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Are we any closer to understanding why fish can die after severe exercise?

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Abstract

Post-exercise mortality (PEM) may occur when fish exercise to exhaustion and are pushed so far beyond their physiological limits that they can no longer sustain life. Although fish exercise to overcome a variety of natural challenges, the phenomenon of PEM is most often observed as the result of interactions between fish and humans. The seminal work of Black (Can J Fish Aquat Sci, 15:573, 1958) and Wood et al. (J Fish Biol, 22:189, 1983) provided a foundation for exploring the potential causes of PEM in fish. With no "silver bullet" explaining PEM being apparent, contemporary research has continued to focus on physiological mechanisms of exhaustion in fish, including factors such as oxygen delivery, ion regulation, hormone signalling, and cardiac function. This paper provides an overview of these studies, and reviews the continuous improvement in data collection methods, tools, and experimental protocols used to examine the PEM phenomenon. These studies of exhaustion have played an important role in informing management actions for activities such as bycatch revival and fish passage. Since the contribution of Wood et al. (Journal of Fish Biology, 22(2):189–201, 1983), the combined efforts of fundamental and applied research have yielded a greater understanding of why fish die after severe exercise, yet much remains to be explored through future work.

KEYWORDS
catch-and-release fishing, emerging technologies, exhaustion, physiology, post-exercise mortality

1 | INTRODUCTION

Throughout the course of their life, fish must overcome many natural challenges that test their physiological systems, yet the greatest challenge is often represented by their interactions with humans (Brownscombe, Cooke, et al., 2017). Fish–human interactions come in various forms and may include fish encountering a dam during migration, which requires them to locate and ascend a fishway (Silva et al., 2018), swimming in areas with dynamic flows (e.g., downstream from peaking hydropower facilities or during floods induced by human land use change; Murchie & Smokorowski, 2004; Taylor & Cooke, 2012), avoiding entrainment in various types of water intakes (e.g., turbines, cooling intakes, industrial use, irrigation; Mussen et al., 2014; Harrison et al., 2019), or during fisheries interactions where fish are initially captured but escape or are released (e.g., as bycatch in commercial fisheries or so-called catch-and-release in recreational fisheries; Davis, 2002; Brownscombe, Danylchuk, et al., 2017). The extreme exercise that fish perform during these interactions with humans or human infrastructure can push individuals beyond the threshold of their physiological systems and, in severe cases, may result in their death: a phenomenon called post-exercise mortality (PEM) (Figure 1). All of these interactions, whether in marine or freshwater systems, are further modulated by other human-induced stressors such as climate change-related increases in water temperature or hypoxia that collectively make recovery from human interactions more challenging (Arthington et al., 2016; Reid et al., 2019; Whitney et al., 2016).

The ability of fish to navigate these physiological challenges governs their behaviour and fitness (Ricklefs & Wikelski, 2002). Therefore, the appropriate management of physiological resources represents the cornerstone of maintaining, or returning to, homeostatic conditions. The demands of metabolically active tissues increase as fish are pushed to their homeostatic threshold before reaching exhaustion (Kieffer, 2000). Although it is thought to be uncommon that wild fish are pushed beyond their homeostatic threshold due to natural activities or conditions (Rodnick & Planas, 2016), interactions with humans can disrupt this balance. This emphasizes the importance of understanding the physiological consequences of extreme exercise to seek solutions that minimize our impact on the well-being of exhausted fish.

From the onset of exhaustion, fish are tasked with managing physiological and biochemical disruptions, including increases in stress hormone secretion, high rates of cellular energy consumption, depletion of tissue energy stores, reduction in phosphorylation potential, and severe acidosis (Rodnick & Planas, 2016). The impacts of exhaustion can also be exacerbated by external factors including temperature (Clark et al., 2013; Farrell et al., 2008), oxygenation (Eliason & Farrell, 2016; Sinha et al., 2012), food availability, nutritional status (Alsop & Wood, 1997; Gingerich et al., 2010), and even individual phenotypes (Killen et al., 2015; Metcalfe et al., 2016). Exposure to multiple stressors, in the context of exhaustive exercise, may act synergistically (Folt et al., 1999), and can have significant impacts on the maintenance of oxygen delivery to tissues, the ability to return to acid–base homeostasis, the regulation of ion and osmotic balance, and the recovery of cellular energy stores (Kieffer, 2000; Wendelaar Bonga, 1997; Wood, 1991). When the summative action of internal and external stressors exceeds coping capacity (Romero et al., 2009; Schreck, 2010) and individuals are unable to re-establish homeostasis/steady-state, PEM can occur (Black, 1958). The phenomenon of PEM is complex because surviving must involve the integration of multiple metabolically active tissues to sustain normal functional capacity (Wood et al., 1983). Stressors affect several important tissues, including gills, muscle tissue, liver, kidneys, and heart, to disrupt normal function and impede recovery from exhaustive exercise. Physiological challenges associated with the downstream effects of exhaustion can have direct implications on the recovery, behaviour, and subsequent performance of fish following bouts of exhaustive exercise (Burnett et al., 2014; Eliason & Farrell, 2016). However, quantifying these effects can be challenging as the multitude of processes involved can make pinpointing the mechanistic causes behind PEM difficult.

Situations that potentially expose fish to PEM in the wild are remarkably common. For example, during the upriver migration of Pacific salmon on regulated rivers, entire assemblages of fish may have to navigate dynamic flows (as a result of hydropower releases; Murchie et al., 2008) and ascend multiple fishways (e.g., in the Columbia River; Caudill et al., 2007). The entire migration of Pacific salmon in such highly regulated systems (which can easily be on the order of millions of fish) requires that a sufficient number of fish survive to reach spawning grounds and produce the next generation of offspring. Fisheries interactions are also common. Cooke and Cowx (2004) estimated that the annual global catch of recreational anglers was ~47 billion fish of which ~30 billion

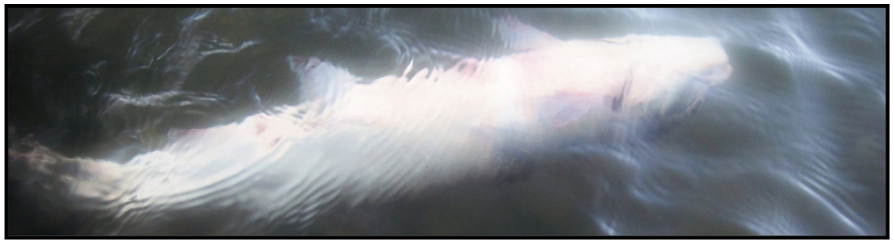

FIGURE 1 An exhausted fish that can no longer maintain equilibrium after a catch-and-release angling event. The exhaustion experienced by this fish could result in post-exercise mortality (PEM), though specific mechanisms behind this phenomenon remain unclear. Photo credit: Cooke Lab.

are released. The number of individual fish captured and released (or that escape capture) from commercial fisheries is difficult to estimate (Alverson et al., 1994), but presumably exceeds the number of recreationally angled fish by severalfold. These are but a few examples of the relevance of PEM given the sheer numbers of fish that interact with humans and human infrastructure on an annual basis (tens of billions of fish, if not hundreds of billions): all of which have the potential to be subjects of PEM. Clearly, there is a need to better understand this phenomenon in an attempt to predict mortality relevant to different contexts and to consider mitigative strategies to reduce PEM.

The goal of this review is to examine the information presently available on fish PEM in an attempt to understand the underlying physiological mechanisms that cause death after exhaustive swimming. We also aim to contextualize the impact of exhaustion on mortality by bridging the gap between the fundamental physiology and the applied ecological consequences. This review is intended to provide conservationists, fish biologists, and managers alike with a brief history of exhaustion in fish, an updated perspective on modern PEM research, identification of knowledge gaps that exist, potential solutions, and novel research that seeks to further our understanding of PEM.

2 | A BRIEF HISTORY OF PEM IN FISH

Researchers have continued to pursue and develop a greater understanding of PEM since Black (1958) postulated that extreme exercise led to a significant disruption in acid–base balance caused by the production of lactic acid as a byproduct of anaerobic glycogen metabolism. Twenty-five years later, Wood et al. (1983) published a paper that aimed to elucidate the mechanism of PEM in rainbow trout (*Oncorhynchus mykiss*, Salmonidae) following bouts of severe exercise. The study postulated that "lactic acid" was an artificial concept since lactate and metabolic protons (H^+_m) appeared at different rates in the blood and suggested that PEM could be a consequence of intracellular acidosis of unknown origin. The authors also presented indirect evidence for the appearance of an unknown anion in the blood of dying fish. These theories are based on the original concept that lactic acid produced by anaerobic glycolysis appears in the blood (Hill et al., 1924) but have now been replaced by a more nuanced perspective.

3 | UPDATED PERSPECTIVES ON HISTORICAL WORK

Research suggests that lactate and metabolic protons move across cell membranes at different rates and with different mechanisms (Wood & Wang, 1999). Research also indicates that the lactate anion is produced via glycolysis while metabolic protons are produced during the breakdown of adenosine triphosphate (ATP) (Robergs et al., 2004). More relevant, perhaps, is the production of metabolic protons produced during ATP catabolism. This could initiate a degenerative cascade of events where fish are challenged to exercise and must use energy to escape. Energy (i.e., ATP) must be acquired and broken down/utilized to facilitate the demands of exercise, therefore increasing the production of metabolic protons. This leads to a decreased intracellular pH and a compromised ability to regulate intracellular ion balance. Furthermore, ATP would be required to regulate ion shifts and achieve homeostasis, yet no ATP would be readily available, leaving fish unable to return to homeostasis and ultimately perish. This warrants further investigation as to the role of metabolic protons and energy utilization in fish that die from severe exhaustion.

In addition to an updated perspective on lactate and metabolic protons, other research groups have explored the intricacies of exhaustive exercise in fish metabolism and bolstered our understanding of the roles of aerobic scope (see Clark et al., 2013, 2017; Farrell et al., 2008), acidosis (Perry & Gilmour, 2006; Wang et al., 1994; Wood & Wang, 1999), muscle energy dynamics (Gleeson, 1996; Weber, 2009), ion transport (Evans et al., 2005; Wood, 1988), catecholamine responses (Hanson et al., 2006; Wood, 1994), and downstream cardiorespiratory effects (Wood & Munger, 1994; Farrell et al., 2009; Prystay et al., 2017). Moreover, in an effort to reduce mortality among exhausted fish (e.g., from fisheries interactions), much effort has been devoted to evaluating potential recovery strategies that can be informative for understanding the potential mechanisms associated with mortality (Brooke et al., 2019; Gingerich et al., 2010; Gleeson, 1996; Raby et al., 2012; Suski et al., 2006; Suski, Cooke, Danylchuk, et al., 2007). When comparing such studies where fish are exercised in the lab (e.g., Wood, 1991) versus exercised in the field (e.g., immediate capture and chase, or using a fishing interaction as a form of exercise), it is important to recognize that laboratory settings create more control over nutrition, environment,

and health such that fish are presumably more similar in starting point and thus response. This includes the use of fish reared in hatcheries, which can have different fitness capacities compared to wild fish (Araki et al., 2008). It must therefore be presumed that data involving fish recently collected from the field should have more inherent variability given the uncertainty in conditions that individual fish have experienced prior to experimentation. These influences must all be given consideration when exploring the mechanistic causes of PEM in hatchery and wild fish populations. Nonetheless, studying the response to exhaustive exercise in a laboratory setting allows researchers to arm themselves with fundamental knowledge that can be applied to the study of wild fishes, particularly their responses to a fisheries interaction.

In recent years, a novel means of exploring mortality resulting from fisheries interactions has emerged through one of North America's most popular sportfish. Black bass (*Micropterus* spp., Centrachidae) are the target species for approximately 80% of fishing tournaments that occur in North America each year (Schramm Jr. et al., 1991). Different angling approaches can be used during tournaments compared to average recreational angling, but it is notable that many fishing tournaments have adopted a catch-and-release strategy for captured fish. Regardless, captured fish are still susceptible to mortality arising from physiological challenges (and injury). Although several tournaments report high survival rates of black bass (Edwards et al., 2004), some competitions have reported an excess of 50% mortality (Neal & Lopez-Clayton, 2001; Wilde et al., 2002). This has led to research exploring the influence of potential interventions intended to reduce mortality in tournament-angled fish. Studies focused on tournament angling for black bass have examined the potential causes of post-release mortality and could provide valuable insight into the mechanistic causes of PEM in fish.

With respect to its relevance to PEM, tournament fish experience additional stressors including confinement in livewells, transportation during that confinement, and the additive stress from handling during the weigh-in process. Tournament fish are also (sometimes) released far from their site of capture. When compounded, these stressors have the potential to increase the severity of physiological responses and prolong the stress of exercised fish (Cooke et al., 2002; Suski et al., 2003; Suski & Philipp, 2004). With respect to mortality, hooking location is the leading source of capture-related injury that results in black bass mortality (Siepker et al., 2007). Though this could represent an important driver of mortality in recreationally angled fish, it has not currently been linked to a fish's ability to recover from an exhaustion event. Perhaps more relevant are the examples of smallmouth bass (*Micropterus dolomieu*, Centrachidae) that require more time to recover if they are angled for longer durations (Schreer et al., 2001), indicating the effect that environmental variables may play on recovery from exhaustive exercise.

Several attempts to reduce tournament fishing mortality in black bass have been studied and include modulating livewell temperatures, introducing additives to alter water chemistry or the addition of supplemental oxygen. However, results vary depending on the method applied to controlling the livewell environment. Recent work has demonstrated that the most effective means of reducing mortality in fish include providing a continuous supply of fresh, aerated water to avoid hypoxia (Suski et al., 2006). Interestingly, the prospect of allowing captured fish to swim at low velocity for the first hour (only) of recovery has also shown promise (discussed more in Section 5) although not for largemouth bass (*Micropterus salmoides*, Centrachidae; Suski, Cooke, & Tufts, 2007), thus emphasizing the contextual nature of such interventions. This work underlines the importance of understanding the interaction between variables associated with the stress of recreational angling in addition to the temporal aspect of delayed mortality, as it is likely these factors that modulate the ability of fish to recover, not the severity of exercise itself. Indeed, several factors have the potential to influence the exercise response (e.g., phenotype, Norin et al., 2016; thermal tolerance, Farrell & Franklin, 2016; sex, Eliason et al., 2020, Hinch et al., 2021); and the response to exercise can vary greatly between individual fish. Nevertheless, we still lack a definitive answer to the question of what causes fish to die after severe exercise.

4 | EXPLORING THE ROLE OF METABOLISM IN PEM

During rest, fish supply working tissues with oxygen to support aerobic energy production (Randall & Brauner, 1991). This energy supply is dedicated to fulfilling the requirements for routine activities where oxygen demand is matched by oxygen delivery, allowing fish to maintain internal homeostatic conditions. Exhaustion occurs when burst swimming requires the use of both red and white muscle, and the supply of oxygen is surpassed by the demand (Burnett et al., 2014; Moyes et al., 1992; Wood et al., 1983). This flux in oxygen demand is sometimes described as metabolic rate: a fluid measure of how much energy a fish is using at a given time and at a given activity level. The measurement of oxygen consumption is used to determine the exercise-induced maximum metabolic rate (MMR), and it should be noted that MMR is highly influenced by environmental factors and species-specific variation (Norin et al., 2016). This maximal oxygen consumption response can be time-dependent, and may not occur until several hours after the exhaustive event (Clark et al., 2013).

As exercise intensity increases, so do the tissue demands for oxygen. Thus, the ability to transport oxygen during these times of increased demand becomes essential to maintaining homeostasis and potentially avoiding PEM. When environmental conditions compromise oxygen delivery, fish may be more susceptible to PEM. Hyndman et al. (2003) showed that 90% (n = 10) of triploid brook trout (*Salvelinus fontinalis*, Salmonidae) exposed to elevated temperatures (19°C) died within a 4 h recovery period following exhaustive exercise. The researchers suggest that although ploidy does not affect muscle pH regulation in brook trout, it is possible that triploid fish are less efficient at delivering oxygen to metabolically active

tissues. Their inability to maintain the high oxygen uptake (MO_2) required at high temperatures could make them more susceptible to PEM during the recovery period when oxygen demand is high. Valuable insight regarding the role of oxygen transport in PEM also came from a study on leopard coral grouper (*Plectropomus leopardus*, Serranidae) in which summer temperatures (30°C) combined with high exercise intensity resulted in elevated post-exercise baseline metabolism and PEM that occurred 3–13 days post-exercise (Clark et al., 2017). A further increase in temperature (to 33°C), combined with high exercise intensity, caused immediate mortality (1.8–14.9 h post-exercise), suggesting a different or at least hastened mechanism of PEM with increased temperature (Clark et al., 2017). It was also observed that mortality was different between low-performing and high-performing individuals, with the latter being more likely to perish from PEM. This study presented clear evidence that certain phenotypes within a population may be more susceptible to PEM and that the interaction between exhaustion and high temperatures represents a lethal threat. Additionally, it highlights the impact of disrupting physiological processes associated with severe exhaustion and underscores the urgency in continuing to investigate the mechanisms of PEM in fish.

Exercise increases the demand for greater oxygen consumption and, subsequently, CO_2 perfusion at the gills. This demand must be met via a compensatory suite of changes including increasing the functional surface area of the gills, reducing the distance for gases to diffuse, increasing blood perfusion, and increasing the rate of ventilation (Randall et al., 1987). These changes facilitate gas exchange, however, they also magnify the potential for osmoregulatory disturbances to occur—that is, "osmorespiratory compromise" (Onukwufor & Wood, 2018; Wood & Eom, 2021). The increased effective permeability of the gills consequently increases the rate of ionic diffusion across the gills and, in turn, branchial energy consumption as compensatory mechanisms attempt to mediate the loss of extracellular and intracellular ions (Evans et al., 1999).

To fully understand the compensatory measures undertaken to overcome oxygen debt (or exercise post-oxygen consumption; Scarabello et al., 1991), consideration must be given to the primary sites of oxygen consumption. This process demands a high amount of cellular energy to restore cellular oxygen stores, high-energy phosphates, metabolites, and ions (Wood, 1991). During extreme exercise, both red and white muscle tissues consume oxygen, albeit for different reasons through disparate mechanisms. Studies have shown that there is a significant difference between red muscle oxygenation and arterial oxygenation during fatigue in rainbow trout (McKenzie et al., 2004), where the partial pressure of arterial blood oxygen (P_aO_2) decreases significantly during fatigue with a relatively small decrease in red muscle ($P_{RM}O_2$). This suggests that oxygen supply to the red muscle is not a limiting factor in the recruitment of white muscle, or a limiting factor during exhaustion. It should be noted that venous oxygen (P_vO_2) remains low following exhaustion, while $P_{RM}O_2$ returns to normal in rainbow trout (McKenzie et al., 2004). This indicates that, during fatigue, venous return from red muscle contributes little oxygen to venous blood and that other metabolically active tissues could account for the bulk of oxygen being consumed following exhaustion (Portz et al., 2006). The increased demand for oxygen could be attributed to other metabolic processes focused on returning the fish to homeostatic conditions following exhaustion, like ion regulation, osmoregulation, glycogen store replenishment, or digestion. Notably, this increased demand for oxygen following exhaustive exercise can compound fish responses to extraneous variables, particularly air exposure, making them more susceptible to PEM. Ferguson and Tufts (1992) demonstrated that fish that were exercised exhaustively and then exposed to the air were much more likely to perish compared to fish that were only exhaustively exercised. This is likely because fish that are exposed to the air are no longer able to perform gas exchange at the gills, limiting their ability to excrete CO_2, decreasing their blood pH, and most importantly, reducing the oxygen content of their blood. Ferguson and Tufts (1992) showed that 60s of air exposure resulted in an 81% reduction in PO_2 and an 87% reduction in the amount of oxygen bound to haemoglobin; a significant difference when compared to fish that only experienced exhaustive exercise. In normal circumstances, fish employ compensatory measures to enhance oxygen delivery and create a favourable environment for haemoglobin to unload oxygen to active tissues. During air exposure, the lack of oxygen during the post-exercise period undoubtedly alters the dynamics of tissue metabolism during the critical recovery period (Cook et al., 2015). Data from the latter study show that only 28% of fish that were exposed to air for 60s following exercise were able to survive for 12 h compared to an 88% survival rate for fish that were only treated with exercise. These animals died between 4 and 12 h after treatment, even though the trajectory of their acid–base status indicated levels were returning to normal. This delayed mortality emphasizes the fact that fish that are released into the wild may not always survive, despite their ability to swim away after a fisheries interaction. However, it is also important to note that fish in this study were cannulated so mortality levels observed in the lab may not be representative of fish in the wild.

Hypoxia may also influence how fish navigate changes in oxygen handling during exhaustion. It is suggested that high-performance swimmers, defined as fishes that can sustain elevated levels of aerobic metabolism (Bernal et al., 2001), have a low tolerance for hypoxia because of ionic diffusion and transport limitations that are linked to haemoglobin's ability to carry and deliver oxygen to metabolically active tissues (Nilsson, 2007). High-performance swimmers capitalize on having haemoglobins with low affinities for oxygen to quickly unload oxygen from their red blood cells (RBCs) at the tissue level (Rummer et al., 2013). This effect can also be achieved without low affinity in normoxic conditions, or through allosteric modulation (Rasmussen et al., 2009). In hypoxia, the partial pressure of oxygen is reduced and thus only haemoglobins with high affinity will be able to bind oxygen. Although these haemoglobins may be able to bind oxygen more readily, their high affinity, coupled with the low partial pressure of oxygen, can make it more difficult to unload the oxygen. Consequently, this makes it more difficult for tissues to receive the oxygen they require (Rummer & Brauner, 2015). Additionally, fish

in hypoxic conditions will have a lower overall capacity for oxygen transport. The limited oxygen supply during hypoxia hinders physiological processes responsible for metabolizing and recycling lactate, while concurrently inhibiting the mobilization of glucose and thus the replenishment of glycogen stores (Gleeson, 1996; Weber et al., 2016; Wood, 1991). Wang et al. (1994) demonstrated that exhaustive exercise elicited a significant increase in intracellular lactate while muscle glycogen reserves were depleted by more than 90%, yet white muscle glycogen concentrations remained low (40%) 2–4 h post-exercise. This suggests that intracellular lactate is more important as a substrate for on-site glycogenesis, and the rate at which lactate can be used as a means of replenishing muscle glycogen stores could play an important part in mitigating the pathological effects of exhaustion and preventing subsequent PEM. Indeed, gaining a greater understanding of the restoration of energy stores could provide valuable insight into the mechanistic causes of PEM in exhausted fish.

The effect of altered oxygen transport on captive fish has received considerable attention, as captive fish provide the opportunity to manipulate environmental variables and examine physiological mechanisms in fine detail (Clark et al., 2013). Comparatively, the study of wild fishes presents unique research challenges, yet wild fish represent an important part of informing those tasked with managing fisheries (Brownscombe, Cooke, et al., 2017; Holder, Jeanson, et al., 2020). Thus, it is important to integrate the fundamental knowledge acquired through the study of captive fish to inform the application of management techniques on wild fish. Pelagic fish depend heavily on aerobic metabolism to swim continuously, and thus locomotion is considered to be the dominant physiological process that requires a constant supply of oxygen (Clark et al., 2013). Having said that, the process of specific dynamic action, which represents the energy expended in the digestion, absorption, and assimilation of food (Secor, 2009), can contribute a substantial proportion of the daily energy budget in fish (Clark, Brandt, et al., 2010). Comparatively, fish that exhibit a lower dependency on aerobic metabolism may have less available capacity to fulfill the demands of physiological processes that require oxygen (Fu et al., 2009), potentially interacting with PEM.

5 | POTENTIAL LINKS BETWEEN EXHAUSTION, ION REGULATION, OSMOREGULATION, AND PEM

Exhaustive exercise depletes endogenous energy reserves, especially substrates which generate ATP anaerobically (e.g., free ATP, phosphocreatine [PCr], and glycogen), threatening long-term survival post-exercise. The demand for energy is proportional to the intensity of exercise associated with behaviours like foraging, escaping predation, pursuing migration, or engaging in reproduction (Moves & West, 1995). As exercise intensity increases, so does the oxygen consumption associated with exertion (Lee et al., 2003; McKenzie et al., 2004). Exercise can also cause shifts in fluid compartments, pH, changes in intra- and extra-cellular ion concentrations, and increases in the relative concentration of metabolic by-products (Wang et al., 1994; Wood, 1991). Relatedly, fish must also expend energy to return to homeostasis that has shifted because of increased activity levels. In response to these changes, fish utilize fast-acting signalling hormones, catecholamines, to rapidly effect change in energetic demands by mobilizing and transporting energy stores to metabolically active tissues that require fuel to continue functioning (Randall & Taylor, 1991; Reid et al., 1998). Catecholamines are mobilized prior to exhaustion (~80%–90% of critical swimming speed) and play a key role in modulating muscle activity, oxygen transport, and managing acid–base disturbances (Vermette & Perry, 1987, 1988). Examples of their capability to quickly modulate cellular function include mobilization of glucose from stores in the liver, increasing arterial blood pressure, and increasing red blood cell affinity for oxygen (Perry & Bernier, 1999; Polakof et al., 2012).

Catecholamines also have a significant influence on osmotic regulation and represent a key signalling mechanism to control ionic balance (Vermette & Perry, 1987; Marshall, 2007). These signalling hormones are responsible for increasing the rate of ventilation, increasing branchial blood flow, increasing gill surface area for diffusion, decreasing diffusion distances, and increasing ion permeability (Sardella & Brauner, 2007). Although this facilitates increased oxygen uptake, it is done at the cost of compromising the ability to control passive ion flux (i.e., ionic loss or uptake). There are also species-specific responses to osmoregulatory compromise, with some being more resilient to compromise compared to others. Gonzalez and McDonald (1994) found that fish with higher average activity levels had relatively greater increases in oxygen consumption and lower ion loss compared to fish with low average activity levels (i.e., low-activity fish lost more sodium per mole of oxygen absorbed compared to high activity fish). Gonzalez and Mcdonald (1994) postulated that high-activity fish were able to resist changes in the ion gas ratio because they exhibited a higher degree of regulation of their tight junctions: a means of establishing connections between a variety of cells of the gill. Gonzalez and Mcdonald (1994) theorized that fish exhibiting a higher activity level could resist ionic loss with increased oxygen demand because of the ability of their tight junctions to effect change.

Studies investigating physiological disturbances associated with recovery from exhaustion have shown increased levels of plasma sodium and mortality in fish treated with exercise and air exposure (Suski, Cooke, Danylchuk, et al., 2007), with the likelihood of mortality increasing as the duration of air exposure increases (Davis & Parker, 2004). However, it should be noted that sodium loss was not measured in these studies. It is well documented that ionic loss, particularly a 30% decrease in whole body sodium, results in mortality in rainbow trout (Laurén & McDonald, 1985; Wood, 2001) and is attributed to exposure to low pH or metals such as copper and silver. Although some degree of ion loss has been associated with exhaustive exercise (Wood, 1991), it is unlikely that the magnitude of that loss would meet the 30% threshold determined by previous studies. Increasing activity levels, or training, could bolster

the ability to resist large ion losses and deliver oxygen to working tissues, representing a potential protective mechanism. Improving signal transduction via tight junction function could allow fish to respond rapidly to large changes in homeostatic conditions, thus it is an important consideration when exploring ways fish resist ion loss.

The effect that environmental conditions have on modulating responses to exercise must also be considered in PEM. Kieffer et al. (2002) showed that mortality in Atlantic salmon (*Salmo salar*, Salmonidae) was related to water hardness. Softer water fish had a lower initial HCO_3 concentration, significant elevation in plasma Na^+ and Cl^-, and an increase in hematocrit following exercise indicating osmoregulatory compromise. Fish that were acclimated to softer water were also more likely to perish after a bout of exhaustive exercise when compared to their counterparts acclimated to hard water. These results suggest that salmon in softer water did not survive because they were unable to correct the exercise-induced acidosis and return to homeostasis. Perhaps this evidence discounts the existence of a "silver bullet" of PEM, yet determining the impact of ion loss associated with exhaustion remains an important part of understanding PEM in fish.

Stressors that perturb energy supply-and-demand dynamics have the potential to initiate stimulus-dependent signalling cascades intended to mobilize energy stores. The endocrine-mediated release of catecholamines into the circulation is a tightly regulated process that must ultimately satisfy the demands of metabolically active tissues (Perry & Bernier, 1999; Reid et al., 1998). These hormones can include adrenaline and noradrenaline, which act on both alpha- and beta-adrenergic receptors of cells resulting in altered ion dynamics and changes in osmoregulation during periods of acute stress. In cases where oxygen uptake must be increased, such hormonal influences can manifest as increases in RBC sodium-hydrogen exchange transport, increases in gill permeability, and increased diffusion capacity (Evans, 2002; Fabbri & Moon, 2016). In teleost fish, catecholamine secretion is stimulated via acetylcholine release at the terminal end of nerve fibres of chromaffin cells located in the posterior cardinal vein and the head kidney (Nilsson et al., 1976; Porteus et al., 2012). Hypoxia (Porteus et al., 2012), hypotension (Perry et al., 2011; Sandblom et al., 2010), air exposure (Lennox et al., 2016; Sopinka et al., 2016), metabolic acidosis (Wood, 1991, 1994), and changes in water temperature (Currie et al., 2013) can lead to increased levels of circulating catecholamines. While sustained aerobic exercise is not correlated with changes in circulating catecholamine levels (Butler et al., 1986), increases have been observed following burst swimming exercise of fish (Primmett et al., 1986). It should be noted that significant secretion only occurs with severe stress. The energetic cost of locomotion is proportional to exercise activity and is affected by aerobic and anaerobic energy production (Bennett, 1991). This ultimately affects swimming performance, where evidence suggests that fish experiencing exhaustive events can be impaired for 6 h post-exercise (Lee-Jenkins et al., 2007). Although this information begins to hint at potential mechanisms for relatively immediate PEM, detailed studies examining the connection between dysfunctional catecholamine signalling and death in teleost fishes remain largely unexplored. Additionally, the discipline would also benefit from a rigorous examination of the mechanisms responsible for delayed PEM, specifically the relationships shared between PEM and the stress caused by exhaustion, injury, or disease.

Cortisol has classically been measured as a metric of stress in fish. When fish experience a significant perturbation in homeostasis or perceive a threat stimulus in the environment (e.g., predator interaction and increased swimming demand), cortisol is produced and secreted by the hypothalamic–pituitary–interrenal (HPI) axis and typically increases in the blood following a diversity of stressors (reviewed in Wendelaar Bonga, 1997; Barton, 2002) and challenges including recovery from exhaustive exercise (see De Boeck et al., 2001; Milligan, 1997). The main target tissues of cortisol following a stressor include the liver and skeletal muscle, as these are areas where cellular energy turnover is high during and after bouts of exercise (Gingerich et al., 2010; Milligan, 2003). Overall, the primary function of cortisol is to facilitate the mobilization and use of energy substrates and restore the hydromineral balance following the stress response (Chasiotis & Kelly, 2012) while also diverting energetic resources away from non-essential functions (e.g., growth and reproduction; see Schreck & Tort, 2016). Notably, cortisol is not necessarily the primary glucocorticoid across all teleost fish and is different for elasmobranchs (subclass; Elasmobranchii), lamprey (Petromyzonidae), and hagfish (Myxinidae).

While chronically elevated cortisol has not demonstrated significant effects on aerobic swimming performance in fish (Butler et al., 1986; Gregory & Wood, 1999), it has been correlated to an increase in standard metabolic rate (SMR) (Lawrence et al., 2019) in addition to other negative effects on feeding performance and growth (Gregory & Wood, 1999). In some instances, elevated cortisol has also been associated with the inhibition of glycogen synthesis in skeletal muscle (Milligan, 2003), implying that the recovery process is impeded as glycogen stores are not replenished at their optimal rate, though cortisol mechanisms of action in fish are highly dynamic (Mommsen et al., 1999). Interestingly, Carbonara et al. (2010) found that fish with elevated cortisol showed a significant reduction in their ability to physiologically recover from exhaustion, though the implications for the potential for impairment should be explored further. This has led to a significant examination of the role of cortisol in metabolic performance. Evidence suggests that elevation in cortisol post-exercise is correlated to the inhibition of glycogenesis, where (net positive) glycogen synthesis does not occur until plasma cortisol levels decline (Pagnotta et al., 1994). Furthermore, pharmacological inhibition of cortisol synthesis and/or release (Eros & Milligan, 1996; Pagnotta et al., 1994) increases the rate of post-exercise muscle glycogen resynthesis. In addition to pharmacological intervention, low-intensity post-exercise swimming has also shown beneficial effects on muscle glycogen resynthesis. Milligan et al. (2000) reported that swimming slowly after exhaustive exercise significantly reduced the typical post-exercise increase in circulating cortisol in rainbow trout. These rainbow trout also showed an increased rate of glycogen resynthesis and lactate clearance

compared to their counterparts held in still-water post-exercise. Additionally, Farrell et al. (2001) also tested this by placing captured fish in a recovery box designed to force fish to sustain a low grade of swimming and found that it promoted partial metabolic recovery and recovery of swimming performance. This also suggests that periods of inactivity after exhaustive exercise may prolong recovery time in fish, decreasing their capacity to perform anaerobic activity during recovery and making them more susceptible to PEM. While this finding is not universal across all teleost fish (see Kieffer et al., 2011; Suski, Cooke, & Tufts, 2007), such evidence underlines the relevance of post-exercise swimming performance to the recovery profile of fish, where low-intensity post-exercise swimming could represent a novel means of enhancing recovery and reducing PEM.

Researchers have attempted to investigate the influence of metabolic distress on post-exercise recovery in varying capacities, with the recent focus shifting towards potential indicators of physiological distress in the blood profile of angled fish. This work often involves quantifying changes in blood chemistry using simulated and real angling events to collect blood samples. In blue sharks (*Prionace glauca*, Carcharhinidae), it was noted that moribund animals (animals that eventually succumbed to the negative effects of angling and died) showed significant increases in five variables compared to their surviving counterparts: magnesium, lactate, potassium, calcium, and Hsp70 mRNA (Moyes et al., 2006). The increased circulating concentrations of these variables are indicative of strenuous muscular activity and are potentially linked to tissue damage, though the extent and location of that damage were not examined in the study. The potential tissue damage is similar to a phenomenon described as "capture myopathy" or "exertional myopathy" described in birds and mammals by Spraker (1993), where captured animals experience damage to muscle tissue as a result of capture.

In recent decades, inorganic ions have been implicated as important indicators of stress, particularly in elasmobranchs. Several studies have observed marked increases in plasma potassium concentrations following the induction of stress (Brooks et al., 2012; Mandelman & Farrington, 2007; Manire et al., 2001; Moyes et al., 2006), indicating a potential loss of potassium ions from intracellular compartments. The mechanism of increased circulating potassium has not been clearly defined, though some researchers identify concurrent increased circulating potassium and intracellular acidosis in sharks that perish after being released (Whitney et al., 2021). Additionally, it is also suspected that hyperkalemia plays a role in pathological cardiac function during stress in elasmobranchs, suggesting a potential connection between potassium concentrations and dysfunctional myocardial function (Skomal & Mandelman, 2012). Consideration must also be given to the concentration of other circulating ions, as increases in stress in elasmobranchs have also been linked to increases in circulating concentrations of magnesium, calcium, and sodium ions (Cliff & Thurman, 1984), though the mechanisms surrounding this phenomenon have not been defined. This begs the question: are these variables linked to exhaustion and PEM? Can we use them as metrics to gauge when we consider a fish "exhausted" and use that metric to determine the likelihood of mortality? Much remains to be discovered with respect to the roles that ion regulation and osmoregulation play in PEM.

6 | EFFECTS OF EXHAUSTION ON CARDIAC FUNCTION

Cardiac tissue oxygen demand varies greatly between species (Farrell, 1991), making it difficult to define the overall oxygen demand of cardiac myocytes in fish. As the intensity of exercise increases, so does the myocardial demand for oxygen (Farrell et al., 2009). To facilitate this increased demand, an increase in overall cardiovascular activity is observed via changes in cardiac output. This effect can be quantified by the Fick equation (Fick, 1870):

$$\left[O_2 \text{ consumption} = CO^{*}\left(C_aO_2 - C_vO_2\right)\right].$$

where oxygen consumption is dictated by a factor of cardiac output (CO) and arteriovenous oxygen content difference (C_aO_2 - C_vO_2). Importantly, oxygen demand is also dictated by the amount of oxygen uptake at a tissue level, where increases are inversely proportional to arterial blood oxygen content. Thus, fish must increase cardiac output to maintain the same oxygen delivery with an increased oxygen demand (Farrell, 2007). This increase is achieved by elevating heart rate, elevating stroke volume, or by mobilizing RBCs from the spleen and modulating their ability to load/unload oxygen. The ability to affect such changes in cardiac output differs between species and can also be dictated by heart morphology. Classically, there are four types of fish hearts (I, II, III, and IV) that have different proportions of spongy and compact myocardium (Davie & Farrell, 1991). These classifications are loosely based on activity level and are correlated to the proportion of compact myocardium (i.e., Type I has no compact myocardium and is commonly found in fish that exhibit low activity levels versus Type IV which has the highest proportion of myocardium and is usually found in high-performance fish). However, it should be noted that there is no classification for ventricular mass relative to whole-heart mass, and there is substantial variation among species because the activity levels/demand for oxygen varies so greatly (Brill & Bushnell, 1991; Icardo, 2012). As such, it is difficult to determine how heart morphology dictates changes in cardiac output, and in turn, the overall responses of different fish species to exercise. Some studies have examined the influence of hypoxia (Gamperl et al., 2020), disease (Powell et al., 2002), and environmental change (Incardona et al., 2004), yet it remains difficult to find a definitive link between exercise, cardiac morphology, and mortality in fish. Future research should focus on the discovery of variables that contribute to the exacerbation of pathological cardiac function during exercise that can lead to heart failure and/or PEM.

Parasympathetic tone serves to lower the intrinsic heart rate via acetylcholine signalling (Axelsson et al., 1987). This effect may be more useful when fish must quickly elevate heart rate because

removing the negative chronotropic effect caused by cholinergic signalling may be more efficient than initiating an alternate signalling cascade. Sympathetic tone serves to increase heart rate in times of increased physical demand as hormonal influence is dictated by circulating catecholamines (Axelsson et al., 1987; Sandblom & Axelsson, 2011). The most influential of these is likely those that act on beta-adrenergic receptors of cardiac myocytes. It should be noted that the increase in circulating catecholamines has only been correlated with maximal swimming or instances where fish must exhibit burst swimming (Kieffer, 2000; Randall et al., 1987). This means that the increase in heart rate before maximum swimming speed or burst swimming is likely attributed to the removal of parasympathetic tone (i.e., cholinergic signalling) from the heart.

Responses to increased swimming demand are also heavily influenced by positive ionotropic agents, specifically hormones that lead to an increase in contractility. These hormones are released into the bloodstream during exercise and marked increases in circulating hormones are observed when fish are stressed (Kieffer, 2000). When studying post-exercise blood profiles, it becomes evident that significant changes to circulating hormones have an impact on cardiac myocyte function. Studies have shown that fish experience a state of combined venous acidemia, hyperkalemia (Danylchuk et al., 2014), and hypoxemia commonly after exhaustive exercise (Holk & Lykkeboe, 1998; Milligan, 1996; Milligan et al., 2000; Domenici et al., 2013). Additionally, teleost fish rely on passive diffusion to oxygenate cardiac tissue as venous blood comes into contact with their myocardium (Davie & Farrell, 1991), thus the heart tissue itself is extremely susceptible to influence from these ionotropic agents. This can severely impact cardiac performance and have negative consequences for fish experiencing stress. Interestingly, Wood et al. (1983) measured the heart rate and arterial blood pressure of one dying fish and found that there was no evidence of cardiac failure and that a strong heartbeat and normal aortic blood pressure were observed for at least 15 min following the cessation of ventilation. Although this observation contradicts the idea that heart failure could be responsible for PEM in fish, it would be beneficial to continue exploring links between cardiac function and PEM in more species and with higher sample sizes.

In addition to the oxygen supplied to the heart via passive diffusion, some high-performance fish have a dedicated cardiac supply of oxygenated blood via a coronary artery (Davie & Farrell, 1991). This coronary circulation provides a consistent supply of oxygenated blood to the compact myocardium of fish with high oxygen demands (Clark & Seymour, 2006; Davie & Farrell, 1991; Farrell, 1991). Some fish benefit from this "dual" supply of oxygen; however, coronary supply is also subject to changes in flow and efficiency as demand on the cardiovascular system changes with stress (Farrell, 1991). Increases in coronary blood flow led to a significant decrease in coronary vascular resistance, although the mechanistic cause of this change is difficult to discern. This could be a result of shear stress initiating an endothelium-dependent adrenergic signalling cascade (Niebauer & Cooke, 1996). This is likely an effect caused by a cascade of signalling hormones intended to decrease peripheral resistance and deliver more oxygen to working tissues, including cardiac myocytes. Recovery from this signalling cascade may be challenging, as hormone signalling is commonly associated with receptor-mediated channel operation and is an energy-taxing process. It is likely that the cellular cost of increasing coronary blood flow creates energetic deficits by creating a larger demand for receptor-mediated signalling. It is also likely that these deficits contribute significantly to exhaustion and shift fish further away from their homeostatic conditions, thus increasing their susceptibility to PEM. Nonetheless, this is speculative and requires additional research to elucidate the links between cardiac function and mortality in exhausted fish.

There are also several cardiac hallmarks that are commonly observed during the recovery from burst exercise, mainly an increase in cardiac output and an increase in heart rate. Most significantly, fish must recover from the acid load created by exhaustive exercise/burst swimming. In cardiac myocytes, metabolic acidosis is a major cause of the decrease in extracellular pH (Wood & Perry, 1985). The efflux of protons changes concentration gradients (Randall & Brauner, 1991), potentially preventing receptor-mediated calcium channels from functioning properly. This dysfunctional calcium handling potentially leads to a calcium deficit that inhibits the rate of excitation-contraction coupling, and ultimately, cardiac dysfunction. This could have negative chronotropic and ionotropic effects, leading to a decreased force of contraction and a decrease in resting cardiac output. This becomes most evident at higher cardiac workloads. Some work with perfusion preparations has indicated that treatment with adrenergic receptor agonists on heart tissue has protective effects from the negative chronotropic effects seen at higher cardiac workloads (Hanson et al., 2006). This work is promising and could be useful if the principles of increased cardiac demands are applied within the context of exhaustive exercise. Although no current link to PEM exists, more remains to be discovered.

7 | EMERGING TECHNOLOGIES—TOOLS FOR STUDYING EXHAUSTION AND KEY FINDINGS

Since the seminal work of Black (1958) and Wood et al. (1983), our capacity to understand PEM has been bolstered by technological advances in field data collection. Significant progress in field physiology and behaviour has also fostered the integration of physiological measurements into field applications, enhancing our ability to identify the mechanisms involved in PEM.

One of the most practical means of evaluating exhaustion is monitoring oxygen uptake and consumption in fish using respirometry (Clark et al., 2013; Roche et al., 2013). In this process, fish are placed in a sealed chamber where researchers can measure changes in oxygen uptake (MO_2) in real-time (Clark et al., 2013). Notably, measurements of MO_2 may provide a holistic measure of the overall physiological status of fish, given the requirement for oxygen to support the ATP used in basically all physiological processes (including recovery from anaerobic exercise). This method of measuring

metabolism in fish has already provided valuable insight into potential mechanisms of PEM (Mochnacz et al., 2017; Clark et al., 2017; see Section 4, paragraph 2). Researchers have full control over experimental conditions and can test the effects of temperature, hypoxia, sensory cues, and many more variables to observe their effect on exhaustion and recovery (Clark et al., 2013, 2017). Respirometer units have become more portable over the years, granting researchers the opportunity to bring their labs to the field (Byrnes et al., 2020; Farrell et al., 2003). With knowledge of resting and maximal oxygen uptake rates in fish, it becomes possible to quantify the magnitude of exhaustion in the context of their aerobic metabolic scope (Clark et al., 2017; Cooke et al., 2014).

The ability to evaluate the physiological condition of fish upon release is paramount in understanding the stressors that compromise their fitness, yet accurately measuring changes in blood profiles can present significant challenges, especially when collecting data in the field (Costa & Sinervo, 2004; Stoot et al., 2014). Discerning the aspects of exhaustion that elicit changes in the physiological profile after extreme exercise is difficult. In addition to interspecies variability, several factors influence changes in the metabolite profile during the collection and handling processes (Clark et al., 2011). Although this impact can be minimized in a controlled laboratory setting, the difficulty is magnified during data collection in the field where collecting physiological measurements presents several challenges. For researchers to acquire blood, fish must be handled: a process that inherently changes the blood metabolite profile (Stoot et al., 2014). Confinement stress (Kiilerich et al., 2018), handling time (Sopinka et al., 2016), and technical ability (Lawrence et al., 2020) represent a small portion of factors that must be managed to minimize the stress fish experience during data collection. Although classical metrics for gauging post-release survival have relied on measuring changes in blood metabolite profiles, these results are conflicting. Our hope is that technology continues to evolve, and that researchers will eventually be able to collect reliable, robust data using point-of-care devices in the field or even being able to quantify metabolites remotely in free-swimming fish. Moving forward, field physiology would benefit from a shift in perspective away from the on-site measurement of blood metabolites towards more rigorous, non-invasive experimentation focused on diagnosing the causes of PEM. In particular, the comparison of ion, acid–base, ammonia, and stress hormone fluxes in the water (Ellis et al., 2004; Wood, 1992) of surviving versus dying fish would help elucidate the potential mechanisms of PEM and add immense value to the discipline. This could also provide insight into behavioural changes observed in surviving versus dying fish as new methods of quantifying behaviour in fish continue to emerge.

One such method is the use of tri-axial accelerometry which has presented itself as a novel means of measuring post-release swimming activity and the survival of fish (Brownscombe, Gutowsky et al., 2014; Brownscombe, Marchand et al., 2014; Wilson et al., 2006). Recent research efforts have focused on applying tri-axial accelerometer technology to quantify energetics and swimming performance in fish (Brownscombe, Gutowsky et al., 2014; Brownscombe, Marchand et al., 2014; Holder, Griffin, et al., 2020; Lennox et al., 2018). This has allowed researchers to quantify behavioural states and estimate energy expenditures in fish, where accelerometer metrics have been linked to cardiac function, spawning behaviour, and feeding/foraging patterns (Brownscombe, Gutowsky, et al., 2014). Depending on the sampling frequency used, researchers can measure broader activity patterns (low frequency) or more detailed behaviours (high frequency). Using high-frequency sampling to measure short-duration post-release swimming performance, researchers can correlate how predictors like fight duration and temperature affect the tail beat frequency and tail beat magnitude in exhausted fish (Brownscombe, Gutowsky, et al., 2014). The accuracy of this assessment is magnified when the post-release swimming performance assessment is combined with other impairment testing methods. For example, using accelerometer metrics in concert with reflex action mortality predictor (RAMP) testing provides a unique means of quantifying impairment and predicting the likelihood of PEM (Davis, 2010; Brownscombe, Gutowsky, et al., 2014). The RAMP protocol is a tool that allows anglers to rapidly assess the condition of captured fish by testing for the presence or absence of five key reflexes correlated with fish survival (Davis, 2010). It is an easy, applicable method for estimating mortality in fish and has great potential to elucidate the causes of PEM when paired with fine-scale measurements of post-release swimming performance. The research focused on the correlations between post-release swimming performance and PEM also has the potential to provide valuable information to managers tasked with protecting fish populations, especially those that are more susceptible to post-release mortality or post-release predation (e.g., Holder, Griffin, et al., 2020; Raby et al., 2014). Lennox et al. (2018) provide a strong context for the application of accelerometry in current fisheries research. When examining the post-release swimming performance of angled arapaima (*Arapaima gigas*, Osteoglossidae), they observed 3 post-release mortality events (of 27 total individuals tagged and measured) where fish could not be revived and ultimately perished. In this case, researchers suspected moribund fish were experiencing increased vulnerability to seasonal environmental changes, paired with the potential loss of energetic reserves from spawning and/or migration. Although the sample size was too small to detect significance regarding the main effects on mortality, it underlines the need for continued exploration into the mechanistic causes of PEM and the importance of understanding how those mechanisms relate to behaviour in a broader ecological context.

Bioenergetics information can be used to track and assess a variety of physiological functions including growth, metabolism, energy management, and response to predation. Researchers have been able to estimate energy use and the physiological consequences of stress using in-body biologgers to measure changes in temperature and heart rate (Clark, Sandblom, et al., 2010; Cooke et al., 2016; Donaldson et al., 2010; Prystay et al., 2017). The use of these biologgers provides specific information regarding how changes in heart rate and temperature correlate with increases in the physiological disturbance. The measurement of heart rate allows researchers to examine the cardiovascular scope (min and max), quantify levels of

stress, and define the duration of time for heart rate to return to normal (i.e., recovery). These loggers also grant researchers the ability to observe the behaviour and swimming performance of captured fish following catch-and-release angling events, and how factors like exhaustion and air exposure modulate heart rate and cardiovascular performance. These loggers, in concert with physiological measurements, aid in refining our understanding of the consequences of exhaustion and other human-induced stressors that fish experience. Although causes of PEM have not been directly assessed using internal biologgers, this technology represents an enormous amount of potential where researchers could use these measurements to correlate variables of exhaustion with fish mortality.

8 | FUTURE DIRECTIONS AND CONCLUSIONS

Since 1958 (Black, 1958), we have discovered more potential factors that may contribute to PEM in fish, yet the PEM "silver bullet" remains elusive. Research has come a long way in elucidating why fish experience impairment following an exhaustive event, though much remains to be discovered regarding PEM (Figure 2). Furthermore, an exploration into the consequences of exhaustion reveals our relative lack of knowledge regarding the ways in which fish can effectively recover from extreme exercise. Perhaps, the most significant consideration is the potential that lies within the employment of emerging technologies to aid in our search for the "silver bullet" of impairment and mortality in fish. Future studies that attempt to find the causes of PEM would benefit from the employment of non-invasive measures that focus on determining whether fish will live or die following exposure to severe exercise. This will further our understanding of energy balance and the role that energy utilization plays in modulating the physiological ability of fish to maintain internal homeostasis. This also opens avenues to explore the role of ion regulation in PEM, specifically acidosis resulting in tissue damage at a muscular level. Moving forward, significant consideration must also be given to the influence and/or interaction between exhaustion and anthropogenic changes in environmental factors like temperature and acidification.

As our knowledge of exhaustion in fish continues to grow, so do our strategies for determining the best ways to measure and observe these changes. What factors contribute to the fragility or resilience of a species to PEM? How do ecophysiological factors affect responses to exhaustion? What influence do these factors have on the ability of fish to recover from exhaustion? Our examination of PEM has given us valuable insight into the answer to some of these questions, yet more remains to be studied. The existence of one key mechanism responsible for explaining such mortality is unlikely. Thus, it may be more appropriate to study individual fish that

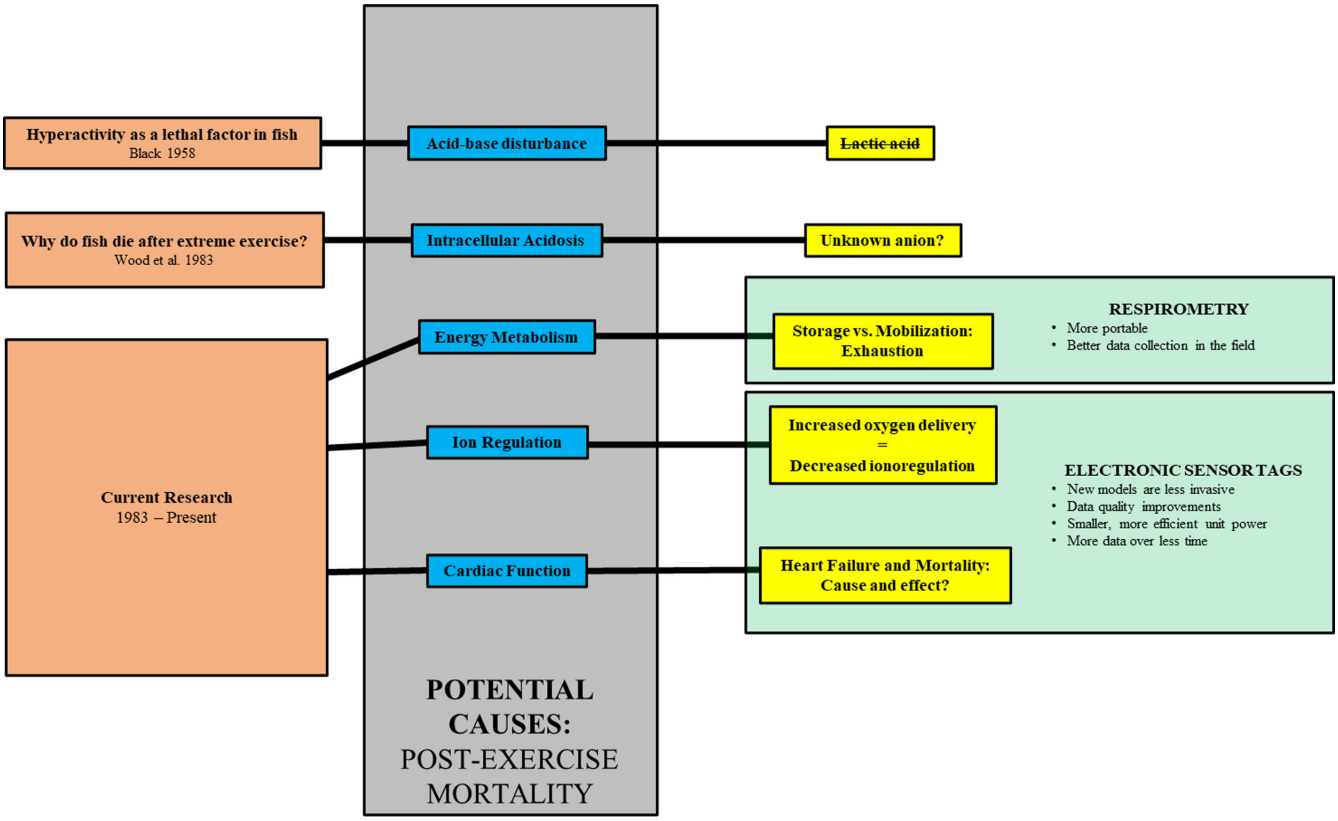

FIGURE 2 A conceptual diagram of themes relevant to the progression of post-exercise mortality (PEM) research in fish. Orange represents work responsible for the inception of PEM research. Blue represents a significant mechanism connected to PEM research (via solid line). Yellow represents key concepts linked to PEM. Green represents technological developments and improvements that have been instrumental in augmenting physiological research related to exercise and exhaustion in fish.

are exposed to a combination of specific variables associated with a fisheries interaction and subsequently experience PEM. Nearly 40 years after Wood et al. (1983) considered why fish die after severe exercise, the number of questions that remain is remarkable. Nonetheless, we are confident that given our evolving understanding and rapidly expanding toolbox, these questions will not require another 40 years to resolve.

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DATA AVAILABILITY STATEMENT

Data sharing is not applicable to this article as no new data were created or analysed for the purposes of this review.

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