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Response of Acetone in Expired Air During Graded and Prolonged Exercise

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Abstract

SASAKI, H., ISHIKAWA, S., UEDA, H. and KIMURA, Y. Response of Acetone in Expired Air During Graded and Prolonged Exercise. Adv. Exerc. Sports Physiol., Vol.16, No.3 pp.97-100, 2011. Ketone bodies consisting of acetoacetate, 3-hydroxybutirate and acetone are synthesized from fatty acids in the liver and released into the circulation. A portion of acetone from the circulation is then expired into the air. The purpose of this study was to determine the response of acetone in expired air during graded and prolonged exercises and the relationship between acetone and other respiratory parameters. Twelve female college students carried out graded and prolonged exercises during which expired gas was collected and acetone, oxygen intake and carbon dioxide excretion were analyzed. In the graded exercises, the acetone began to increase at approximately 35% $\dot{V}o_{2max}$, which followed the maximal fat oxidation rate at approximately 40% $\dot{V}o_{2max}$ and pulmonary ventilatory threshold (VT) at 46% $\dot{V}o_{2max}$. Significant correlations were found between intensity of the acetone threshold and those of the maximal fat oxidation rate ($r = 0.667$, $p < 0.05$) and the VT ($r = 0.871$, $p < 0.001$). In prolonged exercise, the acetone and the fat oxidation rate increased gradually and a logarithmically significant correlation was found ($r = 0.883$, $p < 0.001$). Therefore, the acetone in expired air increases from a relatively lower intensity during graded exercise and does so gradually during prolonged exercise. These results suggest that the expired acetone level correlates with maximal fat oxidation rate and ventilatory threshold during graded exercise. The acetone level similarly correlates with the fat oxidation rate during prolonged exercise.

Keywords: ketone bodies, fat oxidation, acidosis

Introduction

Lipid metabolism plays important roles for diabetes, starvation (6,7,9,10) and prolonged exercise in which the role of free fatty acid (FFA) in blood is studied exclusively, however, that of ketone bodies consist of acetoacetate (AcAc), 3-hydroxybutirate (3-OBH) and acetone are not so much as FFA (4,8). Ketone bodies are generated from fatty acids (FA) in the liver. FA partially oxidized in the liver generate 3-hydroxy-3-methylglutoryl-CoA (HMG CoA) and hydrogen ions (H^+) (6,10), furthermore, the former generates AcAc and the latter is released into the circulation. The AcAc is converted into 3-OBH by reduction and acetone by spontaneous decarboxylation (7,10). These ketone bodies, which are strongly acidic and water-soluble, are easily released into plasma and reach tissues, while a portion of the acetone is expired into the air (5). The acetone level in expired air correlates with plasma AcAc, 3-OHB (9) and probably FA concentrations because FA mobilized from adipose tissue increase blood ketone body levels (5). The acetone will be an indicator for ketoacidosis (9). Therefore, ketone bodies seem to play important roles for lipid metabolism during exercise, which may be reflected by acetone in expired air. Recently, it was showed that acetone in expired air increased gradually during graded exercise performed at three kinds of workloads. The increase at 990 kgm/min was significantly higher than that of the basal level (14).

The purpose of this study was to investigate the response of acetone in expired air during multistage workloads testing and prolonged exercise, and to examine the relationship between the acetone and other respiratory parameters.

Materials and methods

Subjects

Twelve female college students volunteered for this study (Table 1). Each subject gave written informed consent before participating in this study, which was approved

Table 1. Physical characteristics of the subjects.

Subject	Age (yr)	Height (cm)	Weight (kg)	Body fat (%)
Mean	20.4	160.2	53.1	21.6
SD	1.0	5.0	3.7	5.1

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by the Ethics Advisory Committee at our university.

Procedure and measurements

The subjects visited our laboratory twice after 12 hours of fasting to avoid any effect arising from their diets. They rested in a sitting position for 5 minutes with electrodes mounted on their chests for ECG monitoring and recording and were wearing a gas mask to collect the expired air. Thereafter, they exercised on a bicycle ergometer (Monark 818) starting from warm-up at 0 watt for 3 minutes and continuing with the workload increasing by 17 watts every minute until exhaustion. The pedal frequency of the bicycle ergometer was stabilized at 70 revolutions per minute. ECG was continuously monitored and recorded during the final 15 seconds of the rest, warm-up and at each stage of the exercise testing. Expired air was collected into Douglas bags during each period.

About 1 week after the graded testing, each subject carried out walking or running for 2 hours on a treadmill because all the subjects were not accustomed to prolonged bicycling but were to walking or running. The intensity determined by the heart rate was set at which the maximal fat oxidation rate was found during the preceded graded testing. When the heart rate was attained a steady state after 5 to 10 minutes of exercise, the velocity of the treadmill was fixed and the exercise was continued. Heart rate was monitored and expired air was similarly collected into Douglas bags for 2~3 minutes every 15 minutes during the prolonged exercise.

Analysis

A portion of the expired air (250ml) in the Douglas bags was transferred into a sampling tube in order to analyze for acetone concentration. The sampling tube was placed in an incubator kept at a constant temperature of 40℃ for 10~20 minutes to eliminate vapor. Thereafter, a portion of expired air (2.5ml) in the sampling tube was drawn into a syringe, which was injected into a gas chromatograph (Biogas Acetone analyzer, BAS 2000, Osaka, Japan) calibrated with a known concentration of acetone gas. The acetone concentration was automatically calculated in ppm from the area of the acetone peak indicated on the display, with a retention time of 7.5 minutes. The quantity of the rest of the expired air in the Douglas bags was measured using a dry gas meter and a portion was directed into a gas analyzer (Respina IH26, SAN-EI, Tokyo, Japan) calibrated with known concentrations of O_2 and CO_2 gas to analyze for O_2 and CO_2 concentrations. Oxygen intake ($\dot{V}o_2$), carbon dioxide excretion ($\dot{V}co_2$) and respiratory exchange ratio (R) were calculated, and thereby, fat oxidation rate was similarly calculated using the caloric equivalent of R and the percent kilocalories from fat with the assumption that the urinary nitrogen rate was negligible. The acetone concentration and pulmonary ventilation ($\dot{V}_E$) values of each subject were plotted against the work intensity so that those changing points were determined by the 3 students. Ventilatory threshold (VT) was determined from the initial increasing point of the $\dot{V}_E$ value.

Statistics

Values obtained from the measurements and analysis were presented as means ± SD. Differences in the values were tested by analysis of variance (ANOVA). Post-hoc analysis of Turkey-Kramer was applied when applicable. The relationships between the acetone and other respiratory parameters were analyzed with Pearson's coefficient factor. Significance was recognized at $p<0.05$.

Results

Maximal oxygen intake ($\dot{V}o_{2max}$) was 44.7 ± 7.7 (33.1～59.8) ml/kg/min. The acetone concentration in expired air at rest was 0.18 ± 0.18 (0.04～0.49) ppm. During the incremental bicycle exercise testing, the acetone remained at the resting level until an intensity of 34.3 ± 5.5% $\dot{V}o_{2max}$ (0.19 ± 0.19, 0.03～0.68 ppm), but thereafter it increased in a linear fashion (Fig. 1). The increase in $\dot{V}_E$ value facilitated initially and the VT was identified at an intensity of 45.8 ± 5.9% $\dot{V}o_{2max}$ (Fig. 1). Intensity at which the acetone level began to increase was significantly lower than for that where the $\dot{V}_E$ value was beginning to increase ($p<0.01$) and it correlated significantly ($r=0.871$, $p<0.001$) (Fig. 2). The fat oxidation rate showed as a parabola against the exercise intensity and the peak value was found at an intensity of 39.6 ± 6.0% $\dot{V}o_{2max}$ (Fig. 3) which correlated significantly with the intensity of the acetone threshold ($r=0.667$, $p<0.05$). Furthermore, individuals' values of the peak fat oxidation rate correlated with the acetone concentrations at which began to increase ($r=0.609$, $p<0.05$) (Fig. 4). During prolonged exercise, acetone level and the fat oxidation rate increased gradually (Fig. 5) and a logarithmically significant correlation was found ($r=0.883$, $p<0.001$, Fig. 6).

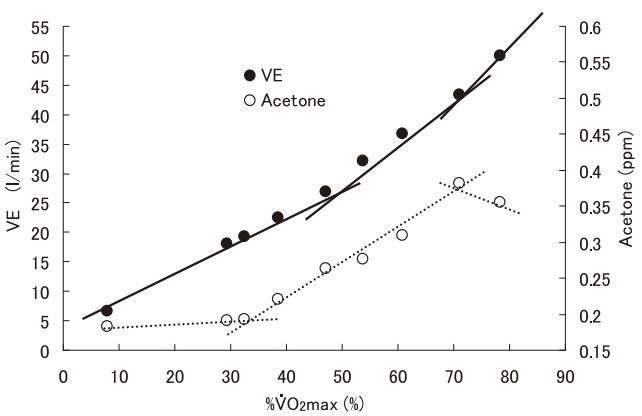

Fig. 1. Responses of acetone level in expired air and pulmonary ventilation ($\dot{V}_E$) during graded exercise testing.

Discussion

This study demonstrated that acetone level in expired air increased rapidly from an intensity of 34.3% $\dot{V}o_{2max}$ during graded exercise, which followed the facilitated increase in $\dot{V}_E$ i.e. ventilatory threshold at an intensity of 45.8% $\dot{V}o_{2max}$.

The result of acetone in this study would be in agreement with that from recent study. Yamai et al. (14) investigated on acetone in expired air during graded exercise performed at the workloads of 360, 720 and 990 kgm/min. As a result, they found a significant increase in the acetone at the 990 kgm/min, compared with basal level. Therefore, the result of acetone in this study would demonstrate that the acetone in expired air was influenced by exercise intensity and suggest that there was a possibility of acetone threshold during graded exercise.

Anaerobic threshold (AT) is a well-established phenomenon in which lactate from working muscles leaks into the circulation and causes metabolic acidosis, which is buffered and stimulates the respiratory center so that the parameters of pulmonary ventilation are influenced to compensate for metabolic acidosis (12). However, previous studies (3,11) have also pointed out that the initial chang-

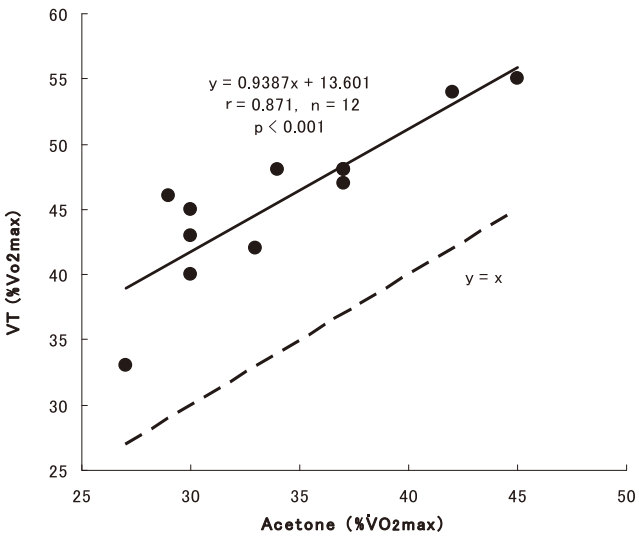

Fig. 2. Relationship between intensity at which acetone level in expired air begins to increase and that inducing ventilatory threshold during graded exercise.

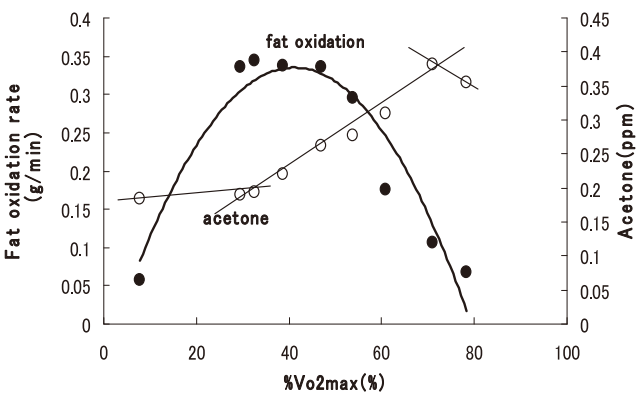

Fig. 3. Responses of acetone level in expired air and fat oxidation rate during graded exercise.

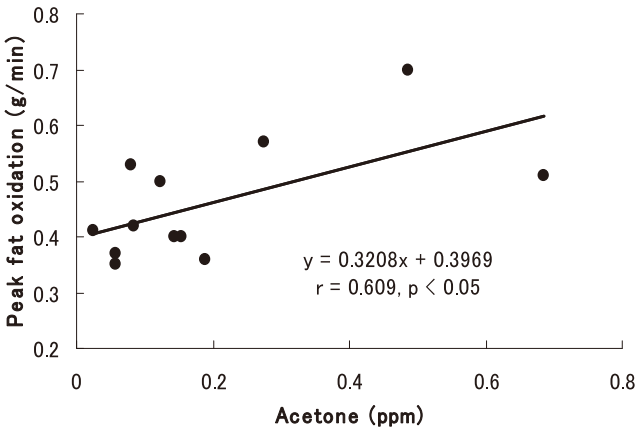

Fig. 4. Relationship between acetone level in expired air at which begins to increase and value of the peak fat oxidation rate during graded exercise.

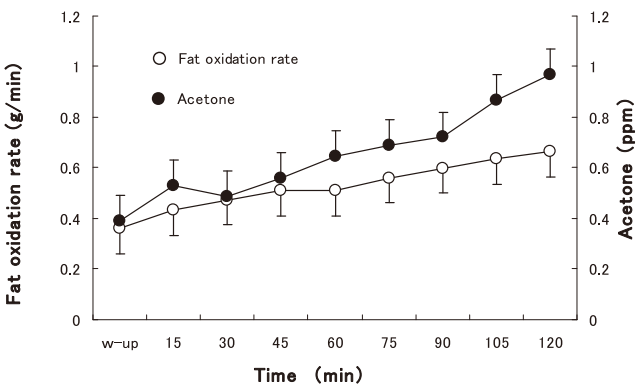

Fig. 5. Responses of acetone level in expired air and fat oxidation rate during prolonged exercise.

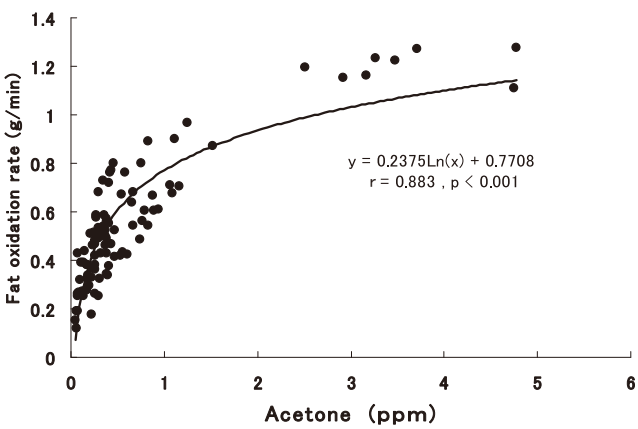

Fig. 6. Relationship between acetone level in expired air and fat oxidation rate during prolonged exercise.

ing point in the parameters preceded the increase in blood lactate from the resting level, suggesting that the other metabolic acids are buffered (3). VT is the initial increasing point of the $\dot{V}_E$ value and regarded as the intensity compensating for metabolic acidosis. The acetone in expired air, reflecting ketone bodies in the circulation (9), increased prior to increase in the $\dot{V}_E$ value, suggesting the possibility that the ketoacidosis induced by ketone bodies contributes to respiratory compensation. Therefore, the results of this study may indicate that ketone bodies are generated from the early stage of the exercise and strongly acidic ketone bodies and hydrogen ions (H^+) released from the liver into the circulation will be buffered and stimulate the respiratory center.

The fat oxidation rate showed a parabolic response during graded exercise and the peak value was detected at an intensity of 39.6% $\dot{V}o_{2max}$, which was partly in agreement with the previous study. Achten and Jeukendrup (1) reported that fat oxidation rate formed a parabola against the intensity, the peak value of which was detected at an intensity of 63% $\dot{V}o_{2max}$ in endurance-trained individuals.

The relative intensity inducing the maximal fat oxidation rate in this study was much lower than that in the previous study (1). An increase in blood lactate inhibits fat mobilization from adipose tissue and its utilization in working muscles (2). Furthermore, it is well known that the exercise intensity inducing the increase in blood lactate from a resting level in the trained individuals is higher than that in the untrained ones (13). The discrepancy of the relative exercise intensity inducing maximal fat oxidation rate resulted from the training level of the subjects (1,5). Another explanation for the discrepancy may be gender of the subjects and/or the duration of each stage in the graded exercise. All the subjects in this study were females, while these in the previous study (1) were males. The workload was increased by 17 watts for every minute in this study, while it was 35 watts every 3 minutes in the previous study (1).

This study also demonstrated that the acetone level in expired air began to increase, which followed the maximal fat oxidation rate at the intensity of 39.6% $\dot{V}o_{2max}$. The acetone level increased gradually and correlated with the fat oxidation rate during prolonged exercise.

It is known that FFA and ketone bodies in blood increase gradually during prolonged exercise and the elevated mobilization results in increasing the utilization (4, 8). Water-soluble ketone bodies are easily released into plasma and reach the tissues (5). The acetone level in expired air correlates with the concentrations of plasma AcAc, 3-OHB (9) and probably FA. Ketone bodies, as described above, would be generated at an early stage of the exercise and are rapidly released. In the present study, the intensity at which the acetone level in expired air began to increase correlated significantly with that which induced the maximal fat oxidation rate (Fig. 4). Therefore, the results of this study may indicate that fat oxidation including ketone bodies is elicited during graded and prolonged exercise. However, this study cannot clarify which ketone bodies or FFA are utilized predominantly during the exercises.

Conclusions

Acetone in expired air begins to increase from relatively lower intensity during graded exercise and does so gradually during prolonged exercise. The intensity at which the acetone level begins to increase correlates significantly with those levels that induce maximal fat oxidation rate and ventilatory threshold during graded exercise. Furthermore, the acetone level similarly correlates significantly with the fat oxidation rate during prolonged exercise.

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