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Review

Understanding the adaptive response in vertebrates: The phenomenon of ease and ease response during post-stress acclimation

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ABSTRACT

Vertebrates have evolved mechanisms to perceive stressors that arise either from their body or from the environment. Consequently, a state of stress and stress response occur in fish which is characterized by a disturbed physiological homeostasis. The pattern of stress response becomes complex as a result of neuroendocrine involvement and shows varied magnitudes in fishes depending on the nature and the severity of stressors. The integrated and compensatory physiological modifications in fishes during their early phase of adaptive response favor them to accommodate the imposed stressor through the process of stress acclimation. In contrast, with the direction of neuroendocrine signals, a phase of recovery often called post-stress acclimation occurs if the animal gets away from the stressor exposure. During this late phase of adaptive response, physiological modifications operate in favor of the animal that reduces the magnitude of stress response and finally to a phase of normality as animals possess the urge to correct its disrupted homeostasis. The phenomenon of ease and its response thus reduces the allostatic load, resets the homeostatic state through physiologic processes and corrects the stress-induced homeostatic disturbance with the aid of neuroendocrine signals. Ample evidences are now available to support this novel concept of ease and ease response where mitigation of the intensity of stress response occurs physiologically. Treatment of fish with melatonin or serotonin precursor tryptophan can modify the magnitude of stress response as evident in the pattern of tested physiological indices. In addition to cortisol, thyroid hormone as a major stress modifier hormone is involved in the regulation of ease response in fish probably due to the mechanisms involving inter-hormonal interference. Understanding the mechanisms of adaptive responses in vertebrates thus warranties more studies on the physiology of ease and its response.

1. Introduction

Perception of stressors and the subsequent physiological and behavioral responses in animals are a major part of stress adaptation that can promote their survival in the dynamic environment. The disturbed homeostatic state in animals often called stress evokes a set of stress responses [55,17,32] and that demands physiological correction. A wide variety of extrinsic or intrinsic stressors impose physiological modifications particularly on the rate of energy utilization, osmotic and metabolic functions which can adversely affect the growth in vertebrates including fishes [2,33,21,32]. For instance, environmental toxicants can modify the pattern of metabolic regulation [12,13,23,43], energy balance [33,55,38] and mineral and water balance in fishes [49,43,23]. Evidence have been presented that neuroendocrine signals that arise from the endocrine axes directs physiological mechanisms leading to stress responses in fishes. These signals can thus play a major role in integrating and modulating the network of physiological processes during stress and post-stress acclimation.

As an early phase of adaptive response, stress acclimation demands involvement of neuroendocrine signals that require physiological machinery. The resulting pattern of stress response thus reflects integrated physiological processes including osmotic and metabolic regulations. Extensive studies have indicated that endocrine stress axis is sensitive to both toxic and non-toxic stressors [43,39,44,40,31,32] which are also known as endocrine interrupting agents (EIAs) [19]. For example, fish interrenals are shown to have a higher sensitivity to many biological and environmental stimuli as many EIAs interrupt the cortisol release and its actions in fish [50–53,27].

Unlike stress acclimation that shows stress response, post-stress acclimation reveals a recovery response. Moreover, specific neuroendocrine signals are directed to correct the disturbed homeostasis particularly during the phase of post-stress acclimation when the animal gets off from the stressor exposure. Physiological analyses of stressed fish kept for post-stress acclimation have yielded an interesting pattern of recovery response. In this

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late phase of adaptive response, as a result of specific physiological processes a reduction in the intensity of stress response and a reversal of adverse response to normality occur at least in fish. In this review a noval concept of ease has been proposed which could explain the process of post-stress adaptive mechanism in fish. Mitigation of adverse effects of stress response is essential in all animals as it can promote survivalship in challenging environment. Catecholamines and corticosteroids, the important players of endocrine stress axis and thyroid hormone as a major stress modifier hormone are involved in ease response. In addition, specific extrinsic and intrinsic factors have also been identified in the regulation of ease responses in fish models.

2. Stress and post-stress acclimation

Extrinsic or intrinsic stimuli evoke physiological and biochemical corrections in animals. Consequently, animals accommodate these challenges with the help of physiological response pattern, which is commonly called stress acclimation [36]. In this phase of adaptive response, animal respond to stressors by showing a classic pattern of stress response because of the disturbed physiological competence. Mounting evidences underscore the role of stress hormones in the modification of physiological processes during stress acclimation particularly in fishes. Hypothalamo-pituitary-interrenal and brain sympathetic chromaffin cell axes are the primary endocrine axes that regulate stress responses in fish.

Studies on fish species have yielded a greater complexicity of adaptive responses as these aquatic vertebrates respond to an array of intrinsic and extrinsic stressors which are known for its direct and indirect effects on many physiological processes. For example, evidence is available to suggest that non-toxic stressor like net confinement and its recovery can modify the osmotic and metabolic responses of tilapia and perch [30,42]. In spite of these studies that focus on the physiological mechanisms of adaptive responses during stress acclimation, little is known about the physiological basis of post-stress acclimation which can be seen in the recovery phase of animal after a stressor exposure. For instance, keeping the climbing perch for 96 h recovery in a clean environment can trigger a series of physiological responses that can correct the disturbed homeostasis. Compensatory regulation of Na^+ K^+ ATPase activity in the organs of this fish would help them to coordinate and correct the disturbed osmotic homeostasis when kept for recovery.

Consequently, the process of stress acclimation as the first phase of adaptive response leaves a physiological recovery phase often called post-stress acclimation. During this late phase of adaptive response, animals again rely on their own physiological processes and correct its disturbed homeostatic status [36]. The process of post-stress acclimation can easily be identified physiologically in fish models when they were kept for recovery for varied time slots. With modified neuroendocrine functions these fish regain their basic homeostatic status by triggering compensatory adaptive response. This physiological response pattern in the post-stress acclimation thus clearly accounts for an adaptive phase which appears to be a prerequisite for stress adaptation.

3. Concept of ease and ease response

It is a fact that animals possess an urge to overcome the physiological and behavioral disturbances when they are confronted by a stressor or challenge. The response of fish to stressor involves the entire physiological machinery and its complex regulatory network. Consequently, the integrated and compensatory physiological modifications in animals during their early phase of adaptive response favor them to accommodate the imposed stressor through the process of stress acclimation. On the other hand, with the direction of neuroendocrine signals, a phase of recovery often called post-stress acclimation occurs if the animal gets away from the stressor exposure. During this late phase of adaptive response, the same physiological machinery operate in favor of the animal that can reduce the magnitude of stress response and finally to a phase of normality. This phenomenon of ease and its response can thus reduce the allostatic load, resets the homesostatic status and corrects the stress-induced homeostatic disruption with the help of specific neuroendocrine signals and physiological processes. In other words, ease and ease response are the innate mechanisms that work along with the stress response.

3.1. Physiological implications of ease response

Like stress acclimation, post-stress acclimation or recovery phase produces a pattern of physiological responses in fish. Altered physiological and biochemical machineries during stress and post-stress acclimation have been identified in fishes [For e.g. [11,13,38,43]. Similarly modifications of energy status in these fish due to modified hormonal actions including THs have also been demonstrated [14,16,22] which may enable them to accommodate the direct effects of stressors particularly during recovery phase [39]. For example, THs have been shown to modify the intermediary and oxidative metabolism in fish challenged by either nimbicidine or rotenone [35,39,44].

The sensitivity of thyroid to non-toxic endocrine interrupting agents (EIAs) like net confinement, air exposure, and high stocking density has been demonstrated in many fish species [20,54,5,55,38,30,32]. A modifier role of THs in stress response has now been well recognized in fish species [36,30,32] and convincing evidence for a role of TH in stress response presented recently in fishes [32]. Modification of physiological processes by THs in stressed fish clearly indicate that the status of thyroid is crucial in stress response as it modifies the stress-induced physiological alteration in fish particularly the osmotic and metabolic regulations [16]. For instance, net confinement and air exposure of perch and tilapia produced declined plasma T_3 [30,31], though many toxic stressors can either promote or produce neutral thyroid responses [42]. Many synthetic chemicals, including plant products [42] can produce thyroid disruption in fishes [7]. For example exposure of catfishes to malathion and endosulfan causes disturbances in circulating THs [56,48]. Declined T_3 has been found in rainbow trout exposed to acidic water [6].

A role for TH in the compensatory mechanism during recovery phase has been reported in fish. On the contrary, activated thyroid axis as evident in the rise of TH release during acidic water exposure and recovery phase has been demonstrated in air-breathing fish [36]. Similar activated thyroid has also been found in fish when kept for recovery after kerosene exposure [43]. Likewise, keeping the fish for recovery after carbaryl exposure (Peter MCS, unpublished) or exposure to the effluent of coconut husk retting [21,34] can stimulate thyroid activity. These differential TH responses during recovery phase indicate that both THs homeostasis and its actions are essential for the fish to adapt to environmental challenges. Similar to thyroid axis, cortisol axis also shows a greater sensitivity to many environmental contaminants. Many pollutants interfere and act through many sites including receptor levels, transporters, cellular uptake and metabolism [3,46] and adversely affect the endocrine function in fish. Induction of stress by stressors produces substantial rise in plasma cortisol in many fish species [30,32,39].

3.1.1. TH-Cortisol interference during ease response

The differential cortisol and TH responses in fish during stress and post-stress phases strongly indicate its interaction or interference at the physiological level. These hormonal interactions due to