REVIEW PAPER

Cold shock and fish

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Rapid decreases in water temperature may result in a number of physiological, behavioural and fitness consequences for fishes termed 'cold shock'. Cold-shock stress occurs when a fish has been acclimated to a specific water temperature or range of temperatures and is subsequently exposed to a rapid decrease in temperature, resulting in a cascade of physiological and behavioural responses and, in some cases, death. Rapid temperature decreases may occur from either natural (*e.g.* thermocline temperature variation, seiches and storm events) or anthropogenic sources (*e.g.* varied thermal effluents from power generation and production industries). The magnitude, duration and frequency of the temperature change as well as the initial acclimation temperatures of individuals can influence the extent of the consequences of cold shock on fishes. Early research on cold shock focused on documenting mortality events associated with cold shock. However, in recent years, a shift in research has occurred where the focus of cold-shock studies now involves characterizing the sublethal effects of cold shock in terms of the stress response in fishes. This shift has revealed that cold shock can actually be used as a tool for fisheries science (*e.g.* to induce polyploidy). The cold-shock stress response offers opportunities to develop many exciting research questions, yet to date, cold-shock research has been largely unfocused. Few studies attempt to link laboratory physiology experiments with ecologically relevant field data on behaviour, growth, bioenergetics and fitness. Additional research will allow for the development of more focused and robust management policies and conservation initiatives. This review synthesizes the sublethal physiological and behavioural consequences of cold-shock stress on fishes, identifies natural and anthropogenic sources of cold shock, discusses the benefits of cold shock to fisheries science and describes mitigation and management efforts. Existing knowledge gaps and opportunities for future cold-shock research are presented.

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INTRODUCTION

Temperature controls and limits all physiological and behavioural parameters of ectotherms (Fry, 1947). In fact, water temperature has been described as the 'abiotic master factor' for fishes (Brett, 1971). Optimal temperature ranges, as well as upper and lower lethal temperatures, vary widely between and among species and are dependent on genetics, developmental stage and thermal histories (Beitinger *et al.*, 2000; Somero, 2005). Within a range of non-lethal temperatures, fishes are generally able to cope with gradual temperature changes that are common in natural systems (*e.g.* diel variation, tidal activity, currents and seasonal cooling). However, rapid increases or decreases in ambient temperature may result in sublethal physiological and, or behavioural responses. Cold shock can be defined as an acute decrease in ambient temperature that has the potential to cause a rapid reduction in body temperature, resulting in a cascade of physiological and behavioural responses. A cold shock that reduces body temperatures to the lower limit of an organism's thermal range can result in severe sublethal disturbances and mortality. The magnitude of the cold-shock response is dependent on both the rate of temperature decrease and the magnitude of change in relation to thermal tolerance limits (Crawshaw, 1977; Tanck *et al.*, 2000; Van den Burg *et al.*, 2005). For example, deleterious sublethal and lethal consequences can result from an individual's body temperature being decreased rapidly towards the lower end of the species-specific thermal tolerance range. While the cold shock stress response is generally considered to be an adaptive response to maintain homeostasis, prolonged or severe temperature changes that occur outside of a specific tolerance to change may ultimately result in mortality (Fry, 1947) or otherwise affect the organism's health and fitness.

Cold shock can occur under natural conditions, such as thermocline temperature variation (*e.g.* when a fish swims up through in the water column), rapid changes in solar heat, abnormal water movements, rapid precipitation events or rapid changes in seasonal temperatures, yet these sources and their consequences on fish populations are poorly understood. Anthropogenic (*i.e.* human-influenced) sources of cold shock include changes in thermal effluents from power generation and production industries, various water control projects and fish handling practices. Coutant (1977*a*) adequately synthesized early developments in the field enabling this contemporary review to focus on more recent developments. The majority of research to date has examined the consequences of exposure to thermal effluents as a source of cold-shock stress response (reviewed in Coutant, 1977*a*).

In recent years, a shift in research has occurred where response to cold shock is measured in terms of sublethal effects to fishes rather than just mortality. Cold-shock response may be a beneficial tool for fisheries science (*e.g.* for induction of polyploidy) and future cold-shock research may reveal other novel opportunities. Yet, to date, cold-shock research has been diffuse and disparate. Accordingly, the scope of this review is intentionally broad. The purpose of this review is to synthesize recent literature to assess the sublethal and lethal consequences of cold shock on temperate fish. In addition, this review highlights the applications of cold-shock research to fisheries science, identifies

directions for future research and describes key management implications of cold shock.

RESPONSES OF TEMPERATE FISH TO COLD SHOCK

Cold shock can be characterized in terms of the general stress response in fishes (Fig. 1 and Table I). In this review, the operative definition of stress, as described by Pickering (1981) and Schreck *et al.* (2001), will be used. Schreck *et al.* (2001) characterized stress as a physiological cascade of responses that occur when an organism attempts to re-establish homeostasis following an insult. Responses to environmental stress are broadly grouped into three categories (Mazeaud *et al.*, 1977; Barton, 2002): primary (*e.g.* neuroendocrine response and corticosteroid–catecholamine release), secondary (*e.g.* metabolic, cellular, haematological, osmoregulatory and immunological changes) and tertiary (*e.g.* whole organism physiological and behavioural stress responses). Cold-shock stress initiates a neuroendocrine response at the central nervous system (CNS), and the primary stress response triggers a release of corticosteroid and catecholamine hormones. Secondary responses include metabolic, haematological and osmoregulatory changes. Cellular responses, including expression of heat shock proteins (HSPs), are grouped with the secondary response. Tertiary responses refer to stress on individuals as a whole (*e.g.* changes in growth and development rates, disease resistance and behavioural modifications (reviewed in Mazeaud *et al.*, 1977; Wendelaar Bonga, 1997; reviewed in Barton, 2002). Behavioural
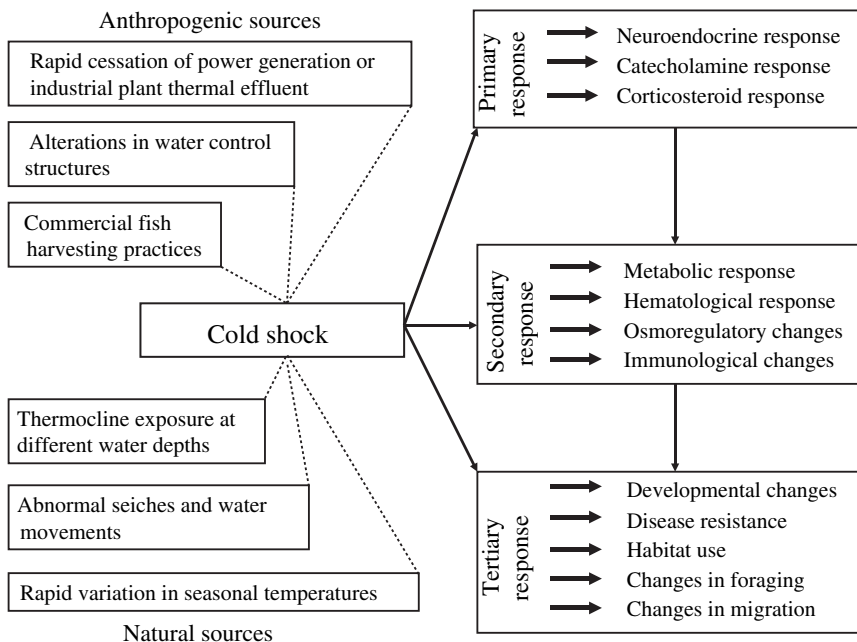

FIG. 1. Schematic representation of natural and anthropogenic sources of cold shock as well as the primary, secondary and tertiary stress responses to cold shock.

TABLE I. Key data sources and current knowledge gaps for each of the primary, secondary and tertiary consequences of cold shock

Cold-shock response	Key references	State of knowledge	Key points and knowledge gaps
Primary			
Brain and central nervous system response	Friedlander et al. (1976); Peeters et al. (2001); Ju et al. (2002); Van den Burg (2005)	Low	Need to investigate the response of neuronal activity in relation to the magnitude of cold shock. Use of fMRI to examine the mechanisms of cold shock-induced stress response
Catecholamine and corticosteroid response	Tanck et al. (2000); Hyvarinen et al. (2004)	Medium	Cortisol release is a sensitive and well-studied indicator of cold-shock stress. The role of catecholamine response to cold shock is poorly understood and demands further investigation
Secondary			
Haematological and metabolic response	Datta et al. (2002); Zarate & Bradley (2003); Lermen et al. (2004)	Medium	The discrepancies regarding the sensitivity of haematocrit, leucocrit and plasma concentrations of lactic acid require further examination
Protein expression and molecular responses	Zakhartsev et al. (2005); Takle et al. (2005)	Medium	Need to address the effects of different HSP families in various tissues to determine their sensitivity to cold shock. Relationship between stress-induced HSP expression and cortisol release needs to be elucidated
Osmoregulatory changes	Houston & McCarty (1978); Gonzalez & McDonald (2000); Mackie et al. (2005)	Medium	The genetic basis of osmoregulatory changes following cold shock is poorly understood and requires further study to examine inter and intra-species-specific differences for the set point of osmoregulation. Examination of the consequences of compromised osmoregulation following cold shock are required to determine the extent to which the whole body is affected by acute decreases in water temperature

TABLE I. Continued

Cold-shock response	Key references	State of knowledge	Key points and knowledge gaps
Tertiary			
Immune function	Engelsma et al. (2003); Tierney et al. (2004)	Low	The majority of studies that have investigated immune function relative to water temperature have focused on gradual, rather than acute, temperature changes
			Future research needs to examine the interactive effects of cold shock and other stressors on the immune function of fishes
Developmental rates	Tang et al. (1987); Hubert & Gern (1995)	Low	Several studies have characterized the mortality rates associated with cold shock for all developmental stages of fishes, but few studies have examined the underlying mechanisms that lead to mortality
			Future research must address the specific genetic differences that account for inter and intraspecies-specific variations in development following cold shock
Whole-body health and disease	Tilney & Hocutt (1987); Black et al. (1991)	Low	Mortality is the general end point in the majority of cold-shock studies and sublethal end points are rarely examined
			Future research needs to examine the compromised immune function following cold shock to determine the consequences on whole-body health and disease susceptibility
Foraging and predation	Clarkson & Childs (2000); Smith & Hubert (2003); Ward & Bonar (2003)	Low	Future research needs to address differences in swimming ability and equilibrium following cold shock
			Juvenile fishes are particularly vulnerable to predation following cold shock, yet little is known about the mechanisms of compromised predatory escape responses following cold shock

TABLE I. Continued

Cold-shock response	Key references	State of knowledge	Key points and knowledge gaps
Microhabitat selection in natural and modified habitats	Smythe & Sawyko (2000)	Low	Future research needs to address microhabitat selection at the broad scale and include a specific focus on cold shock when examining temperature as a limiting factor in selecting microhabitats Few studies have incorporated the whole-body consequences of cold shock in microhabitat selection (*i.e.* compromised swimming ability and loss of equilibrium)
Population, community and ecosystem interactions	N/A	Very low	The majority of cold-shock studies have focused on the primary and secondary stress responses as well as mortality rates, but no studies have examined the population and community level consequences of cold shock Future research must examine the effects of cold shock at the ecosystem level, as the effects of acute temperature decreases are highly variable between species In order for specific guidelines and recommendations to be developed, a greater knowledge base on the site-specific consequences of cold shock is required

fMRI, functional magnetic resonance imaging; HSP, heat shock protein; N/A, none available.

modifications include changes in microhabitat use, abundance and distribution, feeding, predation, migration and spawning behaviours.

PRIMARY AND SECONDARY STRESS RESPONSES TO COLD SHOCK

Present knowledge of primary responses to stress, including temperature stress, has largely been gained by studying responses in the hypothalamic–pituitary–interrenal (HPI) axis and the brain. For vertebrates, the preoptic area integrates perceived body temperature fluctuations and the CNS initiates physiological and behavioural responses (Crawshaw *et al.*, 1985). Brain primary stress response studies often involve the examination of brain function relative to different degrees of temperature variation and often integrate the study of the mechanistic basis of brain response with behavioural impairments. For example, Friedlander *et al.* (1976) examined the behaviour and cerebellar function of goldfish *Carassius auratus* (L.) acclimated to 20° C and exposed to stepwise reductions in temperature from 20 to 2° C over 50 min. Behavioural responses varied with decreasing temperatures as fish showed normal swimming behaviour at 18° C, began to show uncoordinated swimming (*e.g.* head down and bumping into tank walls) at 6–7° C, had difficulty maintaining equilibrium at 5° C and had a complete loss of equilibrium and induction of coma at 2° C. In a second experiment, fish were acclimated to 25° C and held at 21° C and the cerebellum of individuals was cooled by thermode (deep brain, 2 mm) from 19 to 6° C over 42 min. With decreasing temperatures, behavioural responses included hyperresponsiveness to tactile stimuli at 10–12·5° C, bumping into tank walls and spontaneous circling at 8–9° C, partial loss of equilibrium at 6·5–7° C and total loss of equilibrium at 6·0–6·5° C. At a recovery time of 47 min, where temperature was raised to 14–16° C, there appeared to be a behavioural recovery. The authors found that cerebellar interneuron inhibition is most sensitive to acute temperature changes and can be modified by acclimation to specific temperatures. The mechanisms of thermoregulation in whole brain functioning are not clearly understood (Van den Burg *et al.*, 2005), but compensation mechanisms are thought to exist within the vertebrate brain to maintain coordinated sensorimotor function following cold shock (Montgomery & MacDonald, 1990).

Following cold shock, neuronal activity is dependent on the availability of oxygen-rich blood and requires reliable synaptic transmission (Preuss & Faber, 2003). Preuss & Faber (2003) found that a 10° C cold shock over 50 min changed the activation of auditory afferents on the Mauthner cell that triggers an escape response in 18° C acclimated goldfish. This caused a shift in the startle-escape behaviour to an acoustic stimulus, resulting in an increased escape response, but towards the signal rather than away. Thus, cold shock may influence the reliability of synaptic transmission and the reliability of cellular and network responses and could compromise the predatory escape response.

The response of the fish brain to cold shock has recently been explored using functional magnetic resonance imaging (fMRI; Peeters *et al.*, 2001; Van den Burg *et al.*, 2005). Peeters *et al.* (2001) used fMRI to examine the stress-induced neuronal activity in the HPI axis of 25° C acclimated common

carp *Cyprinus carpio* (L.) that were subjected to a rapid decrease of 9° C to an end temperature of 16° C. These authors found decreases in both blood oxygenation level-dependent (BOLD) and cerebral blood volume (CBV)-sensitive fMRI protocols. A change in activation states of the pituitary gland and the hypothalamus were observed because of these decreases in BOLD and CBV. It is hypothesized that these changes are explained by the HPI axis stimulating the release of a cascade of hormones, including the stress-induced hormone, cortisol. Similar findings were observed by Van den Burg *et al.* (2005), who examined the whole brain responses (*i.e.* changes in CBV and oxygenation of haemoglobin) of 25° C acclimated *C. carpio* that were exposed to a 10° C temperature decrease of 5 min. The authors found that after 90 s, brain blood volume was reduced, consequently limiting the entry of cold blood from the gills, resulting in a slower decrease in brain temperature. A cold shock can affect the neuronal activity of fishes by limiting the flow of oxygen-rich blood to the brain, similar to vasoconstriction in mammal skin (Van den Burg *et al.*, 2005). The decreased blood volume in the brain is thought to slow the rate of cooling of the brain following the application of the cold shock, consequently allowing the fish to maintain sensorimotor activity initiation of the physiological stress response, and subsequent behavioural responses. In the study by Van den Burg *et al.* (2005), the preoptic area was activated within 30 s of the onset of the temperature drop. The authors suggest that this region may stimulate neighbouring neuroendocrine neurons that lead to a primary neuroendocrine stress response through the release of corticotropin-releasing hormone (CRH).

The expression of many genes in the brain may be adjusted at low temperatures (Ju *et al.*, 2002), but few studies have examined the expression of genes in the brain following a rapid decrease in temperature (*i.e.* cold shock). However, Ju *et al.* (2002) found that the expression of several genes in the brain of channel catfish, *Ictalurus punctatus* (Rafinesque) were altered within 2 h after a decrease in temperature from 24 to 12° C over a 3 h period. The rapid induction of genes that code for proteins involved in signal transductions and chaperones suggests that both *de novo* synthesis of cold-induced proteins and modification of existing proteins are required for adaptation and tolerance of *I. punctatus* to rapid decreases in environmental temperatures. These induced genes indicate that multiple signal-transduction pathways are involved in cold acclimation (Ju *et al.*, 2002).

The primary stress response in fish is accompanied by rapid changes in plasma concentrations of catecholamine and corticosteroid stress hormones (reviewed in Mazeaud *et al.*, 1977; Barton, 2002). While the catecholamine response has received relatively little attention in the cold-shock literature, a study by Chen *et al.* (2002) found that cold shock modulates catecholamine and cortisol concentrations in tilapia *Oreochromis aureus* (Steindachner) subjected to cold-shock treatments where temperatures decreased from 25 to 12° C over either 15 or 30 min. Here, elevations in plasma norepinephrine and epinephrine were detected earlier than that of cortisol. The release of cortisol begins in the HPI axis with the release of CRH from the hypothalamus. This release stimulates corticotrophic cells to secrete adrenocorticotropin hormone, which stimulates kidney interrenal cells and releases corticosteroids into

circulation (Barton, 2002). The process of cortisol synthesis and release can take several minutes and varies widely both interspecifically and intraspecifically because of differences in genetic, developmental and environmental factors (Barton & Iwama, 1991; Wendelaar Bonga, 1997; Barton, 2002). Plasma cortisol levels can be detected *in vitro* even before the HPI axis has developed enough to respond to a stressor (Barry *et al.*, 1995). There is also evidence for genetic variation in the cold-shock response, as plasma cortisol levels tend to vary when using different genotypic strains (Tanck *et al.*, 2000).

The cold-shock response varies depending on the stage of development. The HPI axis is responsive to stressors within 5 weeks of hatching or even earlier for some species (Barry *et al.*, 1995). Jentoft *et al.* (2002) exposed 15° C acclimated embryonic and larval yellow perch *Perca flavescens* (Mitchill) to 0° C for 30 s. The authors found that cortisol levels were significantly elevated for stressed fish at 8 days post-hatch and the magnitude of stress response was greater in 2–5 week-old fish than in 1 week-old fish. Post-stress cortisol levels were associated with changes in interrenal morphology. The authors concluded that cortisol levels and HPI maturation can be observed within 1 week post-hatching. This study contributes to limited evidence that the stress response is heightened during particular stages of development (Barton & Peter, 1982; Jentoft *et al.*, 2002).

Corticosteroids, along with catecholamines, mediate secondary stress responses and are often considered together to understand the relationships between the cold shock and the physiological stress response in fishes. Primary and secondary stress responses are adaptive if they result in a physiological response that allows a fish to maintain homeostasis. These responses are generally short term and will recover over a short period (*i.e.* within minutes or hours) after a cold-shock stimulus (Tanck *et al.*, 2000). However, either cumulative or high-magnitude stress can result in mortality. For instance, slow recovery from a cold shock will result in stressed fish being vulnerable to predation (see Foraging and Predation below). Primary and secondary stress responses are commonly used as indicators of cold-shock stress, but unless tertiary whole-body physiological and behavioural responses are measured, it is difficult to understand the ecologically relevant consequences of rapid temperature declines on fish.

There is evidence to show that the rate of a cold-shock event is one determinant of the magnitude of the response. For example, Tanck *et al.* (2000) evaluated the cold-shock response of *C. carpio* that were exposed to rapid temperature drops of 7, 9 and 11° C from an initial acclimation temperature of 25° C. Plasma cortisol levels were positively correlated to the magnitude of temperature decreases. Cortisol levels were higher in fish that were exposed to a series of rapid temperature decreases over 120 days compared with 60 days. Peak levels of plasma cortisol were recorded 20 min after the initial temperature reduction rather than after 60 min when the final low temperature was reached. The authors found that multiple shock treatments at 20 and 60 min induced lower cortisol levels than single-shocked fish at 120 min, corroborating evidence that fish can habituate to repeated temperature reductions (Einarsdottir & Nilssen, 1996). Contrary to results from a cold-shock study using tilapia *Oreochromis niloticus* (L.) by Sun *et al.* (1992, 1995), Tanck *et al.* (2000) did not

observe a significant increase in plasma glucose. This may be because of the timing of blood sampling, as Sun *et al.* detected hyperglycaemia 24 h after the onset of the experimental procedure.

Fish may be exposed to cold-shock stress from transfer between tanks of variable water temperatures during hatchery and fishery procedures. These rapid transfers generally result in minimal periods of thermal acclimation. To simulate stress from hauling and stocking procedures, Barton & Peter (1982) exposed fingerling rainbow trout *Oncorhynchus mykiss* (Walbaum) to a rapid temperature decrease (from 10–11 to 1° C) and found that plasma cortisol levels increased within 30 min and were maintained up to 4 h after exposure. Even exposure to a 1° C cold shock resulted in a large increase in plasma cortisol levels after 4 h and levels recovered after 24 h. However, Lermen *et al.* (2004) found that a varying temperature regime did not have a significant effect on cortisol levels in the South American silver catfish *Rhamdia quelen* (Quoy and Gaimard). The lack of a cortisol response may be explained by the fact that the temperature regimes occurred over a relatively gradual 12 h period, resulting in fish acclimating to low temperatures without inducing stress.

Suski *et al.* (2006) examined the physiological consequences of variable environmental conditions (temperature and oxygen) in relation to exercise for largemouth bass *Micropterus salmoides* (Lacepède). These authors found that reduced temperature resulted in elevated lactate concentrations, impaired replenishment of white muscle energy stores and elevated plasma cortisol concentrations relative to fish recovered at ambient water temperatures. These authors conclude that, similar to the conclusions suggested by Galloway & Kieffer (2003) and Hyvarinen *et al.* (2004), rapid transfer of fish to cool water reduces the activity of channels, pumps and enzymes that clear lactate and replenish energy stores. Galloway & Kieffer (2003) examined cold shock relative to metabolic recovery from exhaustive exercise in juvenile Atlantic salmon *Salmo salar* (L.). They measured muscle phosphocreatine (PCr), ATP, lactate, glycogen, glucose, pyruvate, plasma lactate and plasma osmolarity during rest and at 0–4 h following exhaustive exercise at 12° C. The authors found that the recovery of metabolites such as muscle PCr, ATP and plasma lactate that took 2–4 h in the control fish (at 18° C) was delayed in fish that experienced a cold shock (6° C) measured at 0, 1, 2 and 4 h post-exercise.

During hatchery and fishery procedures, cold-shock stress may be exacerbated by the interaction among many stressors, including exercise. Hyvarinen *et al.* (2004) studied the effect of temperature reduction on the stress response and recovery time of brown trout *Salmo trutta* following exhaustion with a trawl swimming simulation. Fish were first exhausted against a water flow of 0·5 m s^{-1} for 60 min at a temperature of 13·7–14·1° C and then exposed to a 10 min cold shock of 0·2° C. This study simulated the transfer of fish caught in a trawl net to ice water tanks to maintain the quality of food. The authors found that fish were comatose within 10 min after both cold shock and exercise but were conscious after only swimming exercise. After 10 min of treatment, levels of blood cortisol, lactate and glucose were higher for fish exposed to extreme cold after swimming relative to those that were only exercised. A cold-shock response was evident by the delayed onset of recovery in cortisol and glucose concentrations.

Many studies have found that temperature has an influence on haematological and metabolic processes, but factors such as photoperiod, salinity and developmental stage and body size can pose challenges in interpreting these parameters following an acute temperature decrease (Sun *et al.*, 1995; Ban, 2000). Current evidence indicates that some haematological and metabolic responses to cold temperature stress are highly variable (Lermen *et al.*, 2004) and may not be sensitive indicators of cold-shock stress. Specifically, Zarate & Bradley (2003) found that haematocrit and leucocrit were poor indicators of temperature stress due to their high variability among individuals. They also suggest that plasma concentration of lactate, which can increase with both activity and stress independently, is of less use than primary stress indicators such as cortisol for the measurement cold-shock stress. However, Carruth *et al.* (2002) suggest that the use of cortisol as an indicator of stress response in maturing salmonids is misleading given the high background levels associated with maturation and variable levels of reproductive hormones. Indicators of temperature stress should be interpreted with caution, particularly when multiple, interacting stress stimuli are used experimentally.

PROTEIN EXPRESSION AND MOLECULAR RESPONSES

The biochemical response to low-temperature acclimation is reflected by a suite of cellular changes, including alterations in membrane fluidity and protein translation machinery. Many organisms show a homeoviscous response at the cellular level. This response enables changes in membrane fluidity with shifting temperatures. Low-temperature acclimation results from the redistribution of phospholipid species in the plasma and microsomal membranes to maintain lipid miscibility and increase cryostability (Thieringer *et al.*, 1998). For example, lipid desaturase, which is a microsomal stearoyl-coenzyme A desaturase, showed an increase in activity following cold shock in *C. carpio* by transcriptional and post-translational mechanisms (Tiku *et al.*, 1996).

Primary and secondary stress responses interact at the cellular level, when cortisol indirectly stimulates the expression of HSPs (Deane *et al.*, 1999; Ackerman *et al.*, 2000; reviewed in Iwama *et al.*, 1998; reviewed in Basu *et al.*, 2002*b*; Zhou *et al.*, 2003). HSPs are a family of stress proteins that are expressed in prokaryote and eukaryote cells and tissues both constitutively and in response to biotic and abiotic stressors by acting as molecular chaperones that protect the cell against denatured proteins (Krone *et al.*, 1997; Lele *et al.*, 1998; Zakhartsev *et al.*, 2005). Cold-shock proteins have been described in prokaryotes, but little is known on this topic in relation to the eukaryote cold-shock response (reviewed in Thieringer *et al.*, 1998). HSPs have been found to repair and prevent damage from cellular stress associated with protein denaturation at high and low temperatures (Nakano & Iwama, 2002; Werner *et al.*, 2005). HSPs are thought to play a role in long-term adaptation to extended periods of environmental stress because they increase after the initial exposure to stress and protect tissues from structural damage during subsequent exposures (Sharp *et al.*, 1994). Primary cell culture studies have found that temperature shocks can induce HSPs over a range of molecular masses (reviewed in Iwama *et al.*, 1998). HSPs are organized by molecular mass: for example HSP90 (85–90 kDa),

HSP70 (68–73 kDa) and low-molecular-mass proteins (16–47 kDa) (Takle *et al.*, 2005). A number of studies have examined the role of HSPs in the temperature stress response of fishes (Yu *et al.*, 1994; diIorio *et al.*, 1996; Iwama *et al.*, 1998; Currie *et al.*, 2000; Zakhartsev *et al.*, 2005), and there has been a recent shift in research that focuses more specifically on the HSP response to cold-shock exposure (Tanck *et al.*, 2000; Zarate & Bradley, 2003; Takle *et al.*, 2005).

Several techniques have been used to examine HSP expression in fish in response to cold shock. Cell line studies provide information on the range of HSPs that may be induced by stressors. The majority of HSP cell-line studies tend to focus on heat shock or other stressors (Airaksinen *et al.*, 1998) rather than cold-shock sensitive. A number of fish HSP studies have looked at tissue and organ-specific responses as well as whole-organism responses to temperature shock (DuBeau *et al.*, 1998; Wood *et al.*, 1999; Currie *et al.*, 2000; Das *et al.*, 2005; Zakhartsev *et al.*, 2005). The majority of whole-fish HSP and temperature studies have focused on adults, but recent evidence suggests that HSPs are important for reducing temperature-induced damage and deformities of fish embryos (Takle *et al.*, 2005).

HSP70 is temperature inducible at different embryonic stages and is known to assist in the folding of nascent polypeptide chains and the remediation of damaged proteins (Krone *et al.*, 1997; Takle *et al.*, 2005). Takle *et al.* (2005) subjected hot and cold treatments to *S. salar* at four embryonic stages from gastrulation to the completion of somiogensesis and found a 14% incidence of vertebral deformities. Following the cold treatment, HSP70 expression was highest at the 45th somite stage with a four-fold increase (compared with a 12 fold increase after heat shock). The findings of this study indicate that HSP70 confers protection in *S. salar* embryos. The authors suggest that although heat shock induces a higher HSP response than cold shock, the rate of deformities may result from the magnitude rather than the direction of temperature change. Werner *et al.* (2005) conducted field and laboratory studies to examine the expression of HSP72 and HSP78 in the white muscle of steelhead *Oncorhynchus mykiss* (Walbaum) parr. A sigmoidal relationship and a linear relationship with temperature change were found for HSP82 and HSP78, respectively. These authors also found that the highest HSP72 levels in juvenile *O. mykiss* were measured at warmwater sites with diurnal temperature fluctuations $\geq 6\cdot5°$ C. These results suggest that HSPs were expressed in response to both stressful water temperatures and daily temperature fluctuations.

A number of studies support the potential for using HSPs as indicators of cellular stress, but the relationship between cellular and physiological stress is difficult to characterize because of differences between tissues, HSP families, thermal histories and acclimation temperatures, as well as the magnitude and the duration of the thermal treatment (Currie *et al.*, 2000; Zakhartsev *et al.*, 2005). Recent evidence shows that even within a single species, rearing conditions and genetic variation can influence an organism's cellular response to thermal stress (Werner *et al.*, 2006). HSP expression is highly variable and does not occur in some coldwater species. For example, Hofmann *et al.* (2000) found that HSP expression was absent in the Antarctic teleost fish, emerald rockcod *Trematomus bernacchii* Boulenger. The loss of a heat shock response in some

coldwater species may be indicative of evolution at cold and constant temperatures (Hofmann *et al.*, 2005). In contrast, most temperate fishes studied express HSPs in response to thermal stress (Iwama *et al.*, 1998). Recent evidence suggests that HSP70 may not be a sensitive indicator of thermal stress, and Iwama *et al.* (2004) warn that the use of HSPs as cellular indicators of stress may still be premature. Zakhartsev *et al.* (2005) found evidence that temperature-related changes in HSP70 expression are small in fishes by studying the effects of various high and low-temperature stimuli on Atlantic cod *Gadus morhua* (L.) tissues. These authors conclude that HSPs are not a suitable indicator for determining long-term temperature stress at the organismal level as levels of HSP70s did not differ significantly between optimal acclimation temperatures for growth (10–13° C) and long-term stressful temperatures (*i.e.* 4 and 15° C). The constitutive HSP70 level in *G. morhua* may be high enough to protect against molecular disruptions (Yu *et al.*, 1994). Zarate & Bradley (2003) found similar results when investigating the upregulation of HSP30, HSP70 and HSP90 after exposure to a number of hatchery-related stressor simulations, including hot and cold temperature. Cold temperature, crowding and capture stress had no influence on HSP90 mRNA levels but did increase following heat-induced stress.

Further investigation of HSP response to various hormone stimuli is required to determine if HSP expression is a sensitive indicator of cold-shock stress. The relationship between elevated cortisol levels and HSP expression has been examined. Deane *et al.* (1999) found that administered cortisol had no effect on hepatic HSP70 expression in a marine teleost, the silver sea bream *Sparus sarba* Forsskål. Although temperature stress was not considered, Vijayan *et al.* (1997*a, b)* found elevated cortisol levels had no effect on HSP70 expression after handling stress. After administering exogenous cortisol, there was no change in HSP70 expression in the liver and gill tissues of *O. mykiss* and Mozambique tilapia *Oreochromis mossambicus* (Peters) (Basu *et al.*, 2002*a*). However, when a 12° C heat shock accompanied the elevated cortisol levels, a significant suppression of HSP70 expression was observed, suggesting that cortisol may mediate HSP70 levels in fish tissues after physiological stress. Deane *et al.* (1999) suggest that other HSPs, particularly HSP90, may be more closely correlated with cortisol levels because they are coupled along the transduction pathway. Sathiyaa *et al.* (2001) provided evidence of this by showing that cortisol modulates HSP90 mRNA expression in primary cultures of trout hepatocytes. Furthermore, increased corticosteroid levels have been correlated with HSP90 expression but not HSP70 expression in mammalian species (Vamvakopoulos, 1993). There is a need for greater research on the expression of different HSP families in various tissues to determine their sensitivity to cold shock. Further research is required to understand the discrepancies in studies employing HSPs as indicators of temperature stress in fishes. The complex and interactive relationship between corticosteroids and HSPs demands further investigation before HSPs can be considered sensitive indicators of cold temperature stress.

There are a growing number of 'omic' approaches that are being used to understand the basis of physiological responses to changing environmental conditions (Cossins & Crawford, 2005). Microarray-based gene expression

is one approach that can be used to reveal new insight into these physiological responses. This approach can be combined with metabolic, proteomic and molecular data, which together can provide new insight into physiological responses to the environment (Gracey, 2007). This approach is useful in understanding the molecular basis of the cold-acclimation process. Gracey *et al.* (2004) acclimated *C. carpio* for 2 months at 30° C. Experimental fish were subjected to a 1° C h^{-1} cooling regime to a maximum of 7° C day^{-1} to 23, 17 or 10° C, over 1–3 days and maintained at that temperature for 22 days. Following treatment, responses across seven tissues were assessed with a 13 440 probe cDNA microarray. The authors identified 3461 cold-regulated cDNAs that were differentially expressed among tissues, but many genes were consistent markers of cold exposure in all tissues. Taken together, this research provides insight into the use of cDNA microarrays to understand system-wide gene expression that underlies the physiological mechanisms of cold adaptation and cold shock.

OSMOREGULATORY CHANGES AND IONIC BALANCE

Cold shock may lead to failure in osmotic regulation such that fish in salt-water will gain plasma electrolytes while freshwater-acclimated fish will lose ions. Metabolically dependent ion transport is more sensitive to temperature decreases than passive ion diffusion (Gonzalez & McDonald, 2000). For freshwater fish, an acute temperature drop reduces the active influx of ions while diffusional efflux remains constant (Allanson *et al.*, 1971; Mackay, 1974; Houston & Mearow, 1982), resulting in a net loss of ions at low temperatures. To compensate for ion loss, either ion uptake can be increased through an increase in Na$^+$–K$^+$ ATPase activity or epithelium permeability can be decreased, resulting in slower down-gradient diffusion (Houston & McCarty, 1978; Gonzalez & McDonald, 2000; Mackie *et al.*, 2005); however, these activities are dependent on given low-temperature acclimation periods. The rapid nature of a cold-shock event often precludes a period of time for fish to acclimate to reduced temperatures. When ionic gradients fail to be maintained, CNS function can be compromised through reduced effectiveness of synapse transmission, resulting in a primary stress response that can affect all physiological functions.

Stanley & Colby (1971) compared the responses of freshwater and seawater-adapted alewife *Alosa pseudoharengus* (Wilson) to acute exposure to cold temperature (a decrease from 16·2 to 3° C at 2·5° C daily stepwise decreases) and found a shift in plasma concentration of sodium and calcium towards environmental conditions. Acute exposure to cold caused a shift in plasma concentrations of sodium and calcium in the direction of environmental concentrations (*i.e.* ion decreases in fresh water and increases in sea water). In both fresh and sea waters, muscle sodium concentrations were reduced, suggesting that shifts of ions or water between cellular and extracellular fluids of muscle occurred after the cold-shock stimulus. The authors suggest that these shifts may affect the function of excitable tissue, possibly resulting in muscle tremors or loss of equilibrium, which occurs during natural cold temperature exposure (Stanley & Colby, 1971). Mortality rates in the laboratory were about equal in

both fresh water and sea water. The authors conclude that salinity does not modify tolerance of acute temperature stress.

Water temperature can affect gill Na^+–K^+ ATPase activity, with a general trend towards reduced enzyme activity at lower temperatures (Muir *et al.*, 1994; McCormick *et al.*, 2000). Na^+–K^+ ATPase is involved in chloride cell secretion by providing an electrochemical gradient for Na^+ and Cl^- movement between blood and water (Mackie *et al.*, 2005). There is evidence that Na^+–K^+ ATPase affects branchial and renal carbonic anhydrase activities that consequently provide relatively thermostable basal rates of Na^+ and Cl^- uptake (Houston & McCarty, 1978). Houston & McCarty (1978) conclude that increased branchial Na^+–K^+ ATPase activity and erythrocyte carbonic anhydrase are associated with Na^+ and Cl^- uptake at temperatures that promote increased electrolyte depletion.

Mackay (1974) examined the restoration of Na^+ uptake over a range of acclimation temperatures for *Carassius auratus* (L.) but did not examine cold shock specifically. Fish that were acclimated to 10° C had 3 times greater excretion than fish acclimated to 30° C, when measured at 20° C. Near the lower lethal temperature of 6·5° C, renal Na^+ and Cl^- reabsorption was inhibited. Urine osmolality and urine Na^+, K^+ and Cl^- concentrations were compensated in fish that were acclimated to 10 and 30° C. Rates of Na^+ and Cl^- excretion were seven to eight times higher in fish acclimated to 10° C than 30° C, while 30° C acclimated fish had a 1·7 times greater rate of excretion than 10° C acclimated fish. When compared at 20° C, the 10° C acclimated fish showed the highest rate of net uptake of Na^+. The results of this study corroborate previous findings by Houston *et al.* (1970), which suggests that plasma Na^+, Cl^- and osmolarity are reduced with decreasing temperatures. Similar results were found by Reaves *et al.* (1968) who studied the effects of acute (+10° C, −10·5° C) and moderate (+5° C, −6° C) temperature shock on osmoregulation in *O. mykiss* acclimated to 11° C. These authors found that, regardless of the direction or the magnitude of temperature change, there were initial decreases in plasma Na^+ and Cl^- levels, followed by a decline in tissue water content and an expansion of extracellular fluid volume. These results corroborate an early study by Threadgold & Houston (1964), suggesting that the intensity of regulatory response is correlated with the magnitude of departure from the original regulatory set point. Consequently, any stressor that elicits a sharp deviation from regulated homeostasis will result in a stress response that will equally counteract the stressor or even overshoot it (Reaves *et al.*, 1968). However, the relationship between temperature and ionoregulation is difficult to predict (McCarty & Houston, 1977), and there are indeed species-specific differences Burton (1986).

Gonzalez & McDonald (2000) found that freshwater-acclimated stenothermal *O. mykiss* experienced a significant drop in Na^+ influx and efflux when exposed to temperatures of 5 and 10° C after being acclimated to 15° C. Na^+ influx normalization was apparent after 2 weeks. The authors tested these findings with another freshwater species, eurythermal common shiner *Notropis cornutus* (Mitchill), and observed a slight decrease in Na^+ influx with similar temperature exposures, but Na^+ efflux remained constant at all temperatures and no signs of Na^+ influx acclimation were observed during the subsequent

2 weeks. The Gonzalez & McDonald (2000) study supports the findings of a synthesis by Burton (1986), providing further evidence that species-specific differences may exist for the set point for osmoregulation.

IMMUNE FUNCTION

The ability of fishes to resist disease is mediated by temperature-dependent immune function. A large body of literature illustrates that low temperature affects specific immune responses (reviewed in Hurst, 2007), but few studies have examined this relationship with respect to the rate or frequency of the change (Le Morvan *et al.*, 1998). In addition, few studies have characterized the response to multiple stressors on immune function (Schisler *et al.*, 2000), despite the fact that combinations of stressors, which often accompany cold-shock stress, may potentially increase the susceptibility of fish to pathogens. Engelsma *et al.* (2003) observed changes in leucocyte populations and antibody responses to cold-shock stress following single and multiple 3 h 9° C temperature reductions on the leucocyte population dynamics in the *C. carpio*. Within 4 h of the onset of single or multiple cold shocks, a decrease in circulating β-lymphocytes in the total leucocyte population was observed. The effects were reversed within 24 h. An increase of β-lymphocytes was measured in the head kidney, but the leucocyte distribution responded differently *in vivo* because granulocyte percentages in the total leucocyte population doubled in circulation and decreased in the head kidney. There was a higher percentage of apoptotic lymphocytes in blood compared with controls because of the β-lymphocytes and Ig^- lymphoid cells. Besides the β-lymphocytes, part of the Ig^- lymphoid-cell population was determined to be glucocorticoid receptor positive. The authors found that the T-lymphocyte-independent antigen showed lower antibody titres in stressed carp during the onset of the immune response, indicating that there is a slower development of an antibody response against the T-lymphocyte-independent antigen.

Bennett & Gaudio Neville (1975) exposed *C. auratus* (that had been acclimated between 22 and 24° C) to 2° C for short durations (3·5–4·0 min). The authors found that relative to white blood cell counts of controls, experimental fish exhibited lymphopenia and neutrophilia within 2 h of the cold-shock event. White cell counts recovered in 4 h following treatment. Engelsma *et al.* (2003) suggest that the stress response induced by acute temperature decreases allows fish to adapt in most cases, if adequate levels of specific antibodies are available after infection. However, immune-compromised fishes may be more susceptible to disease following an acute temperature decrease as there is often a slight reduction in antibody production (Engelsma *et al.*, 2003). Little is known about the time course of reduced immune function following cold shock. To date, disease or mortality associated with compromised immune function has not been linked to cold temperature treatments in either the short or the long-term following exposure, likely as an effect of the fact that most fish diseases are temperature sensitive as well.

TERTIARY RESPONSES

GROWTH AND DEVELOPMENT

Temperature controls the pace of development at all life stages of fishes (Fry, 1947). A number of studies have investigated the role of water temperature variation on egg and larval survival and development rates (Allbaugh & Manz, 1964; Neitzel & Becker, 1985; Tang *et al.*, 1987; Ojanguren *et al.*, 1999; reviewed in McCullough, 1999), but few studies have examined the effects of rapid declines in temperature on survival and development of early life stages of fish, although early developmental stages may be particularly sensitive to cold shock.

Studies that have examined the development rates of different species with respect to acute decreases in temperature have found variable results. For example, Neitzel & Becker (1985) found that the survival of cleavage eggs, embryos, eleutheroembryos and pre-emergent alevins of Chinook salmon *Oncorhynchus tschawytscha* (Walbaum) were not significantly reduced when temperatures were dropped from 10 to 0° C. However, a non-linear effect of temperature on development rate was observed. Relatively small decreases in water temperature resulted in large delays in development rates, particularly at low temperatures, where development time increased by >100 days when temperatures were decreased towards the lower lethal limit (see also Brannon, 1987). In contrast, Alderdice & Velsen (1978) found that there was increased compensation in development rate of *O. tschawytscha* embryos with declining temperature as embryos required fewer thermal units to hatch in cold climates in years with cooler than average incubation temperatures. Alderdice & Velsen (1978) observed faster and more successful development in low-temperature environments when incubation temperatures varied with ambient temperatures rather than remaining constantly low. Hubert & Gern (1995) examined the response of cutthroat trout *Oncorhynchus clarki* (Richardson) embryos fertilized at 7° C and exposed to temperatures of 5 and 3° C over multiple acclimation and exposure times. The authors found that mean survival to hatching was lowest in a group that was exposed immediately to a sudden temperature drop from 7 to 3° C, without a period of acclimation but highest when embryos were acclimated to 7° C for 15 days before being subjected to the treatment. The acclimation period prolonged hatching by 31 days, but the survival rate was 15% improved from a control group that was raised at a constant temperature of 7° C. Cold temperatures within lethal limits, during the later developmental stages, may not reduce the probability of survival (Alderdice & Velsen, 1978, Hubert & Gern, 1995). This trend was demonstrated with coho salmon *Oncorhynchus kisutch* (Walbaum) embryos by Tang *et al.* (1987) who demonstrated that mortality approached 100% at temperature tolerance limits above 14° C and below 1·3° C, but abrupt temperature changes within these extremes resulted in little increased mortality.

Cold shock occurring from diel temperature variation can influence growth. Spigarelli *et al.* (1982) examined the feeding, growth and fat deposition in *S. trutta* at three temperature regimes including an artificially fluctuating diel temperature cycle. The regimes simulated fluctuations experienced by fish in

thermal effluents in the spring (*i.e.* 9–18° C; mean 12·5° C), a naturally fluctuating and arrhythmic temperature regime (*i.e.* 4–11° C; mean 7·7° C) and a constant optimum growth temperature for *S. trutta* (*i.e.* 13° C). Food consumption and growth rates were highest in the thermal effluent simulation treatment which also had higher lipid deposition than in fish reared at a constant temperature. Exposure to large or small diel temperature fluctuations did not affect energy conversion efficiencies (Spigarelli *et al.*, 1982). In thermal effluent discharges, *S. trutta* can select water temperature that is optimal for their metabolism. However, in the event of a rapid cessation of thermal effluent, these fish may experience cold-shock stress, particularly during cold weather.

Few studies have attempted to elucidate the genetic components involved in the relationship among tolerance to temperature change, survival and development rates. Future research efforts must incorporate the specific genetic components that account for interspecies- and intraspecies-specific variations in development following temperature treatment. Furthermore, although several studies have characterized reaction to temperature variation by developmental stage for many fish species, the underlying mechanisms remain elusive.

GAS EXCHANGE AND ALTERED GILL STRUCTURE

Temperature influences respiratory function in fish. Although there is growing evidence that cold-shock stress affects ionoregulation, it has not been linked to structural changes in gill tissue. Black *et al.* (1991) found that respiratory function and O_2 consumption decreased in *O. mykiss* when temperature was reduced from 17 to 8° C. It was attributed to changes in gill membrane permeability and O_2 demand. Tilney & Hocutt (1987) exposed *O. mossambicus* to rapid temperature decreases of 20, 15, 10 and 5° C after being acclimated to 25° C to examine cellular damage and mortality. Some shrinkage in gill tissue was detected, but there was no evidence of cellular damage. Future research needs to address these questions specifically to gain a better understanding of the linkages between cold shock and gas exchange.

FORAGING AND PREDATION

Temperature reduction can result in a suite of consequences that may affect swimming behaviour, compromise foraging and impede predator evasion and consequently, starvation and predation are often linked with mortality from winter kills (Hurst, 2007). Cold-shock stress can result in starvation for fish with compromised locomotory or foraging ability. Reduced swimming ability from cold shock can also result in elevated predation susceptibility, particularly in instances where predators have broader temperature tolerances than prey.

Cold-shock responses in fishes include periods of hyperactivity followed by slowed movement, compromised swimming ability, reduced responsiveness, loss of equilibrium, onset of cold coma and respiratory failure (Friedlander *et al.*, 1976; Muir *et al.*, 1994; Fuiman & Batty, 1997; Smith & Hubert, 2003; reviewed in Coutant, 1977*a*). For example, Smith & Hubert (2003) found that fingerling *O. mykiss* exposed to a temperature reduction from 8 to

0·3° C displayed short-term sublethal behavioural anomalies, including erratic swimming behaviour, followed by lethargy and temporary loss of equilibrium. The loss of equilibrium is a common cold-shock response and may result in reduced response speeds and predator perception and ultimately higher susceptibility to predation (Griffith, 1978; Fuiman, 1991). Griffith (1978) acclimated threadfins *Dorosoma petenense* (Günther) at 15° C and exposed them to hourly 1° C decreases in water temperature over a period of 72 h. At temperatures below 9° C, fish began to lose equilibrium and were less sensitive to external stimuli. When exposed to a more rapid decline (2–5° C decrease over 4 h), equilibrium loss occurred rapidly and in proportion to the magnitude of temperature decrease. Griffith (1978) concluded that these behavioural impairments would result in a reduced ability to escape predation in nature.

Clarkson & Childs (2000) studied the effects of cold shock on the larval and early juvenile life stages of razorback sucker *Xyrauchen texanus* (Abbott), flannelmouth sucker *Catostomus latipinnis* Baird and Girard, humpback chub *Gila cypha* Miller and Colorado squawfish *Ptychocheilus lucius* Girard. These fish were subjected to a temperature decrease from 20 to 10° C to simulate movement from warm-water tributaries to cold tailwaters. Their findings were corroborated by Berry (1988) where early larval stages were found to be highly susceptible to cold coma and accordingly were at an increased risk of predation. The length of cold comas is highly variable and may last between 5 and 90 min after an acute temperature decrease of 10° C. Generally, a reduction of 10° C results in higher cold coma and direct mortality of larval and juvenile fish than less extreme reductions of 6° C (Berry, 1988; Clarkson & Childs, 2000). Johnson & Bennett (1995) examined the escape behaviour of *C. auratus* and killifish *Fundulus heteroclitus* (L.) following cold shock. These authors found that, at 10° C, maximum escape velocity increased by 35%, distance moved increased by 52% and the maximum angular velocity increased by 42% in *C. auratus* acclimated to 10° C *v.* those first acclimated to 35° C and subsequently cold shocked to 10° C.

Fish with a broad tolerance may have competitive advantages over other species, particularly with regards to predator and prey interactions. Ward & Bonar (2003) acclimated age 0 *C. latipinnis* and their predator (*i.e. O. mykiss*) to 20° C and then rapidly transferred them to a 10° C tank. Successful attacks were lower at the acclimation temperature of 20° C (5·8%) than at 10° C (39·1%), suggesting that *C. latipinnis* may be less tolerant to temperature change than *O. mykiss* in regards to swimming response (Ward *et al.*, 2002).

Following acute drops in temperature, prey may also suffer from a reduction in antipredator reaction distance (Coutant, 1973; Webb & Zhang, 1994) and evasive movement increasing their susceptibility to capture (Coutant, 1973; Coutant *et al.*, 1974). For example, Coutant *et al.* (1974) found increased predation of juvenile *I. punctatus* and *M. salmoides* following cold temperature exposure treatment. Furthermore, the authors noted that predation rates increased as treatment temperature differential increased. If juvenile *I. punctatus* were allowed to acclimate to cold temperatures for 1 h before being exposed to predation, they were slightly less susceptible.

CONSEQUENCES OF NATURAL AND ANTHROPOGENIC SOURCES OF COLD SHOCK

NATURAL SOURCES

Cold-shock stress may be induced from either natural or anthropogenic sources (Fig. 1 and Table II). Natural sources include horizontal thermoclines (Steiner & Olla, 1985), seiches (Emery, 1970), rapid changes in solar heat exposure in shallow areas (Newell *et al.*, 1959; Overstreet, 1974) or rapid changes in seasonal or diel temperatures (reviewed in Coutant, 1977; Larimore, 2002). Cold-shock stress is thought to be linked with naturally occurring 'fish kills' (Emery, 1970; Overstreet, 1974; Holt & Holt, 1983; Hurst & Conover, 1998; Hoag, 2003). Causal factors implicated in fish-kill events tend to be proximate and brief, and historically, evidence has often been anecdotal and indirect. Recently, a review on the causes and consequences of winter mortality on fishes was conducted by Hurst (2007), but this review did not directly focus on the effects of cold shock on fish species. Hurst (2007) identified that the sources of these stressors remain unidentified for many of the populations that have been affected by these phenomena.

Natural occurrences of cold shock are thought to be quite common, yet there are relatively few examples of these events that have been described in the literature. For example, Emery (1970) implicated declining temperatures from a seiche as a cause for high mortalities of sculpins *Cottus bairdi* Girard in Georgian Bay, Lake Huron, Canada. Overstreet (1974) identified a seasonal rapid decline in water temperature as responsible for the loss of striped mullet *Mugil cephalus* Linnaeus in the state of Mississippi, U.S.A. Newell *et al.* (1959) described a similar event in shallow bays and estuaries of The Bahamas where rapid declines in water temperature resulted in fish mortality. The authors implicated a naturally occurring rapid decrease in temperature coupled with low-salinity water, which may have affected ionoregulation, starvation and responses to high pesticide loads. These studies were retrospective to the event, and the authors could only speculate that temperature was the stimulus or at least a factor interacting with many others in their prognosis. An improved understanding of the processes linking environmental factors to behavioural and physiological responses, including all associated interactions, is required to identify the role of temperature in the pathway of effects leading to fish mortality.

Fish species are affected by the chronic and cumulative stress of natural fluctuations in temperature associated with diel temperature variation (Baum, 2004). A number of studies have examined the role of diel water temperature fluctuations on behaviour (Walker *et al.*, 2000), physiology (Cairns *et al.*, 1974; Lyytikainen *et al.*, 2002), development (Bestgen & Williams, 1994) and survival (Hartwell & Hoss, 1979; Thomas *et al.*, 1986; Dickerson & Vinyard, 1999).

Water temperature heterogeneity and patchiness provides fish an opportunity to seek thermal refugia and evolve behaviours to preferentially select thermal microhabitats. Recent evidence shows that certain fish species distribute within thermally stratified waters to avoid water temperature that

TABLE II. Key anthropogenic and natural sources of cold shock

Source type	Key research published	Context of research	Relative amount of knowledge about this source
Anthropogenic sources			
Thermal effluent from power generating plants	Smythe & Sawyko (2000)	Field and laboratory study on the effects of cold shock at a thermal discharge	High
Thermal effluent from industrial plants	Birtwell & Kruzynski (1989)	Pulp and paper mill effluents; metal production effluents	Medium
Alterations in water control structures	Lamadrid-Rose & Boehlert (1988)	Effects of cold water mixing during ocean thermal energy conversion on different life stages of fish	Low
Fish harvesting practices	Hyvarinen et al. (2004)	Simulated trawl exercise followed by acute temperature decrease	Low
Natural sources			
Thermocline exposure at different water depths	Steiner & Olla (1985)	Behavioural variation with temperature differences in a laboratory-simulated thermocline	Low
Abnormal seiches and water movements	Emery (1970)	Rapid temperature decrease caused by an abnormal seiche	Low
Rapid temperature changes in shallow areas	Overstreet (1974)	Rapid temperature decrease in freshwater bayous	Low
Diel changes in water temperature	Larimore (2002)	Examination of the behaviour and temperature tolerance of fry following acute temperature decreases from flood and storm conditions	Low

approaches thermal tolerance limits (Birtwell *et al.*, 2003), but prey abundance (Munson *et al.*, 1980) or dissolved oxygen levels (Birtwell & Kruzynski, 1989; Bauer & Schlott, 2006) may act to modify microhabitat distribution. For example, to simulate juvenile hake *Urophycis chuss* (Walbaum) migrations from the warm surface waters (20° C) to the cooler temperatures (10° C) adjacent to the benthos, Steiner & Olla (1985) created an artificial thermocline in an experimental tank. The authors found that these fish, which had been acclimated to 20° C, preferentially selected water temperatures of 15° C. When fish were first introduced to the tank, they immediately made vertical descents to cooler water but displayed behavioural signs of cold shock. The response to cold shock involved individuals shaking side to side and having spasmodic movements (lasting 5–10 s). Fish that failed to return to warmer temperatures lost equilibrium and ceased movement (including opercular movements). However, within 2 h, these fish each showed slight opercular movements, regained equilibrium and swam vertically to warmer waters. The authors suggest that the ability to recover from this cold shock reflects a physiological predisposition for colder temperatures that would be encountered during vertical migrations in nature.

ANTHROPOGENIC SOURCES

In aquaculture or commercial harvesting operations, the transfer of live fish from ambient temperatures to temporary cold-water storage facilities can result in a cold-shock response (Barton & Peter, 1982; Fletcher *et al.*, 1988; Hyvarinen *et al.*, 2004). Furthermore, warm-water adapted aquaculture species may be exposed to cold currents in winter (Hsieh *et al.*, 2007). Heated thermal effluents from pulp and paper production (Birtwell & Kruzynski, 1989), food processing (Hyvarinen *et al.*, 2004), metal production (International Pacific Salmon Fisheries Commission 1952, 1953) and power generation plants (reviewed in Coutant, 1977; Schreer & Cooke, 2002) commonly attract fish, particularly in colder climates and expose fish to rapid declines in temperature following a cessation in operations. Hypolimnetic draws from reservoirs can influence downstream temperature depending on the reservoir's thermal stratification and the degree to which the drawdown changes the effective location of the draw within the reservoir (Webb & Walling, 1997). Generally, downstream temperatures will be cooler in the summer and warmer in the winter following dam construction (Edwards, 1978; Jensen, 1987; Preece & Jones, 2002), but site-specific factors including operational regime (storage and peaking policies), proximity of the outfall and availability of alternative tributary temperature refugia will have a great influence on fishes' susceptibility to temperature stress.

Ocean thermal energy conversion (OTEC), which brings deep, cold water to the surface (Lamadrid-Rose & Boehlert, 1988), is a less common source of cold-shock stress. The temperature differential created at the surface rapidly exposes larval and juvenile fishes to lower water temperatures. Lamadrid-Rose & Boehlert (1988) simulated cold-water entrainment on the egg and larval stages of tropical fish (*e.g.* mahi mahi *Coryphaena hippurus* [L.], manini *Acanthurus triostegus* [L.] and juvenile striped mullet *M. cephalus*) and found that early egg stages were more sensitive to temperature reduction. Mortality increased with increasing

magnitude of temperature change relative to controls but exposure time did not significantly affect mortality. More research is required to assess the role of cold upwelling water from OTEC on all development stages of other marine life.

Power generating plants are common sources of cold-shock stress (Cooper, 1973; Ash *et al.*, 1974; reviewed in Coutant, 1977; Keefer *et al.*, 2004) but little is known about the biological consequences of variable temperatures (Murchie *et al.*, 2007). In the U.S.A., 52% of the steam-electric power plants use 'once-through' technology for power-plant cooling (Baum, 2004). These systems release waste heat from the power-plant condensers creating effluents that are well above ambient temperature (reviewed in Coutant, 1977). As water temperature decreases in winter, many fishes demonstrate positive thermal taxis, aggregating in these warm thermal-effluent plumes (Coutant, 1970; Cherko *et al.*, 1976; reviewed in Coutant, 1977; Stauffer *et al.*, 1988). Fishes that acclimate to the higher water temperature are at risk of cold shock if the release of heated effluent is abruptly stopped (Smythe & Sawyko, 2000). The volume of heated effluent depends on maintenance schedules, mechanical reliability and the demand for power. In the event of a shutdown, temperature fluctuations downstream may be rapid and unpredictable, resulting in stressful conditions for aquatic organisms (Ash *et al.*, 1974; Coutant & Talmadge, 1977; Miller, 1977; Talmage & Coutant, 1980).

Anupkumar *et al.* (2005) while mapping the thermal discharge from an electric power plant found that temperature increases were restricted to the surface 2 m of the water column. Historically, the majority of cold-shock incidents have involved power-plant designs, which feature low-velocity surface discharge to lake, river or estuary shorelines (reviewed in Coutant, 1977). Fish can generally cope with gradual temperature variability, provided that the upper and the lower temperatures remain within their specific temperature tolerance zones, but the risk of stress increases with the rate of temperature change.

Field studies, less common than those in the laboratory, have generally focused on thermal effluents from power production. Ryan & Witzel (1993) observed fish kills during times of low-power production and consequent low discharge temperatures in the outflow from the Nanticoke thermal generating station on Lake Erie from 1983 to 1993. Ash *et al.* (1974) described a large fish kill near a lake discharge canal following a rapid (30 min) temperature drop from 21·8 to 4·8° C. Cold-shock stress can be challenging to study in the field, but technological advancements in videography and biotelemetry have been used in a few studies to assess fish behaviour in industrial settings. Cooke & Schreer (2002) used underwater videography to monitor the community composition and abundance patterns of fish in a discharge canal and the thermal plume of a generation station on Lake Erie. Similarly, Cooke & McKinley (1999) used radio telemetry to examine the distribution of fishes in a thermal-discharge plume. Schreer & Cooke (2002) and Cooke & Schreer (2003) used physiological telemetry to assess *in situ* fish responses to variable thermal conditions in a power-plant effluent. The authors reported that the activity levels of fish tended to be highest during periods of rapid changes in temperature (both higher and lower). These technologies show promise as methods to understand the complex interactions among fish species and their habitat in thermally heterogeneous habitats. Thermal discharge may have direct or indirect

implications on the physiology and behaviour of individual fish, populations, communities and ecosystem structure and composition (Crawshaw, 1977).

Numerous studies have examined abundance, distribution and mortality of fishes to examine physiological and behavioural response to power plant thermal plumes. Schreer & Cooke (2002) monitored cardiac function, movement, distribution and abundance of *Micropterus dolomieu* Lacepède in the discharge canal of a thermal generating station during winter and summer months. Cardiac function was highly responsive to temperature, but few behavioural adjustments were observed. Using telemetry, the authors found 90% of *M. dolomieu* in the warmest and most thermally variable areas of the discharge canal during winter. Residency in warm-water areas may increase growth and survival of some species but increases the risk of cold-shock stress in the event of a sudden decrease in temperature as a result of operational practices. Future research could make use of physiological telemetry to examine cardiac function, movement, distribution and abundance of fishes during operational actions to specifically examine cold-shock response.

Micropterus dolomieu parental care activity was elevated in fluctuating thermal regimes of an effluent canal relative to non-thermally altered environments but was highly variable among individuals (Cooke *et al.*, 2003). Similarly, differences in distribution of *I. punctatus* and *C. carpio* in a thermal-discharge canal suggested that risk to temperature declines is species specific and dependent on response to thermal effluent (Cooke & McKinley, 1999). Similarly, Astles *et al.* (2003) found evidence of species-specific differences in growth, survival, distribution and activity of juvenile Murray cod *Maccullochella peeli* (Mitchell) and silver perch *Bidyanus bidyanus* (Mitchill) in an experimental cold-water facility below the Burrendong Dam on the Macquarie River, Australia.

Smythe & Sawyko (2000) evaluated the effects of cold temperature on fishes captured in and around the Lake Ontario Ginna nuclear power station thermal-effluent plume. *Salmo trutta*, *O. mykiss*, walleye *Sander vitreus* (Mitchill) and gizzard shad *Dorosoma cepedianum* (Lesueur) were considered. This research, along with two other studies that used both laboratory and field components, examined the abundance and distribution of fishes at a thermal plume (Sawyko & Smythe, 1983; Sawyko, 1985). The authors found that *D. cepedianum* showed the lowest survival rates, while *S. trutta*, *O. mykiss* and *S. vitreus* were more tolerant to a sudden drop in temperature.

Birtwell & Kruzynski (1989) examined surface water orientation behaviour in juvenile Pacific salmon (*Oncorhynchus* spp.) in an estuary that received a surface discharge of treated pulp mill effluent. Behavioural bioassays showed that juvenile *O. tschawytscha* avoided cold deep water in preference for surface water exposing them to low dissolved O_2 and other water quality problems. Individuals may expose themselves to rapid temperature change by moving from thermal plumes to adjacent areas with ambient water temperature, in an attempt to reduce exposure to suboptimal currents and dissolved oxygen, and to improve shelter (reviewed in Coutant, 1975). Furthermore, there is evidence that abundance and availability of prey species may be an important factor in determining aggregation of certain species in the discharge canal as prey fish may become injured or disoriented while passing through condenser cooling and tempering pumps (Cooke *et al.*, 2000; Schreer & Cooke, 2002) and may be

attracted to warm-water discharges, resulting in higher prey concentrations for predatory species.

MANAGEMENT CONSIDERATIONS

Since the 1970s, many recommendations have been proposed and implemented to mitigate the events that lead to cold-shock stress and technologies have evolved to reduce the effects of anthropogenic sources such as power stations. However, in the U.S.A., once-through cooling remains the most common technology for steam electric-power generation and has the greatest potential for causing cold-shock stress (Baum 2004). Coutant (1977a) suggests that siting power stations away from zones of high biological activity may minimize the number of fish that are attracted to the heated effluent. Site-specific recommendations tend to be the most effective way to protect aquatic life from the effects of cold-shock stress. Coutant (1977) identified three principle plant design options: (1) restrict the volume of heated water accessible to organisms; (2) reduce the frequency of complete shutdown through multiple-unit designs; and (3) reduce discharge temperatures to limit cold-shock stress in the event of a shutdown. Coutant (1977) recommended that plant shutdowns be scheduled when organisms are least susceptible to cold shock from the cessation of thermal effluents (*i.e.* summer months) in an effort to minimize the temperature differential between the effluent and the ambient waters. However, discharge temperature reduction in the winter may be difficult because plants increase their cooling efficiency in the winter. Although accidental shutdown can rarely be avoided, planned outages with a slow reduction in discharge temperature would reduce the risk of cold-shock stress on aquatic organisms.

Astles *et al.* (2003) suggested that dam facilities need to be managed following well-researched guidelines that are reflective of both site-specific and species-specific differences in susceptibility to declining temperature. If water release depth can be controlled, release temperatures can be manipulated to simulate natural diurnal fluctuations (Hubbs, 1991) or to manage downstream habitats to benefit a particular species or meet ecological targets (Horne *et al.*, 2004; Macdonald *et al.*, in press). Some reservoirs have airlift systems installed to destratify the water column in an attempt to minimize downstream temperature effects but their effectiveness has been questioned (Webb & Walling, 1997).

Becker *et al.* (1977) suggest that the management of ecological interactions of thermal discharge from power stations should be based on sublethal consequences rather than considering only lethal endpoints, a consideration echoed here. In natural systems, a sublethal outcome such as equilibrium loss may ultimately lead to death from elevated predation risk or exposure to cooler temperatures or anoxic conditions if fish are forced to the lower strata of a water body. Managing to prevent cold-shock stress is very difficult as causal events tend to be temporally and spatially unpredictable. Currently, there are several mitigation measures that can be taken to reduce the risk of cold-shock stress and mortality on fishes (Wilde, 1988; Nickel *et al.*, 2004), but until more research can be conducted, management bodies that take a precautionary

approach should consider rapid declines in temperature as a threat to fish species. Although a number of guidelines for protecting fish from deleterious temperature regimes have been proposed (Oliver & Fidler, 2001; US EPA, 2003), the majority of these guidelines fail to specifically consider cold shock. Among the few recommendations to protect fish from rapid temperature changes, there is a greater emphasis on protecting fish from acute temperature increases rather than decreases (Oliver & Fidler, 2001; US EPA, 2003). Recommendations tend to be generalized and rarely consider site-specific (*e.g.* flow conditions and seasonal variation) or species-specific (*e.g.* intraspecific or interspecific, population, life stages, *etc.*) responses to acute temperature decline. Developing and implementing scientifically grounded guidelines to manage the causes of cold-shock stress are a challenging task, as there are considerable knowledge gaps. Future initiatives need to use scientifically grounded research and address site-specific or species-specific concerns.

APPLICATIONS OF COLD SHOCK IN FISHERIES SCIENCE

Although cold-shock stress has traditionally been viewed as a cause of sublethal and lethal effects on fish and fish populations, a recent shift in research has focused on the application of cold-shock stress as a powerful tool in fisheries science. It has seen applications as a short-term anaesthetic (Hovda & Linley, 2000), as a means to alter embryonic sex ratios (Craig *et al.*, 1996) and, most commonly, as an agent in the induction of polyploidy (Peruzzi *et al.*, 2007).

In the past decade, a number of studies have examined the application of temperature shock as an inexpensive method of inducing polyploidy (triploidy and tetraploidy) in many species of fishes (Pandian & Koteeswaran, 1998). Induced triploidy is considered to be the most widely accepted and effective method to disrupt sexual maturation and produce sterility (reviewed in Felip *et al.*, 2001; reviewed in Tiwary *et al.*, 2004). Whereas heat shock is more commonly used to induce polyploidy in cold-water species, cold shock is more common than warm shock in experiments with warm-water fish, such as *O. aureus*. For example, Valenti (1975) reduced the temperature from 32 to 11° C over a period of 1 h shortly after fertilization and found a 75% incidence of polyploidy. However, Don & Avtalion (1988) used both heat and cold treatments to induce polyploidy in *O. aureus* and *O. niloticus* and found 50% induction for *O. niloticus* and 60% for *O. aureus*. The authors suggest that cold temperature is more efficient at inducing triploidy in *O. niloticus*, but both hot and cold temperatures were equally efficient at inducing triploidy in *O. aureus*. Peruzzi & Chatain (2000) induced triploidy in the European sea bass *Dicentrarchus labrax* L. by altering the timing, intensity and duration of cold temperature applications. They achieved a higher percentage of success with treatments of 0–1° C during a 15–20 min duration, 5 min post-fertilization.

Acute cooling induces polyploidy by causing the retention of the second meiotic polar body or blocking the first mitotic division of a fertilized egg (reviewed in Tiwary *et al.*, 2004). In general, triploid fish are more heterozygous, have larger and fewer cells in most tissues and organs and have

underdeveloped gonads, relative to diploid fish (reviewed in Benfey, 1999). Polyploid fish can have a faster growth rate and larger body size than diploid fish (Valenti, 1975), possibly because precocious maturation is eliminated and they convert food more efficiently, which may have benefits for aquaculture (Lemoine & Smith, 1980; Wolters *et al.*, 1982). However, triploidy may have no effect or more commonly cause a reduction in growth rates in salmonids (Devlin *et al.*, 2004; Johnson *et al.*, 2004; Wagner *et al.*, 2006). The aquaculture and sport-fishing industries see the sterile nature of triploids as a means to achieve reproductive containment in situations where wild and hatchery stocks may coexist and thus preserve the genetic integrity of wild populations. It also allows for larger fish to be achieved in semelparous species (Wolters *et al.*, 1982). Unfortunately, few studies have examined the effects of cold-shock procedures on the general condition of the fish. Increased mortality often occurs when attempting to induce polyploidy, but future research must assess the general condition and stress associated with polyploid individuals throughout each developmental stage.

CONCLUSIONS AND FUTURE DIRECTIONS

The application of cold shock to fisheries science represents a new and exciting direction of research. However, the most substantive developments in this field are in the direction of comparative biochemistry and physiology and associated behaviour. A gap still exists in the understanding of the interaction among physiological and ecological processes that are responsible for the direct and indirect mortality associated with acute cold temperature exposure. For example, there are still few studies that investigate multiple endpoints, such as linking physiology and behaviour. Previously, it has been difficult to link physiological and behavioural endpoints; however, there is a growing body of field research that combines approaches from multiple disciplines through the integration of biosampling to non-invasively assess physiological indices and biotelemetry to assess fish behaviour and survival (Cooke *et al.*, 2005; Donaldson *et al.*, 2008). For this approach to be relevant in a temperature context, telemetry gear can have on-board temperature sensors or thermal loggers to assess temperature in relation to physiology and behaviour. This would facilitate the extent to which individuals experience rapid temperature decreases when exposed to either natural or anthropogenic sources of cold shock. Understanding these linkages between cold shock and physiology, behaviour and ecology is fundamental in managing anthropogenic sources of cold shock (Baum, 2004).

Acute cold temperature is a stressor that has consequences for fish at all biological levels (*i.e.* cellular to population level effects). Accordingly, future studies must attempt to link physiological and ecological research from both the field and the laboratory, with many measures of fitness, including growth, bioenergetics, survival and population effects (Young *et al.*, 2006). Corroborative linkages between laboratory and *in situ* studies have and will continue to strengthen the understanding of this integration (Table III).

Many studies have investigated the relationship between acute temperature change and survival of fishes (Felip *et al.*, 1999; Tanck *et al.*, 2000; Hyvarinen

TABLE III. Suggestions for future cold-shock research sorted by priority

General area of research	Research required	Type of study
Sources of cold shock	A detailed characterization of natural and anthropogenic sources of cold shock. There is a need to understand the relative frequency, magnitude, and consequences of variation in thermal conditions	Field
Differences in cold shock sources	Research initiatives must examine site-specific differences with respect to cold shock	Field
Differences in cold shock response	Research initiatives must examine the interspecific and intraspecific differences in response to cold shock. An emphasis should be made on characterizing the cold-shock response relative to the life-history stage of each species	Field/laboratory
Applied cold shock research	Research initiatives must be undertaken at key cold-shock sources that have been identified as high risk to mitigate cold shock	Field
Primary, secondary and tertiary stress responses	The cold shock stress response is highly interactive, yet research fails to simultaneously consider the three responses in each study. Using collaborative research, it would be possible to study the entire suite of stress responses with respect to cold shock. Ideally, this research activity would couple behaviour and physiology in the laboratory and field. There is immense opportunity to use biotelemetry for this type of research	Field/laboratory
Policy development and guidelines	In order to suggest more specific guidelines and develop robust policy, additional research and monitoring is required. These data could be used to develop generalized models of effects and risk-management strategies	Field
Primary brain response	With advances in fMRI technologies, future studies should be conducted to elucidate the mechanisms of stress response in the brain	Laboratory
Heat-shock and cold-shock proteins and molecular responses	Future research is required to determine the species-specific expression of HSPs from cold shock. Studies that assess the role of HSP response to both temperature increases and decreases are needed	Laboratory
Immune function	Research on the longitudinal effects of cold shock on immune function and disease susceptibility is required	Field/laboratory
Cold-shock effects at the community, population and ecosystem levels	Few studies have examined cold shock at higher levels of biological organization. Future studies must characterize the *in situ* effects of cold shock at these levels	Field

fMRI, functional magnetic resonance imaging; HSPs, heat shock proteins.

et al., 2004), but only recently have studies examined the primary and secondary physiological mechanisms that lead to tertiary physiological and behavioural responses. While the primary stress responses in fishes are generally well understood, the primary brain responses to cold temperature stress have not been thoroughly studied (reviewed in Barton 2002), although technology such as fMRI is beginning to allow a more detailed understanding of neural compensatory mechanisms related to declining temperatures (Peeters *et al.*, 2001; Van den Burg *et al.*, 2005). Increases in corticosteroids and catecholamine hormones have been associated with cold shock, but the mechanisms by which acute changes in temperature are integrated by the CNS and the innervation of chromaffin tissues, which release catecholamines, remain elusive (Reid *et al.*, 1996, 1998). The majority of secondary stress response studies have focused on cellular changes, such as HSP expression, or changes to metabolic, haematological and immune function. A number of studies have examined the role of HSPs in the stress response of fishes. However, few studies have clearly demonstrated the effect of acute temperature reduction on HSP expression. The limited number of studies that have been conducted show conflicting results, highlighting the need for more research. Where possible, studies should integrate secondary stress response with long-term tertiary consequences such as growth and development.

The linkages between certain secondary responses to acute reduction in temperature and tertiary responses are not well understood. For instance, temperature is widely known to influence immune function in poikilotherms (Le Morvan *et al.*, 1998), but few studies examine immune suppression, with the associated physiological and behavioural effects, following one or multiple acute temperature declines over varying time periods. This work will entail long-term observations, which are also required for the examination of the integration of secondary and tertiary physiological responses and recovery rates. Examination of stress responses at the organismal or at the community level requires studies that span months or years. The suite of primary, secondary and tertiary sublethal responses to cold shock generally occur in concert, and it is unclear how the cumulative and interactive nature of these responses affect the organism as a whole.

This review illustrates that although cold-shock stress research is increasingly prevalent in the literature, key knowledge gaps still exist in understanding primary, secondary and tertiary stress response mechanisms. Predicting the magnitude of many possible responses is made more difficult because of differences in developmental stage, genetic composition and thermal histories of the subject. However, research efforts to date and in the future have and will facilitate improved management guidelines, policies and laws, allowing enlightened conservation initiatives and development techniques. Currently, management and monitoring activities tend to be specific to a given infrastructure project, but with sufficient research more generalized impact guidelines, and risk-averse management strategies may be developed. For natural sources of cold-shock stress, there is need for greater understanding of the frequency and magnitude of the event as well as their role in structuring ecological communities. Through improved scientific knowledge of the effects of cold shock on fish, the large uncertainty surrounding the effects of different management actions

can be reduced, and thereby provide managers with better information to mitigate the risks.

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