences with baseline (P<0.001)
† Significant differences between groups (P<0.001)

4. Discussion

The aim of present study was to examine the effects of intensive exercise after one night's sleep deprivation on muscle damage indexes in the male runners. The results in line with Twist and Eston (2005) study indicated that serum CK, LDH and AST increases after intensive exercise (P<0.001). Twist and Eston (2005) also demonstrated that serum CK increases after maximal intermittent sprint performance (26). CK and LDH are fragments of the myosin heavy chain (i.e., troponin I and myoglobin) and are related to muscle damage, this is because these molecules are cytoplasmatic and do not have the capacity to cross the sarcoplasmic membrane barrier (27). For this reason, increased serum concentrations of these molecules are used as an indicator of damage to muscle membrane and other tissue structures (28). Exercise-induced muscle damage is a common occurrence following activities with a high eccentric component, such as resistance training, plyometrics, distance
Sleep deprivation and exercise induced-muscle damage

running and prolonged, intermittent shuttle running (29,30). Exercise-induced muscle damage is associated with muscle soreness or discomfort and a marked decline of muscle strength during the first 12–72 hours post-exercise (24). Furthermore, exercise-induced muscle damage leads to the onset of an inflammatory response that is associated with the activation of leukocytes, muscle edema, deterioration of muscle function, delayed-onset of muscle soreness, and several intracellular events that aim to restore the integrity and function of the affected muscle (25).

The results of present revealed that the increase of CK, LDH and AST concentrations after the intensive exercise were higher in sleep deprivation than the adequate sleep condition (P<0.001). The effects of sleep deprivation on a person’s rating of perceived exertion, mood and cognitive functions are well documented (31). However, when it comes to the effect of sleep deprivation on physical or physiological performance, the findings are conflicting and less conclusive. The reason for the discrepancies may be that various exercise modes, frequencies, intensities and durations have been employed, as well as different evaluation procedures (32). Other factors that may explain the differences among such studies are varying durations of sleep deprivation and subjects’ ages (20). A few studies of short-term maximal effort have reported how supramaximal performance can be maintained despite sleep deprivation. Symons et al. (1988) demonstrated no significant change in the isometric strength of flexors and extensors, peak isokinetic torque at 3.14 rad/s, muscular endurance, peak power output, fatigue index or blood lactate after the Wingate anaerobic test (32). Takeuchi et al. (1985) further showed that 64 h without sleep did not impair isometric hand-grip strength or peak torque for leg extension at 3.14 and 5.22 rad/s. However, sleep deprivation did impair vertical jump and knee extension torque at low velocity (1.04 rad/s) (33). Conversely, Bulbulian et al. (1996) reported that sleep loss of up to 30 h affected peak torque but had no effect on the fatigue index (34).

There is a very little information about the effects of sleep deprivation on exercise induced-muscle damage. In only available study, recently Dátillo et al. (2020) was studied the effects of sleep deprivation on acute skeletal muscle recovery after the eccentric contractions of the knee extensor muscles using an isokinetic dynamometer. Muscle damage
protocol was followed by 48 h of total sleep deprivation and 12 h of normal sleep. In the other condition, the same muscle damage protocol was conducted, followed by three nights of regular sleep. Blood samples were collected serially throughout the protocol to analyze the CK level, hormonal changes and to indicate the inflammatory and anti-inflammatory markers. Dáttilo et al. (2020) found that muscle voluntary contraction and serum CK increased equally over the study period in both conditions (35).

5. Conclusion
In summary, our results suggest that intensive exercise may lead to muscle damage recognized by the increase of serum CK, LDH and AST levels. Indeed the results indicate that prolonged sleep restriction can aggravate muscle damage processes as a result of intensive exercise. Thus having required sleep time is advised to all of the athletes who may be at the risk of exercise induced-muscle damage.

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Conflict of interests: No conflict of interests amongst authors

References


