Effects of Thermal Stressors on Growth-Related Gene Expressions in Cultured Fish

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Abstract

Growth in fish is regulated by the growth hormone (GH)-growth hormone receptor (GHR)-insulin-like growth factor-1 (IGF-1) axis. However, the effect of severe acute stressors on the GH-IGF-1 axis in fish is not well understood. The present study determined the changes in mRNA expression of growth-related genes *gh*, *ghr*, and *igf* and the redox state in coho salmon (*Oncorhynchus kisutch*), in response to severe acute stress. Severe stress consisted of exposure to heat shock (adequate rearing temperature +11 °C for 2 h). The plasma expression patterns of redox state-related biomarkers, such as glutathione, lipid peroxides, and superoxide dismutase, in response to heat shock suggest that heat shock might induce oxidative stress in fish. After exposure to heat shock, *ghr* mRNA levels in the pituitary glands and liver increased, whereas levels decreased 48 h post-stress. Hepatic *igf1* mRNA expression levels gradually decreased in response to the stressor. On the other hand, the pituitary *gh* mRNA expression did not change in response to the stressor. These findings showed that a heat shock-induced oxidative stress could affect the redox state and the expression of several growth-related genes in coho salmon. The results of this study also suggest that the expression of several growth-related genes in fish may be affected differently by the types and strength of stress.

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1 Introduction

It is well known that fishing and aquaculture are very important to world food production. World market demand for high quality products has stimulated much of the growth in aquaculture, especially for salmonid, shrimp, and shellfish species (Nakano 2007). Cultured fish are exposed to biotic and abiotic stressors, such as toxicants and acute changes in temperature, which can increase the chances of these fish succumbing to infectious disease (Pickering 1993; Nakano and Takeuchi 1997; Nakano et al. 1999a; Iwama et al. 2006; Nakano 2007, 2011; Pankhurst 2011; Ellis et al. 2012; Prunet et al. 2012). Furthermore, perturbations due to global climate change, typhoon, tsunami, and artificial factors such as environmental pollutants, radioactive contaminants derived from nuclear power plant accident, in aquatic biological systems have recently become a serious problem (Pörtner 2002; Lesser 2006; Valavanidis et al. 2006; Hofmann and Todgham 2010; Urushihara 2013; Hara 2014).

In response to a particular stressor, a series of biochemical and physiological changes occur at both the cellular and organismal levels. These stress responses in fish can affect their general health, disease resistance, growth, and reproduction (Barton and Iwama 1991; Pickering 1993; Pickering and Pottinger 1995; Pankhurst and Kraak 1997; Barton 1997; Nakano 2011; Prunet et al. 2012).

The growth of fish is regulated to a large extent by liver-derived insulin-like growth factor (IGF)-1 in response to pituitary-secreted growth hormone (GH) binding to GH receptor (GHR) in the liver. The GH-IGF-1 axis has a critical role in regulating both fish growth and development (Kopchick and Andry 2000; Moriyama et al. 2000; Björnsson et al. 2002; Reineck et al. 2005; Klein and Sheridan 2008; Deane and Woo 2009; Reineck 2010).

Fish growth is genetically regulated and is also influenced by cellular, endocrinological, and environmental factors. The responses of endocrine tissue are affected by the integration of external stimuli with internal signals according to the physiological state (Peter 1979; Barton and Iwama 1991; Pickering 1993; Pickering and Pottinger 1995; Duan 1998; Moriyama et al. 2000; Mommsen and Moon 2001; Iwama et al. 2006; Kameda et al. 2008; Deane and Woo 2009; Reineck 2010; Nakano 2011; Prunet et al. 2012).

The physiological states of ectothermal organisms, such as fish, depend on the environmental temperature. Studies on thermal stress in fish have primarily focused on cellular molecular chaperones, heat shock proteins (HSPs), expression, and characterization (Iwama et al. 1998; Feder and Hofmann 1999; Basu et al. 2001, 2002; Pörtner 2002; Nakano 2011; Iwama et al. 2006). Little is known about the effects of severe acute stressors, such as heat shock, on the expression levels of genes that are related to growth in fish (Pörtner 2002; Lushchak and Bagnyukova 2006a; Kameda et al. 2008; Deane and Woo 2009; Reineck 2010; Nakano 2011; Beckman 2011; Nakano et al. 2013, 2014). Therefore, it is important to determine the effects of thermal stress on fish fitness and tolerance in order to improve their production and health in both natural and cultural conditions.

In this study, we examined changes in mRNA expression levels of the *gh*, *ghr*, and *igf1* genes in response to a severe acute stress derived from heat shock in coho salmon (*Oncorhynchus kisutch*). Coho salmon is known to be one of the most valued species used in aquaculture. We discuss the relationships between the thermal stress responses, expressions of growth-related genes, and the oxidative stress in fish in the context of our findings.

2 Materials and Methods

2.1 Fish, Rearing Conditions, Stress Performance, and Sampling

Coho salmon were purchased from a local hatchery, Sakai Hatchery Co., in Zao town,

Miyagi, Japan. After acclimatization for 2 weeks at the aquarium facility of Tohoku University, fish (approx. body weight, 144 g) were reared in 60-L flow-through glass tanks at 8 °C (light/dark = 12 h/12 h). The fish were fed by hand to apparent satiation twice a day with commercial feed (Nosan Co., Japan). Food was withheld for over 48 h before each sampling period. Fish were exposed to heat shock (+11 °C for 2 h) and sampled at 2.5, 17.5, and 48 h post-stress. Blood was collected from the caudal vessels under MS222 (m-aminobenzoic acid ethyl ester methanesulfonate) anesthesia. The plasma was separated by centrifugation and frozen at −80 °C. Fish were gutted; the tissues were quickly removed and frozen at −80 °C in RNA later (Ambion, Life Technologies, Austin, TX) until analysis.

Our experiments were conducted in accordance with the principles and procedures approved by the Animal Care Committee at Tohoku University (Sendai, Japan).

2.2 Plasma Cortisol and Glucose Levels

Plasma cortisol levels were measured using an enzyme-linked immunosorbent assay kit (Oxford Biomedical Research, UK) (Basu et al. 2001). Plasma glucose was measured using an enzymatic assay method with a Glucose CII-Test Wako kit (Wako Pure Chemical Industries, Ltd., Japan).

2.3 Plasma Lipid Peroxides, Glutathione, and Superoxide Dismutase Levels

Lipid peroxides (LPO) were determined as thiobarbituric acid-reactive substances (TBARS) by a HPLC-fluorescence method (Wong et al. 1987; Morliere et al. 1991). Glutathione (GSH) levels in plasma were determined by a glutathione reductase-recycling method with a Total Glutathione Quantification Kit (Dojindo Laboratories, Japan). This kit can measure the total amount of reduced GSH and oxidized form of GSH. The superoxide dismutase (SOD) activity was assayed by the formazan-WST method (Total SOD Assay Kit, Dojindo Laboratories, Japan).

2.4 RNA Extraction and cDNA Synthesis

Tissues were suspended in TRIzol Reagent (Invitrogen, Life Technologies, CA) and immediately homogenized using a polypropylene pestle. The resulting RNA pellet was dissolved in RNase-free water (UltraPure, Gibco, Life Technologies, NY), quantified by spectrophotometry (V-630-Bio, JASCO, Japan), and then diluted to 500 ng/μL for use in reverse transcription reactions. RNA samples were stored at −80 °C. Complementary DNA (cDNA) was synthesized using a ReverTra Ace qPCR RT Kit (Toyobo, Japan) with a mixture of random hexamers and oligo-dT primers or with a gene-specific primer for salmon GH (gh-reverse primer) and 250 ng of RNA (Nakano et al. 2013).

2.5 Real-Time qPCR for *gh, ghr, igf1,* and *arp* mRNA Levels

The mRNA expression levels of *gh*, *ghr*, and *igf1* in tissues were determined by a real-time quantitative PCR (qPCR) with an ABI Prism 7300 Sequence Detection System (Applied Biosystems, Life Technologies, Foster City, CA) using acidic ribosomal phosphoprotein P0 gene (*arp*) as an internal standard (Pierce et al. 2004; Nakano et al. 2013). mRNA values for *gh, ghr*, and *igf1* were normalized to those for *arp*. Accordingly, each sample amplification value for each gene was expressed as a relative gene expression ratio (relative mRNA level).

2.6 Statistical Analysis

All samples were run in duplicate and results were expressed as means ± SEM. All data were subjected to one-way analysis of variance

(ANOVA). Multiple comparisons between groups were made by the Tukey-Kramer method.

3 Results

3.1 Plasma Cortisol and Glucose Levels

Plasma cortisol levels increased at 2.5 h post-heat stress compared with those in control fish, but returned to basal levels at 17.5 h post-stress (Fig. 1a).

Plasma glucose levels increased at 2.5 and 17.5 h post-stress compared with those in control fish. However, at 48 h post-stress, plasma glucose levels in stressed fish decreased and were not significantly different from those in control fish (Fig. 1b).

3.2 Plasma LPO, GSH, and SOD Levels

Plasma LPO and GSH levels are shown in Fig. 2. As shown in Fig. 2a, the plasma LPO levels in stressed fish gradually increased after heat shock treatment and increased significantly compared with those in control fish at 17.5 and 48 h post-stress.

Plasma GSH levels decreased at 2.5 h post-heat stress, but returned to basal levels at 17.5 h post-stress (Fig. 2b). At 48 h post-stress, plasma glutathione levels in stressed fish increased significantly as compared to those in control fish.

Plasma SOD activity in stressed fish increased significantly compared with that in control fish at 17.5 h post-stress, but returned to basal levels at 48 h post-stress (data not shown).

3.3 *gh*, *ghr*, and *igf1* mRNA Levels

The mRNA expression levels of *gh, ghr*, and *igf1* in the pituitary glands and livers of stressed and control fish were compared (Figs. 3 and 4).

In the pituitary glands, *gh* mRNA levels were not significantly different between the control and stressed fish (data not shown). *ghr* mRNA expression levels in the pituitary glands of stressed fish gradually increased after heat stress treatment, with the highest level at 17.5 h post-stress. At 48 h post-stress, *ghr* mRNA expression levels returned to their basal levels (Fig. 3).

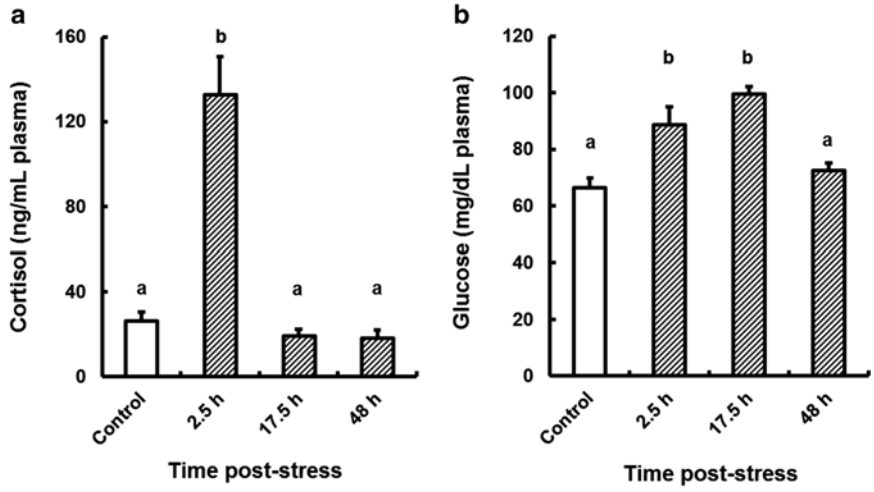

Fig. 1 (**a** and **b**) Effect of thermal stress on cortisol (**a**) and glucose (**b**) levels in plasma from coho salmon *O. kisutch*. Data represent means ± SEM ($n = 8$). Statistical relationships between groups are indicated by *letters* where significant differences were detected ($p < 0.05$)

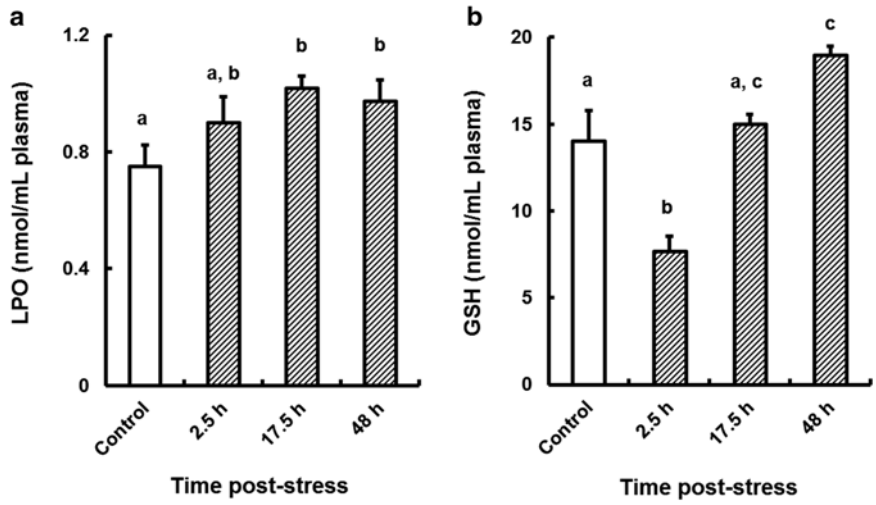

Fig. 2 (**a** and **b**) Effect of thermal stress on LPO (**a**) and GSH (**b**) levels in plasma from coho salmon *O. kisutch.* Data represent means ± SEM ($n = 5$). Statistical relationships between groups are indicated by *letters* where significant differences were detected ($p < 0.05$)

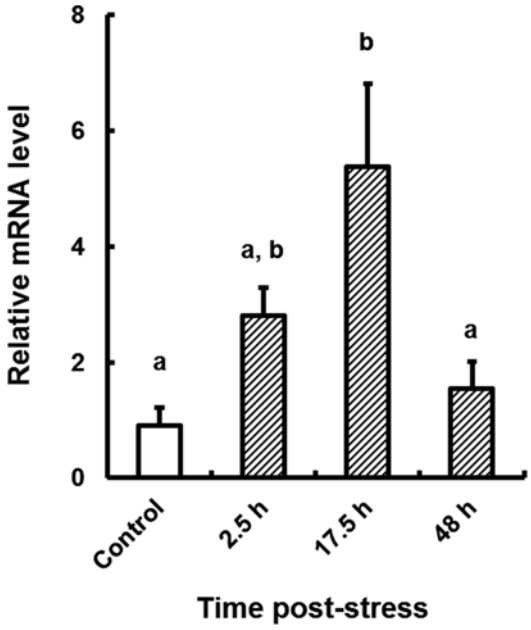

Fig. 3 Effect of thermal stress on expression level of *ghr* mRNA in the pituitary from coho salmon *O. kisutch*. The expressions of target gene were normalized by *arp* expressions. Data represent means ± SEM ($n = 4$). Statistical relationships between groups are indicated by *letters* where significant differences were detected ($p < 0.05$)

As shown in Fig. 4a, *ghr* mRNA levels in the livers of stressed fish increased. In contrast, the *igf1* mRNA levels in stressed fish livers decreased gradually after heat stress and decreased significantly as compared with those in the control fish at 48 h post-stress (Fig. 4b).

4 Discussion

The results of this study demonstrated that a severe acute thermal stressor could affect the redox state and the expression of growth-related genes in coho salmon.

The temperature can induce numerous changes in the biological functions of organisms. Increased environmental temperature results in increased oxygen consumption and stimulates various metabolic processes on the basis of known thermodynamic principles (Pörtner 2002; Lesser 2006; Lushchak and Bagnyukova 2006a, b; Lushchak 2011).

LPO, expressed as TBARS, in plasma are considered to be metabolites derived from various damaged tissues (Parihar and Dubey 1995; Nakano and Takeuchi 1997; Nakano et al. 1999a, b; Rau et al. 2004; Lushchak et al. 2005a, b; Olsen et al. 2005; Heise et al. 2006; Bagnyukova et al. 2007). In this study, the plasma TBARS levels of fish exposed to heat shock increased. TBARS levels in fish tissues

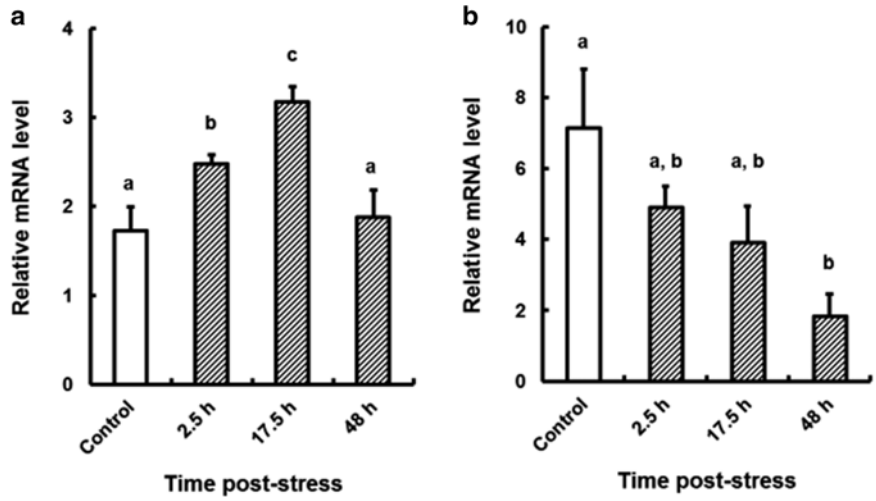

Fig. 4 (**a** and **b**) Effect of thermal stress on expression levels of *ghr* (**a**) and *igf1* (**b**) mRNA in the liver from coho salmon *O. kisutch*. The expressions of target gene were normalized by *arp* expressions. Data represent means ± SEM ($n=4$). Statistical relationships between groups are indicated by *letters* where significant differences were detected ($p<0.05$)

can change under several stressful conditions, such as heat exposure, handling stress, hyperoxia, oxidized oils administration, and heavy metal intake (Parihar and Dubey 1995; Nakano et al. 1999a; Ali et al. 2004; Rau et al. 2004; Lushchak et al. 2005a, b; Martínez-Álvarez et al. 2005; Olsen et al. 2005; Heise et al. 2006; Lesser 2006; Valavanidis et al. 2006; Bagnyukova et al. 2007).

The major nonprotein cellular thiol, reduced GSH, is a tripeptide (Glu-Cys-Gly) with reducing and nucleophilic properties that is one of the major regulators of the intracellular redox state (Niki 1988; Nakano and Takeuchi 1997; Arrigo 1999; Sies 1999; Lesser 2006; Valavanidis et al. 2006). GSH can act as a chain breaker of free radical reaction and is the substrate for glutathione peroxidase, an enzyme that scavenges reactive oxygen species (ROS) and LPO generated within cells. The plasma GSH levels observed in this study were similar to those in the livers of fish that were administered with an oxidant, such as t-butyl hydroperoxide, after heat exposure (Ploch et al. 1999; Ali et al. 2004; Lushchak and Bagnyukova 2006a, b; Heise et al. 2006; Valavanidis et al. 2006; Bagnyukova et al. 2007). At the initial post-heat stress stage, GSH may be consumed to eliminate ROS generated in blood.

Plasma SOD activity in heat-shocked fish showed a transient increase at 17.5 h post-stress in this study. Antioxidative enzymes, such as SOD, glutathione peroxidase, and catalase, can scavenge radicals and contribute to the body's antioxidative defenses. In particular, SODs are considered to have key roles in the first line of the enzymatic antioxidative defense system against oxidative injuries, as they catalyze the removal of oxygen radical, superoxide (Asada 1988; Oyanagui 1989; Nakano and Takeuchi 1997; Taniguchi and Endo 2000; Zelko et al. 2002; Martínez-Álvarez et al. 2005; Lesser 2006; Lushchak 2011). Hence, increased SOD expression may neutralize the harmful effects of superoxides in tissues. The changes in the expression of antioxidative enzymes, such as SOD, in fish have been observed with regard to stress (Poly 1997; Pörtner 2002; Martínez-Álvarez et al. 2005; Valavanidis et al. 2006; Craig et al. 2007; Lushchak 2011).

Heat exposure and enhanced oxygen consumption are considered to promote ROS generation in the tissue. The resulting ROS attack almost all cell components (Asada 1988; Nakano and Takeuchi 1997; Beckman and Ames 1998; Droge 2002; Lesser 2006; Valavanidis et al. 2006; Lushchak 2011). ROS production in cell,

especially in mitochondria where spin-off of superoxides during mitochondria transfer of electrons to oxygen occurs, was increased in exercised mammalian muscle, heat-stressed bivalve gills, heat-shocked chicken muscles, and cultured cells as compared with non-stressed control (Ji 1995; Pörtner 2002; Heise et al. 2003; Martínez-Álvarez et al. 2005; Mujahid et al. 2005; Lesser 2006; Valavanidis et al. 2006; Shin et al. 2008). Inductions of hepatic HSP and GSH in stressed coho salmon have been observed in this study (Nakano et al. 2014). Thus, the present results regarding the plasma levels of cortisol and glucose and the expression patterns of redox state-related biomarkers, such as LPO, GSH, SOD, and HSP, in response to heat shock suggest that heat shock induces oxidative stress in fish. The resulting oxidative stress may enhance oxidation in the body and result in damage to tissues (Nakano and Takeuchi 1997; Nakano et al. 1999a; Martínez-Álvarez et al. 2005; Lesser 2006; Valavanidis et al. 2006; Nakano 2007, 2011; Lushchak 2011). Under oxidative stress conditions, the levels of antioxidative agents, such as GSH, SOD, and HSP, may increase due to their de novo synthesis.

The pituitary is the major organ of the GH-IGF-1 axis in fish (Kobayashi et al. 2002; Takei and Loretz 2006). In the present study, *ghr* gene expression in the pituitary increased in response to heat stress. In contrast, pituitary *ghr* gene expression significantly decreased in response to moderate acute physiological stress induced by handling (Nakano et al. 2013). This may involve interactions between glucocorticoids, such as cortisol and factors of the GH-IGF-1 axis, and be related to the differences observed between heat shock-induced oxidative stress and mild physiological stress derived from handling. The effects of stress on growth-related gene expression in fish may be affected differently by the strength of stress.

Hepatic *igf1* expression gradually decreased after heat shock treatment, whereas *ghr* expression levels increased after heat shock in this study. Thus, *ghr* and *igf1* genes responded differently to heat stress. Furthermore, pituitary *gh* mRNA expression did not change in response to oxidative stress. These observations suggest that igf1 gene expression may not be directly regulated by circulating GH levels alone.

A decrease in plasma IGF-1 level at 2–24 h post-stress has often been observed in many fish species (Beckman 2011). Our results for the changes in hepatic *igf1* gene expression levels in stressed fish are in agreement with those reported for fish that were administered exogenous cortisol and under confinement stress (Kajimura et al. 2003; Dyer et al. 2004; Saera-Vila et al. 2009). However, our results for hepatic *ghr* and *igf1* gene expression patterns in fish under oxidative stress appear to be different from those in coho salmon under mild physiological stress caused by handling (Nakano et al. 2013). These observations suggest that the hormonal regulation of hepatic both *ghr* and *igf1* gene expression can vary depending on the types of hormones and stress.

5 Conclusions and Perspectives

Oxidative stress could affect the expression of growth-related genes accompanying the changing in circulating glucocorticoids, such as cortisol, and alterations in signal transduction. Intercellular signaling is known to be affected by ROS (Schoeniger et al. 1994; Ji 1995; Franco et al. 1999; Arrigo 1999; Allen and Tresini 2000; Droge 2002; Lesser 2006; Valavanidis et al. 2006; Shin et al. 2008; Nakano 2011; Lushchak 2011). An antioxidative supplement that could dramatically reduce oxidative stress-induced damage in fish has been observed (Nakano et al. 1999a, b, 2004; Bell et al. 2000; Martínez-Álvarez et al. 2005; Nakano 2007, 2011). Accordingly, further studies are required on the possible beneficial effects of antioxidative nutraceutical supplements on growth-related factors in oxidative stressed fish.

Marine ecosystems could provide various products and services, including vital food resources for us (Holmlund and Hammer 1999; Worm et al. 2006). The ability of the ocean is thought to maintain water quality and regulate

perturbations and other essential ecosystem services (Worm et al. 2006). Marine ecosystems can be influenced by exploitation, pollution, biodiversity loss, and habitat destruction or indirectly through global climate change and related perturbations (Worm et al. 2005, 2006; Berque and Matsuda 2013). In the northeastern (Tohoku) Pacific coastal area, Sanriku Coast, where fishing and farming are known to be essential to the industries, the Great East Japan Earthquake caused the perturbations in 2011 (Urushihara 2013; Hara 2014). The impact of the earthquake and massive tsunami on the Sanriku area and the subsequent processes of transition over time are yet to be determined. Facilitation of reconstruction of the coastal environment and fisheries at the Sanriku Coast has been required. Therefore, especially in a disaster-stricken area such as the Sanriku Coast, it is thought to be important to research on perturbations, recovery, and resilience processes in marine ecosystems and the management of socio-ecological system in fishing and aquaculture to take sustainable delivery of environmental benefits linked to human well-being (Hadjimichael et al. 2013). Furthermore, a study on the effect of stress in marine organisms is emerging as a worldwide common theme in relation to the perturbations and resilience on marine ecosystems (Pörtner 2002; Lesser 2006; Valavanidis et al. 2006; Hofmann and Todgham 2010; Nakano 2011; Nakano et al. 2013, 2014). Consequently, the results of this study on stress response in fish can provide information that is useful for improving fish fitness and production of fishing and aquaculture. To determine the relationships between oxidative stress, growth-related factors, antioxidant defenses, and the growth of fish, additional investigations are currently underway.

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