INVESTIGATION OF EXERCISE-INDUCED DELAYED-ONSET MUSCULAR SORENESS AND SERUM CK ACTIVITY

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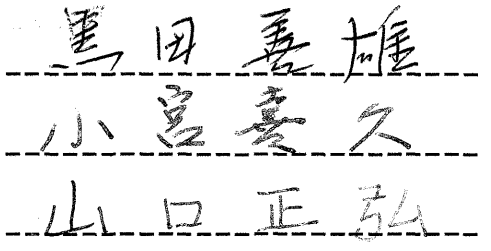

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Abbreviations in the text

CK	=	Creatine kinase
DOMS	=	Delayed-onset muscular soreness
WBC	=	White blood cell
F.I.	=	Fitness index

CONTENS

Chapter I

INTRODUCTION

It is well known that strenuous and unaccustomed exercise is often followed by muscular soreness, that becomes apparent some hours later and may persist for several days. The objective signs of sore muscles are stiffness, firmness, tenderness, and weakness.[5) 10)] The force they are able to exert during maximal voluntary contractions and even when stimulated electrically is decreased.[49)]
Although almost every healthy individual has experienced such a type of muscle discomfort at some time, the cause of muscular soreness occurs 1-2 days after exercise has still not been firmly established. Because of the temporary nature of exercise-induced muscle soreness, little research has been presented to elucidate its mechanisms and its relief.

But it is clear that muscular soreness is one of the important problems in carrying out an effective training and rehabilitation programs. Staton[63)] regretted that the occurrence of this condition is a prominent deterrent to the attainment of optimal physiological fitness, particularly when a high degree of fitness must be achieved within a relatively short period of time.

Recently many studies have suggested that exercise-related delayed muscle soreness is induced by some sort of muscle tissue damage, which is especially noticeable in muscles which have performed eccentric contractions.[21-23)]

Furthermore, the onset and time course of the damage seemed to be consistent with the degree of perceived muscle soreness (cf.Chap.II). It has also been demonstrated that damage of muscles could lead to a large efflux of muscle enzymes into the blood (specifically creatine kinase[EC 2.7.3.2.]; refferred as CK in the following). The release of CK has been taken commonly as a valuable means of asessing the extent of muscle damage, because more than 96% of this enzyme exist in skeletal muscles,[65] especially at the M-line in skeletal myofibrils.[74]

It is tempting to hypothesize that exercise-induced muscle damage is a common trigger of intramuscular enzyme release and soreness sensation, and high correlation might be also conceivable between both events. If this is the case, the degree of soreness sensation would represent the amount of muscle damage as indicated by serum CK activity. And according to the level of serum CK activity, in some extreme cases, more attention must be paid to the treatment of muscular soreness and to the pursuit of exercise programs.[76] Although most exercise enthusiasts believe that exercising the sore muscle must be the most effective way of reducing the soreness, there is little experimental evidence concerning it.

Thus, the main purpose of this study is; to test whether or not the changes in post-exercise delayed muscle soreness correlates with that in serum CK activity. Along with them, the experiment has been designed to confirm the location and degree of muscle soreness in different types

of muscle contraction and to observe the time course of the changes in serum CK activities and in the occurrence of muscle soreness after a stepping exercise using well-conditioned young subjects.

Chapter II

BIBLIOGRAPHICAL SURVEY

1) Description of delayed-onset muscular soreness (DOMS)

a. Definition

When discussing exercise-induced muscle discomfort, it is necessary to distinguish two different types; acute (temporary)pain, and delayed(residual) soreness.[2) 20) 44) 69)] Muscular pain may occur during fatiguing exercise or immediately afterwards, whereas soreness and stiffness usually appear several hours or days later.

Acute type of pain, which as its name implies occurs during and immediatelly following the exercise, is thought to be related to lack of adequate blood flow to the working muscles (ischemia).[17)]

The second type of exercise-related muscle discomfort, delayed-onset muscular soreness (refferred as DOMS in the following) has been defined by Armstrong.[5)] According to his definition, DOMS is the sensation of discomfort or pain in the skeletal muscles that occurs following unaccustomed muscular exertion. The soreness usually does not appear until hours after the initial exercise, lasts for one or two days and then gradually subsides in a week. Other kinds of muscle pain, which can also result from exercise, such as muscle strain or tear,[46)] are not included in DOMS.

b. Differences in muscle contraction type

During dynamic exercise, muscles contract in one of the two ways; either concentrically or eccentrically. Many studies have indicated that any form of exercise which involves eccentric contractions, such as when descending stairs, running downhill or lowering a weight, results in more substantial DOMS than do corresponding concentric contractions.[7) 58) 69)] During eccentric contraction the energy expenditure of generating a given tension is lower, probably because fewer motor units are activated to perform the same amount of work.[49)] Therefore, in eccentric contractions the tension exerted per cross-sectional area of muscle is supposed to be larger, which could cause structural damage in the muscles.[36) 38)]

c. Treatment

Armstrong[5)] and Hough[31)] stated that exercising the sore muscle may be the most effective way of reducing the soreness, even though the soreness returns if the exercise is stopped. It is doubtful that creams containing menthol, salicylate, or anti-inflammatory drugs per se are effective in reducing muscular soreness.[5) 39)] But the sensation of warmth imparted by medication, or stimulation of massage, is thought to be helpful in reducing DOMS.[24)] Application of cold is a quite common method to reduce pain, but the efficacy of ice massage alone on DOMS has not been supported by Yackzan et al.[75)]

The prevention of DOMS is thought to be much easier than the treatment.[10)] Graded exercise intensity, previous training, "warm-up" and "cool-down" before and after exercise could help to minimize DOMS.

2) Hypotheses about mechanisms of DOMS

In considering muscle discomfort in athletes, findings in various disease states provide some insight. In pathologic conditions, muscle pain is associated with failure of muscle energy.[56] A depletion of muscle energy and resultant ischemic necrosis of muscle fibers may cause inflammatory changes,[6) 26) 28) 62)] swelling of muscles,[9) 69)] edema,[11)] release of some chemical substances,[73)] all of which could be linked to soreness sensation. Soreness following severe activity may have similar mechanisms in some cases.[43)]

At the present time, predominant explanations for possible cause of DOMS could be classified as follows:

(a) Structural damage of muscle fibers per se

(b) Damage to connective tissue within muscles

(c) Muscle spasms

(d) Metabolic waste product accumulation

As noted earlier(cf. 1)-b), the fact, that eccentric contractions are most likely to result in DOMS makes it plausible that an eccentric component of dynamic work, such as increased tension, plays an important role in causing the structural changes either in the muscle fibers per se and/or in the connective tissue element in the muscle.

It was first hypothesized by Hough,[31)] that microscopic ruptures or tears in the muscle fibers were causative factors for soreness. Recently some experimental evidences have been obtained of considerable damage to the muscle fibers themselves,

which have performed eccentric work.[4) 7) 21-23)] From the observations of muscle biopsies with electron microscope, they have demonstrated that broadening, streaming and occasional total disruption of Z-bands occurs in some fibers. These were more apparent 2-3 days after exercise, when the subjects were suffering from DOMS.[21) 23)]

In 1977 Abraham[1)] induced soreness in 11 subjects and examined the three different hypothesis (a),(b) and (c) mentioned above. He used Hydroxyproline/creatinine(OHP/Cr) in urine as a marker to assess connective tissue damage, the presence of myoglobinuria for assessing muscle fiber trauma, and surface electromyograms to evaluate the muscle spasm. His experiments support the opinion that DOMS is most likely correlated to disruption of the connective tissue elements located parallel to muscle fibers and/or in the tendons at the end of the muscle fibers. But quite recently, conflicting data has also been presented at the ACSM(American College of Sports Medicine) 33rd Annual Meeting.[30)]

Based upon the observation of a greater resting EMG-activity in sore muscles than in that of controls, a quite different mechanism for DOMS has presented by De Vries.[16)] His Spasm Theory goes on as follows: Exercise promotes ischemia which leads to the accumulation of some particular pain substance(P-substance) that stimulates in the pain nerve endings within the muscle. Then the pain sensation causes more reflex spasms which induce more ischemia, and the entire cycle is repeated. As evidence of his theory, he has shown that stretching a sore muscle reduses DOMS, although another

researcher[42] found no benefit from this form of exercise. In the literature, contradictory results are reported about the resting EMG level of sore muscles.[1) 9) 35) 49)]

Metabolic waste products are undoubtedly the most popular and traditional idea of the cause of DOMS.[5) 28)] However, there is considerable experimental evidence against this hypothesis.[10)] For example, Schwane et al.[58] reported that instead of lower metabolic cost and less lactic acid production as compared with concentric contraction, eccentric contraction resulted in more sever DOMS. He also mentioned that lactic acid concentrations in muscle and blood return to resting level within an hour; that is long before the onset of soreness sensation. Consequently he concluded that lactic acid was not directly related to the DOMS.

3) Increase in serum CK activity following exercise

Numerous studies have reported increases in serum CK levels following exercise.[15) 18) 27) 53) 61)] Many of them demonstrated that the effects of exercise on serum enzyme may persist over several days.[8) 37) 41) 64)] A significantly higher level of CK was also found in athletes, with continued physical activities.[29) 45) 55)] Due to the diagnostic importance in various types of disease, serum enzyme activity has been studied intensively by many workers during recent years.[3) 66)] But the mechanism of enzyme release from cell to the blood remains obscure.

Mayer et al.[41] suggested that cellular injury with subsequent change in membrane permeability would cause a mechanical disruption of the fiber and promote CK release. Thomson et al.[70] have reported that intracellular levels of ATP must depleted before the release of intracelluar enzymes will occur.

The following factors have been thought to be related to exercise-induced CK efflux: Change in membrane permeability,[27) 52] mechanical disruption of the muscle cell,[8) 33) 57] muscle hypoxia,[15) 68] depletion of muscle energy sources.[32) 70] These factors have an additive effect, so that when all of them are affecting synergistically, they may account for the total enzyme release.

About the time course and the magnitude of changes in CK activity following exercise, there is much controversy in the literature.[29) 52) 59) 64) 72] The disagreements must be the result of such factors as the investigators using either trained or untrained, male or female subjects, applying long or short, hard or mild exercises, and following a short-term or a long-term course.[37]

4) Relationship between post-exercise serum CK activity and magnitude of DOMS

As has been stated previously, both DOMS and increases in the activities of intramuscular enzymes in the blood are induced by physical activity. These occurrences have been

interpreted to be evidence of some sort of muscular damage following exercise. Thus a common cause-and-effect relationship is imaginable between DOMS and CK elevation.

Schwane et al.[57] compared DOMS and plasma CK and LDH activities after downhill running with those of level running. Their results showed that running down an incline, during which muscles performed primarily eccentric contractions, caused stronger DOMS and marked increases in plasma CK activity. Furthermore changes in plasma CK activity were closely associated with muscle soreness ratings. They suggested that structural changes in muscle tissue were responsible for both events..

Tiidus and Ianuzzo[71] examined the effects of intensity and duration of exercise on serum enzyme activities and DOMS by means of dynamic leg-extension. Their results indicated that intensity of exercise is of greater importance in causing both changes than the duration. Moreover they found a relationship between work performed, post-exercise serum CK activity, and magnitude of soreness senstion in untrained individuals.

5) Training effects

A number of authors have shown that both DOMS and serum enzyme increase in response to exercise are less marked in trained subjects than in untrained.[10) 14) 19) 33) 53) Hunter and Critz,[32] who studied the effect of training on

plasma enzyme levels in man, suggested that trained skeletal muscle has an increased availability of ATP, which may better maintain the integrity of the cell membrance during work and thus reduce enzyme efflux. For a given work load, physically fit individuals display a smaller postexercise increase. Nuttall and Jones[52] suggested that the enzyme response to a given amount of exercise might be useful as a biochemical estimate of physical fitness.

Regarding the training effect on DOMS, it has been recently suggested that this efficacy appears to be highly specific, not only for the particular muscles used, but for the type of contractions.[5) 60)] Thus, the DOMS that result from eccentric exercise can only be modified by training, which employs eccentric contractions.

Chapter III

METHOD

1) Subjects

Twelve healthy male volunteers participated as subjects in this study. They were aged between 19 and 24 years, well-conditioned university and graduate students in physical education. Although some of them were on a routine training program, no subject had exercised for 24 hours before the experiment day. None engaged in any regular activities involving their legs in eccentric contractions. Informed consent was obtained from all subjects. Their characteristics are summmarized in Table 1.

2) Blood Analysis

Blood samples were taken from the antecubital vein immediately before and after exercise, then after at intervals of 5,24,48 hours. Some samples of whole blood were used for white blood cell (reffered as WBC in the following) count deternination to observe the presence of inflammation. The remainder were then centrifuged at 3000rpm to obtain blood sera. The resulting sera were refrigerated until all the specimens from an individual over the 48 hours had been collected. Under these conditions, the CK activity in their sera was stable for at least one week.[25] To minimize analytical variation, they were assayed on one occasion.

Total WBC conunts were determined using a Sysmex CC-150 (Tooa Medical Electronic Co.,Ltd.). Total activities of serum CK

were measured spectrophomometrically by means of IATOSET CPK-S kits, which applied the NADPH method[34] described by Rosalki.[54] CK activity was reported in I.U./l (International Units) at 25°C, i.e., micromoles of substrate converted per minute per 1000 ml of serum.

3) Muscle soreness evaluation

In the present study, DOMS was considered as a distinct syndrome as described by Armstrong[5] (cf.Chap.II,1)-a.). Perceived muscular soreness was quantified using the technique described by Talag[69] (Figure 1). Subjects were asked to complete this muscle soreness questionnaire before taking each blood sample. Soreness ratings were obtained from different sites of each leg. The sites included on the questionnaire were the gluteal, quadriceps, hamstrings, anterior leg, and posterior leg. Additionaly, they were instructed to choose at most 3 sore regions throughout the experimental period using a illustration printed on the questionnaire(Figure 2).

4) Exercise-Protocol

In session I, each subjest performed the Harvard Step Test, which has been described by Brouch[12] to estimate the general physical fitness level. This test consists in having the subject step up and down a 20 inch (ca.51cm) platform 30 times/min for 5 minutes. The pulse is counted from 1 to 1.5, 2 to 2.5, and 3 to 3.5 minutes after the work stops. The fitness index (reffered as F.I. in the following) is obtained according to a formula (Figure 3).

After 10 minutes rest, using the same platform, the main exercise (session II) was carried out. It was expected that this would result in DOMS. The subjects were instructed to use one leg to step up, the other to step down throughout the 40 minutes of exercise, so that the muscle of one leg was used mainly concentrically, while the other was used mainly eccentrically. In session II, the stepping frequency was slowed down to 15 times/min.,which was determined by a metronome.

Besides this, one subject, who was not involved in the experiment above, participated in the training experiment to observe the training effects. The step-exercise was repeated during a 4 weeks training period* with lower intensity, but in the same manner. Exactly the same exercise (session I,II) were done before and after training, and values of CK activity and muscle soreness were compared each other.

5) Statistical analysis

The T-test was used to describe the differences in blood parameters. Pearson's product moment coefficient of correlation were calculated to discuss associations between soreness rationgs and serum CK activities in this study.

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* Because of the strong muscle soreness at the beginning, and need of rest before the second experiment, the training was carried out irregularly 7 times during this period.

Chapter IV

RESULTS

1) Muscle soreness

Time course of change in the degrees of soreness sensation and the serum CK activity are shown in Tabele 2. and Figure 4.. Total incidences of muscle soreness were obtained by summing the scores at every site of both legs.

Muscle soreness was predominantly experienced in those muscles that had worked eccentrically, that was the calf of the descending leg in this experiment (Figure 5). Incidence of soreness in different regions of lower limbs is shown in Figure 2. Although the degree of the soreness was very variable, its peak occurred at 24 hours after exercise in all subjects except one (Table 3). At 48 hours, 6 out of 12 subjects showed a tendency for the soreness to decline, the others remained unchanged. When compared with those of immediately after exercise, soreness ratings were higher at 24-48 hours and the delayed onset of soreness sensation was clearly observed in most cases.

2) White blood cell

Changes in total WBC counts are shown in Table 4. As far as determined by change in three subjects, no significant rises were observed at any time following exercise.

3) Serum CK activity

Serum CK activities have reached its highest level between 5 and 24 hours after exercise and they tended to decline by 48 hours in most individuals. But in some cases CK activity remained elevated even at the end of determination. Compared with pre-exercise level, mean increase of CK activities was statistically significant at each sampling time except 48 hours post-exercise ($p<0.01$ at 0h,5h; $p<0.001$ at 24h)(Table 2).

There was a variation between subjects in their mean per cent increase of CK activity, which was not exactly related to general cardiovascular fitness, indicated by F.I. in this study (Figure 6). Correlation between mean per cent increase of serum CK activity and F.I. was $r=-0.19$ (N.S.). The corresponding values are presented in Table 5. The index ranged from 79.8 to 134, with a mean of 101. When judging by method of Brouch,[12] this mean value is ranked as "excellent"(Figure 3).

4) Muscle soreness and serum CK correlation

The relationships between serum CK activity and muscle soreness after exercise are shown in Figure 7. Because of the differences in baseline CK values among subjects, CK values are also reported as a per cent increase to baseline levels (Figure 8). In both cases, no significant relationship could be seen between DOMS and CK activity. Only one significant correlation was observed between degree of soreness at 24 hours and serum CK activity at 5 hours post-exercise ($p<0.05$)(Figure 9).

A case of one subject who has accomplished the training session is presented in Figure 10. Changes in serum CK activity were minimal and similar at the two time points. But the increase in soreness ratings has been considerably diminished after training.

Chapter V

DISCUSSION

1) Muscle soreness

The result, that incidences of delayed muscle soreness were highest at 24 hours after exercise, is very consistent with earlier observations about DOMS.[42) 58)] The perception of the severer soreness in descending leg may suggest that much damage was associated with eccentric type of muscle contraction compared with concentric. This tendency has already demonstrated by others.[4) 23)] In this work it is impossible to assess whether the sore muscles are responsible for the enzyme release, because CK activity has been measured only in the general circulation, so it can not give any precise information about its origin.[67)] However, considering that there was some evidence of morphological change after eccentric exercise followed by intramuscular enzyme efflux,[22)] it seems to be reasonable to expect that the exercise in the present study might cause similar changes in muscle tissue of the descending leg.

In this study, the location of the most affected muscle was the calf of the descending leg. This is quite different from the results reported by Newham et al.,[50)] who used the similar exercise to study isotope uptake in sore muscles. In their experiment muscle discomfort developed in the quadriceps, gluteal and adductor muscles of the descending leg and in the contralateral calf muscles in the following 24 hours. This disagreemant, I think, is primarily due to a slight

difference in the way in which subjects stepped up and down the platform during the experiment. Such a difference could be a contributing factor when the exercise was demanding and prolonged.

The exact location of soreness is documented in literature as being localized in the muscle-tendon junction.[7] Newham et al.[48] suggested that this could be a reflection of the fact that population of muscle pain receptors is greatest in this region and in connective tissue in the muscle. They also referred to unpublished anatomical investigations suggesting that the angle between the muscle fibers and the line of the tendon are greatest in these areas, so they could be more susceptible to mechanical trauma.

However, in our examination the regions of soreness could not be stated certainly. Most of the subjects complained of generalized soreness in the entire muscle area and not especially in the muscle-tendon junction (Figure 2). The sensation of slow and secondary pain in skeletal muscle, such as DOMS, is primarily transmitted by group IV(C) afferent fibers, which are believed to carry dull, diffuse pain.[5) 73] Thus it is sometimes difficult to point out the exact locatio of soreness.

In this study muscle soreness was evaluated subjectively using the method described by Talag.[69] Accordingly further investigation is necessary to examine, to any extent, the degree of muscle damage reflected on the subject's perception of soreness. Some workers have used either a probe fixed to a strain gauge[50] or palpation on the skin[1) 39] to assess the soreness.

The former technique was only suitable for superficial muscles, as recoginized by Newham et al..[50] From the author's personal experiences, as well as those of subjects, it would seen that palpation may not be an adequate method to estimate pain sensation. In the present conditions, because of extremely complex mechanisms of pain, it was not possible to provide perfect assessment of soreness sensation in different individuals.

2) White blood cell

The fact that no significant changes in total WBC counts were remarked in this study might suggest that inflammatory response was not associtated with DOMS. But the conclusions remain tentative, because of small sampling size and lack of controls.

3) Serum CK activity

The extent of the CK rise found in this study ($\bar{X}$=153% of its pre-exercise level) is smaller and faster than has been previously reported following various types of exercise which subsequently cause muscle soreness.[57) 71)] Some investigators have showed large delayed rises[33) 47)] or biphasic changes[68) 72)] of CK activity especially in untrained subjects. Those late phase increases have been considered as a evidence of more serious damage of the tissue.[47) 68)] But it was not the case of this study within the 48 hours time course following.

Enzyme elevation in serum after certain amounts of work were known to be moderated in trained individuals (cf.Chap.II,5)).

Although they were not accustomed to this particular exercise, most of the subjects in this study were active, well-conditioned, and at least none of them could be described as a physically unfit person. So they might be sufficiently trained to prevent such an unusual increase as mentioned above.

Nonetheless, no significant correlation is observed between individuals mean per cent increase of CK activity and F.I.(Figure 6). About this point, there has not been general agreement in the literature. Schwartz et al.[59] reported that increase in enzyme activities correlated very poorly with general cardiovascular conditions represented by $\dot{V}O_2$ max. Therefore it is also conceivable that the training effects observed by others depended on closer training for the specific contractile activity rather than on the general fitness level measured in this study.

4) Muscle soreness and serum CK correlation

It was the main purpose of this study to test ths hypothesis, that DOMS represents the degree of muscle tissue damage as indicated by serum CK changes. From the viewpoint of practical use, it was intended to find out at first the relationship between DOMS and serum CK activity following exercise. Unfortunately the findings generally tends to suppot the concept, that the correlation between both events is too low for the purpose of predictability.

Examples of the three different types of response are shown in Figure 11. In spite of similar changes in serum CK, quite different time courses of soreness were observed. This discrepancy can been seen in Figure 10 too, which displays the training effect of one subject. When the changes in serum CK and DOMS are plotted against each other, low correlation can been seen more clearly (Figure 7.8).

Considering this relationship, the suggestions presented by others[59) 68)] must be taken into account i.e., that immediate and delayed responses of serum enzymes to exercise are the result of different processes occuring in the organs of origin of the enzymes. Therefore, the blood samples and the soreness rating immediately after exercise, which can not regarded as DOMS, are excluded from the results.

Although one significant correlation existed betweem CK activity at 5h and degree of soreness at 24h post-exercise (Figure 9), the magnitude of enzyme release did not exactly reflect the degree of soreness sensation. In other words, muscle soreness was not necessarily accompanied by release of muscular enzymes.

This does not coincide with the findings of Schwane et al.[57)] and Tiidus et al.,[71)] but agrees with others,[13) 14) 51)] who failed to observe a causal relationship between them. Although serum CK has been widely used to quantify muscle damage, Kuipers et al.[40)] have recently reported a low correlation

between serum CK and amount of affected muscle volume in rats. These unexplained discrepancies have also been discussed by others.[14] Newham et al.[50] stated that after intensive eccentric exercise several changes can occur in the muscle. Examples include; development of soreness, disruption of myofibrillar architecture, and changes in membrane permeability that allows muscle enzyme efflux. Their work showed that those changes could occur independently, so that the soreness was not a reliable indicator of muscle damage.

Accordingly it is possible that the high correlation between change in serum CK activity and change in degree of soreness sensation has been accidentaly observed in untrained subjects, who could be more susceptible to physiological demand of exercise. And the failure to find the significant relationship in this study might depend on the smaller and faster increase of CK activity in our relatively active, young subjects.

Although temporary, DOMS are known to accompany the reduction of muscular strength and performance.[23] [38] [49] [69] Hough[31] suggested that muscule strength may be decreased both from reduced voluntary effort due to the sensation of soreness itself and from the lowered inherent ability of the muscle to produce force. Thus, when muscle discomfort exists, it may contribute to loss of interest in continuing exercise and the occurence of injury in the worst case.

Yet the essential meaning of DOMS is not clear. It represents the "over-used" state of muscle to a certain extent. Usually, the sensation of pain is thought to have a function to protect the organ from permanent impairment. But in the case of DOMS, exercising the sore muscle is thought to be effective to reduce the sore sensation (cf.Chap,II,1)). From the review by Armstrong,[5] it can be seen that the endorphin secretion enhanced by exercise might play a role in minimizing the soreness sensation during exercise. On the one hand it can be interpreted that the sensation of DOMS is still a signal to protect muscle from serious damage, and that soreness muscle requires rest. But on the other hand, one must continue to train until the muscle becomes sufficiently conditioned to get higher strength. Such a dilemma or difficulty in muscle conditioning is experienced not only in athletic training but also in the process of rehabilitaion.

Although most athletes have their own measures to judge their physical conditions, objective barometers are lacking to prevent overtraining and give them proper advice. CK efflux in this study was only a poor indicator of DOMS, which has been thought to be induced by muscle damage. But in company with other parameters, serum CK value is still expected to be a useful index in assessing muscle condition.

It therefore remains of great interest to us to know the morphological changes in human muscles when they show high levels of CK in sera.

Because of the restricted results of the work, further discussion is beyond the scope of this paper. Additional data is necessary to determine whether the post-exercise muscle soreness, muscle tissue damage, and CK efflux are inevitably linked.

This study suggested that increases in serum CK activity after exercise might indicate muscle damage in some cases, but that it is not a reliable index of DOMS. This is mainly due to the multiple factors involved in enzyme efflux and the soreness sensation in humans.

Chapter VI

CONCLUSION

After 45 minutes' stepping exercise using 12 well-conditioned young male subjects, delayed-onset muscular soreness (DOMS) in eccentrically worked muscles, and elevation of serum creatine kinase (CK) activity with a mean of 153% to pre-exercise level were observed. When the CK activity increased in sera, soreness rating showed a tendency to be higher, but no significant relationship could be established between change in serum CK activity and change in soreness rating.

Summary

The present study intended to test the hypothesis that the changes in serum CK activity after exercise relate to the change in muscle soreness. In addition to that, it was intended to observe the location and degree of muscle soreness in different types of muscle contraction, and the time course of change in serum CK activity.

Twelve young, well-conditioned university and graduate students in physical education participated in the experiment as subjects.

In exercise-session I, they performed the Harvard Step Test in the same manner as described by Brouch[12] and the general physical fitness level (indicated as F.I.) was estimated by the pulse counts following the work. After 10 minutes rest, the session II was carried out, in which the subjects were instructed to use one leg to step up, the other to step down throughout 40 minutes of exercise period. The stepping frequencies were 30 times/min in session I and 15 times/min in session II. Perceived muscle soreness and serum CK activity were measured at 0,5,24,and 48 hours after exercise and they were compared with pre-exercise level.

Total white blood cell counts were determined in three of the subjects and additional one subject continued training to become accustomed to the stepping exercise.

The following results were obtained:

1) Muscle soreness was notably experienced in the calf of descending leg, which was used mainly eccentrically.
2) The incidence of soreness was highest at 24H after exercise.
3) As compared with pre-exercise level, no significant changes in total white blood cell counts were observed at any time following exercise.
4) Mean increase of CK activities was statistically significant at each sampling time except 48H post-exercise.
5) The highest level of CK activities were observed between 5-24H after exercise.
6) Correlation between mean per cent increase in CK activity and Fitness Index was not statistically significant.
7) After training in one subject, the increase in soreness was considerably deminished, but the increase in CK activity did not change so much.
8) No significant correlation was observed between the degree of soreness sensation and serum CK activity, when the 2 values at same post-exercise time were plotted each other.
9) 5% level of significant relationship could be observed between serum CK activity at 5H and degree of muscle soreness at 24H post-exercise.

The findings generally suggest that the change in serum CK activity after exercise was only a poor indicator of delayed-onset muscular soreness.

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和文要約

運動による筋肉痛と血清CK活性に関する研究

山口樹里

運動後に生ずる筋肉痛と、血清中のクレアチンキナーゼ（CK）活性の変動との関連を探る目的で、以下の実験を行った。あわせて異なる筋収縮形態での筋肉痛の発生率、発生箇所、並びにCK活性値の変動特性を検討した。

本実験の被験者は、比較的高い体力水準を有すると考えられる12人の体育専攻大学生、及び大学院生であった。

実験Ⅰにおいて、Brouha[12]により提唱された〝ハーバードステップテスト〟を実施し、5分間の運動終了後の脈拍数から心肺機能の体力指数（F. I.）を算出した。10分間の休息の後、実験Ⅱとして、実験Ⅰの半分の1分間15回の昇降テンポにて、40分間運動を継続した。この間被験者は、常に同じ一方の足で昇り、他

方の足で降りる様、指示された。

運動直後、5, 24, 48時間後に血清CK値, 並びに筋肉痛の評価・測定を行い、運動前値を対象として比較検討した。内、3人の被験者について全白血球数の変化を、別の1人について同じ運動形態でのトレーニングを行い、前後の変化を観察した。

得られた結果は以下の通りである。

1) 筋肉痛は、主にエクセントリックな収縮を行った下降脚の下腿後面に顕著であった。

2) 筋肉痛は、運動後24時間にピークを示した。

3) 全白血球数は、運動前値に比しどの採血時点でも有意な変化を示さなかった。

4) 平均血清CK値は、48時間後を除く各採血時点で有意な上昇を示した。

5) 血清CK値のピークは、運動後5～24時間の間に観察された。

6) 各人の血清CK値の平均上昇率とF.I.との

間に有意な相関は認められなかった。

7) トレーニングにより筋肉痛の程度は減少したが、血清CK値の変化は僅少であった。

8) 運動直後を除く各測定時点での筋肉痛の程度と血清CK値との間に、有意な相関は認められなかった。

9) 運動後5時間の血清CK値と、24時間後の筋肉痛の程度との間に、5%レベルでの相関がみられた。

以上の結果から、運動後に遅発性の筋肉痛が生じた際、血清中のCK活性値が高値を示す傾向が見られた。しかし、血清CK値の上昇が筋肉痛の程度を正確に反映するとはいいがたい事が示唆された。

TABLES

Table 1. Characteristics of subjects (N=12)

Subject	Age (Yrs.)	Height (cm)	Weight (kg)
1	22	170.5	66.7
2	23	167.8	63.8
3	24	173.2	59.5
4	21	165.0	56.4
5	22	172.0	67.2
6	20	181.6	67.2
7	19	177.3	67.4
8	22	177.0	65.0
9	20	181.0	63.2
10	24	165.8	65.5
11	22	181.0	69.2
12	23	172.0	65.0
$\bar{X}$	21.8	173.7	64.7
SD	1.6	5.9	3.6

Table 2 . Mean values of serum CK activity and soreness rating throughout the experimental period.

Condition	Pre-exercise	-Hours post-exercise- 0h	5h	24h	48h
CK (I.U./l)	70.9 ±25.7	101.9** ±11.8	107.0** ±39.6	116.8*** ±49.6	82.1 ±32.2
DOMS	0 -	1.7 ±1.6	2.3 ±2.2	5.9 ±4.1	5.5 ±3.8

Values are means±SD. Significant difference compared with pre-exercise level: $p<0.01^{**}$, $p<0.001^{***}$.

Table 3. Values of serum CK activity (I.U./l), relative increase of CK (%), and Soreness rating (DOMS)

		1)D.Y	2)T.S	3)H.H	4)S.S	5)Y.Y	6)T.I	7)K.S	8)S.M	9)T.T	10)K.M	11)T.H	12)K.N	$\bar{X}$	SD
Pre.	CK(I.U./l)	82.4	81.3	69.4	37.2	86.9	62.8	119.4	90.9	29.8	88.8	47.7	54.7	70.9	25.7
	DOMS	0	0	0	0	0	0	0	0	0	0	0	0	0	-
	CK(%)	100	100	100	100	100	100	100	100	100	100	100	100	100	-
0h	CK(I.U./l)	114.5	97.3	208.9*	86.7	123.4	92.0	100.0	109.9	99.0	94.0	120.0*	77.5*	101.9	11.8
	DOMS	1	0	3	1	4	5	2	0	1.5	1	1.5	0	1.7	1.6
	CK(%)	139	120	301 *	233	142	146	84	121	332	106	252 *	142 *	158.1	77.2
5h	CK(I.U./l)	148.4	135.4	112.5	91.4	111.4	167.2	128.5	121.6	53.6	291.6*	53.3	54.0	107.0	39.6
	DOMS	2	2	2	1	3	9	1	1	1.5	1.5	2	1.5	2.3	2.2
	CK(%)	180	167	162	246	128	266	108	134	180	328 *	112	99	162.0	54.7
24h	CK(I.U./l)	147.7	115.2	78.9	79.0	154.5	151.5	163.6	179.3	51.7	169.2	50.1	61.4	116.8	49.6
	DOMS	7	10	7	2	6	15.5	2	3	3	9	3.5	3	5.9	4.1
	CK(%)	179	142	114	212	178	241	137	197	173	191	105	112	165.1	43.1
48h	CK(I.U./l)	148.8	72.7	54.0	113.5*	57.0	90.6	291.4*	78.1	76.3	126.9	65.2	51.5	82.1	32.2
	DOMS	7	7	9	2	5	14	1.5	2.5	3	9	3.5	2	5.5	3.8
	CK(%)	181	89	78	305 *	66	144	244 *	86	256	143	137	94	127.4	58.2

CK (%) Per cent increase from pre-exercise level(=100)

DOMS The values were obtained by summing the scores at every sites

* Hemolysis samples were not included in Mean ± SD

Table 4. Changes in total white blood cell counts (WBC)

		Pre.	0 h	5 h	24h	48h
WBC ($10^2/mm^3$)	$\bar{X}$	47	52	58	51	54
	SD	12	18	14	11	7

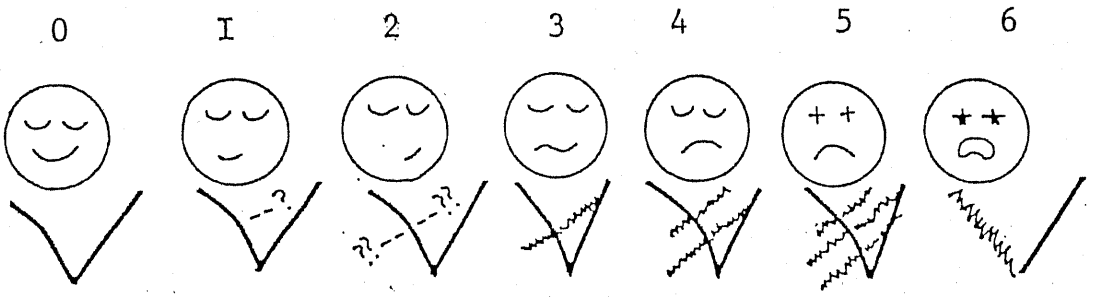
Values are Mean ± SD (N=3)

Table 5. Fitness Index (F.I.) and
Mean per cent increase of serum CK activity
CK(%)

Subject	F.I.	CK(%)
1	96.0	170
2	96.0	130
3	87.7	164
4	98.7	230
5	90.0	129
6	111.0	199
7	108.0	110
8	134.0	135
9	94.3	203
10	79.8	147
11	106.0	118
12	107.0	102
$\bar{X}$	100.7	153
SD	13.9	41

FIGURES

0. No pain
1. Dull vague ache
2. Slight, persistent pain
3. More than slight pain
4. Painful
5. Very painful
6. Unbearably painful



	0	.50	1	.50	2	.50	3	.50	4	.50	5	.50	6	.50
0 H														
5 H														
24 H														
48 H														

Figure 1. Soreness rating scale (Talag[69])

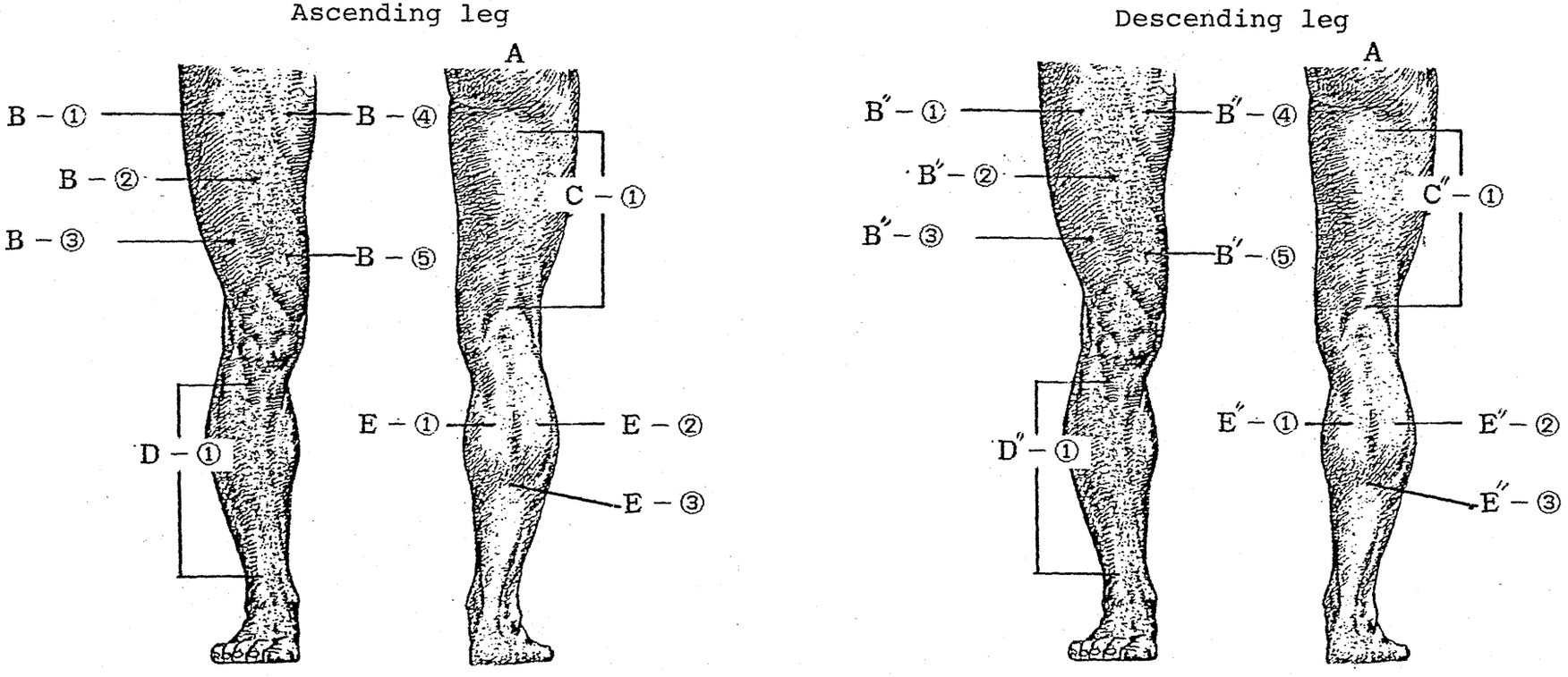


Figure 2. Illustration for determining the sore regions

The soreness was pronounced experienced in entire calf of descending leg (E″ – ①~③); secondly in calf (E – ①~③) and medial thigh (B – ④) of ascending leg.

$$\text{Index} = \frac{\text{Duration of exercise in seconds} \times 100}{\text{Sum of pulse counts in recovery} \times 2}$$

Below 55 ----- poor physical condition
55 - 64 ----- low average
65 - 79 ----- high average
80 - 89 ----- good
Above 90 ----- excellent

Figure 3. A calculating formula for Fitness Index

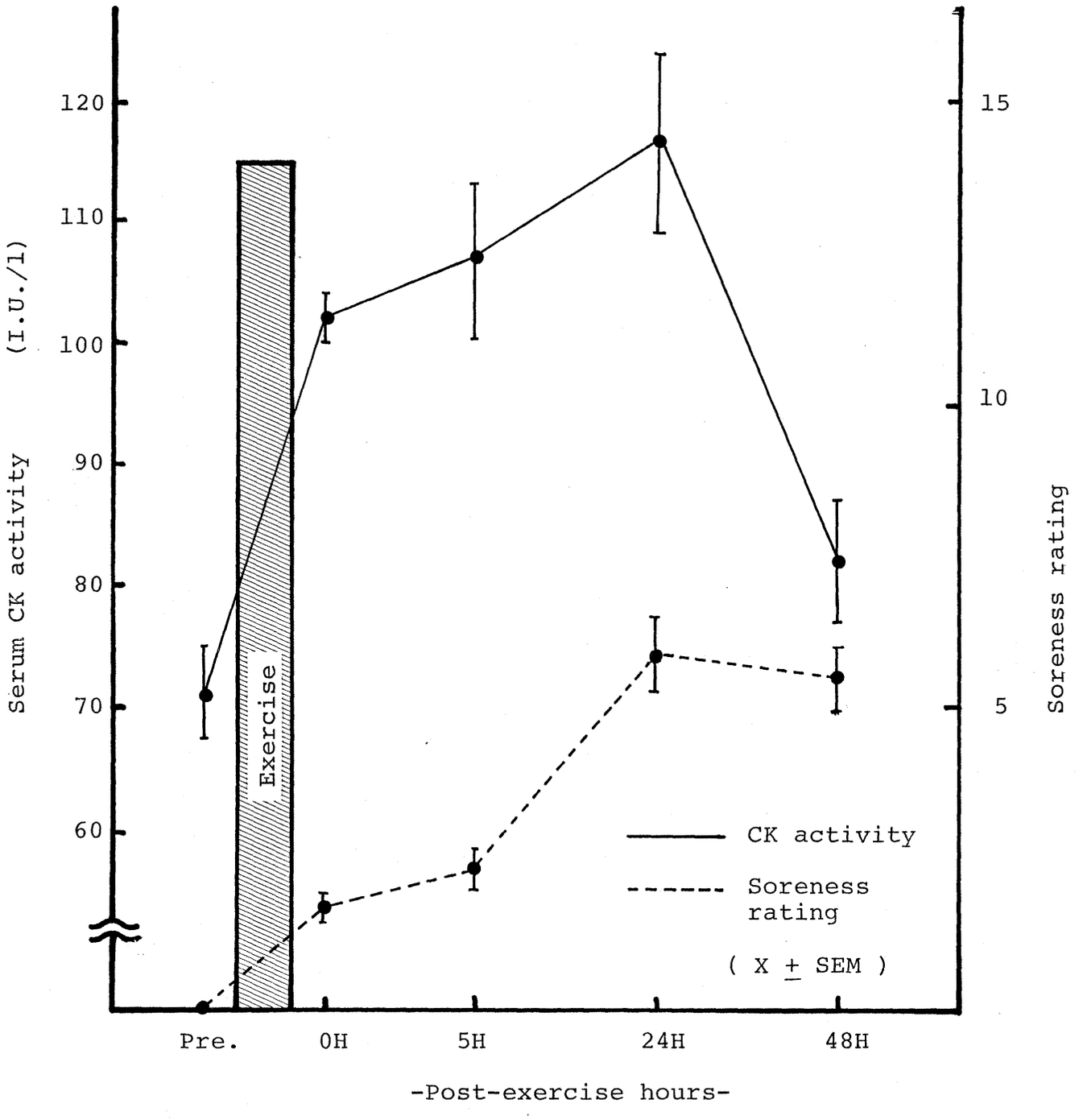


Figure 4. Changes in serum CK activity and soreness rating throughout the experimental period.

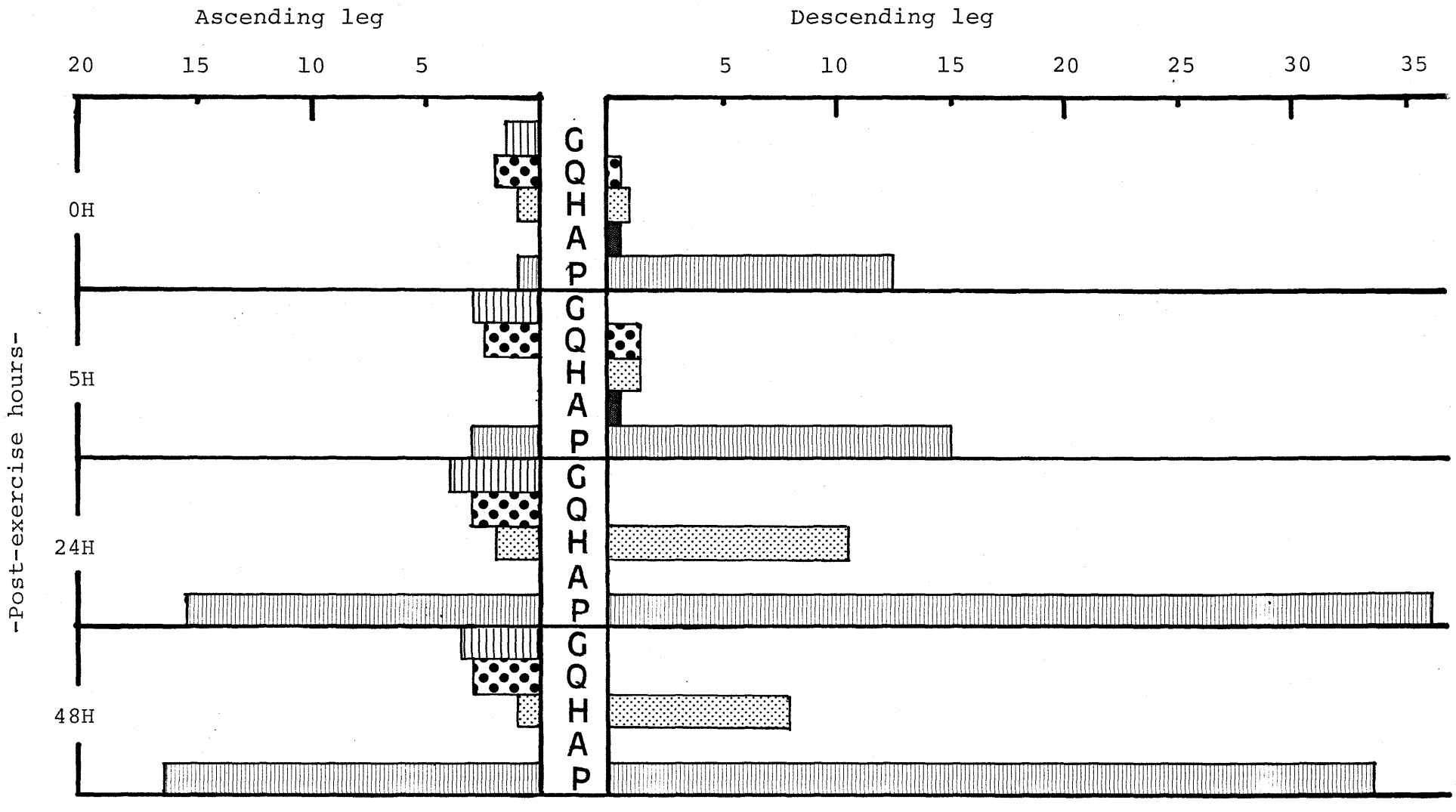


Figure 5. Incidence of muscle soreness after exercise
G=Gluteal; Q=Quadriceps; H=Hamstrings;
A=Anterior leg; P=Posterior leg.

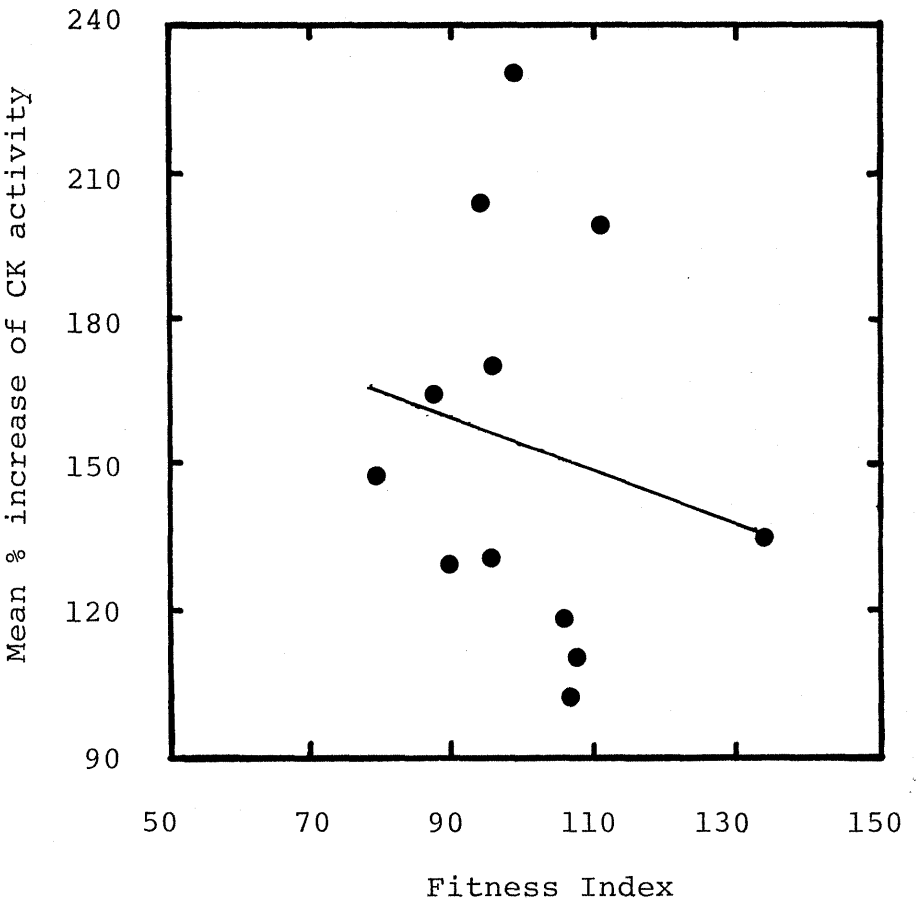


Figure 6. Mean per cent increase of serum CK activity and its relation to Fitness Index (F.I.) (r=-0.19 ;N.S.)

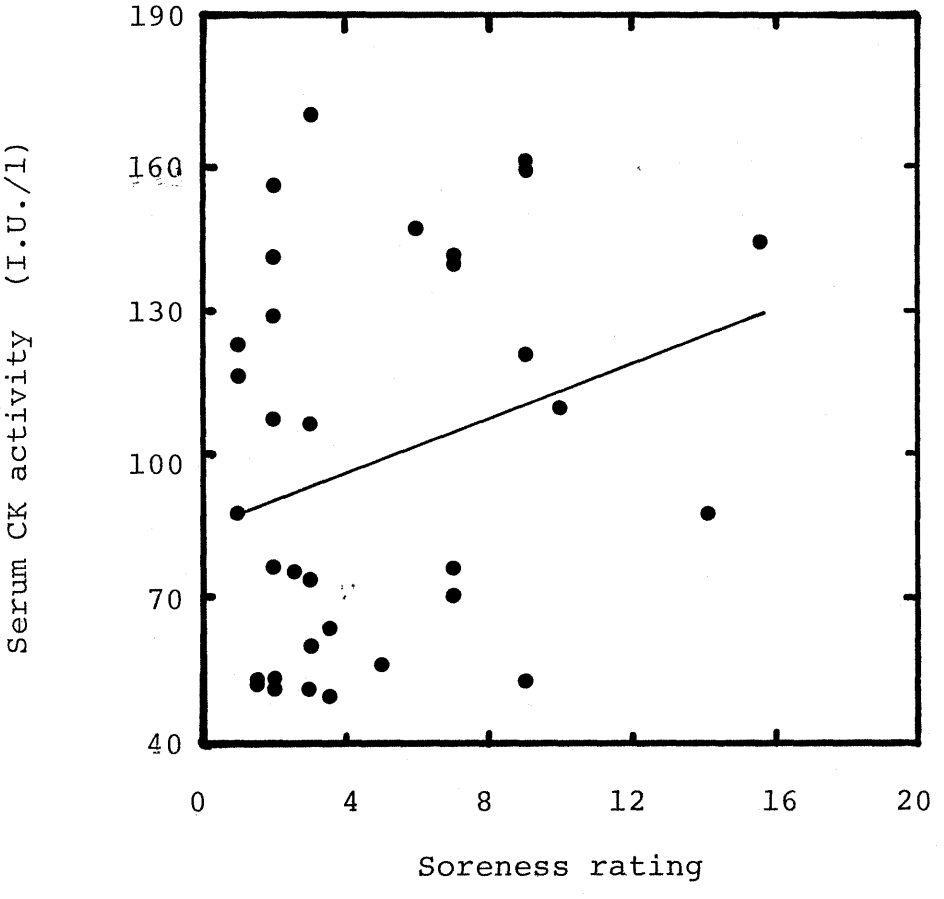


Figure 7. The realtionships between serum CK activity and muscle soreness (r=0.27 ; N.S.)

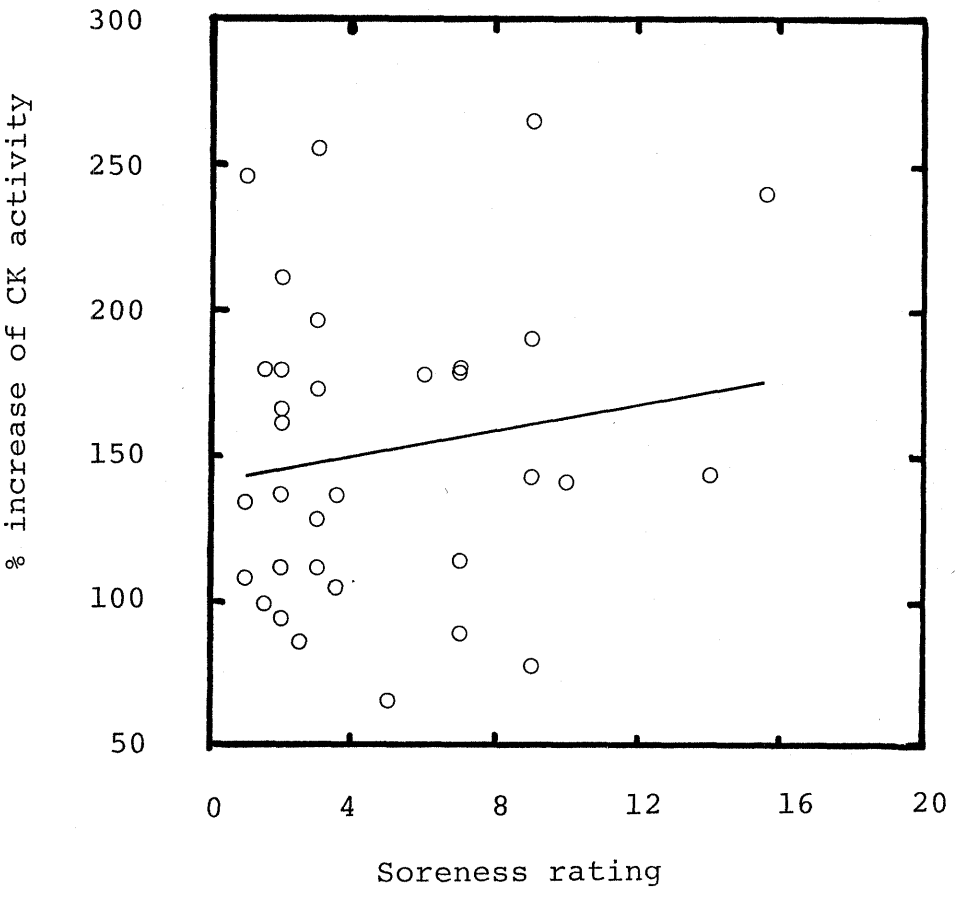


Figure 8. The relationships between per cent increase of serum CK activity and muscle soreness (r=0.16 ; N.S.)

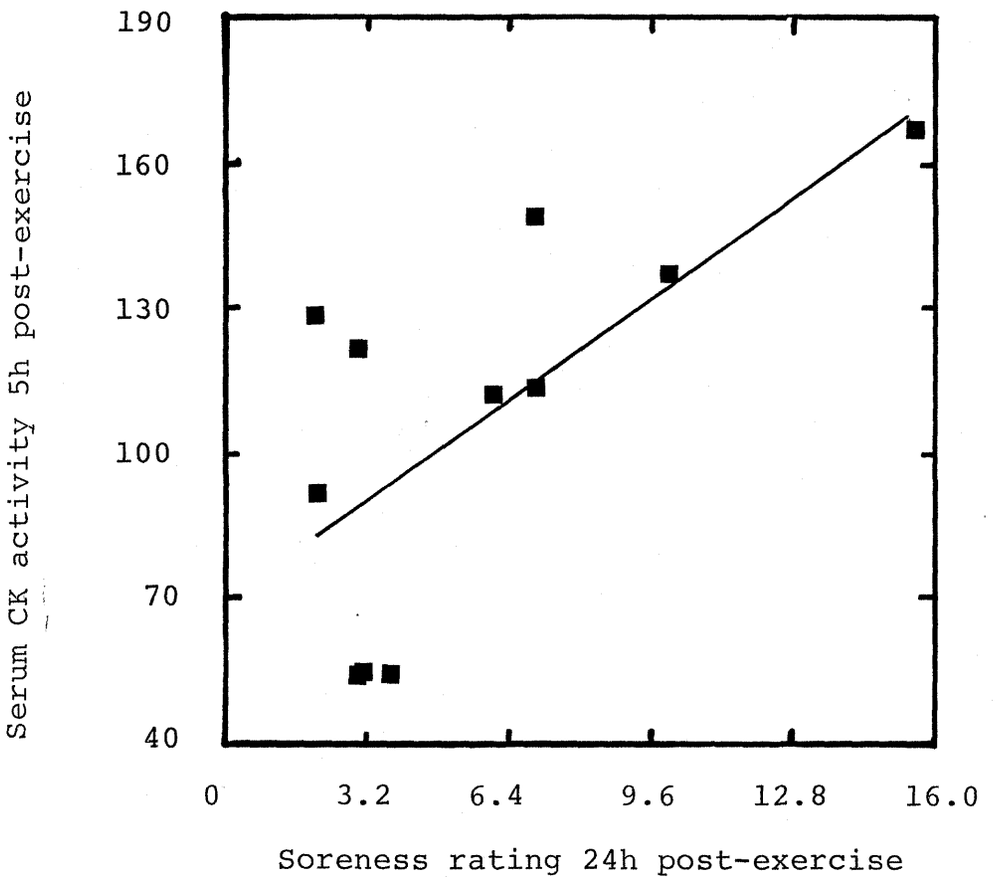


Figure 9. The relationships of post-exercise serum CK activity at 5h to degree of muscle soreness at 24h ($r=0.68$; $p<0.05$)

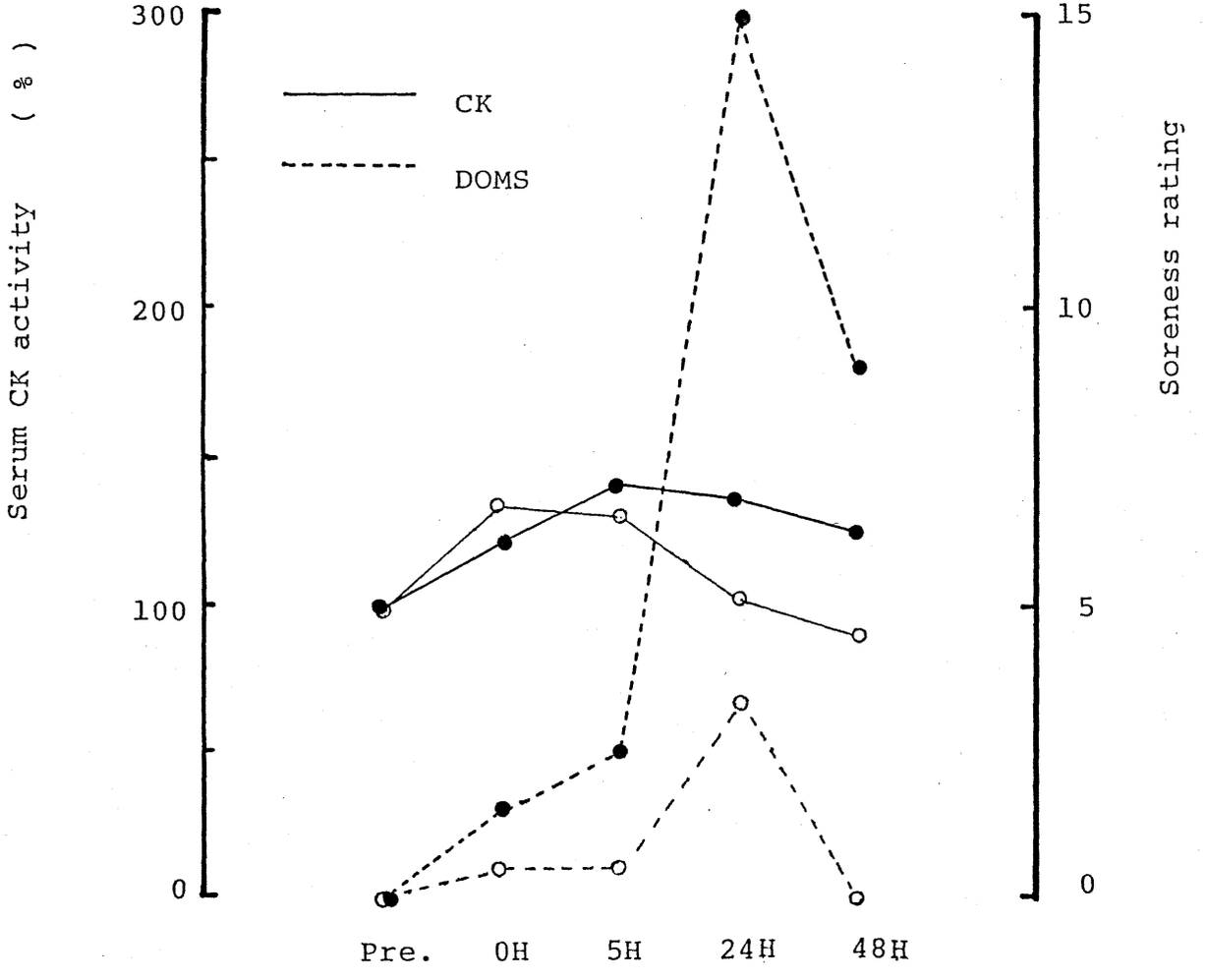


Figure 10. Changes in serum CK activity(CK) and soreness rating(DOMS) before(●) and after(O) training session

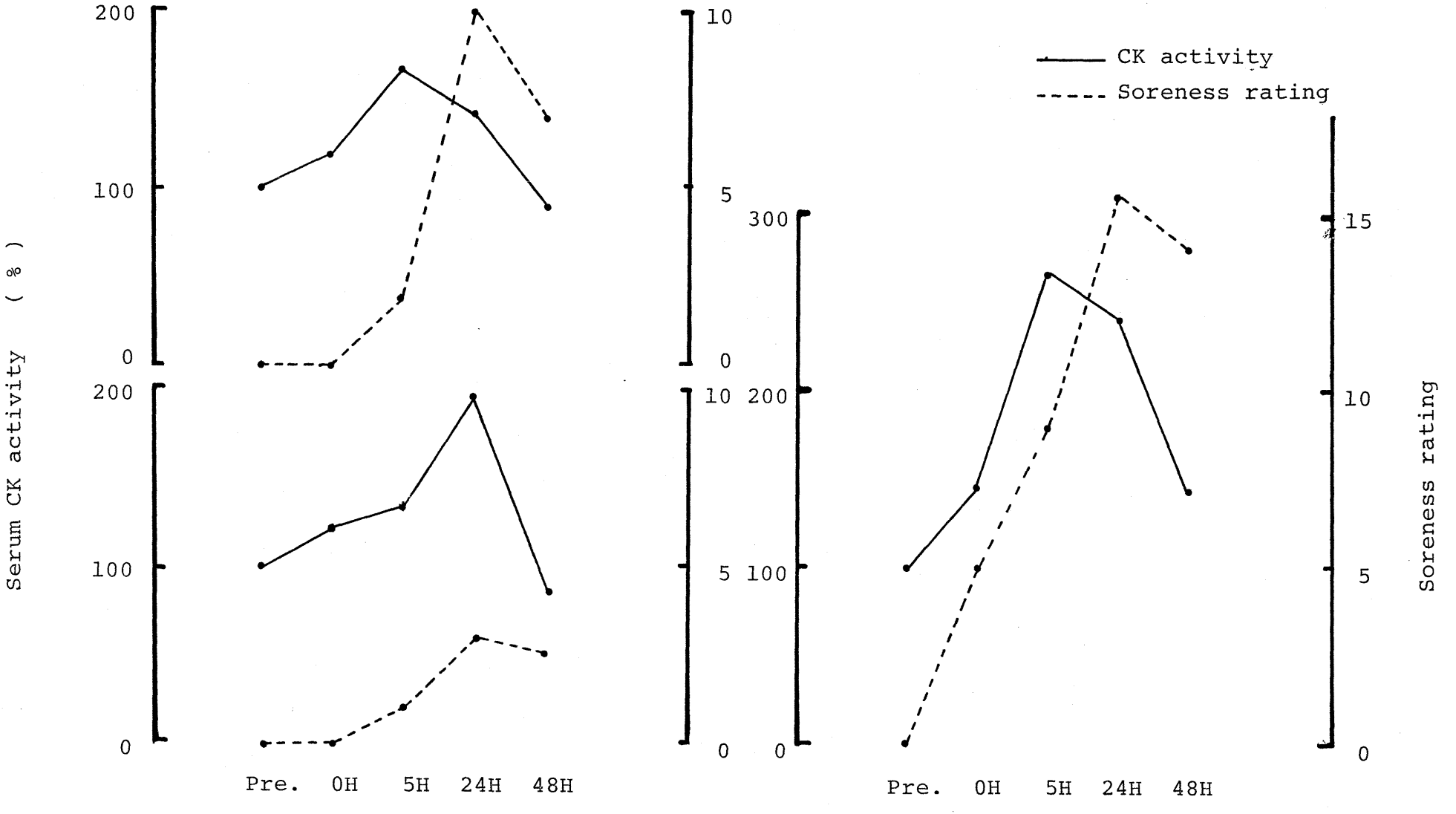


Figure 11. Post-exercise serum CK activity and soreness rating changes in three subjects.