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Muscle damage and soreness following a 50-km cross-country ski race

Running head: Cross-country skiing and muscle damage

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ABSTRACT

This study examined indirect markers of muscle damage and muscle soreness following a 50-km cross-country ski race completed between 2 h 57 min and 5 h 9 min by 11 moderately trained male university students. Maximal strength of the knee extensors (MVC), several blood markers of muscle damage and inflammation, and muscle soreness (Visual analog scale, 0: no pain, 50 mm: unbearably painful) were measured one day prior to, immediately after, and 24, 48, 72, and 144 h following the race. Changes in the measures over time were analyzed using one-way repeated measures ANOVA with a Fisher's post hoc test. MVC decreased significantly ($P<0.05$) immediately after the race (-27 ± 6 %), but returned to the pre-exercise level by 24 h post-race. All blood markers increased significantly ($P<0.05$) following the race, peaking either at immediately (lactate dehydrogenase: 253.7 ± 13.3 $IU \cdot L^{-1}$ and myoglobin: 476.4 ± 85.5 $ng \cdot ml^{-1}$) or 24 h post-race (creatine kinase: 848.0 ± 151.9 $IU \cdot L^{-1}$ glumatic oxaloacetic transaminase: 44.3 ± 4.2 $IU \cdot L^{-1}$, aldolase: 10.0 ± 1.3 $IU \cdot L^{-1}$ and C-reactive protein: 0.36 ± 0.08 $IU \cdot L^{-1}$). Muscle soreness developed in the leg, arm, shoulder, back, and abdomen muscles immediately after the race (10-30 mm), but decreased after 24 h (<15 mm), and disappeared after 48 h post-race. These results suggest that muscle damage induced by the 50-km cross-country ski race is mild and recovery from the race does not take long.

Key words: isometric strength, creatine kinase, myoglobin, aldolase, C-reactive protein

INTRODUCTION

Long distance running, such as a marathon (42.195 km), induces muscle damage indicated by ultra structural alterations of muscle fibers (Hagerman et al., 1984; Hikida et al., 1983), increases in muscle proteins such as creatine kinase and myoglobin in the blood (Nuviala et al., 1992; Warhol et al., 1985), decreases in muscle function (Nicol et al., 1991; Sherman et al., 1984), and development of delayed onset muscle soreness (Liang et al., 2001). It is documented that the recovery from a marathon race takes at least several weeks even for well-trained marathon runners (Hagerman et al., 1984). This may be one of the reasons why marathon runners can participate in only a few marathon races a year.

In contrast, cross-country skiers generally participate in several long distance races in a season. The International Ski Federation (FIS) Marathon Cup consists of eight races over 42 km in distance in four months, and athletes compete for the total points gained from each race. This means that elite cross-country skiers can take part in a race with a relatively short interval compared to marathon runners. Although a similarity exists between a 50-km cross-country ski race and a marathon (42.195 km) race based on the exercise time (approximately 2 hours for the top-level athletes), it is interesting to note that the number of races that athletes can participate in during a season is different between the two events.

One of the reasons making it possible for cross-country skiers to participate in many races in a season may be associated with less muscle damage induced by a cross-country ski race compared with running a marathon race. In fact, changes in blood markers of muscle damage appear to be smaller following a cross-country ski race than a marathon race. Ronsen et al. (2004) reported a small (86%) increase in plasma creatine kinase (CK) activity after a 50-km (male) or 30-km (female) ski race. Changes in muscle function after cross-country skiing have also been reported (Millet et al., 2003); however, few studies have systematically investigated changes in markers of muscle damage after a cross-country ski

race.

Therefore, the purpose of this study was to examine changes in muscle function, blood markers of muscle damage and inflammation, and muscle soreness following a 50-km cross-country ski race.

METHODS

Subjects and a cross-country ski race

Eleven male university students participated in this study as subjects. Prior to involvement in the study, the subjects signed informed consent documents, which were approved by the Institutional Ethics Committee and in accordance with the ethical standards of the Declaration of Helsinki. Their mean ± SD age, body mass, and height were 21.2 ± 1.9 yrs, 63.0 ± 6.7 kg, and 171.9 ± 4.2 cm, respectively. All subjects were moderately trained and belonged to either a cross-country skiing (n=4) or rowing club (n=7) of a university. They had been free from any musculoskeletal disorders and in good health when they participated in the 23rd Sapporo International Ski Marathon (50 km). All of them used skating techniques to compete the race that had the altitude difference of 187 m, and the average air and snow temperature during the race were 1°C, and 0°C, respectively. Although all subjects had participated in a local cross-country ski race prior to the race, the race distance (50 km) was novel for 8 subjects, and 3 subjects had experienced the distance more than 9 months prior to the race. Subjects were requested to refrain from any nutritional supplements or medicines, including anti-inflammatory drugs, before and for a week after the race.

Criterion measures

Maximal voluntary isometric strength of the knee extensors (MVC) measurement and blood samples were taken 1 day prior to the race, within 15 minutes after, and then 24, 48, 72, and 144 h following the race.

MVC was assessed using a load-cell (model 1269, Takei Scientific Instruments Co. Ltd., Tokyo, Japan) attached to a steel wire with a belt surrounding the ankle joint, while the subject was seated on a specially designed chair which adjusts the knee and hip joint angles to 90°, and secures other parts of the body by straps. Subjects were asked to generate maximal force for 3 seconds under verbal encouragement, and this measurement was taken twice with a rest of 60 s between attempts. Peak MVC was displayed on a digital recorder (model 9E54-7D, NEC San-Ei Co. Ltd., Tokyo, Japan) connected to the load cell, and the higher value of the two measurements was used for further analysis. Prior to the pre-exercise measurements, the subjects were familiarized with the MVC measurement protocol, and the reliability of the measure was confirmed by an Intra class correlation (r=0.9).

Approximately 6 ml of blood was drawn from the antecubital vein by a standard venipuncture technique, and centrifuged for 10 minutes to obtain plasma. The plasma samples were frozen and stored at -20°C until analyses for creatine kinase (CK), lactate dehydrogenase (LDH), glutamic oxalacetic transaminase (GOT), and aldolase (ALD) activities, myoglobin (Mb) and C-reactive protein (CRP) concentrations. These measures were chosen based on previous studies (Driessen-Kletter et al., 1990; Nosaka et al., 1992) that assessed muscle damage. CK, LDH, GOT and CRP were measured by an automatic analyzer (AU-5421, Olympus, Tokyo, Japan) using test kits (CK: Nitto Boseki Co. Ltd., Tokyo, Japan; LDH and GOT: Wako Pure Chemical Industries Ltd., Osaka, Japan; CRP: Denka Seiken Co. Ltd., Tokyo, Japan). ALD was measured by a different analyzer (Hitachi 7170, Hitachi, Tokyo, Japan) with a test kit (Wako Pure Chemical Industries Ltd, Osaka, Japan). Plasma Mb concentration was determined using γ-counter (ARC-950, ALOKA Co.

Ltd., Tokyo, Japan) and a test kit (Daiichi Radioisotope labs Ltd., Tokyo, Japan). The normal reference ranges for male adults are CK: 57-197, LDH: 115-245, GOT: 10-40, ALD: 1.7-5.7 $IU \cdot L^{-1}$, Mb: $<60 ng \cdot m^{-1}$, and CRP: $<0.5 mg \cdot dl^{-1}$, according to the information provided in the test kits.

Muscle soreness was assessed by a visual analog scale consisting of a 50 mm line with "no pain" on one end (0) and "unbearably painful" on the other (50). This method has been used in previous studies (Nosaka et al., 2001; Nosaka et al., 2002), and its reliability has been established (Ohnhasu and Adler, 1975). Subjects were asked to mark their subjective soreness level on the line, provided in a questionnaire, when contracting and stretching the following muscles; deltoid, pectoralis, biceps brachii/brachialis, triceps brachii, brachioradialis, rectus/obliquus abdominis, vastus lateralis/quadriceps femoris, biceps femoris, gastrocnemius/soleus, and lower back.

Statistical analysis

Changes in the criterion measures over time were analyzed using one-way repeated measures ANOVA. When a significant time effect was found, a Fisher's protected least significant difference test was performed to locate a difference from the pre-exercise value. Significant level was set at $p<0.05$. The results are presented as mean ± SEM.

RESULTS

All subjects completed the race between 2 h 57 min and 5 h 9 min, and the mean finishing time of the 11 subjects was 3 h 42 ± 13 min. The time of the winner of the race among all participants was 2 h 18 min.

Figure 1 shows normalized changes in MVC from the pre-race value. MVC decreased

significantly immediately after the race (-27 ± 6 %), but returned to the baseline by 24 h post-race.

All blood markers showed significant increases following the race, however, the magnitude of increase and the time course of the changes varied among the measures (Figure 2). Plasma CK activity increased by 230% immediately after, peaked (331%) 24 h after, and returned to baseline by 72 h post-race. Mb showed the largest increase (832%) among the markers immediately after race, but no significant difference from baseline was observed 24-144 h post-race. The magnitude of increase in LDH (44%), GOT (84%), and ALD (71%) was smaller than that of CK and Mb, and the peak occurred either immediately (LDH) or 24 h (GOT, ALD) post-race. CRP was significantly higher than pre-race value at 24 (364%) and 48 h (209%) post-race, but remained in the normal reference range.

As shown in Table 1, muscle soreness peaked immediately after exercise for most of the muscles, but disappeared by 48 h post-race. No muscle soreness existed in the pectoralis, biceps brachii/brachialis, and abdominal muscles. The most profound muscle soreness was observed for the knee extensors (vastus lateralis/quadriceps femoris), but the level of the soreness 1-2 days post-race (<15 mm) was considered light.

DISCUSSION

To our knowledge, this is the first study to systematically investigate indirect markers of muscle damage in a 50-km cross-country ski race. The results showed 27% decrease in MVC (Figure 1), increases in blood markers of muscle damage and inflammation (Figure 2), and development of muscle soreness in the leg and arm muscles (Table 1) following the race. However, a significant decrease in MVC from baseline was evident only immediately post-race (Figure 1), and the increases in the blood markers of muscle damage and

inflammation (Figure 2) were not as large as those reported after a marathon run (Asp et al., 1999; Liang et al., 2001) or the maximal eccentric exercise (Clarkson et al., 1992; Nosaka and Clarkson, 1996). Moreover, muscle soreness did not exacerbate 1-2 days after the race (Table 1). These results suggest that the magnitude of muscle damage induced by the 50-km cross-country race was not severe.

Ronsen et al. (2004) reported a significant increase in plasma CK activity but no change in CRP immediately after a 50-km (male) or 30-km (female) ski race. Increase in plasma CK activity (130%) immediately post-race in the present study was larger than that in the study (86%) by Ronsen et al. (2004). It has been documented that the training level of subjects influences the magnitude of increase in plasma CK activity after exercise, and increases in blood markers of muscle damage are attenuated with training (Eston et al., 1996; Evans et al., 1986; Vincent and Vincent, 1997). The subjects in the present study were moderately trained, while the study by Rosen et al. (2004) used well-trained athletes. This may be the reason why the present study observed larger increases in plasma CK activity and significant increases in CRP (Figure 2). However, it should be noted that the CRP concentration remained in the normal reference range post-race. This suggests that inflammation after cross-country skiing is minimal. Zeppilli et al. (1985) showed a large increase in CK activity in the blood ($2,832 IU \cdot L^{-1}$) immediately after a 189-260 km cross-country ski race in moderately trained athletes. It seems likely that the larger increase in CK in the study by Zeppilli et al. (1985) was due to the longer distance and exercise time (189-260 km/ 24 h) compared to those in the present study.

It is generally accepted that increases in muscle proteins in the blood after exercise indicate plasma membrane damage (Hikida at al., 1983; Hagerman et al., 1984), and delayed large increases in plasma CK activity and Mb concentration are observed following eccentric exercise (Nosaka et al., 1992). Some of the blood markers showed further increases up to 24

h post-race; however, the magnitude of increase was not large (Figure 2). The smaller changes in LDH and GOT compared to CK, Mb, and ALD are likely associated with their less specificity to skeletal muscle damage (Croisier et al., 1999; Driessen-Kletter et al., 1990; Nosaka et al., 1992). Although cardiac muscle and liver damage cannot be ruled out for the source of these proteins, it appears that skeletal muscle damage was the main cause of the increases.

Asp et al. (1999) and Liang et al. (2001) reported that plasma CK activity peaked 24 h after running a marathon (2,655 $IU \cdot L^{-1}$, and 2,437 $IU \cdot L^{-1}$, respectively) in moderately trained marathon runners and triathletes. Another study also showed that plasma CK activity peaked 24 h (1,147 $IU \cdot L^{-1}$), and elevated for 96 h following running a marathon race in moderately trained runners (Kyrolainen et al. 2000). In the present study, plasma CK activity peaked (848 $IU \cdot L^{-1}$) 24 h post-race and returned to baseline by 72 h post-race. It is documented that plasma membrane damage is followed by muscle cell necrosis when the membrane damage is severe (Armstrong et al., 1991; Proske and Morgan, 2001). In fact, it has been reported that a marathon race or a long distance running causes ultra-structural muscle damage (Hagerman et al., 1984; Hikida et al., 1983). Although histological measures were not included in the present study, it seems likely that ultra-structural damage in a 50-km cross-country ski race is less than that in a marathon race. Since trained muscles are less susceptible to eccentric exercise-induced muscle damage (Vincent and Vincent, 1997), it can be assumed that the magnitude of increase in plasma CK activity would have been smaller if more trained cross-country skiers had been used as subjects in the present study. Thus, it seems reasonable to conclude that the magnitude of increase in plasma CK activity following the cross-country ski race is smaller than that of a marathon race.

It is well known that delayed onset muscle soreness (DOMS) is peculiar to eccentric exercise, and muscle damage is associated with DOMS (Armstrong, 1984; Miles and

Clarkson, 1994; Nosaka et al., 2002; Gibala et al., 1995). Muscle soreness after a marathon has been reported to persist for several days post-race (Liang et al., 2001). Although muscle soreness developed in several muscles after the cross-country ski race, it peaked immediately after and disappeared by 48 h post-race (Table 1). The muscles demonstrating highest soreness were the knee extensors, but its magnitude was less than 15 mm at 24 h post-race (Table 1), which is considered light. Cross-country ski skating requires extension movement of the knee, ankle and hip joint (Perrey et al., 1998), and poling generates propulsive force (Smith, 1992). It seems reasonable to assume that these movements induced muscle soreness, but eccentric muscle actions in the movements were limited. It may be that muscle fatigue rather than muscle damage was responsible for the soreness developed immediately after the race (Armstrong, 1984).

Smith (1992) has reported that poling provided most of the propulsive force (about 66%), and suggested that the major uphill propulsive forces are derived from the upper body. Although subjects performed a large number of repetitions of poling in the race, muscle activity of poling are likely to be mainly concentric actions. This is supported by the light development of muscle soreness in the upper limbs (Table 1). The present study did not measure muscle strength of upper body muscles; however, it seems unlikely that a large decrease in muscle strength would have been observed for these muscles after a cross-country ski race.

A prolonged decrease in MVC is an index of muscle damage (Clarkson et al., 1992; Warren et al., 1999). The magnitude of decrease in MVC after the cross-country ski race (27%) in the present study was larger than that reported by Millet et al. (2003) who found 8.4% decrease in knee extensor strength of trained skiers after a 42-km cross-country ski. The larger decrease in MVC in the present study is likely due to the fact that the subjects of the present study were not highly trained, and they were unaccustomed to the distance. It

has been shown that MVC of the knee extensors decreased 22% after a marathon (Nicol et al., 1991). Sherman et al. (1984) reported that maximal peak torque of the knee extensors decreased 36% immediately after a marathon race, remained at that level for 72 h, and recovered to baseline at 168 h post-exercise. The magnitude of strength loss immediately after the cross-country ski race was comparable to that reported after running a marathon race, but the recovery was quicker and MVC returned to the baseline by 24 h post-race (Figure 1). It would appear that the decrease in MVC after the cross-country ski was due to muscle fatigue rather than muscle damage. It is known that endurance exercise results in impairment of neuromuscular function (Gandevia, 2001; Millet et al., 2003), which affects the voluntary force output. The fast recovery of muscle strength after the cross-country ski race suggests that the magnitude of muscle damage in cross-country skiing is less than that in a marathon race.

Gerritsen et al. (1995) reported that ground reaction force in low speed running was 1.5 times the body mass at a heel strike phase. In contrast, in cross-country skiing, ground reaction force is about 0.5 fold of body mass immediately after a ski touches the ground in diagonal technique (Komi, 1987). It seems likely that the higher the grand reaction force, the greater the eccentric load for the muscles to absorb the impact. It has been shown that the lower limb extensor muscles perform stretch-shortening cycle (SSC) actions in ski skating (Perrey et al., 1998). Repeated SSC muscle actions are known to induce muscle damage (Avela et al., 1999; Horita et al., 1999). However, the magnitude of eccentric load in the SSC muscle actions is also likely to be smaller in cross-country skiing than running because of the smaller impact from the ground in cross-country skiing. These findings may explain the less muscle damage induced in cross-country skiing than in a marathon. Further study is necessary to confirm this speculation.

In summary, the present study showed that 50-km cross-country ski race induced

decrement of muscle function immediately post-race, increased blood markers of muscle damage and inflammation, and developed muscle soreness. However, the magnitude of change in these measures was relatively small compared to a marathon race. It is concluded that muscle damage induced by a cross-country ski race is not severe and recovers in a short period of time. This appears to explain why cross-country skiers can take part in several races in a season.

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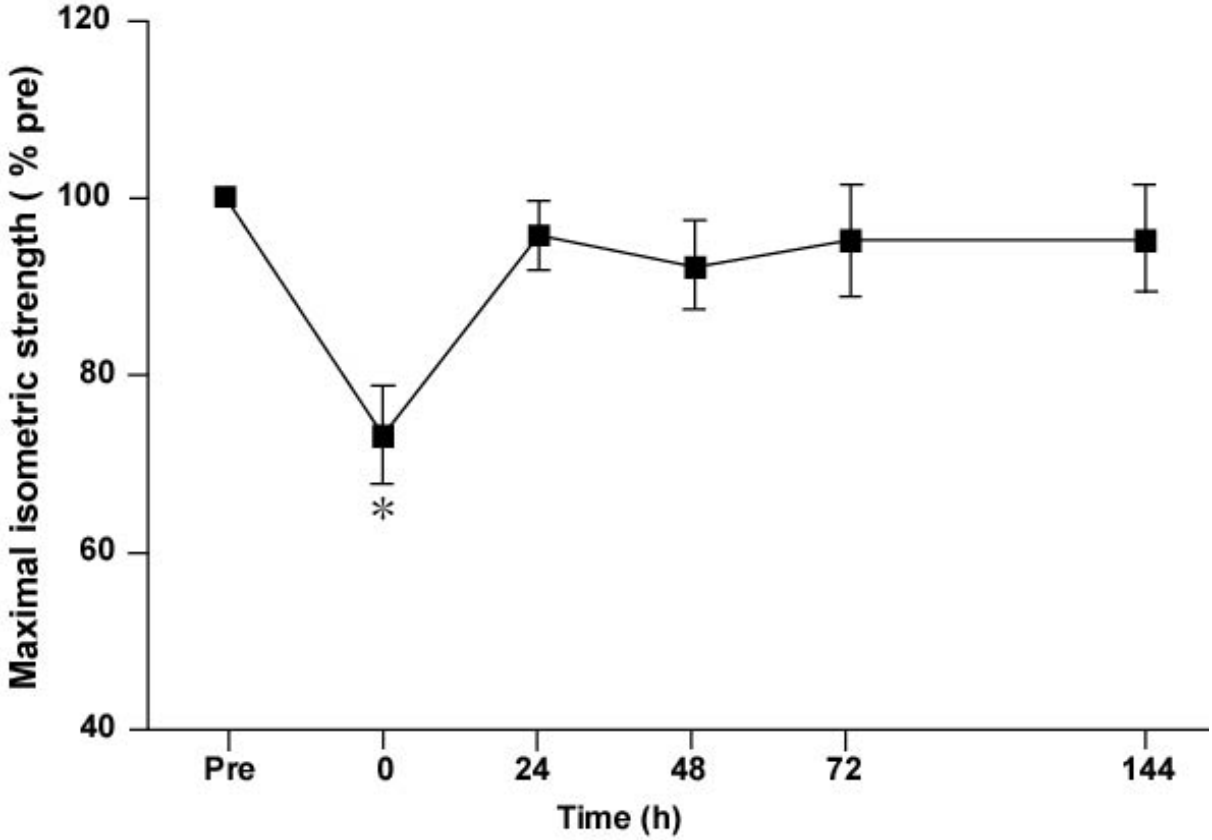
Table 1: Changes in muscle soreness of the listed muscles before (pre), immediately (0) and 24-144 h after the cross-country ski race. Mean (SEM) values of 11 subjects are shown. * indicates a significant ($p<0.05$) difference from the pre value.

Muscle	pre	0	24	48	72	144
deltoid	5.8 (2.8)	10.2 (3.9) *	7.9 (3.8)	7.5 (3.7)	4.6 (2.3)	5.5 (2.5)
pectoralis	4.5 (2.0)	4.5 (2.2)	4.1 (2.3)	2.9 (1.7)	2.7 (1.7)	6.0 (2.2)
biceps brachii/brachialis	6.3 (3.3)	10.4 (3.5)	4.5 (2.1)	3.6 (2.3)	2.4 (1.6)	2. 1 (1.2)
triceps brachii	2.7 (1.2)	14.5 (4.6) *	8.6 (3.0) *	6.5 (2.9)	6.5 (3.0)	6.5 (2.5)
brachioradialis	2.6 (1.1)	9.7 (4.0) *	6.3 (2.6)	3.2 (1.6)	3.1 (1.9)	5.8 (3.0)
rectus/obliquus abdominis	5.4 (2.9)	7.9 (3.2)	6.6 (2.7)	4.9 (2.9)	5.1 (2.8)	6.8 (2.5)
vastus lateralis/quadriceps femoris	6.3 (3.4)	27.5 (4.9) *	14.5 (4.1) *	8.6 (3.1)	8.0 (3.1)	8.6 (2.7)
biceps femoris	3.8 (2.3)	14.7 (4.8) *	9.0 (2.5)	5.7 (2.2)	6.8 (3.3)	9.1 (3.5)
gastrocnemius/soleus	3.0 (1.8)	23.0 (6.2) *	5.8 (2.2)	5.5 (2.8)	5.6 (2.6)	5.0 (2.9)
loerw back muscles	6.8 (2.7)	21.6 (3.5) *	9.0 (2.5)	6.8 (2.6)	3.7 (1.6)	9.1 (2.7)

FIGURE LEGENDS

Figure 1: Changes in maximal isometric strength of the knee extensors before (pre), immediately after (0) and 24-144 h post-race. Mean (SEM) values of the 11 subjects are shown. * indicates significant ($p<0.05$) difference from the pre-race value.

Figure 2: Changes in plasma creatine kinase (A), lactate dehydrogenase (B), glutamic oxalacetic transaminase (C), and aldolase (D) activities, myoglobin (E) and C-reactive protein (F) concentrations before (pre), immediately (0) and 24-144 h after the cross-country ski race. Mean (SEM) values of the 11 subjects are shown. * indicates a significant ($p<0.05$) difference from the pre value.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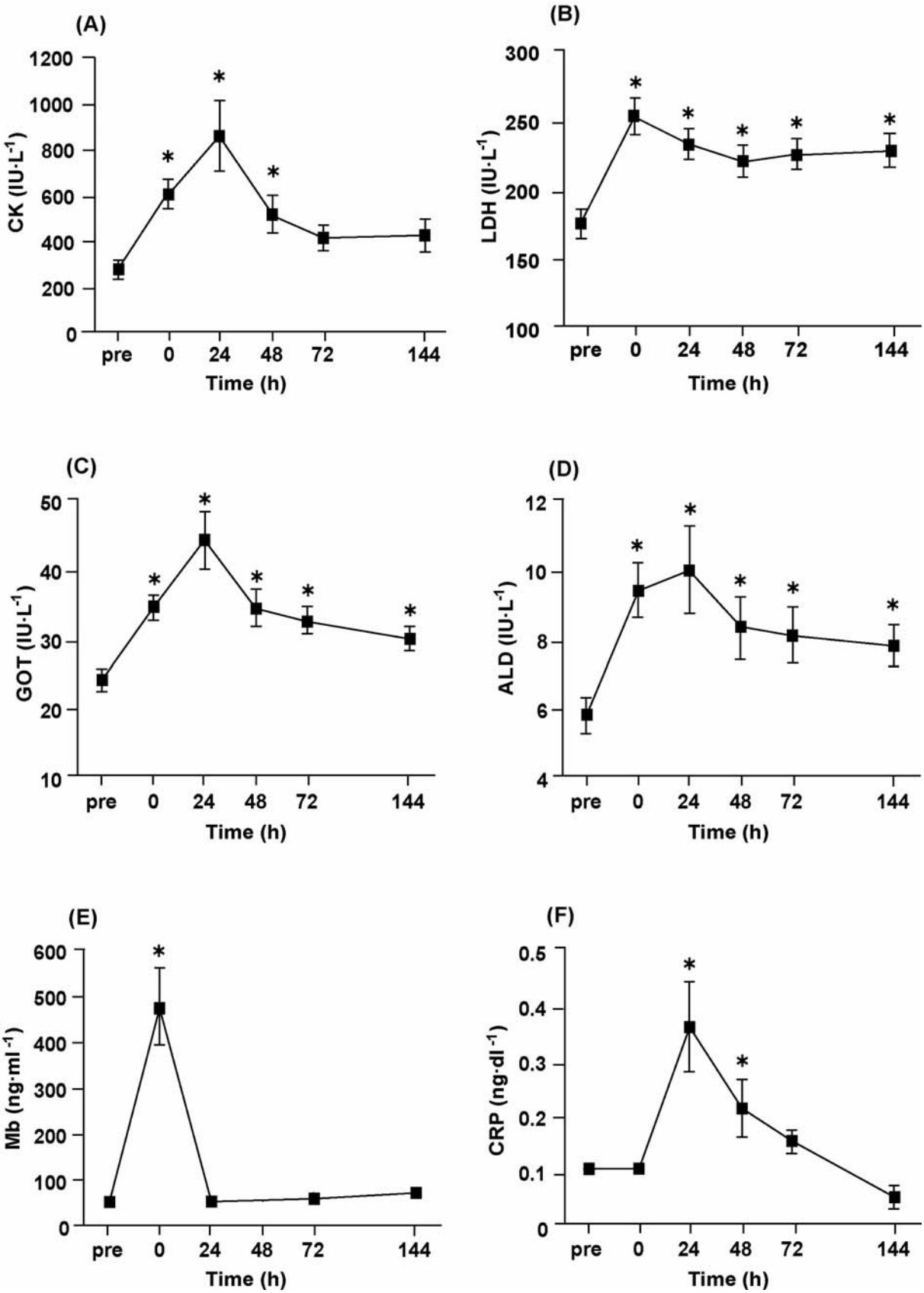
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Time (h)
(B)
LDH (IU·L-1)
300
250
200
150
100
pre
0
24
48
72
144
Time (h)
(C)
GOT (IU·L-1)
50
40
30
20
10
pre
0
24
48
72
144
Time (h)
(D)
ALD (IU·L-1)
12
10
8
6
4
pre
0
24
48
72
144
Time (h)
(E)
Mb (ng·ml-1)
600
500
400
300
200
100
0
pre
0
24
48
72
144
Time (h)
(F)
CRP (ng·dl-1)
0.5
0.4
0.3
0.2
0.1
0
pre
0
24
48
72
144
Time (h)