General and Comparative Endocrinology 165 (2010) 549–556

Contents lists available at ScienceDirect

General and Comparative Endocrinology

journal homepage: www.elsevier.com/locate/ygcen

Stress and fish reproduction: The roles of allostasis and hormesis

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ARTICLE INFO

Article history:
Received 16 December 2008
Revised 2 June 2009
Accepted 3 July 2009
Available online 9 July 2009

Keywords:
Stress
Fish
Reproduction
Allostasis
Hormesis
Cortisol
Hypothalamic–pituitary–interrenal axis

ABSTRACT

This paper is a review of the effects of stress on reproduction in fishes. I hope to further the development of the concepts of *allostasis* and *hormesis* as relevant to understanding reproduction in general and in fish in particular. The main contentions I derive in this review are the following: Stressors affect fish reproduction in a variety of ways depending on the nature and severity of the stressor. The effects are transduced through a hormonal cascade initiated by perception of the stressor and involving the hypothalamus–pituitary–interrenal axis, the catecholamines, and also cytokines. Mounting a stress response and resisting a stressor is an energetically costly process, including costs associated with *allostasis*, attempting to reset homeostatic norms. Responses in emergency situations (e.g., being chased by a predator or a net) can be different from those where fish can cope (e.g., being in a more crowded environment) with a stressor, but both situations involve energy re-budgeting. Emergency responses happen in concert with the onset of energy limitations (e.g., the fish may not eat), while coping with allostatic overload can happen in a more energy-rich environment (e.g., the fish can continue to eat). Low levels of stress may have a positive effect on reproductive processes while greater stress has negative effects on fish reproduction. The concept of *hormesis* is a useful way to think about the effect of stressors on fish reproduction since responses can be nonmonotonal, often biphasic.

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1. Introduction

Stress plays a key role in the ability of vertebrates, including fishes, to perform necessary life functions, including reproduction (Schreck, 1981). The word *stress* has its roots in the physiological definition proposed by Selye (1950, 1973): "stress is the nonspecific response of the body to any demand placed upon it." A variety of definitions have been offered since that time (Pickering, 1981). In any event, it needs to be recognized that stress is the physiological response to a demand, the "stressor." More specific concepts tend to involve an endocrine cascade as part of the nonspecific response. However, I favor a broader view of *stress*, stress being the "... physiological cascade of events that occurs when the organism is attempting to resist death or reestablish homeostatic norms in the face of insult" (Schreck, 2000). To paraphrase Emlen et al. (1998), a common thread to the definition of stress is that it in some way relates to energetic efficiency; this review argues that energy repartitioning is key to how fish reproduction is affected by stressors. This effect of stress on efficiency has obvious animal welfare implications as reviewed by Broom and Johnson (1993).

1.1. The stress response and reproduction

The basic physiological response of fishes to stressors is, in a general way, quite stereotypic. The stress response *per se* has been described numerous times for fishes, initially by Mazeaud et al. (1977), expanded on in Schreck (1981), and reviewed by Barton and Iwama (1991) and Balm (1999). These reviews provide understanding of the physiological basis of the stress response. Knowledge of the genomics of the stress response in fishes is in its infancy, as reviewed by Purnet et al. (2008). I will not delve into the details of the stress response or its molecular basis here. Suffice it to say that the stress response consists of a physiological cascade of events that is initiated by perception of a stressor and communicated to the body via the central nervous system acting both neuronally and hormonally. Primary hormones include the catecholamines and those of the hypothalamic–pituitary–interrenal (adrenal) axis. The increase in circulating levels of hormones result in changes in secondary physiological responses including most organ systems. They reflect ultimately changes in osmotic status, development, immune capacity and learning, and shifts in energy stores, energy biosynthesis, and use efficiency. Particularly the latter also leads to altered reproductive capacity in one form or another (see Schreck, 2007 for brief review). Behaviors of fish are also affected by stressors (reviewed by Schreck et al., 1997). Shifts in behavior can be driven by changes in energy partitioning during the stress response, and behaving also has an energetic cost associated with it, as discussed subsequently, that could affect reproductive processes.

This review focuses on this relationship between stress, bioenergetics and reproduction, particularly in fishes. My intent is not to

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0016-6480/$ - see front matter Published by Elsevier Inc.
doi:10.1016/j.ygcen.2009.07.004

underemphasize the importance of the neuroendocrine stress response and its potential influence on the neuroendocrine control of reproduction or a potential direct effect on reproductive processes of fish. Of course, the bioenergetic affects of stress on reproduction is a consequence of neuroendocrine controlling factors; it is also likely that energetic effects of stress feed back in some direct or indirect way to affect neuroendocrine control of the stress response and the reproductive system. My perception is that the literature is comparatively replete with information on the neuroendocrine control of the stress response and the neuroendocrine control of reproduction compared to that concerning interactions of the two systems. This literature tends to be relatively descriptive, and could allow for good inference; however, it is sparse with regard to direct cause-effect types of studies or observations at the organismic level.

Hormones associated with stress clearly affect reproductive characteristics of fish. Foo and Lam (1993) demonstrated that exogenously administered cortisol resulted in retarded oocyte growth, reduced condition factor, and lowered serum testosterone and 17β-estradiol in tilapia, *Oreochromis mossambicus.* However, Pankhurst et al. (1995), experimenting with goldfish, *Carassius auratus*, carp, *Cyprinus carpio*, and a sparid, *Pagrus auratus*, demonstrated that cortisol does not affect testosterone and 17β-estradiol production, at least at the level of ovarian steroidogenesis. The negative effects of stress on reproduction of the spiney damselfish, *Acanthochromis polyacanthus*, also appears to be attributable to levels above that of steroidogensis (Pankhurst, 2001). The negative cortisol-mediated effects of stress on reproduction also do not appear mediated through inhibition of GTH secretion, as suggested by studies on the rainbow trout, *Oncorhynchus mykiss*, by Pankhurst and Van Der Kraak (2000). Small et al. (2008) reviewed the effects of stress and cortisol on fish reproduction, concluding that it is difficult to tease apart the effects of cortisol on reproduction versus survival under stressful situations.

Certain aspects of the neuroendocrinology of fish stress and fish reproduction have also recently been reviewed. An overview of the endocrine control of the morphological (gamete) and physiological control of reproduction was provided by Rocha and Rocha (2006). Stress effects primarily on hormones of the hypothalamic–hypophyseal–interrenal axis and possible interaction with those of the hypothalamic–hypophyseal–gonadal axis was reviewed by Mosconi et al. (2006). The physiology of the hypothalamic–hypophyseal–interrenal axis of fish is discussed in detail by Norris and Hobbs (2006). They also consider the endocrinology of the stress response and discuss the role of cortisol on fish reproduction. Guerriero and Ciarcia (2006) reviewed stress and reproduction in fish, particularly from the perspective of biomarkers of stress. Arginine vasotocin appears to be an integral part of the stress response of fish that could affect reproduction by playing a regulatory role in ACTH secretion (Balment et al., 2006). Urotensin-I is also corticotropic, likely more than CRF-I and is involved in the regulation of cortisol during reproduction as shown in the Masu salmon, *Oncorhunchus masou*, by Westring et al. (2008).

The bioenergetic aspect of the stress response is best thought about within the paradigm of allostasis. Allostasis is the bodily process of attempting to achieve stability, i.e., homeostasis, by varying physiological and behavior operations (McEwen and Wingfield, 2003). The energy associated with attempting to stabilize the body's functions imposes an energetic load, allostatic load. Given that the physiological and organismic response to stressors can be nonmonotonic, I also include in this chapter discussion of the relevance of hormesis on the effects of stress on reproduction. That is to say, responses are not necessarily unimodal; they often can be bimodal. Hormesis is the phenomenon in which low doses or severities of a challenge cause one response while at a higher level or severity cause a response in the opposite direction, positive to negative, or *vice versa*. Depending on the nature of the parameter being affected, the direction of that response changes. At the organismic level one would suspect that there could be considerable variation in the effects of stress on reproduction, given the exceptionally large amount of variation in the different modes of reproduction amongst the fishes (Breder and Rosen, 1966). Because the information available for fishes is so fragmentary and their modes of reproduction are so diverse, I may be guilty of over generalizing in this review.

This paper does not discuss toxicants as stressors, even though I recognize that they may have reproductive consequences. Reproductive fitness is considered from an organismal, not a population level perspective. While nutritional stressors such as insufficient food or poor diets are often considered in terms of population demographics (see Lambert and Dutil, 1997, for example), they do relate to the energetic status of individual fish and hence have interactive effects with other stressors.

2. Stress and reproduction

Surprisingly little holistic research has been published on the effects of stressors on fish reproductive physiology or genetics. Several generalities can be gleaned from earlier reviews (Schreck et al., 1995, 2001). Stress can affect reproduction in various ways, depending on when in the life cycle it is experienced and the severity and duration of the stressor. It can accelerate ovulation or inhibit reproduction. Constraints involving mate choice can also result in greater number of gametes to compensate for poor-quality of gametes (Gowaty et al., 2007). Also, stressors encountered during one developmental phase can have effect during later phases. Jalabert (2008) nicely depicted the many aspects of physiology as fish mature and reproduce; these "aspects" are potentially vulnerable to effects of stressors. From the previous reviews one would infer that all of these aspects could be affected by stress. Fig. 1 attempts to depict reproductive events that happen during the rearing, maturation and spawning periods. Earlier work proposed that stressors have effects on fish performance in considerable measure due to redirection of energy resources associated with the stress response (Schreck and Li, 1991).

A fish's social environment can also affect reproduction through interactions with the endocrine stress response. Leitz (1987) reported that for the Siamese fighting fish, *Betta splendens*, social factors affected control of testicular steroidogenic capacity. The African cichlid *Haplochromis burtoni* is also stressed by social encounters as judged from increased circulating levels of cortisol. Fox et al. (1997) concluded that the reproductive competence of the fish is also affected by the same social interaction because size of GnRH neurons in the hypothalamo-preoptic area as well as testes size appear inversely related to social status.

Reproductive hormones of brown trout, *Salmo trutta*, are affected by crowding, where stressed fish have elevated plasma ACTH and cortisol and decrease circulating testosterone and 11-keto-testosterone. Interestingly, plasma gonadotropin was elevated by stress. These findings lead to the inference that stress can adversely affect reproduction in fishes (Pickering et al., 1987). Subsequent studies have shown that stress does indeed have a negative effect on reproductive fitness (Campbell et al., 1992, 1994; Contreras-Sanchez et al., 1998; Schreck et al., 2001). Castranova et al. (2005) demonstrated that striped bass, *Morone saxatilis*, that had genetically determined low cortisol responses to stressors, had lower androgen levels and lower spermiation response to gonadotropin treatment when subjected to stressors. Stressful handling of mature jundia, resulted in lowered 17β-estradiol; fewer oocytes could be stripped from the stressed fish and the quality of these eggs appeared reduced (Soso et al., 2008).

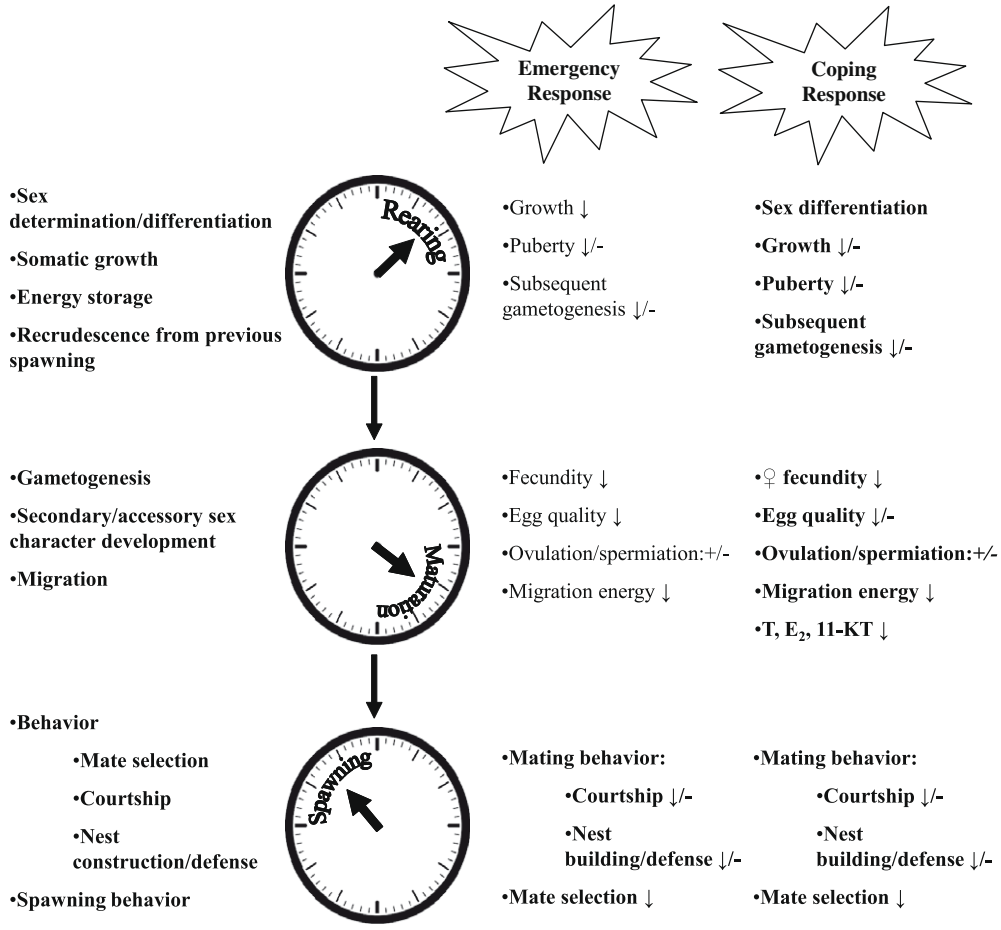

Fig. 1. The main phases of the reproductive clock and the key reproductive events that happen during each phase (shown to the left of each respective clock). The organism-level response to stressors causing emergency responses and coping responses as part of allostatic load are shown to the right of the clocks. Responses in **bold** lettering are based on solid information; responses not in bold lettering are based on strong inference. A down arrow indicates a decrease. A minus sign (−) indicates inhibition.

Most fishes are gonochoristic; therefore, under normal conditions fish remain the sex into which they are first differentiated (Devlin and Nagahama, 2002). Stress can play a large role in the reproductive biology of these fishes, however, it does not affect sex differentiation once the process is complete. However, there are a considerable number of different taxa that are hermaphroditic. The advantage of changing sex and what drives it ecologically is still under debate. It appears advantageous if the fecundity of one sex increases sufficiently under some conditions so as to outweigh the energetic costs associated with the anatomical and physiological redevelopment of the reproductive system (Warner et al., 1975; Warner, 1984). Stress may play a role in the sex change in some of these species. For example, psycho-social constraints (e.g., aggressive social interactions) may play a regulatory role for the sex change in wrasses of the protogynous genus *Thalososoma.* However, the actual mechanisms may be different. The bluehead wrasse, *T. bifasciatum*, is a haremic species where a male is dominant and females have a social hierarchy. Removal of the male head of a harem results in sex change of the dominant female into a male (Robertson, 1972). Sex change in the saddleback wrasse, *T. duperrey*, appears related to population density and growth rate (Ross, 1987). Protandrous hermaphrodites such as clown fish, *Amphiprion nigripes*, also follow a haremic-type system where removal of the dominant female causes a male to change sex (Godwin, 1994). The endocrinology, of the sex change in protogynous (Nakamura et al., 1989; Perry and Grober, 2003) and protandrous (Godwin 1994; Nakamura et al., 1994) hermaphroditic fishes has been nicely worked out. The hormones include neuropeptides, corticosteroids and sex hormones. While the mechanism regarding sex change has a neuroendocrine basis, the ultimate reason (energetics related to fecundity and sex change) or proximate reason (the trigger initiating the neuroendocrione cascade leading to sex change) may well have an energetic underpinning.

2.1. Allostasis and allostatic load

The paradigm of allostasis was introduced by Sterling and Eyer (1988) who also described the endocrine pathways involved. McEwen (1998) and McEwen and Wingfield (2003), as Sterling and Eyer (1988) did, considered allostasis to be "the ability to achieve stability through change." This concept is also relevant for fish (Schreck, 2000). This means that set points and controls for physiological processes essential for life can change due to stress; allostasis can be thought of as a continuum between routine and perturbed states. My interpretation is that the "stasis" of homeostasis is in reality a "kinesis". The physiological status of an animal is never stable, but rather tending in one direction or the other. Cannon (1929) in his original, seminal work proposing the paradigm of homeostasis recognized that bodily systems are constantly in a state of flux. I believe that a useful way to think about stress is that stress simply pushes that physiological and biochemical status beyond normal bounds of the fluctuations. It is not uncommon for

systems to oscillate before stabilizing at some new set point following compensation to a stressor. Of course, life cannot persist if new set points are established beyond certain limits.

In a general way different stressors can be categorized as those that evoke one of two distinct responses by the organism: those that cause emergency-types of responses and those that cause coping-types of responses (McEwen and Wingfield, 2003). Emergency responses would include the "fight and flight" sorts of mechanism as introduced by Cannon et al. (1927) and Cannon (1929). These are referred to as *type 1 allostatic overload*. Coping responses involve stressors that are not as severe immediately or perceived as being as threatening; however, they also negatively affect reproductive fitness; these are referred to as *type 2 allostatic overload* (McEwen and Wingfield, 2003). In a general way I would think of type 1 overload being associated with more acute stressors and type 2 overload being associated more with more chronic stressors. However, it is not that simple, and the distinction between acute and chronic is quite fuzzy. However, for fish, as with other animals, it is difficult to generalize about the effects of various stressors, since more then one stressor is often experienced simultaneously, stressors can happen sequentially rather close temporally, and different stressors can occur in series (Schreck, 2000). I think the effects of different stressors and severities are in reality more of a continuum than falling into two discrete categories. However, using these two paradigms as a way of thinking is useful for understanding the effects of stress. It is also import to recognize that animals in different physiological states exposed to the same stressor might differ in regard to which type, emergency or coping, stress response they have. While the allostatic load associated with stress relates to increasing fitness in the short-term, its effects can reduce long-term fitness.

Emergency responses are induced by a hormonal cascade following perception of a threatening situation, for example, being chased by a predator or captured in a net. Coping-types of responses may also involve a similar hormonal cascade at the onset where the threat may be perceived as being severe but in reality being more moderate-where animals (Precht, 1958) including fish (Schreck, 2000) can compensate to the new conditions. Examples could include living in environmental conditions close to the fish's tolerances or being crowded in fish culture. Both emergency and coping responses are energetically demanding processes as discussed with theoretical models by McEwen and Wingfield (2003); stress is similarly energetically costly for fishes (Schreck and Li, 1991; and Schreck (2000). This energy demand is referred to as *allostatic load*, and while costly, increases the overall fitness of the animal (Wingfield, 2003). One can think about this in terms of immediate benefits, resisting the stressor, which is adaptive. I depict the potential consequences of emergency and coping responses for each reproductive phase for fish in Fig. 1. This is based on known and inferred responses gleaned from the literature referenced above.

To put the cost of stress into some perspective for fish, Barton and Schreck (1987) and Davis and Schreck (1997), found that the cost of a brief handling stressor of steelhead and coho salmon, *O. kisutch*, respectively, amounted to approximately 25% of the total energy available for activity (scope for metabolic activity). Varsamos et al. (2006) is one of the few papers that mention that allostasis and allostatic overload may explain negative effects of stress in sea bass, *Dicentrarchus labrax*.

The energetic cost of reproduction can be visualized mathematically as:

$$E_{\text{Reproduction}} = E_{\text{Food}} + E_{\text{Standard Metabolism}} + E_{\text{Specific Dynamic Action}} + E_{\text{Other Activity}} + E_{\text{Allostatic Load}}$$

where E = energy input or output.

$$E_{\text{Reproduction}} = E_{\text{Maturation}} + E_{\text{Reproductive Activity}}$$

$E_{\text{Maturation}}$ includes energy needed for hyperplasia and hypertrophy associated with growth of gametes and secondary and accessory sex characters.

$E_{\text{Reproductive Activity}}$ includes energy needed for reproductive behaviors such as migration, courtship and spawning.

$E_{\text{Other Activity}}$ includes energy needed for activities such as obtaining food, avoiding predators and social interactions.

Most of the energy associated with allostasis must be attributable to the functions affected by the physiological factors that control the stress response. Wingfield (2005) discussed and presented a model of these for mammals. They include hypothalamic corticotrophin-releasing factor, arginine vasopressin, and oxytocin. These would affect pituitary POMC and the production of adrenocorticotropin, melanocyte stimulating hormone, and endorphin. These subsequently affect adrenal glucocorticosteroid production. Cytokines and the catecholamines are also involved. Wingfield (2005) presents a nice review of allostasis for animals in the wild and the relationship of energy, allostasis, and glucocorticosteroids. These control factors are essentially the same for fish. While interesting, it would be daunting to calculate the energetic part of allostasis associated with the up and down regulation on these control factors.

Interestingly, energetically costly reproductive processes such as vitellogenin production appear to be conserved even during stressful situations or during reduced energy intake via food. This inference is based on work by Schwindt et al. (2007) who found that juvenile rainbow trout primed with 17β-estradiol to elevate plasma levels of vitellogenin maintained high circulating levels even when severely stressed or when fed only a ration at maintenance quantity. One interpretation of this is that maintenance of reproductive fitness is likely supported at the expense of other life functions. This interpretation may contrast with results of Clearwater and Pankhurst (1997) who showed that capture and confinement stress in red gunard, *Chelidonichthys kumu*, induced follicular atresia in vitellogenic oocytes.

Stress affects the timing of events associated with reproduction. One can think of this as the *reproductive clock* of fish. Stress also affects the quality of the reproductive effort. The effects of a stressor depends on when during reproductive development and maturation the stressor is applied and its duration and severity (Schreck et al., 2001). Wingfield (2003) suggests that reproduction *per se* is not stressful, likely referring to homeothermic animals, but no evidence for this is provided in the citation (McEwen and Wingfield, 2003) mentioned in support of this contention. I suspect that activities associated with reproduction could be at least somewhat directly stressful to fishes. Circulating concentrations of cortisol tend to increase during reproduction, and if unchecked can lead to Cushing syndrome-like conditions in fish (Schreck et al., 2001). And clearly, activities associated with spawning migrations and courtship are stressful. That is not to imply that there is evidence indicating that events such as gametogenesis are stressful.

Stress-induced impairment of gamete quality and quantity (fecundity) are paramount, but other activities associated with reproduction may also be affected. Behaviors such as reproductive migration, nest building, and courtship could conceivably be affected by stressors experienced at critical times of the life cycle. For example, Morgan et al. (1999) showed that stressed cod, *Gadus morhua*, had both fewer and altered courtship activities. While realized fecundity appeared unaffected, chronically stressed fish produced abnormal larvae.

Gamete quality and quantity depends on trophic conditions and stress. Nutrition is highly important in determining reproductive performance in fish. Improved nutrition is associated with both egg and sperm quality and fecundity in fish under culture. Lipid and fatty acid composition of diets appear particularly important (Izquierdo et al., 2001). Nutritional condition also has bearing on the reproductive success, as in wild cod, *G. morhua*, with poorer condi-

tion reflected in lower fecundity (Lambert and Dutil, 2000; Lambert et al., 2000). There can be large inter- and intraspecific variation in how fish respond to stressors and in their reproductive development that is habitat related. Pankhurst et al. (2008a,b) found that population density, via trophic relationships, could affect17β-estradiol and testosterone concentrations in females and in addition, 11-ketotestosterone in males in two wild species of damselfish *A. polyacanthus* and *Amblyglyphidodon curacao*, respectively. Confinement stress also can depress 17β-estradiol and testosterone of snapper,*P. auratus* (Cleary et al., 2007). Fish with low levels of steroid had lower steroidogenic capacity, which was correlated with low fecundity. The quality of the rearing phase of fish can affect the age at puberty and the timing of successive reproductive events in iteroparous species. Importantly, the total amount of energy in a fish does not necessarily relate directly to reproductive fitness; other factors are equally or more important. For example, sockeye salmon, *O. nerka*, that were not exercised had higher lipid content and energy reserves than exercised fish yet both groups had equal egg size and gonad composition. In addition, unexercised fish had lower egg deposit rates and higher pre-spawning mortality and their eggs were less likely to survive to the eyed stage than those from the exercised fish (Patterson et al., 2004).

Food availability and quality clearly affect pituitary function and reproduction. A thorough review by Martin et al. (2008) on the effects of caloric intake in the mammal is worth noting. They reported that GnRH, leptin, kisspeptin, ghrelin, growth hormone, the poylpetides YY and NPY, insulin, IGF-1, galanin-like peptide and pro-opiomelanocortin were all involved in affecting reproduction and interacted with hormones of stress and the gonads. They conclude that "The initiation of this [i.e., stress pathway consequent to caloric restriction] reproductive feedback has been suggested to emanate from the upper digestive tract." I suggest that this could be a fertile area for research on fishes.

Pathogens are clearly stressful to fish, and it is known that they can affect reproduction (Schreck, 1996). Endocrine and immune systems interact; this is particularly important during stress (see Maule and VanderKooi, 1999 for a nice review). These two systems can be considered one and the same for fish and other vertebrates (Schreck and Maule, 2001). Recently, Bonnet et al. (2008) showed that pathogen-induced factors associated with the innate immune response in rainbow trout (*O. mykiss*) advanced ovulation and affected gamete quality. LPS and TNF affected granulose cell viability and stimulated thecal testosterone production. These authors speculated that "pathogen-induced activation of the immune system prior to ovulation could increase TNF mediated apoptosis in the trout ovary, advance ovulation and result in potentially important decrease in egg quality." It is interesting that pathogen related stress would advance ovulation, a similar effect found for sequential physical stressors applied during vitellogensis by Contreras-Sanchez et al. (1998).

Parasites can also affect reproduction in fish. Barber et al.'s (2000) review suggests that parasites can be energetically costly to fish. In addition, if the energetic load prevents the fish from breeding, there could be advantages to the parasite since the fish might put energy instead towards somatic growth and thereby render more energy available to the parasite. In addition, the parasite would be protected from predation whilst in the fish. Parasites could prevent gonadal development and effectively castrate the fish. Accessory sex characters, like coloration, are energetically costly to develop, and may be poorly expressed in parasitized fish. This would affect mate selection and courtship. However, parasites do not always negatively affect reproductive fitness as shown by Candolin and Voigt (2001) for male sticklebacks, *Gasterosteus aculeatus*, infected with cestodes.

Reproductive behaviors are energy demanding processes and hence reproductive performance can be affected by both emergency and coping-types of stressors. Reproductive migratory behavior can be a considerable energetic cost and is dependent on energy accumulation during rearing. In fact, the energetic cost of migration may be as great as the energy investment in gonad maturation, as Lambert and Dodson (1990) showed in cisco, *Coregonus artedii* and lake whitefish, *C. clupeaformis*. Anecdotally it is known that courtship activities could also be affected by stress, with stressors preventing breeding during this time. While there may be an energetic attribute to the courtship x stress interaction, stressors could have direct effects almost immediately, likely via neuronal and neuroendocrine pathways. While I know of no such example in fish, interestingly, early developmental stress can affect courtship (male song) of a bird and thereby mate choice (Spencer et al., 2005), and presumably reproductive fitness.

In summary, the resistance of stressors is an energetically costly process. These costs can be thought of as competing with those needed to conduct other functions associated with reproduction including growth, parasite and disease resistance, production of high quality gametes, behavior, and timing of reproduction to coincide with ecologically optimum conditions. The nature of the reassignment of energy from reproductive functions to stressor resistance is ultimately also dependent on nutritional considerations.

2.2. Hormesis

Stressors of differing severities, durations and frequencies can result in various response patterns at the organismic level. The physical fitness of fish can be enhanced by low levels of stress while they are reduced by elevated levels or prolonged durations of stress (see review by Schreck, 2000). Stress that is not very severe may both accelerate and enhance adaptation to a stressor (Emlen et al., 1998). Minois (2000) reviewed the hormetic response of various invertebrates and suggested that both longevity is enhanced and the aging process retarded by mild stress. While *hormesis* is a phenomenon typically associated with the fields of health and toxicology, I argue that in concept it can be generalized to stress and hence the effects of stress on fish reproduction.

"Hormesis" can be "considered an adaptive response characterized by biphasic dose responses of generally similar quantitative features with respect to amplitude and range of the stimulatory response that are either directly induced or the result of compensatory biological processes following an initial disruption in homeostasis" (Calabrese and Baldwin, 2002). The concept of hormesis can be readily appreciated by considering the effects of radiation; low levels of radiation decrease the risk of cancer while higher levels are directly toxic (Johansson, 2003). In a general way one could think about the hermetic response pattern of a variable being a function of the severity off a stressor, however, duration also is involved. In reality, there could be multiple phases of the response and in my opinion it can be best thought of as representing one of the potential response patterns depicted by Precht (1958) when considering compensation to environmental impositions. These are not very dissimilar to those depicted by Calabrese and Baldwin (2002) for hormesis. I think that the key is that hormesis is a "nonmonotonic dose response," as mentioned by Cook and Calabrese (2006). It is also possible, maybe probable, that the beneficial and detrimental responses of the different phases of hormesis involve different mechanisms (Kitchin, 2002).

The nonmonotonic response to stressors and the mediation by cortisol has been nicely depicted and described by Dahbhar and McEwen (2001). While this example derives from the field of immunology and not reproduction, it is extremely heuristic in terms of the message it presents. To wit, low levels of stress are immunoenhancing while higher levels of stress can be considered distress and are immunosuppressive. There is a range in level of stress (resilience) between the former low level of stress and

distress where the organism is attempting to maintain a robust immune capacity in the face of the insult. In essence, slight stress would reduce the likelihood of disease while distress would make the organism more prone to pathogenic effects. I believe that something similar may be said for stress effects on reproduction, where a small amount of stress has a positive effect and more severe stressors have a negative effect on reproductive capacity. For example, psychological and physical conditioning involving low levels of stress, yet sufficient to activate the HPI, can enhance stress-resistance in fishes (see Schreck et al., 1997 for review); one can think of this as "stress hardening" (Schreck, 2000). One could extend this to reason that reproductive performance could be enhanced if fish are exposed to very mild stressors during rearing phases and maturation. Another way to think about hormesis and stress is that animals, including fish, are adapted to low severity of stressors, acute or chronic, and one can think of them as "background levels of stressors". Fish can thrive under those conditions. It is at greater severities that stressors become detrimental. Johansson (2003) discussed this phenomenon with regard to human ecology.

Endocrine responses can often be biphasic and hence can be thought of as part of the response due to hormesis, as implied by Kitchin (2002). Numerous hormonal systems of mammals associated with various aspects of reproduction have biphasic dose responses. These included sex steroids like estrogens (Calabrese, 2001a) and androgens (Calabrese, 2001b), the GTH antagonist dopamine (Calabrese, 2001c), prostaglandins (Calabrese, 2001d) and opiates (Calabrese, 2001e). The biphasic nature of the endocrine response likely can be explained by receptor-mediated processes (Cook and Calabrese, 2006) It is thus reasonable to infer that hormetic stress responses, e.g., those that are biphasic, can affect reproductive control mechanisms, but in different ways depending on the latter's physiological status at the time of the stressful encounter. The effects of corticosteroids on fish reproduction of both sexes appear biphasic, positive or negative. That is the general conclusion from a recent review on the effects of corticosteroids on fish reproduction (Milla et al., 2009). In a broader review of vertebrates, including a few species of fish, Breuner et al. (2008) addressed a possible relationship between the corticosteroid stress response and fitness. My conclusion is that the variation in responses reported can be viewed as driven variable conditions of the animals but also likely hormesis.

Environmental challenges can affect reproduction in fishes in different ways. For example, short-term, low severity stress associated with hatchery practices does not affect gamete quality of stellate sturgeon, *Acipenser stellatus* and Russian sturgeon, *A. gueldenstaedtii*. However, prolonged culture stress and high rearing density has a negative effect on reproduction (Bayunova et al., 2002). Sex determination, while having a strong genetic basis, is also responsive to environmental influence. In some cases environmental extremes can affect sex ratio in a hormetic manner. Devlin and Nagahama (2002) provide an excellent review of sex differentiation in fishes, including environmental effects. Environmentally realistic temperature extremes and pH can also be sex-directing factors.

The mechanisms whereby hormetic stress responses are mediated, particularly those concerning reproduction, are not well understood. The biphasic organismic effects of physical stressors such as handling and crowding can be appreciated when one considers the positive effects of mild stress on improvements in cardiovascular performance, muscle tone, and metabolism. Such enhancements are particularly profound when the physical challenges are coupled with positive psychological conditioning. "Stress hardening" attributable to mild physical stressors and psychological factors has been reviewed earlier (Schreck et al., 1997; Schreck, 2000). The response of fishes to environmental water quality-related stressors that follow a hormetic patterns may, at least in part, be explained by the induction of heat shock (stress) proteins (Hsp). For example, Brown et al. (1992) found that to a point increasing heat made nephric tissue more resistant to temperature, likely via production of the protective heat shock proteins Hsp28, Hsp70 and Hsp90 in winter flounder, *Pleuronectes americanus*. And, DuBeau et al. (1998) demonstrated an increase in Hsp70 during osmotic challenge in Atlantic salmon, *Salmo salar*. Nonmonotonal endocrine responses to gradients of stressors can be envisioned to be due to changes in substrate availability for synthesis of hormones, receptor dynamics, carrier protein abundance, and hormone clearance dynamics.

3. Conclusions

Stress evokes either (1) emergency-types of responses in fish if the stressor is perceived to be severe, called type 1 allostatic overload or (2) coping-types of responses if the fish can resist the stressor for some period of time and have energy available, called type 2 allostatic overload. This overall concept of adapting through change is allostasis. These concepts are important in understanding how stressors affect fish reproduction and concerns the process of re-budgeting energy available to the fish.

The physiological and organismic response to stressors is frequently hormetic. That is, responses can often be nonmonotonic. Slight stress can enhance reproductive performance while more severe or frequent or prolonged stressors have negative effects on reproductive processes. Unfortunately, we just do not know enough yet, in my opinion, to be able to well define when the bounds of homeostatic fluctuations have been crossed, leading to stress. Technically, the term *allostasis* refers to a change in stasis. Given that systems in the body are always in a state of flux, I propose that it might be useful to consider the concept of homeostasis during the stress response as *allokinesis*.

Acknowledgments

I appreciate the thoughts and comments of numerous students and colleagues as I was organizing my thoughts and preparing the drafts regarding this review, in particular Drs. Scott Heppell, David Noakes and Kenneth Rodnick. Tracey Momoda and Ben Clemens assisted with preparation of the figure.

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