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EFFECTS OF CO_2 ON MARINE FISH

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ABSTRACT

To provide basic information useful in assessing the impacts of ocean sequestration of CO_2 on marine fish, we studied 1) acute CO_2 tolerance during early life stages, 2) long-term effects of sublethal hypercapnia, 3) physiological responses of adults to hypercapnia, and 4) compared the effects of hypercapnic water and acidic water. These experiments clarified tolerance levels of elevated CO_2 and gave insights into compensatory mechanisms at different developmental stages.

INTRODUCTION

Increased atmospheric concentrations of CO_2 will cause not only global warming but also raise the partial pressure of CO_2 and lower pH of oceanic water. These environmental alterations are likely to affect shallow-water marine organisms. On the other hand, feasibility studies recently suggested that sequestration of anthropogenic CO_2 in the deep ocean could help reduce atmospheric CO_2 concentrations [1]. However, implementation of this strategy could have significant environmental impacts on deep-sea life. Hence there is an urgent need for detailed studies on the effect of CO_2 on marine organisms. Fish are a central component of the marine ecosystem, and constitute an important protein source, particularly for countries such as Japan. Our literature survey [2] demonstrated that the majority of work concerning effects of hypercapnia on fish has focused on freshwater species and little is known for marine species. Thus, to provide basic information useful for assessing the impacts of ocean sequestration of CO_2, the following studies were conducted.

ACUTE CO_2 TOLERANCE DURING EARLY LIFE STAGES OF MARINE FISH

We adopted a conventional toxicity test design to obtain relationships between CO_2 concentrations and survival of eggs, larvae and juveniles. For the two species we studied, LC_{50} peaked about 10 days after fertilization (preflexion stage, Fig. 1). We conducted an immunocytochemical study on chloride cells using an antiserum specific for Na^+,K^+-ATPase to examine possible involvement of the cells in compensatory mechanisms under hypercapnia. Chloride cell size significantly increased in response to 24 h exposure to 1%CO_2 in juveniles of red sea bream (rsb). A similar trend was confirmed for embryos of silver whiting (sw), though statistical comparison was not conducted because of low N (N = 2), suggesting that chloride cells play a compensatory role under hypercapnia even in embryonic stage (Fig. 2).

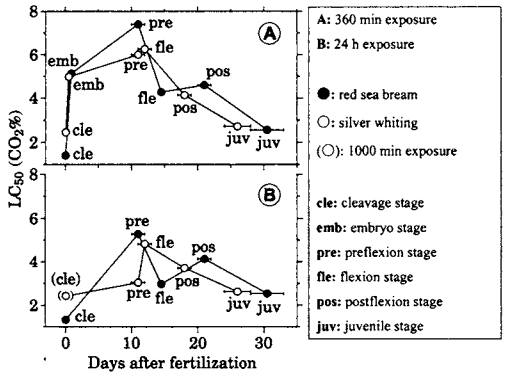

Figure 1: Ontogenetic changes of median lethal CO_2 concentration (LC_{50}) during early life stages.

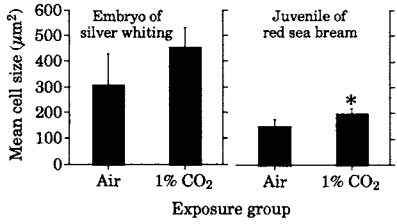

Figure 2: Mean size of chloride cells in yolk-sac membrane (left, N = 2) and in gill filament (right, N = 5) under normal and hypercapnic (1% CO_2) seawater for 24 hours. Vertical bars indicate SD. Asterisk indicate significant difference ($P < 0.05$) from control (one-way ANOVA).

LONG-TERM EFFECTS OF SUBLETHAL HYPERCAPNIA ON FISH

Juveniles of rsb and sw were cultured under sublethal CO_2 concentrations. Although growth of rsb was unaffected by CO_2 concentrations used after one month, branchial chloride cell became significantly larger at higher CO_2 concentrations (Fig. 3). After five months, mean body weight was significantly lower in

hypercapnic groups of sw than in the control group. There was no difference in gonad development between the two groups of sw.

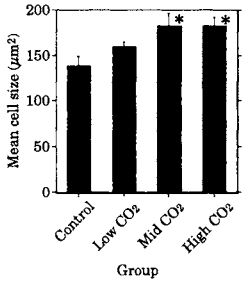

Figure 3: Mean size of chloride cells in gill filament of young red sea bream reared under control (normal seawater, pH 8.1), low CO_2 (0.25% CO_2, pH 7.3), mid CO_2 (0.5% CO_2, pH 7.0), high CO_2 (1% CO_2, pH 6.8) conditions for 4 weeks. Vertical bars indicate SD. Asterisks show significant difference ($P < 0.05$) from control (Dunnet test, N=3).

PHYSIOLOGICAL RESPONSES OF ADULT FISH TO HYPERCAPNIA

Blood acid-base variables were measured under control conditions and subsequent hypercapnic conditions up to lethal level for Japanese flounder, yellowtail and gummy shark. Acid-base disturbances were compensated in all fish while the rate and extent of compensation varied among them (Fig. 4).

COMPARISON OF THE EFFECTS OF HYPERCAPNIC WATER AND ACIDIC WATER

CO_2 exposure resulted in consistently higher mortality than exposure to acidified water (Table. 1). Acid-base responses were markedly different between the two conditions (Fig. 5). The results indicate that experimental data on acidic water cannot be used to assess the impact of Ocean Storage of CO_2.

TABLE 1

COMPARISON OF LETHAL EFFECT OF HYPERCAPNIC WATER (pH6.2) AND ACIDIC WATER (pH6.2) ON FISH

Animal	Exposure time	Mortality by CO_2	Mortality by acid
Adult of Japanese flounder	48h	100%	0%
Egg (embryo) of red sea bream	6h	86%	4%
Larva (preflexion) of red sea bream	24h	62%	1%

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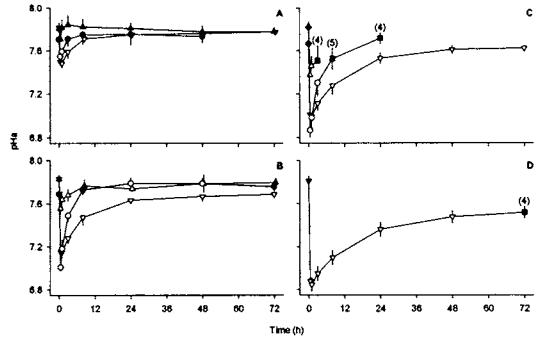

Figure 4: Arterial pH of the Japanese flounder (●), the yellowtail (▲), and the gummy shark (▼) during exposure to hypercapnic water equilibrated with 1% (**A**), 3% (**B**), 5% (**C**) and 7% CO2 in air (**D**) (flounder; $N = 6$, yellowtail and dogfish; $N = 5$). Open symbols indicate significant differences from 0-h values ($P < 0.05$). N was decreased due to mortality at 5% and 7% (■), to which no statistical comparison was applied.

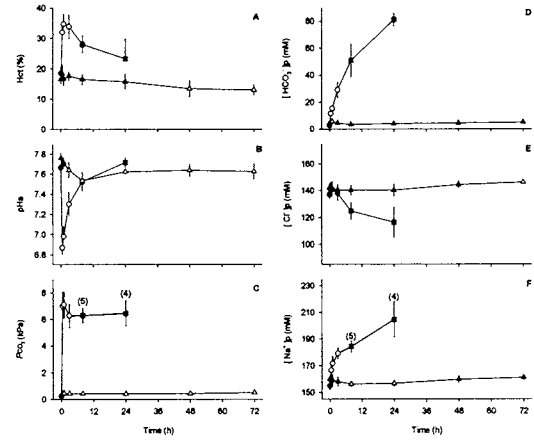

Figure 5: Arterial haematocrit (**A**), arterial pH (**B**), blood Pco_2 (**C**), and plasma $[HCO_3^-]$ (**D**), $[Cl^-]$ (**E**) and $[Na^+]$ (**F**) of the Japanese flounder during exposure to hypercapnic water (●) and acidic water (▲) (hypercapnic water exposure; $N = 6$, acidic water exposure; $N = 5$). Open symbols indicate significant difference from 0-h values ($p < 0.05$). N was decreased due to mortality at hypercapnic water exposure (■), to which no statistical comparison was applied.

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