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Effects of Hypoxic Stress on Energy Metabolism in Red Sea Bream, *Pagrus major*-I

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Abstract: Young red sea bream, *Pagrus major*, were exposed to a gradual decrease in partial pressure of oxygen (PO2) down to 20 mmHg for 5 h, and then sustained under 20 mmHg PO2 for an additional 3 h at 19.9ºC. Fish respiratory frequency slowly increased and attained a peak level at approximately 35 mmHg PO2, and then gradually decreased. Fish sank to the tank bottom about an hour after PO2 fell below 20 mmHg, and respiration began to arrest after another hour. Hematocrit value increased and mean cellular hemoglobin content (MCHC) decreased with increasing hypoxia load. Plasma cortisol and glucose levels were significantly augmented when respiratory frequency decreased. ATP, total adenylate (TA) concentration and energy charge (EC) in the hepatopancreas, in addition to TA in the kidney, were considerably diminished by hypoxia. However, this was not observed in the gill and brain of all fishes prior to respiratory arrest. In the hepatopancreas, levels of ATP, TA and EC notably declined as respiratory frequency decreased. These results show that the cortisol stress response to hypoxia is significantly induced with the decreased respiratory frequency. Decreased energy status in the hepatopancreas appears to be important and occurs with stress response as a metabolic depression in the whole body.

Key words: Red sea bream; Hypoxic stress; Cortisol; Adenine nucleotide concentration

Cultivated marine fish reared in floating net cages are frequently subjected to stress caused by environmental changes because of their inability to migrate to more suitable environments1,2). For example, tidal currents and red tides caused by water pollution that occur from spring to summer induce severe changes in surface water oxygen levels2-4). Heavy rain and river water reduce surface water salinity around cage culture in bays. Within a year, the range of water temperatures in a given location may be inappropriate to rear several fish. It has been also known that the outbreak of parasites, pathogenic microbe and viruses all year round5,6) in addition to overcrowding7) and inadequate diets8) evoke stress responses in fish reared in net cages. Moreover, cultivated fish are continually exposed to fish farming practices such as handling9), grading10) and transport11), which often cause severe stress. These stresses cause reduce inflammatory and immune responses, resulting to increased susceptibility to fish diseases12,13). Understanding the mechanisms of stress responses in cultivated fish is therefore of critical importance, and can be used to reduce fish stress or hasten adaptation to future stresses.

Conversely, various species of freshwater fish such as tilapia14), common carp, *Cyprinus carpio*15), and crucian carp, *Carassius carassius*16), are known to thrive in poor environments such as closed ponds, etc. These species are therefore considered to possess higher tolerance for environmental stressors and stronger
disease resistance because of adaptation to enclosed environments. In particular, a great ability to adapt to hypoxia as a basal stressor is evidenced. Stress responses\textsuperscript{17} and flexible metabolic depression\textsuperscript{17-19} have also been demonstrated with anaerobioses. Moreover, in the Nile tilapia, \textit{Oreochromis niloticus}\textsuperscript{17}, and the grass frog, \textit{Rana temporaria}\textsuperscript{20}, which possess a remarkable tolerance for hypoxia or anoxia, significant differences in adenosine triphosphate (ATP) concentrations are observed between organs subjected to hypoxia. Decreased ATP concentration seems to be equilibrated with the consumption and synthesis of ATP in each tissue. Although marine cultivated fish, particularly the red sea bream, \textit{Pagrus major}, also display stress responses to hypoxia\textsuperscript{21}, little is known about the energy metabolism in fish subjected to low water oxygen levels \textit{in vivo}.

The purpose of the present study was to elucidate the effects of hypoxia on stress responses and energy status in red sea bream by measuring basal and stressed levels of blood composition, circulating cortisol and glucose, in addition adenosine nucleotide content in several tissues.

**Materials and Methods**

*Fish and exposure methods of hypoxic stress*

Young red sea bream used in these experiments were obtained from Kinki University Fish Nursery Center Shirahama Station in 1996. All fish were fed daily with commercial pellets for red sea bream at the same station. Fifty fish (body weight 109.7 ± 11.1 g, body length 15.7 ± 0.6 cm) were acclimated in two 7 m\textsuperscript{3} experimental tanks with running seawater at 20ºC and PO\textsubscript{2} of 120-150 mmHg for a few days after prefeeding. Fish were not fed for 24 h before the start of the experiment.

In the experimental tank, hypoxic stress was induced by bubbling nitrogen gas into the water (Fig. 1). A small quantity of plain water and nitrogen gas-bubbled water were also poured into the tank to adjust oxygen tension, while excess water was allowed to flow out through the drain. Oxygen tension in the experimental tank was reduced gradually from 125 mmHg to 20 mmHg over 5 h and then was maintained below 20 mmHg PO\textsubscript{2} for 3 h at 19.9ºC. PO\textsubscript{2} in the water was made uniform by means of a small submersible pump. A vinyl sheet cover on the top of the experimental tank was used to prevent diffusion of air from the outside. Nitrogen gas between the surface water and vinyl sheet prevented the fish from gasping on the surface during hypoxia load. All fish activities were observed and recorded, and respiratory frequency and number of fish sinking to the tank bottom during the experiment were counted. Six to ten fish were sampled within six stages, namely, (1) during normoxia at 125 mmHg PO\textsubscript{2}, (2) during increased respiratory frequency at around 60 mmHg PO\textsubscript{2}, (3) during peak level of respiratory frequency at about 35 mmHg PO\textsubscript{2}, (4) during decreased respiratory frequency under 20 mmHg PO\textsubscript{2}, (5) upon sinking to the bottom of the experimental tank about an hour and a half after PO\textsubscript{2} fell below 20 mmHg, and (6) just before respiratory arrest approximately 2 h and a half after PO\textsubscript{2} fell below 20 mmHg.

![Fig. 1. Diagram of experimental system. E, experimental tank; H, hatch for fish sampling; NC, nitrogen gas cylinder; O, overflow; OM, oxygen meter; P, submersible pump; R, reserve tank for sea water-bubbled nitrogen gas; S, sea water-bubbled air; V, vinyl sheet.](image-url)
Ten fish under normoxia from another experimental tank were sampled at intervals of approximately 30 min during the study to serve as the initial point of the experiment. This was to eliminate as a factor the effects of sampling stress on the remaining fishes in other experimental stages. Six to ten fish in the other experimental stages were rapidly sampled using a small net inserted through the hatch of the tank cover. To minimize handling stress, anaesthetic treatment and blood sampling from the caudal vessel were completed within approximately one minute of capture for each fish. An aliquot of whole blood was quickly obtained and used for measurement of the hematocrit value and hemoglobin concentration. The remaining blood was centrifuged instantly and the plasma frozen for use in the determination of cortisol and glucose concentrations. After killing the fish instantly, the dorsal ordinary muscle, gill, hepatopancreas, kidney, heart, and brain were isolated from the body on a dry ice block and immediately frozen in liquid nitrogen. All samples were kept at -75°C until adenine nucleotide analysis.

Analysis
Hemoglobin concentration was measured using the alkaline haematin method of Zander et al.\textsuperscript{22} and glucose level was determined according to the enzymatic coupling method with hexokinase and glucose-6-phosphate dehydrogenase of Stein\textsuperscript{23}, using the Vision System (Abbot Laboratory, IL, USA). Plasma cortisol levels were measured using a radioimmunoassay kit (ICN Biomedicals, Inc., Costa Mesa, CA, USA) according to manufacturer’s instructions. The competition curves for the extract and cortisol standard were parallel as shown in Fig. 2. The mean recovery rate of additional cortisol in the assay was 80.2\% (n=5). The coefficients of variation of intra-assay and inter-assay were 7.3 and 12.1\%, respectively (n=5). For measurement of adenine nucleotide content, an acid soluble fraction was extracted from a portion of the frozen tissue following the method of Yokoyama et al.\textsuperscript{20}. Adenine nucleotide analysis was performed by an HPLC method according to Ando et al.\textsuperscript{25}, using a Senshu PEGASIL ODS column (4.6 × 250mm, Senshu Scientific Co., Ltd, Tokyo, Japan). Mean cellular hemoglobin content (MCHC), total adenylates (TA) and energy charge ratio (EC) were calculated from the following equations:

\[
\text{Mean cellular hemoglobin content (g/100ml)} = \frac{\text{hemoglobin concentration}}{\text{hematocrit value}} \times 100.
\]

\[
\text{Total adenine nucleotide} = \text{ATP} + \text{ADP} + \text{AMP}\textsuperscript{26}.
\]

\[
\text{Energy charge} = \frac{(\text{ATP} + 1/2 \text{ADP})}{(\text{AMP} + \text{ADP} + \text{ATP})}\textsuperscript{27}.
\]

Statistics
One-way analysis of variance (ANOVA) was utilized for data analysis as no significant differences were observed in Bartlett’s test (\(p > 0.05\)) for comparison of variances. Duncan’s new multiple range test was considered for significant differences among groups when the significant differences were recognized among the means using ANOVA treatment (\(p < 0.05\)). Significant differences among groups were further analyzed using a Mann-Whitney \(U\)-test if significant differences between groups were shown using the Kruskal-Wallis test (\(p < 0.05\)) subsequent to Bartlett’s test (\(p < 0.05\)).
Results

Fish status exposed to hypoxia

Changes in respiratory frequency and the percentage of fish that sank to the tank bottom in the red sea bream experiment are presented in Fig. 3. The respiratory frequency of fish showed a tendency to increase under hypoxia and attain a peak level around 35 mmHg PO2. Thereafter, respiratory frequency tended to decrease with increasing hypoxia load. Fish sank to the tank bottom about an hour after PO2 fell below 20 mmHg, followed by respiration arrest after another hour. Some fish demonstrated bursts of frantic swimming immediately before respiratory arrest.

Blood characteristics and plasma components

Fig. 4 shows the changes in plasma cortisol, glucose levels, hemoglobin concentration, hematocrit value and MCHC of red sea bream exposed to hypoxia. Hematocrit value increased almost linearly with hypoxia up to the time fish started to sink. Hemoglobin concentration in blood displayed a tendency to increase from the time of maximum respiratory frequency to the time of decreased respiratory frequency. MCHC decreased gradually with increasing hypoxia load. Plasma cortisol concentrations of each fish in every experimental stage did not increase sequentially according to sampling order. Plasma cortisol concentration tended to increase as respiratory frequency peaked. Plasma cortisol and glucose content both significantly increased at 20 mmHg PO2 with the decrease in respiratory frequency. Cortisol concentration then increased to about four times initial values. Afterwards, glucose level further elevated and reached around thirteen times initial values immediately prior to respiratory arrest.

Adenine nucleotide

The effects of hypoxia on adenine nucleotide concentrations and energy charge in tissues of red sea bream are given in Table 1. Just before respiratory arrest, ATP and TA concentrations in the hepatopancreas and TA concentration in the kidney significantly decreased, and ATP and TA concentrations in the dorsal ordinary muscle and heart also tended to diminish with

Fig. 3. Changes in respiratory frequency and percent of fish that sank to the bottom of aquarium of red sea bream exposed to hypoxia. Arrows indicate the sampling points. The area of bias background indicates the period under 20 mmHg partial pressure of oxygen (PO2) and the decrease of respiratory frequency.

Fig. 4. Changes in concentrations of plasma cortisol, glucose, hemoglobin, hematocrit value and mean cellular hemoglobin content (MCHC) of red sea bream exposed to hypoxia. Each value represents the mean ± SD, n = 4 – 10. Values with different letters in the same curves are significantly different (p <0.05). Refer to Fig. 3 for bias background.
Stress Response, ATP of Red Sea Bream in Hypoxia

However, the brain and gill were not susceptible to such changes. In the EC, the hepatopancreatic levels were only affected by hypoxia and reduced to 78% of initial values. ATP, TA and EC levels in the hepatopancreas displayed a tendency to decrease from peak level of respiratory frequency to decreased respiratory frequency, and showed significant differences just before respiratory arrest (Fig. 5). Conversely, AMP concentration in the hepatopancreas markedly increased with hypoxia immediately prior to respiratory arrest.

### Discussion

**Aerobic respiration under hypoxic conditions**

Respiratory frequency in red sea bream exposed to a gradual decrease in PO₂ increased until about 35 mmHg PO₂, and then followed by a gradual decrease (Fig. 3). Under the present experimental conditions, red sea bream appear capable of adapting to hypoxia by increased ventilatory responses up to 35 mmHg PO₂, although the fish cannot fully cope with hypoxia by moving the operculum at levels below 35 mmHg PO₂. Subsequently, fish sank to the tank bottom about an hour after PO₂ fell under 20 mmHg, and fish respiration began to arrest after another hour (Fig. 3). Previous studies in Japanese parrot fish, *Oplegnathus fasciatus* and Nile tilapia have shown the same trend. However, PO₂ at peak respiratory frequency, fish sinking and respiratory arrest in red sea bream and Japanese parrot fish are remarkably higher than that in tilapia. The hypoxic tolerance of red sea bream was found to be sub-

### Table 1. Effects of hypoxia on the adenine nucleotide concentrations and energy charge in tissues of red sea bream

<table>
<thead>
<tr>
<th>Tissue</th>
<th>Sampling point</th>
<th>ATP (\mu\text{mol/g})</th>
<th>ADP (\mu\text{mol/g})</th>
<th>AMP (\mu\text{mol/g})</th>
<th>TA (\mu\text{mol/g})</th>
<th>Energy charge</th>
</tr>
</thead>
<tbody>
<tr>
<td>Dorsal muscle</td>
<td>Initial</td>
<td>3.15 ± 0.98</td>
<td>0.37 ± 0.16</td>
<td>0.14 ± 0.07</td>
<td>3.66 ± 1.11</td>
<td>0.91 ± 0.01</td>
</tr>
<tr>
<td>Heart</td>
<td>Initial</td>
<td>1.40 ± 0.19</td>
<td>0.55 ± 0.33</td>
<td>0.12 ± 0.01</td>
<td>2.49 ± 0.36</td>
<td>0.79 ± 0.04</td>
</tr>
<tr>
<td></td>
<td>Final†</td>
<td>1.27 ± 0.20</td>
<td>0.45 ± 0.17</td>
<td>0.21 ± 0.07</td>
<td>1.99 ± 0.21</td>
<td>0.76 ± 0.03</td>
</tr>
<tr>
<td>Hepatopancreas</td>
<td>Initial</td>
<td>0.91 ± 0.23</td>
<td>0.31 ± 0.09</td>
<td>0.15 ± 0.01</td>
<td>1.37 ± 0.26</td>
<td>0.77 ± 0.05</td>
</tr>
<tr>
<td></td>
<td>Final†</td>
<td>0.36 ± 0.03</td>
<td>0.27 ± 0.03</td>
<td>0.19 ± 0.01†</td>
<td>0.82 ± 0.06†</td>
<td>0.60 ± 0.01†</td>
</tr>
<tr>
<td>Kidney</td>
<td>Initial</td>
<td>0.44 ± 0.07</td>
<td>0.60 ± 0.23</td>
<td>0.46 ± 0.13</td>
<td>1.50 ± 0.13</td>
<td>0.49 ± 0.06</td>
</tr>
<tr>
<td></td>
<td>Final†</td>
<td>0.43 ± 0.05</td>
<td>0.19 ± 0.03†</td>
<td>0.20 ± 0.09</td>
<td>0.83 ± 0.10†</td>
<td>0.64 ± 0.08</td>
</tr>
<tr>
<td>Brain</td>
<td>Initial</td>
<td>0.37 ± 0.17</td>
<td>0.15 ± 0.03</td>
<td>0.18 ± 0.10</td>
<td>0.78 ± 0.08</td>
<td>0.67 ± 0.10</td>
</tr>
<tr>
<td></td>
<td>Final†</td>
<td>0.34 ± 0.12</td>
<td>0.16 ± 0.06</td>
<td>0.12 ± 0.03</td>
<td>0.75 ± 0.08</td>
<td>0.71 ± 0.04</td>
</tr>
<tr>
<td>Gill</td>
<td>Initial</td>
<td>0.23 ± 0.13</td>
<td>0.22 ± 0.09</td>
<td>0.31 ± 0.05</td>
<td>0.77 ± 0.24</td>
<td>0.42 ± 0.09</td>
</tr>
<tr>
<td></td>
<td>Final†</td>
<td>0.23 ± 0.03</td>
<td>0.23 ± 0.05</td>
<td>0.22 ± 0.12</td>
<td>0.68 ± 0.13</td>
<td>0.52 ± 0.12</td>
</tr>
</tbody>
</table>

†, †† Significantly different from the corresponding initial values for \(p<0.05\) and \(p<0.01\), respectively.

*1 Values represent mean ± SD of four individuals.

*2 Total adenine nucleotide = ATP + ADP + AMP.

*3 Energy charge = \((ATP^{1/2} \times ADP)/(AMP \times ADP + ATP)\).

*4 Just before respiratory arrest.

Fig. 5. Changes in concentrations of adenine nucleotide and energy charge in hepatopancreas of red sea bream exposed to hypoxia. Each value represents the mean ± SD, \(n=4\). ■, Energy charge; ○, Total adenine nucleotide; ○, ATP; △, ADP; □, AMP. Refer to Figs. 3 and 4 for bias background and superscript letters, respectively.
stantially lower than that of tilapia\textsuperscript{17-19}, in addition to those of goldfish\textsuperscript{29} and common carp\textsuperscript{15}, which display anoxic tolerance. The hemoglobin-oxygen affinity of fish inhabiting low-oxygen water regions is known to generally be higher than that of fish from normal water regions\textsuperscript{30,31}. These species differences may depend on oxygen affinity to hemoglobin up to a maximum respiration frequency and may explain hypoxic tolerance.

In this study, the hematocrit value of red sea bream increased almost linearly with hypoxia up to the time when the fish sank (Fig. 4). MCHC markedly decreased with increasing hypoxia load. Under hypoxic conditions, the same phenomena were observed in Japanese parrot fish\textsuperscript{2} and Nile tilapia\textsuperscript{17} in previous studies. An increase in cell volume during hypoxia appears to increase hemoglobin-oxygen affinity in lampreys and teleost fish\textsuperscript{31}. The increase in erythrocyte volume is coupled to increases in erythrocyte pH\textsuperscript{32,33}. These changes are caused by catecholamine activation of sodium proton exchange across the erythrocyte membrane during hypoxia\textsuperscript{31,34}. This is suggestive of the same response obtained in red sea bream under hypoxic conditions.

**Stress response and energy supply**

In red sea bream, as respiratory frequency peaked at 35 mmHg PO\textsubscript{2}, a significant decrease in MCHC-related catecholamine release was observed (Fig. 4). Plasma cortisol concentration subsequently peaked at 20 mmHg PO\textsubscript{2}. Glucose levels likewise reached a maximum immediately prior to respiratory arrest, with a lag of only about 2 h from the drastic cortisol elevation. It is well known that stressors elevate energy supply through cortisol release and hyperglycemia in many fish\textsuperscript{35}. On the other hand, Ishioka\textsuperscript{21} reported the stress response in red sea bream to various changes in oxygen level, temperature, and salinity, in addition to transport, fishing, and anesthesia. She was also suggested that catecholamine played an important role in glucose release in the first phase of stress response by \textit{in vitro} experiments. In stress conditions, neuroendocrine controls of cortisol and catecholamine axes are likely to be interrelated\textsuperscript{36}. Vijayan \textit{et al.}\textsuperscript{37,38} hypothesized that adrenaline is involved in the immediate production of glucose after stress, whereas cortisol, either directly or indirectly, or perhaps with other hormones, regulates glucose in fish. In the present study, stress responses to hypoxic conditions occurred by, first, catecholamine release with decreased MCHC at maximum respiratory frequency, followed by cortisol release as respiratory frequency decreased. Up to respiratory arrest, increases in glucose level were due to catecholamine and/or cortisol. While the latter cannot be confirmed in this experiment, energy supply under hypoxic condition is an important factor in hypoxic adaptation of red sea bream.

**Energy status under hypoxic conditions**

Just before respiratory arrest, the ATP and TA concentrations in the hepatopancreas and TA in the kidney of fishes were considerably diminished by hypoxia (Table 1). The ATP and TA in ordinary muscle and heart showed a tendency to decrease under hypoxic conditions and but not in the gill and brain. In Nile tilapia\textsuperscript{17}, a similar trend was observed in each tissue. ATP and TA reduction rates under hypoxia displayed the following order: hepatopancreas > kidney > ordinary muscle > heart > brain and gill. These experimental data on red sea bream, particularly the trends in the hepatopancreas, kidney and muscle were also in accordance with the grass frog, \textit{Rana temporaria}\textsuperscript{39}. However, the effects of stress on ATP and TA levels as well as EC in each tissue were far less than in tilapia and frog (e.g. 0.18 and 0.60 as hepatopancreas EC for Nile tilapia and red sea bream just before respiratory arrest, respectively). This may be one of the reasons why the hypoxic tolerance of red sea bream was substantially lower than that of other fish and frogs.

In the hepatopancreas just before respiratory arrest, the percentage decrease in ATP level compared to initial value was around 60%. Rainbow trout exposed to severe levels of oxygen restriction showed approximately
70% diminution in ATP levels in the liver\textsuperscript{40}. Conversely, in the teleost flounder, \textit{Platichthys flesus}\textsuperscript{41}, common carp\textsuperscript{60} and Nile tilapia\textsuperscript{17} which have higher tolerance to low oxygen, hypoxia caused about 90\%, 90\% and 96\% drops in ATP concentrations, respectively. Moreover, ATP levels of hepatocytes isolated from goldfish were markedly depressed by hypoxia\textsuperscript{42}. The liver in stress-tolerant animals such as frogs, flounder, common carp, tilapia and goldfish may conceivably adapt to severe hypoxia by reducing energy status and metabolic turnover rate\textsuperscript{39}. Although differences in this reduction ability are present between stress-tolerant animals and red sea bream, the hepatopancreas in red sea bream also seems to be capable of adaptation to severe low-oxygen levels by reducing both energy status and metabolic turnover rate.

We measured adenine nucleotide concentrations at each stage of hypoxia to determine aspects of energy status in hepatopancreas with increasing hypoxia load (Fig. 5). The ATP, TA and EC levels in the hepatopancreas steeply decreased from the peak level together with the decrease in respiratory frequency, and showed a significant difference just before respiratory arrest. The adult red sea bream exposed to hypoxic conditions has been shown to significantly decrease oxygen consumption and arterial blood PO\textsubscript{2} to around 20 mmHg as oxygen regulator\textsuperscript{43,44}. Otherwise, anaerobic respiration based on lactate accumulation under hypoxic conditions in red sea bream\textsuperscript{45}, tilapia\textsuperscript{17}, and Japanese parrot fish\textsuperscript{3} appears when the fish sinks. In red sea bream, decreased energy status in the hepatopancreas has been suggested to occur between the maximum and decreased respiratory frequency, indicating total metabolic depression without increasing aerobic and/or anaerobic metabolism.

\textbf{Outline and comparison of hypoxic adaptation methods}

In the previous study\textsuperscript{17}, we reported that Nile tilapia responds to hypoxia by first attempting to maintain oxygen uptake by increasing respiration frequency and erythrocyte volume. Subsequently, tilapia adapted to hypoxia by conserving energy through metabolic depression without increasing aerobic and/or anaerobic metabolism, as well as by stress response through increasing cortisol and glucose. Finally, tilapia utilized anaerobic respiration through lactate and creatine production. Adaptation methods to hypoxia in red sea bream were also observed as three phases, namely, (1) by increasing ventilation rates and erythrocyte volume\textsuperscript{44}, (2) by conserving energy through decreased energy status and oxygen consumption\textsuperscript{43,44} and by stress response through increasing cortisol and glucose, and (3) by anaerobic respiration\textsuperscript{45}. Based on this rough outline, no marked differences may be present in the basal adaptation styles of tilapia and red sea bream. However, adaptation abilities in each species differ considerably. Further studies in metabolic depression and anaerobic respiration due to increasing hypoxic load will be needed to assess the adaptation ability of red sea bream under hypoxic conditions.

\textbf{Acknowledgments}

The authors would like to express our gratitude to Prof. Shigeru Miyashita of Kinki University Fisheries Laboratory, Shirahama Station for his invaluable support on the fish sampling. We are grateful to the staff of Kinki University Fish Nursery Center, Shirahama Station and students of the School of Agriculture, Kinki University, for their assistance during the experiment.

\textbf{References}


低酸素負荷に対するマダイのストレス反応および各種組織のエネルギー状態

石橋泰典・平田八郎・熊井英水

マダイに, 酸素分圧（PO₂）を125から20 mmHgまで約5時間かけて低下し, その後, 20 mmHg以下で約3時間放置するストレスを19.9℃で負荷した。呼吸数は徐々に増加し, 35 mmHg PO₂前後で最大値を示し, その後に低下した。PO₂が20 mmHg以下になると横転魚が増加し, その後に呼吸停止した。Ht 値はストレスの負荷に伴って増大し, MCHC は低下した。血漿コルチゾールおよびグルコース量は, 呼吸数低下時に著しく増加した。肝臓のATP, TA 含量およびエネルギー充足率は, 呼吸数低下時に激減し, 呼吸停止直前に有意な低値を示した。呼吸停止直前の筋肉, 心臓および腎臓のATPおよびTA 含量は開始時よりも低い傾向を示したが, 鰓および腸に著しい差異はなかった。これよりマダイは, 呼吸数低下時にコルチゾール分泌に基づく顕著なストレス反応を示すとともに, 肝臓臓は, 同時期にエネルギー状態を低下させ, 低酸素負荷に対する代謝抑制に貢献することが示唆された。