Received: 2 May 2023 | Revised: 20 March 2024 | Accepted: 25 March 2024

DOI: 10.1111/raq.12915

REVIEW

REVIEWS IN Aquaculture

Towards sustainable water disinfection with peracetic acid in aquaculture: A review

Dibo Liu[1] | David L. Straus[2] | Lars-Flemming Pedersen[3] | Christopher Good[4] | Carlo C. Lazado[5] | Thomas Meinelt[1]

[1]Department of Ecophysiology and Aquaculture, Leibniz-Institute of Freshwater Ecology and Inland Fisheries, Berlin, Germany

[2]US Department of Agriculture, Agricultural Research Service, Harry K. Dupree-Stuttgart National Aquaculture Research Center, Stuttgart, Arkansas, USA

[3]Technical University of Denmark, DTU-Aqua, National Institute of Aquatic Resources, Section for Aquaculture, North Sea Research Center, Hirtshals, Denmark

[4]The Conservation Fund's Freshwater Institute, Shepherdstown, West Virginia, USA

[5]Nofima, The Norwegian Institute of Food, Fisheries & Aquaculture Research, Ås, Norway

Correspondence
Dibo Liu, Department of Ecophysiology and Aquaculture, Leibniz-Institute of Freshwater Ecology and Inland Fisheries, Müggelseedamm 301, 12587 Berlin, Germany.
Email: dibo.liu@outlook.com

Present address
Dibo Liu, Nofima, The Norwegian Institute of Food, Fisheries & Aquaculture Research, Sunndalsøra, Norway.

Funding information
Fiskeri – og havbruksnæringens forskningsfond, Grant/Award Numbers: 901472, 901826; Norges Forskningsråd, Grant/Award Number: 237856; Landwirtschaftliche Rentenbank, Grant/Award Number: 887139; US Department of Agriculture—Agricultural Research Service, Grant/Award Number: 59-8082-0-001

Abstract

Peracetic acid (PAA) has a long history as an efficacious and eco-friendly disinfectant. It was first synthesised in 1902, and since then a wide range of applications has been developed in various industries. Aquaculture is a more recent industry wherein the potential of PAA is significant. As the global demand for sustainable development increases, there has likewise been growing interest in using PAA in aquaculture as an alternative to less environmentally friendly practices. PAA has no carcinogenic risk to humans (unlike formalin), has negligible harmful by-products (unlike chlorine-based disinfectants) and with appropriate precautions, the risks of causing severe human health damage is easier to control than ozone. Fish show strong physiological recovery and adaptation to PAA, whereas susceptible life stages of pathogens are highly vulnerable, enabling a safe and efficacious disinfection of the entire culture water and not the flow-restricted disinfection by such processes as ultraviolet radiation or ozone. The effective concentration of PAA against many fish pathogens is usually below 2 mg L^{-1}, which is tolerable for most fish, and it has very low environmental risk due to rapid degradation. However, such degradation and the hydrodynamics in production-scale aquaculture systems complicate the practical use of PAA. In this review, we summarise key results of safe concentrations of PAA and its effectiveness specifically for fish farmers. We also outline major difficulties and possible solutions for practical uses of PAA. We intend to bring global attention to this compound and inspire future possibilities for its sustainable use as a water disinfectant in aquaculture.

KEYWORDS
aquaculture disinfectant, fish health management, fish pathogen, fish physiology, redox potential

1 | INTRODUCTION

The chemical nomenclature of peracetic acid (PAA) as standardised by the International Union of Pure and Applied Chemistry (IUPAC) is ethaneperoxoic acid,[1] but it is typically known as PAA or sometimes peroxyacetic acid. Other names include acetic peroxide, acetyl hydroperoxide and peroxyethanoic acid. The publication of Schreiner is a great source of information for the earliest history of the chemical

synthesis of PAA, although it was written in German and not disseminated widely.[2] He reported that Freer and Novy first synthesised PAA by hydrolysing benzoyl acetyl peroxide.[3] Almost a half century later, the sulphuric acid-catalysed reaction between hydrogen peroxide and acetic acid was developed to synthesise concentrated PAA,[4] and this method is still in use.

$$H_2O_2 + CH_3COOH \overset{H_2SO_4}{\leftrightarrow} CH_3COOOH + H_2O.$$

Commercial PAA products are mixtures containing acetic acid (CH_3COOH), hydrogen peroxide (H_2O_2), PAA (CH_3COOOH) and water (H_2O); PAA is rarely obtained as a pure substance. The equilibrium and product stability are maintained by stabilisers such as dipicolinic acid, phosphonates,[5] pyrophosphates, polymerised phosphoric acid or sodium stannate in <0.1% (manufacturer's personal communication). Users of PAA products should keep these additional components in mind when considering specific PAA applications. More importantly, users of PAA products should pay attention to the safety datasheets and be aware of the risks, personal protection and first aid measures while handling. None of the components in PAA products is known to have carcinogenic risks (unlike formaldehyde[6]) through airborne exposure[7] or otherwise. Inhaling PAA vapours may cause discomfort (short exposure) and mild irritation (long exposure) of the respiratory tract[8]; however, the strong pungent odour naturally deters prolonged exposure. In comparison, the odour of ozone is much less recognisable through smell alone, and occupational exposure to it may result in lung and central nervous system damage.[9,10] Although PAA and H_2O_2 are both considered as environmental friendly water disinfectants,[11] the latter needs a higher concentration (15 mg L^{-1}) to achieve a similar prophylactic efficacy by 0.2 and 0.5 mg L^{-1} PAA in aquaculture settings.[12]

There are several research reviews in the literature about various aspects of PAA. A review by European Centre for Ecotoxicology and Toxicology of Chemicals provides comprehensive information addressing the environmental fate and toxicology of PAA to microorganisms, animals and humans.[5] Although most fish toxicity values in this review were from unpublished reports that were not peer-reviewed, they provided an approximate range for some preliminary investigations of peer-reviewed studies. The in-depth reviews by Kitis[13] and Luukkonen and Pehkonen[14] are cited for readers to acquire detailed knowledge about wastewater treatment with PAA, including the mode of antimicrobial action and the advantages or disadvantages to using other disinfectants. In a sense, PAA use for wastewater treatment is similar to recent approaches in aquaculture.

The first published use of PAA in aquaculture was surface disinfection rather than water disinfection. The Fish Disease book of Schäperclaus[15] suggested a 15-min treatment with a 1% dilution of a 40% PAA product (i.e., Wofasteril) with the addition of 1% Graham's salt (as protection against corrosion at ≤5°C) for the disinfection of equipment, facility surfaces and fish eggs. He emphasised that 'the scope of disinfection includes fish parasites, bacteria, fungi, viruses but not helminth eggs'. Researchers subsequently demonstrated equivalent effectiveness by applying PAA directly in water at much lower concentrations without control of contact time and in the presence of cultured animals.

Since 2014, the use of PAA has been approved in the European Union (EU) for organic aquaculture (Regulation [EU] No. 1358/2014, which amended the Regulation [European Commission, EC] No. 889/2008) in the presence of aquaculture animals. Although the Regulation (EU) No. 1358/2014 was repealed by Regulation (EU) 2021/1165 from 2022, the transitional provisions in Article 12(1) allow the use of some cleaning and disinfection products approved by Regulation (EC) No. 889/2008, including PAA, until the end of 2023 when further EU regulation is expected. A recent survey revealed that PAA has become the most commonly used surface disinfectant in Norwegian aquaculture industry.[16] In the United States, the Environmental Protection Agency (EPA) approved the use of the PAA product VigorOx® SP-15 (PeroxyChem, LLC, Philadelphia, PA, USA) to disinfect equipment and culture surfaces when fish are not present in 2017. In February 2023, the US EPA expanded label claims for VigorOx® Trident (Evonik Active Oxygens, LLC, Philadelphia, PA, USA; EPA Reg. No. 65402-3) to reduce pathogens in recirculating aquaculture system (RAS) and pond water when fish are present.

In this review, we aim to summarise scientific knowledge about concentrations of PAA that are safe to fish and its effectiveness against various undesirable microorganisms and their excreted substances (i.e., biofilm). Ultimately, this review hopes to provide understanding and recommendations about the application of PAA for the aquaculture industry.

2 | VARIABILITY IN CONCENTRATION REPORTING AND SUGGESTIONS FOR ONSITE MEASUREMENT

Before discussing the safe concentration of PAA, it is necessary to define nominal concentration. In aquatic toxicology, nominal concentration is the theoretical concentration of the tested substance when preparing a test solution; an example would be adding 100 mg of a substance to 1 L water; the nominal concentration is 100 mg L^{-1}. With PAA, the concentration of the working or stock solution must be verified via analytical methods as discussed below. Few published studies incorporate chemical measurements and provide the actual measured concentrations of PAA.[17–23] Even fewer studies provide the actual measured concentrations of H_2O_2.[20,22] Most studies have referred to the product safety datasheets provided by the manufacturers for preparing PAA solutions at the target concentrations. In our experience, these concentrations are usually close to the nominal concentrations as defined above, with a precondition that freshly produced products are used and properly stored under cool and dark conditions. In addition, most published literature reports the concentration in mg L^{-1} of the active compound (PAA), while others used mg L^{-1} of product. In this review, we have converted mg L^{-1} of product to mg L^{-1} of active compound (if the product information is available) for ease of comparison. Considering the global product variety

(Table S1) and the necessity of scientific uniformity, we highly recommend that future PAA researchers provide the concentration of PAA as mg L^{-1} PAA active compound. Research has also shown the importance of H_2O_2 in the product, so we recommend including this concentration in mg L^{-1} of active compound. All PAA concentrations in this review are nominal unless they are specifically labelled otherwise.

Test methods for PAA include test strips, titration, photometry and offsite laboratory testing. Besides the *N*,*N*′-diethyl-*p*-phenylene diamine (DPD) photometric method that we have used and adapted for measuring PAA ≤5 mg L^{-1} (and H_2O_2),[17,18,22–24] there are other methods available for a similar or different detection range.[25,26] Many test kits specific for PAA use this DPD method (i.e., CHEMetrics, Hach, LaMotte, etc.). For aquaculture practice, commercial test kits for determining total chlorine in swimming pools are compatible with the measurement of PAA concentration (D. Liu, personal experience). The key reagents of these kits are DPD, phosphate buffer and potassium iodide in either separate tablets or a single integrated tablet and these reagents can react with PAA in water samples of fixed volume, usually around 10 mL, resulting in intensities of pink that can be compared with a provided colour-concentration indicator. Because chlorine (71 g mol^{-1}) and PAA (76 g mol^{-1}) have similar molar mass, the indicated concentration for total chlorine can roughly represent that of PAA. Although these test kits are easy to use and inexpensive, we discourage excessive use by fish culturists due to the toxic properties of DPD[27] and a lack of proper disposal measures. Alternatively, a PAA test kit by CHEMetrics (Calverton, VA, USA) draws the water sample into Vacu-vials® when the tip is snapped; these vials contain the hazardous chemicals that will still need disposed, but can be read on a relatively inexpensive portable photometer. However, we have preliminary data that suggest the electrochemical measurement of oxidation–reduction (redox) potential might be useful for predicting the disinfection efficacy of PAA. More details are explained in Section 6.

3 | PRODUCT VARIETY AND CHEMICAL DEGRADATION

According to the Globally Harmonized System of Classification and Labelling of Chemicals (GHS), an internationally agreed-upon standard managed by the United Nations, and European Regulation No. 1272/2008, PAA products that contain ≤15% PAA are classified as corrosive compounds and may cause fire upon heating.[28] In comparison, products that contain ≥17% PAA are additionally classified as explosive oxidisers and toxic if inhaled. Due to higher safety measures for storage, handling and transport of the more concentrated products, the majority of PAA products on global market consist of ≤15% PAA. The US Department of Homeland Security enacted the Chemical Facility Anti-Terrorism Standards regulation in 2007 to identify and/or regulate facilities that possess specific high-risk chemicals at certain quantities to ensure they have security measures in place that reduce the risks of their misuse.[29] H_2O_2 (concentrations ≥35%) is on this list and quantities of 400 lb (181 kg) must be reported; this is equivalent to 0.7 of a 55-gallon (208 L) container. PAA is also included in concentrations ≥1% in quantities >10,000 lb. (4536 kg); however, only the amount of PAA in solution is considered. Therefore, for a 15% PAA product, storage of more than 24 totes that contain 330 gallons or 1250 L each must be reported.

Yuan and his colleagues suggested three reactions responsible for the degradation of PAA: spontaneous decomposition, transition metal-catalysed decomposition and hydrolysis.[30] Both decomposition reactions are water-independent and generate predominantly acetic acid and O_2. The concentrated PAA products may undergo this spontaneous decomposition and lose concentration over time; however, when stored under proper conditions (good ventilation, cool and protection from direct sunlight), degradation can be largely ignored within the manufacturer's specified shelf life which is usually 1 year (see product specification sheets). Hydrolysis, which is PAA reacting with water to generate acetic acid and H_2O_2 (the reverse of PAA synthesis), is the dominant reaction of PAA degradation during water disinfection. Any soluble or insoluble impurities can accelerate the hydrolysis of PAA. Among the most common solutes in aquaculture, sea salt was found to have a more significant impact on PAA degradation in deionised water than dissolved organic carbon (DOC) and calcium/magnesium carbonates (i.e., hardness and alkalinity).[23] Among the common insoluble water components in aquaculture waters, bacterial aggregates and bacteria-colonised particles were found to predominantly mediate the H_2O_2 decomposition.[31] According to the law of conversion of mass (i.e., mass can neither be created nor destroyed and that the total mass of any material system is neither increased nor decreased by reactions between the parts), these aggregates and particles are likely to promote the hydrolysis of PAA by scavenging H_2O_2. Suspended solids were found to accelerate PAA degradation more profoundly than soluble matter.[32] Moreover, higher feeding rate and fish stocking density are correlated with faster degradation of PAA.[17,18] Therefore, users may expect faster degradation of PAA in seawater systems and freshwater systems with high microbial activities, high suspended solids, high feeding rates and high fish stocking densities.

We also observed an unusual case at a flow-through trout farm. PAA was applied at 0.6 mg L^{-1} in a 2-m^3 circular tank stocked with rainbow trout (*Oncorhynchus mykiss*) fingerlings. During the static PAA treatment, the flow-through mode was interrupted, while aeration with high-pressure oxygen (5 bars) was retained. Unlike the usual first-order degradation,[17] PAA and H_2O_2 showed minimal degradation and maintained concentrations of 0.4–0.5 mg L^{-1} (concentration measured with DPD photometric method[22]) for 1 h. Meanwhile, the dissolved oxygen constantly increased from 10 to 15.1 mg L^{-1}. We speculate that the aeration with high-pressure oxygen and the resulting oxygen oversaturation generated trace amounts of H_2O_2 under sunlight,[33] which inhibited the hydrolysis of PAA. The farm owner reported unusual fingerling mortality without signs of infections shortly after the PAA treatment. After resuming the flow-through water during PAA treatments, there was no mortality. This example emphasises the need for knowledge transfer from researchers to aquaculturists, and vice versa, to ensure that PAA is safe for fish in a range of culture systems.

4 | CONCENTRATIONS SAFE TO FISH

The primary priority for any treatment is to not cause mortality or detrimental effects. Although PAA is considered to be a disinfectant rather than a therapeutant, exposure to PAA is known to cause adverse effects to aquatic animals. In this case, the concept of a "therapeutic window" from clinical research is compatible to PAA treatments in the presence of fish. The term is defined as the range of drug concentrations that provide therapeutic response without significant adverse effects.[34] For PAA treatments, the therapeutic response corresponds to its effectiveness, and the significant adverse effects corresponds to any impact on fish that may result in impaired welfare and even mortality. In this section, we summarise studies from aspects of toxicology, genotoxicity and fish physiology to provide an overview of the adverse effects of PAA on fish.

4.1 | Toxicity

Toxicologists normally use the median lethal concentration (LC50) value to represent the toxicity of a substance, but that is not overly helpful to fish culturists, who need to know the highest concentration that will not kill their fish; this is called the no-observed-effect-concentration (NOEC), which is reported in some publications. Some ecotoxicologists believe that the 5% lethal concentration (LC5) could be an alternative to the NOEC,[35,36] but statistically this value can exhibit a high degree of variability or inaccuracy; the NOEC represents safe concentrations to the fish under the constraints of the environment in which it was determined. Toxicity values or effects are shown in Table 1.

Disinfecting eggs to prevent saprolegniasis was the earliest use of PAA in the presence of fish. Many preliminary disinfection protocols were like those for surface disinfection recommended by Schäperclaus who suggested a brief bath in a solution of PAA.[15] As researchers gained more knowledge and experience with PAA, concentrations at lower doses proved to be effective. Moreover, limiting the bath duration was unnecessary due to the fast degradation of PAA. For fish, PAA was delivered either in the culture systems or in a separate bath system. Detailed information of studies addressing the acute toxicity of PAA to fish is summarised below. Key information such as safe concentrations and important exposure conditions are enumerated in Table 1.

4.1.1 | Toxicity of PAA in freshwater

The antifungal efficacy and safety of several fungicides were evaluated with rainbow trout eggs.[37] After 1-h exposure at 12°C with a total alkalinity of 105 mg L^{-1} (as $CaCO_3$), PAA was found toxic at 15 mg L^{-1}, resulting in ≥10% reduced hatching rate compared with the control group. While evaluating the ability of PAA to control saprolegniasis on rainbow trout eggs, toxicity tests were carried out at 11°C in petri dishes and 3 mg L^{-1} PAA administered twice daily for 1 h was reported well tolerated by the eggs.[38] The 24-h LC50 values were reported for zebrafish (*Danio rerio*) embryos exposed to 7 PAA products in 1/10-diluted, original and 10-fold concentrated reconstituted water (DIN European Norm [EN] ISO 7346-3: 294 mg L^{-1} $CaCl_2{\cdot}2\,H_2O$, 123.3 mg L^{-1} $MgSO_4{\cdot}7\,H_2O$, 63.0 mg L^{-1} $NaHCO_3$ and 5.5 mg L^{-1} KCl) having total alkalinity values of 7.5, 75 and 750 mg L^{-1}, respectively; the 24-h LC50 values varied between 2.2 and 7.1 mg L^{-1} depending on alkalinity.[39] Similar 48-h LC50 values were reported for these alkalinity levels in another study, where zebrafish embryos were exposed to PAA in 1/10-diluted, original and 5-fold concentrated reconstituted water (DIN EN ISO 7346-3) with and without an additional 10 mg L^{-1} of dissolved humic substance; the presence of humic substance resulted in reduced toxicity of PAA, especially at low alkalinity levels.[40]

Pigmented and glass eels (*Anguilla anguilla*) with an average body weight of 6.6 and 0.3 g, respectively, were exposed to 30 chemicals as a treatment for trichodina infestations in RAS; Detarox AP® (containing 5% PAA) was chosen for further investigations. Mortality of pigmented eels (elvers) occurred when exposed to more than 100 or 70 mg L^{-1} Detarox AP® (containing 5 or 3.5 mg L^{-1} PAA) in 5 or 96 h, respectively, while younger glass eels tolerated 25 mg L^{-1} Detarox (containing 1.25 mg L^{-1} PAA).[43] Juvenile pike perch (*Sander lucioperca*, 3 cm) were exposed to PAA concentrations of 0.5, 0.9, 1.3 and 1.7 mg L^{-1} in reconstituted water (DIN EN ISO 7346-3); the 24-h LC50 value was 1.14 mg L^{-1} PAA and the NOEC was 0.5 mg L^{-1} PAA.[44] Treatments for ichthyophthiriasis were investigated in lab facilities supplied with ground water at 18°C on rainbow trout and common carp (*Cyprinus carpio*) and it was reported that the PAA used was lethal to both species when used at 100 µL product L^{-1} within 30 min[45]; the authors did not report the PAA concentration in this product other than to say it was comparable to the PAA product used in another study which was 13% PAA.[58] Therefore, we calculated that the 100 µL product L^{-1} corresponded to 13 mg L^{-1} PAA. Rainbow trout (450 g) were exposed to the concentrations of 1.5, 3.0, 4.5, 6.0, 7.5 and 9.0 mg L^{-1} PAA at 16°C and an alkalinity of 50–90 mg L^{-1} for 1 h. All trout survived in the 1.5 and 3.0 mg L^{-1} PAA treatments, whereas 14%–17% mortality was observed during the 24-h recovery period in the 4.5, 6.0 and 7.5 mg L^{-1} treatments, and 85% mortality was reported during recovery in the 9.0 mg L^{-1} treatment.[47]

Static toxicity tests with PAA were carried out on channel catfish (*Ictalurus punctatus*) fry in well water with an alkalinity of 217 mg L^{-1} at 23°C and the 24-h LC50 values for yolk-sac fry and swim-up fry were determined to be 2.6 and 1.6 mg L^{-1} PAA, respectively; the 24-h NOEC was 2.2 mg L^{-1} PAA for yolk-sac fry and 1.3 mg L^{-1} PAA for swim-up fry. Histopathology indicated there was severe gill damage in fish treated with 2.2 mg L^{-1} for 1 h and moderate degeneration of renal tubule epithelium within the posterior kidney in fish treated with 1.7 mg L^{-1} PAA for 48 h.[42] This study was a 48-h study, but only one additional fish died after 24 h, therefore the optimal duration for an acute toxicity study with PAA is 24 h. The most extensive research to date on the toxicity of PAA was conducted with economically important fish in the United States; the 24-h LC50 values and NOEC concentrations of PAA in well water to fingerlings of 12 fish species in

TABLE 1 The lethal or non-lethal concentrations of PAA to various fish species in the order of life stages after acute exposures in freshwater and seawater.

Species	Stage/ size (mean)	Alkalinity (mg L^{-1})[a]	Exposure	Effect (mg L^{-1} PAA)[b]	24-h NOEC (mg L^{-1} PAA)	Ref.
	Freshwater					
Rainbow trout (*Oncorhynchus mykiss*)	Embryos	105	1-h static	Lethal at 15	NA	37
	Embryos	75	2 × 1-h static per day	Safe at 3	NA	38
Zebrafish (*Danio rerio*)	Embryos	7.5	Single static, 7 products	24-h LC50 = 2.2–4.5	NA	39
		75		24-h LC50 = 3.0–6.1	NA	
		750		24-h LC50 = 4.2–7.1	NA	
		7.5	Single static	48-h LC5 = 2.5	NA	40
		75		48-h LC5 = 2.7	NA	
		375		48-h LC5 = 5.4	NA	
Channel catfish (*Ictalurus punctatus*)	Embryos	243	Twice daily in flow-through trough	Safe at ≤5	NA	41
	Yolk-sac fry	217	Single static	24-h LC50 = 2.6	2.2	42
	Swim-up fry			24-h LC50 = 1.6	1.3	
European eel (*Anguilla anguilla*)	0.3 g glass eel	NA	5-h static, followed by 91-h recovery	Safe at 1.25	NA	43
	7 g pigmented eel	NA		Safe at 3.5	NA	
Pike perch (*Sander lucioperca*)	3 cm juvenile	75	Single static	NA	0.5	44
Common carp (*Cyprinus carpio*)	7 cm (Ich-infected)	178	Single static	Lethal at 13 (estimated) within 30 min	NA	45
Black fathead minnow (*Pimephales promelas*)	5.0 cm juvenile	200	Single static	24-h LC50 = 2.8	1.9	46
Golden shiner (*Notemigonus crysoleucas*)	6.2 cm juvenile			24-h LC50 = 4.1	2.8	
Rainbow trout (*On. mykiss*)	6.0 cm juvenile			24-h LC50 = 4.2	2.8	
Bluegill (*Lepomis macrochirus*)	6.4 cm juvenile			24-h LC50 = 4.8	2.8	
Grass carp (*Ctenopharyngodon idella*)	7.7 cm juvenile			24-h LC50 = 4.2	3.3	
Goldfish (*Carassius auratus*)	7.6 cm juvenile			24-h LC50 = 4.6	4.0	
Channel catfish (*Ict. punctatus*)	11.4 cm juvenile			24-h LC50 = 5.6	4.0	
	11.4 cm juvenile	118		24-h LC50 = 4.8	3.3	
	11.4 cm juvenile	225[c]		24-h LC50 = 5.8	4.8	
Largemouth bass (*Micropterus salmoides*)	8.4 cm juvenile	200		24-h LC50 = 5.9	4.0	
Walleye (*Sander vitreus*)	8.4 cm juvenile			24-h LC50 = 5.9	4.0	
Hybrid striped bass (*Morone chrysops* × *Morone saxatilis*)	5.3 cm juvenile			24-h LC50 = 5.3	4.0	
Black-nose crappie (*Pomoxis nigromaculatus*)	8.0 cm juvenile			24-h LC50 = 5.9	4.8	
Blue tilapia (*Oreochromis aureus*)	7.0 cm juvenile			24-h LC50 = 9.3	5.8	

(Continues)

TABLE 1 (Continued)

Species	Stage/ size (mean)	Alkalinity (mg L^{-1})[a]	Exposure	Effect (mg L^{-1} PAA)[b]	24-h NOEC (mg L^{-1} PAA)	Ref.
Rainbow trout (*On. mykiss*)	8 cm (Ich-infected)	178	Single static	Lethal at 13 (estimated) within 30 min	NA	45
	6 cm	200	Single static	NA	2.8	46
	450 g	50–90	1-h static	Safe at 1.5 and 3	NA	47
Atlantic salmon (*Salmo salar*)	11.3 g parr	20	2 × 1-h static	Safe at ≤1.6	NA	48
	Eyed embryos	264 (RAS water)	Single static for 5 and 10 min	24-h LC50 = 782 and 485	500 for 5 min 300 for 10 min	49
	0.17 g fry	70 (RAS water)	Single static	24-h LC50 = 4.0	NA	
	16.3 g juvenile	55 (RAS water)	Single static	24-h LC50 = 5.3	NA	
	47 g parr	184 (RAS water)	Single static	24-h LC50 = 4.3	NA	50
	66.5 g smolt	170 (RAS water)	Single static	24-h LC50 = 4.3	NA	
	176.7 g post-smolt	131 (RAS water)	Single static	24-h LC50 = 4.8	NA	
	Brackish water/seawater					
Atlantic halibut (*Hippoglossus hippoglossus*)	Embryos	NA (seawater)	1-min static	Safe at 200	NA	51
Almaco jack (*Seriola rivoliana*)	Embryos	NA (seawater)	1-min static	Safe at 15.7	NA	52
Atlantic Cod (*Gadus morhua*)	Embryos	NA (seawater)	1-min static	Safe at 180	NA	53
Atlantic salmon (*Sal. salar*)	~90 g post-smolt	NA (12‰ salinity)	Repeated every 3 days for 6 weeks	Safe at 1	NA	54
	80–90 g smolt	NA (35‰ salinity)	Single static to 10 mg L^{-1} PAA for 15 or 30 min, repeated every 15 days over 45 days	Mortality = 10% (15-min group) and 10.83% (30-min group)	NA	55
	150 g smolt	NA (seawater)	5-min + 30-min static with 2-week recovery interval	Safe at 0.6 and 2.4	NA	56
	131 g smolt	NA (seawater)	Single static for 30 min post-crowding stress	Safe at 4.8	NA	
Lumpfish (*Cyclopterus lumpus* L.)	46.9–297.2 g fish	NA (seawater)	Single static for 40 min after euthanasia	2 and 10 caused rougher epidermal boarder, removed epidermis from bony plates	NA	57

Note: All concentrations are nominal.

Abbreviations: NA, not available; NOEC, no-observed-effect-concentration; PAA, peracetic acid; RAS, recirculating aquaculture system.

[a]As $CaCO_3$.

[b]All are lethal concentrations values.

[c]Water had 10 mg L^{-1} Huminfeed to add dissolved organic content.

static systems were reported.[46] Varying tolerance to PAA among species was reported, and LC50s ranged from 2.8 mg L^{-1} PAA for fathead minnow (*Pimephales promelas*) to 9.3 mg L^{-1} for blue tilapia (*Oreochromis aureus*); NOECs for these fish ranged from 1.9 to 5.8 mg L^{-1}. In tests with fingerling channel catfish, greater toxicity was shown in 50% deionised water-diluted well water that had lower alkalinity/hardness, while a 10-mg L^{-1} increase in dissolved humic substances had no effect on PAA toxicity. These two studies were carried out in fresh well water and organic load was minimal; these should be considered a worst-case scenario for acute toxicity.

Atlantic salmon (*Salmo salar*) parr (11.3 g) in individual freshwater RASs were exposed to 0.05, 0.1, 0.2, 0.4, 0.8, 1.6, 3.2 and 6.4 mg L^{-1} PAA (AquaDes™) for a 1-h static treatment. Mortality was 0% when PAA was ≤1.6 mg L^{-1}, 20% at 3.2 mg L^{-1} and 100% at 6.4 mg L^{-1}. Swimming behaviour was normal when PAA was ≤1.6 mg L^{-1}, but increased erratic swimming was observed in fish exposed to 3.2 and 6.4 mg L^{-1} PAA; substantial damage to skin and gills was also reported at these higher concentrations. The NOEC of PAA was reported ≤1.6 mg L^{-1}.[48] Eggs with eyed embryos of Atlantic salmon were exposed to PAA in recirculating spring water for 5 or 10 min and reported LC50 values were 781 and 485 mg L^{-1}, respectively. NOEC values for eyed embryos were 300 and 500 mg L^{-1} PAA for 5- and 10-min bath, respectively. The 24-h LC50 values to fry (∼0.2 g) and fingerlings (∼16 g) were reported to be 4.0 and 5.3 mg L^{-1} PAA, respectively, in mature RAS water.[49] In a follow-up study, three late freshwater life stages (parr, 47 g; smolt, 67 g; post-smolt, 178 g) of Atlantic salmon were exposed to PAA in mature RAS water. The 24-h LC50 values were 4.3, 4.3 and 4.8 mg L^{-1} PAA, respectively.[50]

In summary, the toxicity of PAA to fish in freshwater showed high variation depending on the mode of exposure, fish species, life stages and water alkalinity. Multiple exposures are more toxic than single exposure. Static exposures are more toxic than exposures in a flow-through setup. Fry is less tolerant to PAA than the other life stages. Increase of alkalinity could enhance the tolerance of fish to PAA, and vice versa.

4.1.2 | Toxicity of PAA in seawater

The first research using PAA in an aquaculture setting was conducted to disinfect eggs of Atlantic halibut (*Hippoglossus hippoglossus*) to prevent mass mortalities during egg and early larval rearing.[51] It was determined that a 1-min exposure to 200 mg L^{-1} PAA at 6°C and 33‰–35‰ salinity resulted in a strong antibacterial effect without decreasing hatching rate. This concentration is considerably higher than in freshwater conditions probably because of the very short exposure time and promoted PAA degradation by salinity, as discussed in Section 3. Disinfecting eggs of almaco jack (*Seriola rivoliana*) were evaluated with formalin, H_2O_2 and PAA at 26°C and 35‰ salinity to reduce bacterial load and improve hatch rate[52]; in preliminary tests, a previous study was referred, where eggs of Atlantic cod (*Gadus morhua*) were disinfected with 180 mg L^{-1} PAA for 1 min,[53] but the same treatment was lethal to almaco jack eggs. Therefore, the concentration was decreased until mortality was no longer observed, resulting in a 1-min treatment of 15.7 mg L^{-1} PAA in seawater. These results suggest that sensitivity of embryos to PAA can vary greatly among species.

4.2 | Disinfection by-products and genotoxicity

The toxic effect is the combination of the concentration and duration of exposure.[59] Chronic exposure to PAA may cause adverse effects on fish that are undetected after an acute exposure. The earliest studies addressing the chronic adverse effects of PAA on fish were from wastewater treatment researchers. These studies shared a common hypothesis that the disinfection by-products (DBPs) from PAA may induce genotoxicity in aquatic animals. A recent review summarised studies concerning the types of DBPs formed during PAA dinfection as well as their formation mechanisms and genotoxic properties.[60] In freshwater scenarios, DBPs formed during PAA disinfection are mainly carboxylic acids and aldehydes in μg L^{-1}. Carboxylic acids are generally considered non-genotoxic. Aldehydes need to reach the mg L^{-1} level to be hepatotoxic and can be further oxidised into carboxylic acids. Despite minor conflicting results, most genotoxicity studies using fish suggest that chronic exposure to PAA-treated sewage or lake water, at ≤1.0 mg L^{-1} PAA or with PAA completely degraded, had limited genotoxic effects on fish.[61–68] Therefore, freshwater aquaculture has low genotoxic risks of using PAA as a water treatment.

In halide-rich brackish and sea water scenarios, the halides can be oxidised into halogenated DBPs (mostly hypohalous acids at the μg L^{-1} level) after extended contact to PAA at high concentrations (5–150 mg L^{-1}).[60] In marine aquaculture settings, high concentrations of PAA are rarely sustained for an extended period, especially in the presence of fish. Furthermore, the presence of H_2O_2, especially when its concentration exceeds PAA, can reduce the hypohalous acids to halides.[69] As mentioned above, the hydrolysis of PAA primarily generates H_2O_2 and favours the subsequent dominance of H_2O_2 in the equilibrium for a certain period. Therefore, the genotoxic risks of PAA disinfection in marine settings can be reduced by limiting the PAA concentration and length of the disinfection.

4.3 | Physiological responses of fish

Studies addressing the physiological response of fish to PAA disinfection were mostly conducted in aquaculture settings. During these studies, either the fish were transferred to a separate container for a PAA bath, or PAA was applied periodically or continuously in the fish culture system with or without interruption of water flow. PAA can induce stress responses slightly during the first exposure, but this is followed by adaptively reduced stress responses during subsequent exposures. Minor morphological changes of mucosal surfaces may occur post-PAA exposure and this is followed by quick recovery. Evidence of oxidative stress and metabolic or transcriptional reactions

in the blood, mucosal surfaces and internal organs have been detected post-PAA exposure. Details of these studies are summarised below for several fish families.

4.3.1 | Cyprinidae

Red garra (i.e., doctor fish; *Garra rufa*) were transferred from recirculating aquaria (hardness = 290 mg L^{-1}) to 40-min static baths of PAA 6 times day^{-1} for 1 week.[70] For each bath, either 15 or 45 μL L^{-1} of a PAA product was tested. Neither the authors nor the manufacturer provided the PAA concentration in the product; if the product contained 15% PAA, then the PAA concentrations in the baths were 2.25 and 6.75 mg L^{-1}, respectively ($\text{Final concentration}_{[\text{mg L}^{-1}\text{ PAA}]} = \text{Dose}_{[\text{or μL PAA product L}^{-1}\text{ water}]} \times \text{Product strength}_{[\%\text{PAA in product}]}$). The authors observed an increase of mucous cells and unchanged mucin types on epidermis and gills after treatment at both concentrations; a decrease of club cells was only observed after treatment at high concentrations. They also reported feeding behaviour of fish treated with the low PAA dose was not affected, but fish treated with the high dose of PAA had poor appetite, especially towards the end of the week. In practice, users must consider lowering the PAA concentration if irritation on fish appears. Juvenile grass carp (*Ctenopharyngodon idella*, ~72 g) were exposed to single doses of 1 and 3 mg L^{-1} PAA in static tanks twice per day for 10 days; water in tanks was changed daily.[71] Repeated exposure to 3 mg L^{-1} PAA resulted in 71.5% mortality. Repeated exposure to 1 mg L^{-1} PAA resulted in a decrease of anti-oxidative enzymes but unchanged lipid peroxidation in gill and liver. It is noteworthy that the water used in this study was soft (total Ca^{2+} and Mg^{2+} was 14 mg L^{-1}) and the control fish were not healthy considering the presence of protozoan parasites on the gills and suboptimal gill health. In this case, the observed effect of PAA should only be valid for fish with impaired health under similar low-hardness conditions. Other than the effect on redox balance, exposures to single doses of 2 mg L^{-1} PAA twice per week in a pilot RAS system induced an adaptable stress in adult mirror carp (*Cy. carpio*, ~649 g). The stress response was strong during the first exposure but progressively decreased with subsequent exposures.[21]

4.3.2 | Salmonidae

Clinically healthy juvenile rainbow trout (~6 g) were transferred from culture tanks to 20-min baths of 2.4 mg L^{-1} PAA 3 times day^{-1} for 3 days. No changes of anti-oxidative enzymes and lipid peroxidation were observed in the gill, heart and liver.[72,73] PAA was applied either twice per week with single doses of 1 mg L^{-1} PAA or continuously via a peristatic pump at 0.2 mg L^{-1} in the inflow water, flow-through tanks stocked with juvenile rainbow trout (~115 g); the concentrations of PAA were measured 5 min post-application and were 0.7 mg L^{-1} and below detectable range, respectively. Stress was induced by the first dose of 1 mg L^{-1} PAA but was followed by progressive adaptation of the fish to subsequent doses[19]; this was confirmed by serotonin activity in the brain.[74] In comparison, the continuous dose of 0.2 mg L^{-1} PAA in the inflow water quickly degraded in the tanks due to the strong dilution effect and spontaneous degradation, and therefore did not induce stress in the fish. In a follow-up study, it was observed that endogenous total free radicals in rainbow trout were elevated by PAA exposure in both dosing methods; the total antioxidant capacity (TAC) in gill and serum also showed corresponding elevation as well. The cutaneous and humoral immunity-related enzyme activities were mostly unaffected, with the exception that serum ceruloplasmin and antiprotease activities being lower in fish exposed to inflow water continuously dosed with 0.2 mg L^{-1} PAA.[75]

In another study, PAA was applied in the RASs sump before entering the culture tank via peristaltic pumps. The pump was set in semi-continuous mode (repeated cycles of 0.5 min on: 4.5 min off), and increasing PAA concentrations of 0.05, 0.1 and 0.3 mg L^{-1} were maintained for approximately 1 month. The growth performance of the sub-adult rainbow trout (~407 g) showed no difference between PAA-treated and -untreated RAS.[76] Rainbow trout fries (1 g) were exposed to a single dose of PAA at concentrations from 0.125 to 2 mg L^{-1} in unchlorinated tap water (450 mg L^{-1} alkalinity). No erratic swimming was observed within 24 h post-exposure. Expression of inflammatory cytokines and acute phase reactants were upregulated by PAA exposure in both gills and fins. Hyperplasia of mucous cells were present in fish 2 h post-exposure, followed by quick recovery 24 h post-exposure.[77]

PAA exposure studies in Atlantic salmon revealed some of the most extensive physiological investigations in a fish species. In a study, Atlantic salmon smolts (~150 g) were exposed to a 5-min static bath with 0.6 and 2.4 mg L^{-1} PAA; fish were exposed to a second 30-min bath of the same PAA concentrations 2 weeks later. Compared with the control fish, the TAC and cortisol in plasma were significantly higher in fish 2 h after the second exposure to 2.4 mg L^{-1} PAA. Gene expression of antioxidant enzymes was upregulated predominantly in the gills rather than in the skin after the second exposure to 2.4 mg L^{-1} PAA; there was partial upregulation in the group of 0.6 mg L^{-1} PAA treatment.[56] A supporting paper further revealed marginal histological changes in the skin, though signs of altered expression of genes coding for proteolytic enzymes were reported after the second exposure to both PAA concentrations. In addition, fin damage and scale loss were more prevalent in PAA-exposed fish than unexposed fish.[78] The first transcriptome-wide profiling of PAA-exposed Atlantic salmon revealed the regulation of genes predominantly related to immunity, metabolism and tissue integrity in both skin and gills, while the former showed more profound changes. The metabolomic profiling of plasma from these Atlantic salmon showed that PAA exposure resulted in changes of a few antioxidative functional metabolites, nonetheless, no global metabolomic disturbances were documented.[79] The dynamic morphological analysis of gill mucous cells in lamella and filaments revealed transient hypertrophy of gill mucous cells post exposure to PAA up to 2.4 mg L^{-1} followed by quick recovery. Histopathological scoring was similar between PAA-exposed and unexposed fish.[80]

Exposure to stress could interfere with the physiological responses prior to PAA treatment. Crowding stress (by reducing water volume to 20%) was employed to Atlantic salmon smolts (~131 g) prior to a single 30-min seawater bath with 4.8 mg L^{-1} PAA. The crowding stress significantly altered the stress and systemic antioxidant responses to 4.8 mg L^{-1} PAA. Histology revealed that crowding affected skin integrity, and as a result, made the epidermal layer more vulnerable to PAA. Histological features of the gills were generally in good condition and crowding, with or without PAA exposure, showed no substantial impact. The transcriptomic profiling revealed that the gills were more responsive to PAA than the skin, as demonstrated by the magnitude of changes on some key mucosal biomarkers following exposure. However, crowding stress prior to PAA exposure reversed this response profile and transcriptional changes were more pronounced in the skin than the gills. Plasma stress metabolites were affected by crowding and sampling time and not by PAA exposure.[81]

Repeated exposure may occur when administering treatments in fish. Atlantic salmon smolts (80–90 g) were exposed to 10 mg L^{-1} PAA for 15 or 30 min every 15 days over 45 days. The PAA exposures resulted in strong stress behaviour in fish, which began with erratic swimming, followed by diminished swimming activity with increased opercular ventilation and finalised with 10% loss of balance. Minor fish mortality was observed in PAA-exposed groups only. Transcriptomic analysis revealed predominant altered gene expression in gills and liver in response to PAA. The main transcriptional changes in gills were related to immunity and ribosomal function, while those in the liver were related to oxidation–reduction processes. Skin morphology was unaffected by PAA, while hypertrophy of gill mucous cells was observed in fish after the final 30-min PAA exposure. Plasma total reactive oxygen species was higher in PAA-exposed fish than unexposed fish indicating that PAA triggered systemic oxidative stress. Several plasma metabolites, plasma indicators for hepatic and renal health and ribosomal proteins in skin mucus significantly differed between PAA-exposed and -unexposed fish. One week after the final PAA exposures, the fish were given a crowding stress by reducing the water volume in tanks to 10% for 1 h. Results revealed that repeated exposures did not impede the ability of the fish to mount a response to a secondary stressor.[55] This repeated exposure study was supported by another study, where changes in the brain were investigated. Regulation of genes related to vasotocinergic and isotocinergic systems and the corticotropin-releasing factor signalling system was affected, indicating interference of the stress axis but could also suggest an anxiolytic effect. The total reactive oxygen species in the brain was unaffected by the repeated PAA exposures.[82]

Atlantic salmon smolts (~90 g) were treated with 1 mg L^{-1} PAA in a brackish water RAS every 3 days over a 45-day exposure period. These treatments resulted in the downregulation of antioxidants, cytokines and mucin genes in the gills, while upregulation was the prominent response in the skin. Histological changes were present at all mucosal surfaces, though the most profound impact was on the gills. Excessive production of nasal mucus was observed in PAA-exposed fish. The stress response in fish against additional netting and confinement was not significantly altered by PAA. It was also demonstrated that the olfactory organ was responsive to intermittent PAA administration. Compared with pre-exposure level, expression of several erythrocyte-related genes in olfactory rosette tissue was upregulated, while expression of several immune response-related genes was downregulated.[54,83] Atlantic salmon parr (~25 g) in 2‰ salinity RASs were exposed to 1 mg L^{-1} PAA applied in either periodic (every 3 days) or continuous (daily dose was delivered every 3 h over a 24 h period) mode for 4 weeks. PAA was administrated via a peristaltic pump at 1 mg L^{-1} into the sump before entering the culture tank. The periodic application was intended to result in a shorter exposure to a higher concentration of PAA than the continuous application. Neither application mode caused systematic changes in transcriptional regulation of key antioxidant enzyme genes nor DNA/protein damages in mucosal organs and liver. The total free radicals and TAC in plasma and skin mucus were affected by PAA. Minor histological changes in gill, skin and olfactory organ were present in exposed fish but these showed recovery.[84]

PAA is a strong immune modulator. Freshly isolated olfactory leucocytes were exposed to PAA, H_2O_2 and acetic acid at 100 μM (equivalent to 7.6 mg L^{-1} PAA, 3.4 mg L^{-1} H_2O_2 and 6 mg L^{-1} acetic acid, respectively) for 30 min. Leucocyte proliferation was inhibited by all stimulants at 24 h post-exposure, with signs of recovery at 48 h to PAA and H_2O_2. The leucocyte migration was promoted by exposure to H_2O_2, while unaffected by other exposures. All exposures triggered the increase of intracellular reactive oxygen species and correspondingly the upregulation of antioxidant genes. The upregulation of several cytokines and heat shock proteins was also detected in olfactory leucocytes exposed to PAA and H_2O_2.[83]

4.3.3 | Sparidae

Juvenile gilthead seabreams (*Sparus aurata*, 20 g) were exposed to a 5-min seawater bath of PAA at 4 mg L^{-1}. Transcriptional regulation of antioxidant enzyme genes in the gills and skin were mostly unaffected by PAA exposure. Mild stress was induced by PAA exposure and followed by recovery 24 h post-exposure. The TAC in plasma was mildly enhanced 8 h post-exposure and showed tendency of recovery from 24 h to 1 week.[85]

4.3.4 | Cyclopteridae

Lumpfish (*Cyclopterus lumpus* L.) are used in the sea cage production of Atlantic salmon as a biological control strategy against the ectoparasitic salmon louse (*Lepeophtheirus salmonis*). Using an in vitro skin model, the effects of PAA and H_2O_2 on lumpfish skin were compared. PAA exposure (2 and 10 mg L^{-1}) resulted in morphological alterations in the microarchitecture of skin such as rougher epidermal border and absence of bony plates in the epidermis. H_2O_2 treatments (2000 and 10,000 mg L^{-1}) resulted in more pronounced effects than PAA treatment in lumpfish skin.[57]

5 | TREATMENT EFFECTIVENESS

The effectiveness of PAA against many aquaculture bacteria, oomycetes, viruses, parasites and unfavourable bio-synthetics has been evaluated both in vitro and in vivo. Depending on the safe concentrations discussed above, the in vitro effective concentration of PAA might be incompatible for the disinfection in the presence of fish. Details of these studies are provided below and summarised in Table 2.

5.1 | Fish Bacteria

5.1.1 | Environmental bacteria

Planktonic *Escherichia coli* carrying *nfxB* plasmids were exposed to serial concentrations of PAA in vitro in phosphate-buffered saline for 15 min. The regrowth of disinfected cells within 64 h was completely halted by 8 mg L^{-1} PAA. However, a high abundance of *nfxB* plasmids could be retained from cells disinfected with up to 25 mg L^{-1} PAA with minimal loss of their transforming activity.[86]

In production-scale RAS stocked with common carp (~649 g), twice per week applications of 1 mg L^{-1} PAA in the culture tanks could reduce 90% colony forming units (CFUs) of total heterotrophic bacteria in water. Despite the short-term effects of oxidation after the PAA application, long-term improvement of the gill health was observed as indicated by fewer inflammatory cells.[87]

5.1.2 | Pathogenic bacteria

It was reported that in vitro exposure of wild-isolated and lab-cultured *Pseudomonas aeruginosa* to 3.0 mg L^{-1} PAA for ≥1 h in suspensions resulted in a reduction in total number by 5 $\log_{10}$. It was also reported that 6.1 mg L^{-1} PAA was necessary to achieve the same effect for a 5-min exposure.[88] A similar study was conducted using a standard strain of *Ps. aeruginosa* (ATCC 15442). It was reported that 5-min exposure to 2.9, 5.1 and 10.3 mg L^{-1} PAA reduced the total number of bacteria by 2 $\log_{10}$, 3 $\log_{10}$ and 5 $\log_{10}$, respectively.[89]

The in vitro bactericidal activity of PAA against Gram$^-$ *Aeromonas salmonicida* subsp. *salmonicida* and *Yersinia ruckeri* and the Gram$^+$ *Carnobacterium piscicola* and *Lactococcus garvieae* was investigated with a contact time of 30 min at 4°C. It was reported that the Gram$^-$ bacteria were more sensitive to PAA than the Gram$^+$ bacteria; 100 mg L^{-1} PAA was bactericidal against all four organisms and this equated to >5 $\log_{10}$ reduction in total bacterial numbers. The exposure was conducted in 'high-level soiling conditions" by supplementing 10 g L^{-1} yeast extract and 10 g L^{-1} bovine albumin in the exposure solutions.[90]

The in vitro efficacy of PAA products with various PAA:H_2O_2 ratios against *Flavobacterium columnare* was compared. The tests were conducted on agar plates inoculated with 4-day-old bacterial broth and applied PAA solutions in a central 6-mm diameter hole where the agar was aseptically removed. Concentrations of the PAA solutions (100–1000 mg L^{-1}) were 100-fold the described exposure concentrations (1–10 mg L^{-1}), as the volume of each agar was 100-fold the volume of each PAA solution. Therefore, the exposure concentrations should be understood as mg PAA L^{-1} agar (assuming no instant PAA decay) instead of regular mg PAA L^{-1} solution. The inhibition was defined as the radius of the inhibition zone without visual bacterial growth divided by the radius of the agar. The authors found 40% inhibition 24 h post-exposure to 1 mg PAA L^{-1} agar for two of the tested PAA products with the lowest PAA:H_2O_2 ratios. Other PAA products showed similar inhibition at concentrations of 2 or 4 mg PAA L^{-1} agar. Products with a lower molecular PAA:H_2O_2 ratio showed better growth inhibition and the authors suggested that H_2O_2 played an important additive effect[95]; however, the definition of exposure concentrations lacked supporting data. The penetration and degradation of PAA and H_2O_2 through solid agar has never been studied. Assuming the penetrating speed is constant, and the decay follows first-order kinetics as in organic-enriched aqueous solutions,[17] the concentration of PAA and H_2O_2 (prior to complete decay) on agar plates should be decreasing in radial gradients instead of overall. Therefore, the inoculated bacteria at different radial locations received different exposure intensities.

A cultivation-based method was employed to test the inhibitory effect of PAA on the in vitro growth of planktonic and biofilm cells of *Aeromonas hydrophila*. A 30-min exposure to 10 mg L^{-1} PAA reduced the planktonic cell density from $10^{6.3}$ to $10^{3.7}$ CFUs mL^{-1}. A 20-min exposure to 25 mg L^{-1} PAA reduced the planktonic cell density from $10^{6.1}$ to $10^{1.9}$ CFUs mL^{-1}. In comparison, a decrease of biofilm cell density from $10^{6.2}$ to $10^{1.3}$ was reported after a 30-min exposure to 100 mg L^{-1} PAA, suggesting higher tolerance of biofilm cells to disinfection than planktonic cells.[96]

Ae. salmonicida and *Y. ruckeri* in suspensions were exposed in vitro to PAA products of various PAA:H_2O_2 ratios, and the effect was evaluated by inoculating the exposed suspensions on agar plates. Differences in efficacy among the products tested and a significant influence of concentration and time of exposure were determined. Specifically, the products with higher molar PAA:H_2O_2 ratios were more effective in reducing bacterial growth. Except for one product, exposure for 5–10 min at 0.5–1 mg L^{-1} PAA was sufficient to reduce bacterial growth. An increase of 1 mg L^{-1} PAA reduced the CFUs by >6 $\log_{10}$ for *Ae. salmonicida* and >7 $\log_{10}$ for *Y. ruckeri*. An increase of exposure time by 1 min decreased the CFUs by >5 $\log_{10}$ for *Ae. salmonicida* and >6 $\log_{10}$ for *Y. ruckeri*. The influence of exposure time was less important than the concentration of the product. This implies that the antibacterial effect of PAA/H_2O_2 is very rapid and only short exposure times are necessary.[91] The quick onset of PAA in bacterial suspensions may benefit from the instant full contact with individual cells. In contrast, in the aforementioned agar diffusion method,[95] the contact of PAA to bacterial cells was hindered by the diffusion process and fast PAA degradation. H_2O_2 may retain longer in agar and had a stronger contribution to the total antibacterial effect of a PAA product.

The effect of PAA on two strains of *Y. ruckeri* was studied in vitro via visual turbidity of bacterial broths. For a 20-min exposure, a

TABLE 2 The effectiveness of PAA against pathogens from several trophic levels, their interactions and resulting adverse products.

Target	Exposure	Effect	Ref
Environmental bacteria			
Escherichia coli	15-min exposure to 8 ± 1 mg L^{-1} PAA in suspension	No regrowth; plasmid remained transferable up to 25 mg L^{-1} PAA	86
Total heterotrophic bacteria	Single exposure of RAS water to 1 mg L^{-1} PAA	90% CFU reduction	87
Fish-pathogenic bacteria			
Pseudomonas aeruginosa (Gram$^-$)	≥1-h exposure to 3 mg L^{-1} PAA in vitro in suspension	5 $\log_{10}$ reduction in total numbers	88
	5-min exposure to 6 mg L^{-1} PAA in vitro in suspension	5 $\log_{10}$ reduction in total numbers	
	5-min exposure in vitro in suspension	2 $\log_{10}$, 3 $\log_{10}$ and 5 $\log_{10}$ reduction in total numbers at 2.9, 5.1 and 10.3 mg L^{-1} PAA, respectively	89
Aeromonas salmonicida (Gram$^-$)	30-min exposure to 0.1 g L^{-1} PAA in vitro in organics-enriched suspension	>5 $\log_{10}$ reduction in total numbers	90
	Single exposure in vitro in suspension	>6 $\log_{10}$ CFUs reduction with concentration increase by 1 mg L^{-1} PAA; >5 $\log_{10}$ CFUs reduction with an increase of exposure time by 1 min	91
Yersinia ruckeri (Gram$^-$)	30-min exposure to 0.1 g L^{-1} PAA in vitro in organics-enriched suspension	>5 $\log_{10}$ reduction in total numbers	90
	Single exposure in vitro in suspension	>7 $\log_{10}$ CFUs reduction with concentration increase by 1 mg L^{-1} PAA; >6 $\log_{10}$ CFUs reduction with an increase of exposure time by 1 min	91
	20-min exposure to 3.91 mg L^{-1} PAA in vitro in suspension	Inhibited growth (indicated by turbidity)	92
	15-min exposure to 0.6–2.2 mg L^{-1} PAA in vitro in suspensions with various levels of alkalinity and humic substances	EC50 = 0.64–1.33 mg L^{-1} PAA (lower at lower alkalinity levels and in the absence of humic substances)	40
	2/5/10-min exposure to 1–10 mg L^{-1} PAA in vitro in suspensions prepared with RAS water	negligible CFU reduction after 10-min exposure to 2 mg L^{-1} PAA; >6 $\log_{10}$ CFU reduction after 5-min exposure to 5 mg L^{-1} PAA	93
	Single exposure to 0.3–1.2 mg L^{-1} PAA in vitro in reconstituted water at 4°C in darkness	Up to 48-h lag phase between exposure and bacterial cell death; many survived bacterial cells are viable but non-culturable	94
Carnobacterium piscicola (Gram$^+$)	30-min exposure to 0.1 g L^{-1} PAA in vitro in organics-enriched suspension	>5 $\log_{10}$ reduction in total numbers	90
Lactococcus garvieae (Gram$^+$)	30-min exposure to 0.1 g L^{-1} PAA in vitro in organics-enriched suspension	>5 $\log_{10}$ reduction in total numbers	90
Weissella ceti (Gram$^+$)	2/5/10-min exposure to 1–10 mg L^{-1} PAA in vitro in suspensions prepared with RAS water	Negligible CFU reduction after 10-min exposure to 2 mg L^{-1} PAA; >6 $\log_{10}$ CFU reduction after 10-min exposure to 10 mg L^{-1} PAA	93
Flavobacterium columnare (Gram$^-$)	Single application of 100–400 mg L^{-1} PAA solution in a central hole on inoculated agar plates in vitro (0.1 mL PAA solution on 10-mL agar)	Average 40% circular area outside central hole on agar plate without visual bacterial growth for 24 h; Higher efficacy from PAA products with lower PAA:H_2O_2 ratios	95
	2/5/10-min exposure to 1–10 mg L^{-1} PAA in vitro in suspensions prepared with RAS water	>3 $\log_{10}$ and >6 $\log_{10}$ CFU reduction after 5-min exposure to 1 and 3 mg L^{-1} PAA, respectively	93

(Continues)

TABLE 2 (Continued)

Target	Exposure	Effect	Ref
Aeromonas hydrophila (Gram$^-$)	20-min exposure to 25 mg L^{-1} PAA in vitro in suspension	4.2 $\log_{10}$ CFU reduction	96
	30-min exposure to 10 mg L^{-1} PAA in vitro in suspension	3.6 $\log_{10}$ CFU reduction	
	30-min exposure as biofilm	4.9 $\log_{10}$ CFU reduction at 100 mg L^{-1} PAA	
Piscirickettsia salmonis (Gram$^-$)	1-min exposure to 10 mg L^{-1} in vitro in organics-enriched suspension	No CFU formation	97
Vibrio anguillarum (Gram$^-$) *Vibrio harveyi* (Gram$^-$) *Vibrio alginolyticus* (Gram$^-$) *Photobacterium damselae* subspecies *piscicida* (Gram$^-$)	10-min exposure to 2–4 mg L^{-1} PAA in vitro in suspension and as biofilm	Inhibited growth for all four species (indicated by turbidity)	98
Cyanobacteria			
Microcystis aeruginosa	Single exposure in culture media	45% microcystin MC-LR reduction at 3 mg L^{-1} PAA; 2 $\log_{10}$ reduction of chlorophyll-a concentration and the viable cell density at 7.5 mg L^{-1} PAA	99
Aquaculture pond water with cyanobacteria	Single exposure to 1.5 mg L^{-1} PAA (supplemented with 10% culture media)	Eradication of phycocyanin and mild reduction of chlorophyll-a concentration	100
Nitrifying bacteria			
Mixed nitrifying bacterial culture	Single exposure to 1–3 mg L^{-1} PAA in suspension	Inhibition of enzymes for ammonia removal and nitrate production; harms *Nitrosomonas* but favours *Nitrosospira*	101
Oomycetes			
Saprolegnia parasitica	1-h exposure in vitro in suspension	In vitro: inhibited growth at 3 mg L^{-1} PAA and negligible growth at 5 mg L^{-1} PAA; In vivo: both concentrations were ineffective to prevent saprolegniasis in rainbow trout eggs	37
	Single exposure in vitro in agar containing 1–6 mg L^{-1} PAA	Average 40% growth reduction within 24 h; Higher efficacy from PAA products with lower PAA:H_2O_2 ratios	95
	Single exposure to 0.4–5 mg L^{-1} PAA in vitro in suspensions with various levels of alkalinity and humic substances	EC50 = 1.2–2.5 mg L^{-1} PAA (lower at lower alkalinity levels and in the absence of humic substances); resumed growth 24 h post treatment with all PAA concentrations	40
	Single exposure in vitro in agar containing ≥50 mg L^{-1} PAA	Inhibited visual mycelium growth for 6 days	102
	1-h exposure to ≥5 mg L^{-1} PAA in vitro in suspension followed by inoculation on PAA-free agar	Inhibited visual mycelium growth for 6 days	
Saprolegnia delica	Single exposure in vitro in agar containing ≥50 mg L^{-1} PAA	Inhibited visual mycelium growth for 6 days	
	1-h exposure to ≥5 mg L^{-1} PAA in vitro in suspension followed by inoculation on PAA-free agar	Inhibited visual mycelium growth for 6 days	
Saprolegnia hypogyna	1-h exposure in suspension	In vitro: inhibited growth at 3 mg L^{-1} PAA and negligible growth at 5 mg L^{-1} PAA; In vivo: both concentrations were ineffective to prevent saprolegniasis in rainbow trout eggs	37

TABLE 2 (Continued)

Target	Exposure	Effect	Ref
Multiple strains of *Aphanomyces* and *Saprolegnia*	5-min exposure to 100 mg L^{-1} PAA in vitro in suspension	No visible growth	103
Saprolegniasis	Daily double exposures to 2.5–5 mg L^{-1} PAA in vivo with channel catfish eggs	Inhibited visual mycelium growth and 50% increase of egg survival	41
	Daily exposure to 0.2, 0.5 and 1 mg L^{-1} PAA in vivo with Atlantic salmon parr post-vaccination against bacterial infections	Reduced Saprolegniasis and increased fish survival over 6 weeks	104
	Daily (weekdays) 30-min exposure to 0.2 and 0.5 mg L^{-1} PAA in vivo with Atlantic salmon fries	Insignificantly improved fish survival and unaffected concentration of genomic DNA and culturability of *Saprolegnia* spp. in rearing water over 4 weeks	12
Fish viruses			
Infectious salmon anaemia virus	5-min exposure to 200 and 600 mg L^{-1} PAA in vitro in suspension	>4 $\log_{10}$ titre reduction	105
Infectious pancreatic necrosis virus	30-min exposure to 138 mg L^{-1} PAA in vitro in suspension	>4 $\log_{10}$ titre reduction	90
Koi herpesvirus Viral haemorrhagic septicaemia virus	Single exposure in vitro in suspension	Complete viral deactivation at ≥400 mg L^{-1} PAA and 2 $\log_{10}$ titre reduction at 40 mg L^{-1} PAA	106
	Twice per week 1-h exposure to 1 mg L^{-1} PAA in vivo with rainbow trout	Retarded fish mortality probably through inhibition of bacterial co-infections; no direct evidence for viral inhibition	107
Fish parasites			
Neoparamoeba perurans	15- or 30-min exposure to ≥2.4 mg L^{-1} PAA in vitro in suspension	Reduced viability	108
	30-min exposure to 5 mg L^{-1} PAA in vivo with infected fish	Reduced abundance of *Ne. perurans* DNA in gill swabs after 4 weeks	109
	30-/60-min exposure to 5 mg L^{-1} PAA in vivo with infected fish	Neither reduced infestation nor improved histology within 2 weeks	110
Vannella spp.	Single exposure to ≥0.03 mg L^{-1} PAA in vitro in suspensions	100% mortality within 21 h	111
Pfiesteria spp.	Single exposure to ≥1.5 mg L^{-1} PAA in vitro in suspensions	Better efficacy at higher PAA concentrations; more profound cyst formation and resumed growth (in the present of feed) at lower PAA concentrations	112
	Repeated daily exposures to ≥1.5 mg L^{-1} PAA in vitro in suspensions for 5 days		
Ichthyophthirius multifiliis (theronts)	Single exposure to 0.4 mg L^{-1} PAA in vitro	up to 95% mortality within 1 h	113
Ich. multifiliis (tomonts)	Single exposure to 0.9 mg L^{-1} PAA in vitro	82% mortality in freshly harvested tomonts at within 48 h; reduced efficacy with longer settling and development period	44
Ichthyophthiriasis	3 times $^{-1}$week^{-1} exposures to 5.2 mg L^{-1} PAA in vivo with Atlantic salmon in concrete tanks	No effect on the number of infested trophonts	58
	Single exposure to 0.5, 1 and 2 mg L^{-1} PAA in vivo with experimentally infested pike perch in aquaria	No effect on the number of infested trophonts	44
	Continuous exposure to 1 mg L^{-1} PAA in vivo with experimentally infested carp in aquaria for 4 days	Reduced infestation	114
	Daily continuous in vivo exposure to 0.1–0.15 mg L^{-1} PAA for 10 h in spring and summer	Successful prevention of ichthyophthiriasis and improved gill health over two consecutive years	115

(Continues)

TABLE 2 (Continued)

Target	Exposure	Effect	Ref
Cryptocaryon irritans (theronts)	1-h exposure to ≥10 mg L^{-1} PAA	~80% mortality	116
Trichodina jadronica	Single exposure to 2.25 mg L^{-1} PAA in vivo in an eel RAS with 1-h isolation of biofilter	Effective killing of parasites yet followed by reoccurrence in a few days	43
Trichodina spp.	Daily exposures to 1 mg L^{-1} PAA in vivo in a carp RAS for 1 week with reduced feeding	Effective killing of parasites without reoccurrence	Authors' experience
Ichthyobodo necator	Daily exposures in vivo in channel catfish flow-through tanks for 3 days	No effect on reducing fish mortality at 1.5 mg L^{-1} PAA; Increased fish mortality after first exposure to 3 mg L^{-1} PAA	117
Ich. necator	2-h exposure to 0.3 mg L^{-1} PAA in vivo with experimentally infested rainbow trout fries	Over 90% reduced infestation	118
Lepidotrema bidyana	Single exposure in vitro	40% and 60% mortality at 1 and 2 mg L^{-1} PAA within 100 min, respectively	119
Co-infections			
Ich. multifiliis, *Trichodina centrostrigeata* and *Ae. hydrophila*	Repeated exposures to 1 mg L^{-1} PAA in vivo at a Nile tilapia farm Week 1: twice per day Week 2–4: twice per week	Eradicated infestation of *Ich. multifiliis*/*T. centrostrigeata* and infection of *Ae. hydrophila* from Day 7; Improved tissue morphology and reduced mortality over 4 weeks	120
Microbial community			
Biofilm and water in duplicate rainbow trout RASs	Double applications of 1.1 mg L^{-1} PAA in pump sumps; repeated 1, 2 and 4 times $^{-1}$week^{-1} for 13 weeks	No selective effect on microbial community; reduced total ammonia concentration and increased fish mortality with higher frequency of PAA application (IPNV-infection reported in the first 2 weeks)	121
Biofilm and fish gill mucus in triplicate rainbow trout flow-through raceways	Continuous application of 0.2 mg L^{-1} PAA in raceway; twice per week application of 1 mg L^{-1} for 1 h (w/ and w/o pH-neutralisation) in raceway	No selective effect on microbial communities over 6 weeks (VHSV-infection reported from the second week)	107
Off-flavour			
Rainbow trout RAS	Double applications of 1.1 mg L^{-1} PAA in pump sumps; repeated 1, 2 and 4 times $^{-1}$week^{-1} for 13 weeks	Lower concentrations of geosmin and 2-methylisoborneol in biofilms, water and fish filets at higher PAA application frequencies; no effect on temporal increase of geosmin synthase DNA, geosmin and 2-methylisoborneol in all RAS units at any PAA application frequencies	122

Abbreviations: CFU, colony forming unit; IPNV, infectious pancreatic necrosis virus; MC-LR, microcystin-LR; PAA, peracetic acid; RAS, recirculating aquaculture system; VHSV, viral haemorrhagic septicaemia virus.

minimum of 3.9 mg L^{-1} PAA was needed to effectively inhibit bacterial growth.[92] Suspensions of *Piscirickettsia salmonis* were exposed to 10, 500 and 3000 mg L^{-1} PAA in vitro for 1, 5 and 30 min in the presence of interfering substances following British Standards EN 1656:2009. The authors did not specify whether the tests were conducted in a low- or high-level soiling condition. The low-level soiling condition corresponds to 3 g L^{-1} bovine albumin, while the high-level soiling condition corresponds to 10 g L^{-1} yeast extract and 10 g L^{-1} bovine albumin. The 1-min exposure to 10 mg L^{-1} PAA resulted in zero formation of CFUs. This concentration, however, was selected for the disinfection of facilities and equipment in the absence of fish.[97]

Planktonic and biofilm cells of *Vibrio anguillarum*, *Vibrio harveyi*, *Vibrio alginolyticus* and *Photobacterium damselae* subspecies *piscicida* were treated in seawater with a single dose of 2, 4 and 20 mg L^{-1} PAA. The minimum inhibitory concentrations (MICs) of PAA against both planktonic and biofilm bacteria were assessed by the turbidity of cell suspensions and were between 2 and 4 mg L^{-1} PAA for all tested species; a quick effect within 10 min was also reported.[98]

Planktonic cells of *Y. ruckeri* were exposed to serial concentrations of PAA (0.6–2.2 mg L^{-1}) in reconstituted water with three levels of total alkalinity (7.5, 75 and 375 mg L^{-1}) and two levels of humic substances (0 and 10 mg L^{-1}) for 15 min, and subsequently inoculated the bacterial cells on agar plates. The bacterial growth was assessed by counting CFUs after 96 h incubation. The EC50 values of PAA (causing 50% reduction of CFUs) varied between 0.64 and 1.33 mg L^{-1} and were lower at lower alkalinity levels and in the absence of humic substances.[40]

The in vitro effect of PAA at 1–10 mg L^{-1} was investigated after an exposure period of 2–10 min on the growth of *Y. ruckeri*, *Weissella ceti* and *F. columnare*, respectively. The exposure was conducted in cell suspensions prepared with water freshly collected from two stocked and fully operating RASs. Water from RAS 1 had a total alkalinity of 141 mg L^{-1} and was used for *Y. ruckeri* and *W. ceti*. Water from RAS 2 (with a higher exchange rate than RAS 1) had a total alkalinity of 255 mg L^{-1} and was used for *F. columnare*. The exposed bacterial suspensions were inoculated on agar plates and cultivated at 30°C for 24 h. A 10-min exposure to 2 mg L^{-1} PAA resulted in negligible CFU reduction for *Y. ruckeri* and *W. ceti*, but >4 $\log_{10}$ CFU reduction for *F. columnare*. Greater CFU reduction (>6 $\log_{10}$) was reported with at least 5, 10 and 3 mg L^{-1} PAA with at least a 5-min exposure for *Y. ruckeri*, *W. ceti* and *F. columnare*, respectively; the authors suggested that the different susceptibility of the three bacterial species to PAA was related to the protection of the cell wall, since *W. ceti* was the only $Gram^{+}$ pathogen assessed.[93]

5.1.3 | Cyanobacteria

Suspensions of *Microcystis aeruginosa* in culture media were treated with 1.5, 3 and 7.5 mg L^{-1} PAA and the kinetic changes of chlorophyll-a, cell viability and microcystin-LR (MC-LR, determined in ultrasonicated cell suspensions) were observed at 0, 24, 48 and 72 h post-treatment. Results showed that only 7.5 mg L^{-1} PAA reduced the chlorophyll-a concentration and the viable cell density by 2 $\log_{10}$; the effect of PAA was more profound on chlorophyll-a than cell viability. In comparison, the reductive effect of PAA on microcystin MC-LR was stronger; a reduction of 45% was caused by 3 mg L^{-1} PAA.[99]

The effectiveness of PAA against cyanobacteria was tested in a 14-day lab study and a 35-day field study. The lab study was conducted with 500-μm filtered water collected from eutrophic aquaculture ponds and supplemented with 10% BG-11 media. Treatments were 0.3, 0.75, 1.5 and 1.8 mg L^{-1} PAA and measurements of chlorophyll-a and phycocyanin were conducted on Days 0, 1, 3, 5, 7, and 14 post-treatments. The field study was conducted in mesocosms floating in a eutrophic aquaculture pond filled with pond water sieved through 200-μm mesh to exclude large debris. A single treatment with 1.5 mg L^{-1} PAA was performed and measurements of chlorophyll-a and phycocyanin were conducted on day 1, 3, 7, 14, 21, 28, and 35 post-treatments. Results of the lab study showed that 1.5 mg L^{-1} PAA reduced the phycocyanin concentration from about 1 mg L^{-1} pre-treatment to an undetectable level on Day 1 post-treatment followed by minimal recovery till Day 14. In parallel, the chlorophyll-a concentration of near 600 μg L^{-1} pre-treatment was reduced progressively by 1.5 mg L^{-1} PAA, but never to an undetectable level. Results from the field study showed a similar effect of 1.5 mg L^{-1} PAA on phycocyanin concentration. Interestingly, PAA's effect on chlorophyll-a was much milder than shown in the laboratory study and the effect of PAA on zooplankton biomass was also marginal[100]; this could be important information for future studies to investigate.

5.1.4 | Nitrifying bacteria

It was reported that 1–3 mg L^{-1} PAA inhibited ammonia removal and nitrate production of nitrifying bacterial suspensions. Transcriptional analysis of the cells revealed that the inhibitory effect was predominantly associated with enzyme inhibition rather than cell mortality. Moreover, *Nitrosomonas* was negatively affected by PAA, while *Nitrosospira* seemed to benefit from it.[101]

5.1.5 | Viable but non-culturable state

The effect of PAA at 0.3–1.2 mg L^{-1} was tested in vitro on the growth and viability of two *Y. ruckeri* isolates. The culturability was analysed via cultivation of bacterial suspensions after 15-min exposure to PAA on agar plates and CFU assessment after 72-h incubation. The viability was analysed via membrane integrity-based differential staining (Sybr Green and propidium iodide) followed by flow cytometry with bacterial suspensions in time series (2, 24, 48 and 72 h) after a single exposure to PAA (suspensions were constantly kept at 4°C in darkness). A distinct lag phase of up to 48 h between the PAA exposure and the onset of viability losses of both bacterial isolates was detected. Abundant viable cells post-PAA exposure were unable to reproduce and form colonies on agar plates.[94] Although this is a study with only one pathogenic bacterial species, the viable but non-culturable (VBNC) state is a general defence strategy in bacteria against sublethal stress and an unfavourable environment. It allows bacteria to survive and resuscitate when the environment becomes favourable again.[123,124] Therefore, PAA disinfection at safe concentrations is bacteriostatic rather than bactericidal. Although species/strain-dependent susceptibility may exist, it is unlikely for PAA disinfection at these concentrations to eradicate the entire bacterial population. The cultivation-based method is unable to detect the VBNC populations, and therefore may provide overly optimistic results.

5.2 | Oomycetes/saprolegniasis

It was reported that 1-h exposure to 3 mg L^{-1} PAA was able to inhibit the in vitro growth of *Saprolegnia parasitica* and *Saprolegnia hypogyna* 48 h post-exposure. Exposure to 5 mg L^{-1} PAA resulted in visually undetectable growth. However, in vivo tests with the same concentrations failed to prevent infection in rainbow trout eggs inoculated with either *Saprolegnia* species. Both concentrations were reported to be toxic to rainbow trout eggs.[37] The inhibitory effect of PAA was tested in vitro on the growth of multiple species of *Aphanomyces* and

Saprolegnia. Exposure to 100 mg L^{-1} PAA for at least 5 min stopped visual growth and similar control was realised by 1 h exposure to at least 500 mg L^{-1} H_2O_2.[103]

PAA products with various PAA:H_2O_2 ratios were tested in vitro to reduce the vegetative growth of *Sap. parasitica* on agar plates. Warm liquid agar was mixed with 10-fold PAA stock solutions in 9:1 ratio to produce 0.5, 1, 2, 4, 6, 8 and 10 mg L^{-1} PAA in the solid agar (assuming no instant PAA decay). The inhibitory effect was assessed with the maximum diameter of the growth area of *Sap. parasitica* 24 h post-inoculation at the centre of the agar plates compared with a negative control. At 1 mg L^{-1} PAA, 40% growth reduction was observed using two PAA products with the lowest PAA:H_2O_2 ratios; other PAA products required 4–6 mg L^{-1} PAA to induce similar growth reduction. The authors suggested that H_2O_2 played an important additive effect.[95] Although mixing PAA with liquid agar resulted in equal concentrations of PAA and H_2O_2 in solid agar, their decay was generally accelerated in agar (DL/TM lab, personal experience). As current analytical methods are compatible in aqueous solutions only, the fate of PAA and H_2O_2 in solid agar remains difficult to assess.

PAA was tested in vivo to control saprolegniasis in flow-through hatching troughs (28-min water exchange rate) on channel catfish eggs until eye development could be identified. Compared with the untreated control, where cumulative survival was ~11%, daily doses of 2.5–5 mg L^{-1} PAA significantly inhibited visual growth of *Saprolegnia* spp. and greatly increased the egg survival by approximately 50%. PAA concentration ≥ 10 mg L^{-1} began to show a toxic effect on the eggs.[41]

The effect of PAA on the in vitro growth of two strains of *Sap. parasitica* and one strain of *Saprolegnia delica* was determined. Tests were performed by either inoculating mycelium of all three strains onto solid agar incorporated with PAA, or by submerging mycelium in PAA solution prepared with sterile, deionised water for 1 h then inoculating on PAA-free agar. The MIC of PAA to mycelium growth for 6 days was determined to be 50 mg L^{-1} PAA incorporated in solid agar and 5 mg L^{-1} PAA in sterile deionised water.[102] This supports the notion of rapid degradation of PAA and H_2O_2 in solid agar.

Single doses of 0.2, 0.5 and 1.0 mg L^{-1} PAA were applied daily for 6 weeks in culture tanks of RAS stocked with Atlantic salmon parr (~94 g) post-vaccination. Natural infection of *Saprolegnia* spp. on fish skin was less often observed in tanks receiving higher PAA concentrations. All PAA-treated tanks resulted in significantly higher fish survival than untreated tanks, although the targeted post-vaccination saprolegniasis outbreaks in control fish, as typically observed in the aquaculture industry, was not achieved under these study conditions.[104]

Either 15 mg L^{-1} H_2O_2 or 0.2 and 0.5 mg L^{-1} PAA was used to reduce early-stage mortality of Atlantic salmon alevin (~0.5 g) associated with saprolegniasis. Fish were exposed to a 30-min static bath of either treatment which were repeated daily (Monday through Friday) for 4 weeks. The alkalinity was 274–276 mg L^{-1}. The H_2O_2 bath significantly increased fish survival compared with control (deionised water sham treatment). The PAA bath at both concentrations appeared to improve fish survival, yet the effect was statistically insignificant. Neither disinfectant affected the abundance of genomic DNA and the ability to culture *Saprolegnia* spp. from the rearing water.[12]

Inocula from 3-day-old *Sap. parasitica* agar were treated in 24-well plates with PAA solutions at serial concentrations (0.4–5 mg L^{-1}) prepared with reconstituted water at 3 levels of alkalinity (7.5, 75 and 375 mg L^{-1}) and at 2 levels of humic substances (0 and 10 mg L^{-1}). Vegetative growth was assessed by mycelium length, and growth reduction was compared with a negative control. The EC50 values of PAA varied between 1.2 and 2.5 mg L^{-1} PAA and were lower at lower alkalinity levels and in the absence of humic substances. Resumed growth was observed 24 h post-treatment at all PAA concentrations.[40]

5.3 | Fish viruses

The in vitro effects of PAA and several other disinfectants on cultured infectious salmon anaemia (ISA) virus were observed in reconstituted hard water at 4°C. PAA was quenched 5 min after ISA exposure with a catalase/sodium thiosulphate solution. A titre reduction of >4 $\log_{10}$ was reported after exposure to 0.02% and 0.06% PAA (200 and 600 mg L^{-1}).[105] The in vitro virucidal activity of PAA against infectious pancreatic necrosis virus (IPNV) was evaluated. Concentrations of ≥0.276% the PAA product (138 mg L^{-1} PAA) produced >4 $\log_{10}$ titre reduction with a contact time of 30 min at 4°C.[90]

The in vitro virucidal effects of various agents, including PAA, against Koi herpesvirus and viral haemorrhagic septicaemia virus (VHSV) were investigated at 24 h and 8°C. It was reported that ≥400 mg L^{-1} PAA (pH = 4.2) resulted in complete viral deactivation and 40 mg L^{-1} PAA (pH 6.9) reduced titres by 2 $\log_{10}$ for both viruses.[106]

A recent study (DL/TM lab; manuscript submitted) indicated that the mortality in rainbow trout flow-through raceways caused by a naturally occurring VHSV infection was slowed by twice per week application of 1 mg L^{-1} PAA in the raceway water for 1 h compared with the untreated control raceways. No direct evidence of viral inhibition by PAA was found in trout gill samples, but a strong non-selective antibacterial effect on both planktonic and biofilm bacteria was detected. Therefore, the slowed mortality was probably through inhibition of bacterial co-infections.[107]

5.4 | Fish parasites

5.4.1 | Protozoa parasites

Amoeba

Neoparamoeba perurans, the causative agent for amoebic gill disease in marine fish, were exposed to 0.6, 2.4, 4.8 and 9.6 mg L^{-1} PAA in vitro for either 15 or 30 min. Significantly reduced viability was observed with PAA concentrations ≥2.4 mg L^{-1} regardless of exposure duration.[108] A freshwater amoebic species, *Vannella* sp., was isolated from Danish rainbow trout farms. An aliquot containing >20 live specimens was exposed in vitro to serial concentrations of 0.01–

0.5 mg L^{-1} PAA (15% Aqua-Oxides) for 21 h. It was reported that PAA at ≥0.03 mg L^{-1} resulted in 100% mortality of the amoeba.[111] *Ne. perurans*-infected Atlantic salmon smolts (80–90 g) were treated with a single dose of PAA either at 5 mg L^{-1} for 30 min or 10 mg L^{-1} for 15 min. Only the treatment with 5 mg L^{-1} PAA for 30 min resulted in significant reductions of *Ne. perurans*-DNA in gill swabs 4 weeks post-treatment.[109] *Ne. perurans*-infected Atlantic salmon smolts (70 g) were treated with single baths of 5 mg L^{-1} of three different PAA trade products for either 30 or 60 min. None of the PAA treatments resulted in significantly reduced infestation. However, PAA-treated groups for 30 min showed lower macroscopic gill scores than the infected-untreated fish. Microscopic scoring of gill injuries showed that amoebic gill disease (AGD)-infected PAA-treated fish had lower scores; however, an overall trend among the different PAA trade products could not be established. The effectiveness of PAA towards AGD is not only dependent on the dose, duration of exposure or type product, but also the severity of infection.[110]

Ichthyophthirius multifiliis *(Ich)*

Several compounds including two PAA products were tested as alternatives to malachite green either alone (Per Aqua, Nordic Breentag, 40 mg L^{-1} product = 5.2 mg L^{-1} PAA) or in combination (Desirox, Finnish Peroxides, 10 mg L^{-1} product = 1.3 mg L^{-1} PAA) with 100 mg L^{-1} formalin to treat natural Ichthyophthiriasis in 1-year-old Atlantic salmon reared in flow-through concrete tanks at two farms. The treatments were performed 3 times $week^{-1}$. During each treatment, the water volume in each tank was reduced from 50 to 15 m^3 before treatment; at one farm water flow was stopped but not at the other farm. The water volume was restored 2 h later. Neither the PAA alone nor in combination with formalin controlled the growing number of trophonts on fish skin during the early infection. However, the combination of PAA and formalin was associated with lower fish mortality than PAA alone and were administered in the first 4 weeks after the start of an infection to allow the fish to develop immunity against these ciliates.[58]

Single doses of 0.5, 1, 1.5 and 2 mg L^{-1} PAA were tested to treat experimentally induced Ichthyophthiriasis in juvenile pike perch (9–12 cm). None of the PAA concentrations reduced the number of trophonts on the mucosal surface or the fish mortality; however, it was proposed that a successful treatment of Ichthyophthiriasis with such low concentrations could possibly be achieved by targeting the free-living stages, such as theronts and tomonts.[44] Theronts were found to be most sensitive to PAA treatments in vitro; despite varied resistance among *Ichthyophthirius multifiliis* strains to certain PAA products, the 4-h LC50 values of the PAA products tested were in the range of 0.1–0.3 mg L^{-1} PAA. Most theronts were killed within 1 h post-treatment, and 0.4 mg L^{-1} PAA caused up to 95% mortality in theronts.[113] The effect of PAA in vitro on the survival of tomonts freshly harvested from fish and the theront-producing ability of tomonts that were allowed to settle for 2.5 and 24 h after harvest were tested. An 82% mortality was observed in freshly harvested tomonts treated with a single dose of 0.9 mg L^{-1} PAA and incubated for 48 h. In comparison, the mortality of control tomonts without PAA treatment was <5%. The settling period increased the resistance of tomonts to PAA. Exposure of 2.5 h-settled tomonts to a single dose of 0.5, 1 and 2 mg L^{-1} PAA resulted in 42%, 75% and 98% fewer released theronts than without PAA treatment. However, treatment with the same PAA concentrations was ineffective to reduce theronts released from 24 h-settled tomonts. Concentrations higher than 2.5 mg L^{-1} PAA were necessary to damage the tomites in the encysted tomonts but had reduced efficacy.[125] For this reason, the most efficient strategy to prevent or reduce losses from Ichthyophthiriasis is to eradicate the theronts and early stage tomonts. This requires continuous treatment with PAA for a minimum number of 4 days. In aquaria, PAA was continuously applied via a peristatic pump containing common carp (~471 g) that were experimentally infested with *Ich. multifiliis*. The use of the peristaltic pump aimed to maintain 1 mg L^{-1} PAA concentration for 4 days but resulted in varied PAA concentrations from below detection limit to 2.5 mg L^{-1} (measured with a test strip from Merck KGaA). The abundance of parasites on the skin and fin were significantly lower in PAA-treated fish than PAA-untreated fish.[114] In a production-scale flow-through trout farm, PAA was applied via a dosing pump at the main inlet. PAA concentrations in culture tanks varied in the range of 0.1–0.15 mg L^{-1} depending on the distance to the dosage pump. The fish farmer reported successful prevention of Ichthyophthiriasis and improved gill health over 2 consecutive years by using this method for 10 h daily in spring and summer.[115]

Cryptocaryon irritans *(marine Ich)*

A study on the efficacy of two PAA products against *Cryptocaryon irritans* showed that a 1-h treatment with ≥10 mg L^{-1} PAA was required to produce ~80% mortality of theronts in vitro.[116] The authors recommended scheduling PAA treatment between dusk and dawn because most *Cr. irritans* theronts and trophonts are released after dark.[126] Compared with freshwater *Ich. multifiliis*, the higher resistance of marine *Cr. irritans* to PAA probably results from excessive PAA degradation in seawater.[23]

Trichodina *spp.*

It was determined that 2.25 mg L^{-1} PAA (calculated from 45 mg L^{-1} of a 5% PAA product, Detarox AP®) is effective in killing *Trichodina jadronica* found in eel RAS after isolating the biofilters for 1 h. However, several days later, it was found that *T. jadronica* had reappeared in the system. The authors hypothesise that some trichodinids were in the biofilters and were therefore not affected by the treatment; they suggest that several more bath treatments may be necessary to keep trichodina numbers low.[43] Taking fish safety into consideration, daily treatment with 1 mg L^{-1} PAA for 1 week accompanied by a reduction in feeding rate was effective against *Trichodina* (re)infestation on common carp in a pilot-scale RAS (DL's experience).

Ichthyobodo necator

Juvenile channel catfish naturally infested with *Ichthyobodo necator* were treated with 1.5 and 3 mg L^{-1} PAA for 3 days in flow-through tanks (213 mg L^{-1} alkalinity). Increased fish mortality was observed after the first treatment with 3 mg L^{-1} PAA. The 1.5 mg L^{-1} PAA treatment was unsuccessful in reducing fish mortality, and the intensity of

infestation was not reduced until 7 days post-treatment.[117] Optimistic results were found in another study, where a single 2-h treatment with only 0.3 mg L^{-1} PAA in static water (108 mg L^{-1} alkalinity) eliminated over 90% *Ich. necator* experimentally infested on rainbow trout fry (2–3 g).[118] We propose that: (1) naturally infested *Ich. necator* might have better adaptation on host fish than experimentally infested ones; (2) treatment in flow-through tanks resulted in a dilution effect on the concentration of PAA and/or (3) higher alkalinity reduced the efficacy of PAA (see Section 6 for more details).

Dinoflagellates

An in vitro trial was conducted with PAA against the harmful dinoflagellate *Pfiesteria* spp. PAA was delivered either in single doses of 1.5 and 7.5 mg L^{-1} or in repeated daily doses of 1.5 mg L^{-1} for 5 days. All treatments killed *Pfiesteria* cells within 5 days, and the most profound effects were observed with the single dose of 7.5 mg L^{-1} PAA and the daily repeated 1.5 mg L^{-1} PAA. However, cyst formation was observed along with cell mortality and was most prevalent in the single dose treatment of 1.5 mg L^{-1} PAA. Unfortunately, when food was supplied after the PAA treatments, growth resumed in all treatments and was highest in *Pfisteria* that were treated with a single dose of 1.5 mg L^{-1} PAA.[112]

5.4.2 | Metazoa parasites

PAA was tested in vitro against the monogenean *Lepidotrema bidyana* isolated from naturally infested silver perch (*Bidyanus bidyanus*). The study showed that the 100-min EC90 value (causing 90% mortality) of PAA to *Lepi. bidyana* was 4.6 mg L^{-1}. Single in vitro treatments with 1 and 2 mg L^{-1} PAA resulted in approximately 40% and 60% mortality in *Lepi. bidyana* 100 min post-treatment, respectively.[119]

5.5 | Ichthyotoxic microalgae

The effectiveness of PAA and other oxidising disinfectants against two euryhaline ichthyotoxic microalgae species, *Prymnesium parvum* and *Heterosigma akashiwo*, were tested. Both species were associated with fish mortality in natural and aquaculture environments. Algal suspensions in standard enriched seawater culture medium (f/2 medium) with an initial density at approximately 10^4 cells mL^{-1} were exposed to a single dose of PAA at a serial concentration of 0.1–2 mg L^{-1}. Significant growth reduction of *P. pavum* was observed within 14 days post-exposure to 1 mg L^{-1} PAA, while *He. akashiwo* was resistant to PAA up to 2 mg L^{-1}. The estimated EC90 values of PAA against *P. pavum* and *He. akashiwo* was 0.58 and 28.14 mg L^{-1}, respectively. Faster degradation of PAA was observed in *He. akashiwo* culture than in *P. pavum* culture.[127]

5.6 | Co-infections in fish

Mass mortality at a Nile tilapia (*Oreochromis niloticus*) farm caused by concurrent co-infection with *Ich. multifiliis*, *Trichodina centrostrigeata* and *Ae. hydrophila* was reported. The fish (~80 g) were maintained in concrete tanks with 10% daily water exchange and treated with PAA to attempt to control the infection and reduce fish mortality. In the first week, 1 mg L^{-1} PAA was applied in tank water (828 mg L^{-1} total alkalinity) twice per day. Afterwards, 1 mg L^{-1} PAA was applied twice per week. The infestation of *Ich. multifiliis* and *T. centrostrigeata* on the skin of PAA-treated fish showed reduction beginning on Day 2 and disappearing by Day 7. The infection of *Ae. hydrophila* in the inner organs of PAA-treated fish were also absent by Day 7. In contrast, the untreated control fish showed unchanged external parasitical infestation and internal bacterial infection. After PAA treatment for 1 month, the PAA-treated fish showed improved morphology of gill, skin, liver and kidney as well as lower mortality compared with untreated fish.[120]

5.7 | Microbial community

Twice-daily doses of 1.1 mg L^{-1} PAA were applied in the sumps of pilot-scale rainbow trout RASs. Compared with the negative control treatment, repeating the daily PAA application 1, 2 and 4 times $^{-1}$week^{-1} for 13 weeks did not impact the microbial community composition in the fish culture tank or biofilter biofilms. Higher frequency of PAA application was correlated with lower concentration of total ammonia nitrogen in water and higher fish mortality. Transient decrease in the abundance of ammonia-oxidising bacteria and nitrite-oxidising bacteria was observed at the beginning of PAA treatments but was followed by adaptive recovery. The water alkalinity varied between 40 and 70 mg L^{-1} and pH values varied between 7.1 and 7.4. IPNV-associated fish mortality was reported in all RAS units in the first 2 weeks. The clinically unhealthy fish were subsequently removed and the stocking density in tanks were equalised at 12.5 kg m^{-3}.[121]

Liu and his colleagues (DL/TM lab; manuscript submitted) reported that the temporal shift of the microbial communities on biofilms attached on submerged glass slides and trout gill mucus at a flow-through farm was affected by the microbial community in the upstream water. The gill mucus microbial community had a close relationship with the upstream water microbial community, while the biofilm retained a specific microbial community. PAA applied either continuously at 0.2 mg L^{-1} or twice per week at 1 mg L^{-1} with and without pH neutralisation (via 1:1 mixture of a 15% PAA product with 10%–11% NaOH) for 1 h had no selective effect on the microbial communities of both biofilm and gill mucus over 6 weeks. VHSV-infection in all raceways was reported from the second week.[107]

5.8 | Off-flavour

Twice-daily treatments of 1.1 mg L^{-1} PAA were applied in the sumps of RAS and repeated 1, 2 or 4 times per week; water from sumps flowed directly into fish culture tanks. The concentrations of geosmin and 2-methylisoborneol (the muddy/earthy off-flavour metabolites

produced by a range of bacteria) in the water of fish tanks and pump sumps 8 and 13 weeks after treatments showed a significant decreasing trend along with the increase of PAA treatment frequency. A similar trend was also observed with geosmin and 2-methylisoborneol in fish filets 13 weeks after treatment. Regardless of the PAA application frequency, the concentrations of geosmin and 2-methylisoborneol showed general increases in all RAS units. The geosmin synthase DNA, mainly from *Streptomyces*, was most abundant in the biofilm of biofilters but was also detectable in the biofilm and water from other RAS units. None of the PAA application frequencies affected the abundance of geosmin synthase DNA and its general increase in all RAS units.[122]

6 | REDOX POTENTIAL AND ITS POTENTIAL USES IN PRACTICE

6.1 | Definition and impact from pH

According to the IUPAC Gold Book definition[128]: 'Any oxidation-reduction (redox) reaction can be divided into two half-reactions: one in which a chemical species undergoes oxidation and one in which another chemical species undergoes reduction. If a half-reaction is written as a reduction, the driving force is the reduction potential. If the half-reaction is written as oxidation, the driving force is the oxidation potential related to the reduction potential by a sign change. So, the redox potential is the reduction/oxidation potential of a compound measured under standard conditions against a standard reference half-cell. In biological systems, the standard redox potential is defined at pH 7.0 versus the hydrogen electrode and partial pressure of hydrogen = 1 bar'.

Caution was raised in a review article for using the redox potential values under standard conditions to predict the microbial kill-rate of a disinfectant in realistic conditions; a large variation of standard redox potential values was reported in the literature for PAA, and these may have been determined under varying reaction conditions (i.e., different pH values, etc.).[129] Water and wastewater disinfection are usually practiced at near neutral pH (i.e., 7.0), so this review calculated and compared redox potential values under biochemical standard state conditions (pH 7, 25°C and 101,325 Pa [i.e., 1 atmosphere]) versus the standard hydrogen electrode (SHE) and denoted *E*' as shown in Table 3. Biochemical standard state conditions best represent the conditions in aquaculture systems. This table also includes redox potential values under standard state conditions (pH 0.0 and 101,325 Pa [i.e., 1 atmosphere; most references also assume 25°C] vs. the SHE). Redox potential values under standard state conditions (denoted E°) are included because these values are often reported in the literature; however, it is important to note that they do not represent redox values in aquaculture environments. Redox potential values at biochemical standard state conditions are lower than at standard state conditions with the difference solely in pH values. Even within a small range of pH (7–9), any changes in pH values will strongly affect redox potential values measured in water.[141]

6.2 | Relationship with PAA concentration

In practice, the redox potential is measured with an electronic probe integrated with a standard reference electrode. Measurement of redox potential has been broadly used to control the dosage of ozone for the disinfection of RAS water. It was determined that in the range of 0–20 μg L^{-1} (ppb), the concentration of ozone showed a strong linear relationship with the measured redox potential value (330–600 mV) in a freshwater RAS.[142] In the case of PAA disinfection, the

TABLE 3 Redox potentials for common water and wastewater disinfectants under specific conditions.

Oxidant	Half-reaction	*E*'(V)[a]	E°(V)[b]	E°(V)[c]
Hydroxyl radical (·OH)	$OH_{(aq)} + e^- \leftrightarrow OH^-_{(aq)}$	2.386[d]	2.800[e]	2.020
Ozone (O_3)	$O_{3(g)} + 2H^+_{(aq)} + 2e^- \leftrightarrow O_{2(g)} + H_2O_{(l)}$	1.661	2.075	2.076
Peracetic acid (CH_3CO_3H)	$CH_3CO_3H_{(aq)} + 2H^+_{(aq)} + 2e^- \rightarrow CH_3CO_2H_{(aq)} + H_2O_{(l)}$	1.385	1.748	1.960
Chlorine gas (Cl_2)	$Cl_{2(g)} + 2e^- \leftrightarrow 2Cl^-_{(aq)}$	1.361[f]	1.361	1.358
Hydrogen peroxide (H_2O_2)	$H_2O_{2(aq)} + 2H^+_{(aq)} + 2e^- \leftrightarrow 2H_2O_{(l)}$	1.349	1.763	1.776
Hypochlorous acid (HOCl)	$HOCl_{(aq)} + H^+_{(aq)} + 2e^- \leftrightarrow Cl^-_{(aq)} + H_2O_{(l)}$	1.288	1.495	1.482
Chlorous acid	$HClO_{2(aq)} + 3H^+_{(aq)} + 4e^- \leftrightarrow Cl^-_{(aq)} + 2H_2O_{(l)}$	1.259[d]	1.570[g]	1.570
Chlorine dioxide (ClO_2)	$ClO_{2(g)} + H^+_{(aq)} + e^- \leftrightarrow HClO_{2(aq)}$	0.774[d]	1.188[h]	1.277

[a]Biochemical standard state conditions versus the standard hydrogen electrode (SHE).[129]
[b]Standard state conditions versus the SHE.[129]
[c]Standard state conditions versus the SHE.[14]
[d]Calculated using the E° value in the middle column and the Nernst equation[130] referring to Zhang, personal communication.
[e]Value reported in the literature.[131–139]
[f]H^+ is not involved in this reaction, therefore E° is equal to *E*'.
[g]Value reported in the literature[140] referring to Zhang, personal communication.
[h]Referring to Zhang personal communication, when chlorine dioxide is applied during water or wastewater disinfection, it is ultimately reduced to chloride ions (Cl^-), while chlorous acid could be an important intermediate.[140]

measured redox potential values in freshwater RAS water increased from 248 to 290 mV as the PAA concentration increased from 0.05 to 0.3 mg L^{-1}.[76] Unlike ozone, the relationship between PAA concentration and redox potential value is logarithmic instead of linear. In reconstituted water with an alkalinity of 7.5, 75 and 375 mg L^{-1}, increasing PAA concentration from 0 to 6 mg L^{-1} resulted in an increase of measured redox potential values from 250 to 280 mV to 470, 400 and 350 mV, respectively. The redox potential plateaued at lower PAA concentrations in lower water alkalinity.[40]

6.3 | Predicting effectiveness of PAA

The impact of pH on redox potential values and the logarithmic relationship between PAA concentrations and redox potential values hamper the measurement of redox potential to indicate the PAA concentration in complicated aquaculture conditions. Factors such as alkalinity, humic substances and suspended solids were found to affect the disinfection efficacy of PAA.[32,40] A small data set suggests it may be inappropriate to predict the disinfection efficacy of PAA with the concentration since there are other factors to consider. Perhaps, redox potential values seem to be in agreement with the disinfection efficacy of PAA. It was reported that varied EC50 values of PAA after a short exposure against the in vitro growth of *Y. ruckeri* and *Sap. parasitica* assessed at various alkalinity, pH and organic load conditions corresponded to a similar redox potential value; ~300 mV for *Y. ruckeri* and ~ 320 mV for *Sap. parasitica*.[40] This suggests that the redox potential could be the main driving force for the antimicrobial toxicity of PAA. If this is the case, the measurement of redox potential could be used to determine the disinfection efficacy of PAA instead of its active concentrations in aquaculture practice. Redox potential values might also be used to predict negative effects on fish. For example, in ozonated RAS, sustained elevation of redox potential at 300–320 mV was reported to induce stress in sea bass (*Dicentrarchus labrax*); redox potential over 320 mV resulted in fish mortality.[143] An ideal range of sustained redox potential between 270 and 300 mV was recommended as the best compromise between antimicrobial efficacy and fish welfare.[144] In addition, strong pH reduction caused by acidic compounds (mainly acetic acid and sulphuric acid) from PAA products, especially at low alkalinity conditions, showed additive negative effects on fish.[39,40] Therefore, control of pH is necessary in soft water aquaculture systems when using PAA for water disinfection.

In essence, a quick and inexpensive means to measure effectiveness of PAA in a water body would be beneficial to aquaculturists. The authors of this review suggest a method such as the redox potential or a way to measure oxidation potential (e.g., potassium permanganate demand[145]) of the water; however, more research should focus on this predictive method to treat with PAA. Caution is essential when using the redox potential to predict the microbial kill-rate of a disinfectant since the potency of a disinfectant is also affected by numerous other factors such as pH of the solution, concentration of the disinfectant, bacterial encapsulation, and so forth.[129] For instance, H_2O_2 has a fairly high redox potential and is a strong oxidising agent, but it has limited disinfection capabilities.[88,129,146–148]

It was reported that the reaction rate constant of PAA with organic compounds in water follows the order: sulphur-containing compounds > phenolic compounds > nitrogen-containing compounds > alkenes > aromatics with alkene group > aldehydes.[149] Different organisms (or their bio-synthetics) may vary in the amount and composition of these organic compounds, hence differences in susceptibility to PAA (bacteria > viruses > bacterial spores > protozoan cysts).[150] Nevertheless, disparity of susceptibility to PAA is unlikely to exist between similar taxonomic organisms and chemically similar molecules. Therefore, we suggest the clustering of reference effective redox potential values against typical pathogens that are susceptible to PAA at safe concentrations to fish. Based on the literature and our experiences summarised in Table 2, the most susceptible fish pathogens are bacteria and protozoan parasites (susceptible life stages). We suggest that there might be a universal reference effective redox potential value (range) against each cluster of pathogens (e.g., $Gram^+$ bacteria, $Gram^-$ bacteria, amoeba, ciliates and flagellates), or that there is another rapid method that could provide this information.

There are special cautions for the use of redox potential probes in aquaculture practice. The probes should not be constantly submerged in culture water because biofilm formation will result in false lower readings. Regular inspection and calibration are recommended to ensure long-term accuracy of the probes.

7 | HYDRODYNAMIC-DEPENDENT APPLICATION IN AQUACULTURE SYSTEMS

Water disinfection using PAA in production-scale aquaculture systems has not always been successful and has sometimes resulted in expensive lessons (authors personal communication and experience). A major reason for these failures is a lack of understanding of the hydrodynamic properties, which results in uneven distribution of PAA, and hence incorrect PAA concentration in system water. Basic hydrodynamic properties of typical aquaculture systems are summarised below, and the most appropriate PAA treatment strategies are recommended.

7.1 | Flow-through raceways versus tanks

Apart from vertical flows in areas near the inflow and outflow depending on their vertical positions, water generally flows in one direction in simple raceways.[151] In tanks, however, the flow is directed in circular motions for the ease of particle precipitation and removal from the outflow at the bottom centre drain.[152] Due to this difference, mixing of culture water is easier in tanks than in raceways. Therefore, homogenous distribution of PAA treatments in tanks can be achieved through vigorous aeration following a single dose. The best strategy in raceways is to maintain a stable PAA concentration in the inflow water; this is often done in practice via a dosing pump at

the inflow with mixing (via vigorous aeration or mechanical stirring).[107,115] Due to PAA degradation, a progressively lower PAA concentration is expected in areas with increasing distance from the dosing location. Because PAA has a half-life of 5–30 min in aquaculture water, depending on the degree of organic enrichment,[18,24] areas further down a raceway from the dosing point receive an active PAA concentration lower than desired.[115] As suggested previously, the measurement of redox potential may help to detect these areas. If the remote areas are large and stocked with fish, dosing at an intermediate spot might be necessary in tandem with dosing at the inflow.

7.2 | RAS versus flow-through

RAS differs from flow-through system in hydrodynamics by having a longer hydraulic retention time.[153] Depending on the amount of oxidisable content in the culture water, PAA can degrade slowly or quickly in RAS but will be flushed out by water exchange in flow-through systems. PAA disinfection in flow-through systems follows along with the timing of the PAA treatment. The risk of overdosing in flow-through systems is low and controllable. In comparison, PAA disinfection in RAS happens longer than the PAA treatment and may increase the risks of overdosing and affect the performance of biofilters. It was demonstrated that biofilter performance was negatively affected when exposed to over 1 mg L^{-1} PAA in experimental RAS.[17] To avoid this adverse effect, the biofilter can be isolated when PAA is applied to the culture tank at >1 mg L^{-1},[43] or PAA can be applied at <1 mg L^{-1} in the sump prior to the culture units without isolating the biofilter.[76,84,121,122] For the latter case, PAA degradation in culture units and the mechanical filter helps to further lower the PAA concentration in water arriving at the biofilter. PAA will be diluted when water flows to the downstream units, and the degree of dilution is higher when the downstream units consist of larger water volume than the original receiving unit.[21] In addition to monitoring key water quality parameters, we suggest monitoring the redox potential at least in the culture and biofilter units for the control of disinfection efficacy and biofilter bacteria safety.

8 | CONCLUDING REMARKS

Prevention is easier than a cure. This is particularly true in the case of using disinfectants (not limited to PAA) against aquaculture diseases. The purpose (and the capability) of PAA disinfection is to suppress undesirable microbial activities at a low-risk level instead of eradicating them during fish health episodes. This relies on regular disinfection during high-risk production phases, for example, when fish are juvenile and susceptible to opportunistic infections. When applying PAA in the presence of fish, the fish farmer must be confident and gain experience with balancing the tradeoff between fish safety and disinfection efficacy. In regular freshwater settings, 1–2 mg L^{-1} PAA (as the active compound instead of product) is generally safe for fish and effective against microorganisms. In brackish/seawater settings, the safe concentration can be higher due to fast degradation. Caution should be taken with fish fry and low water alkalinity. In either case, the safe concentration for fish must be reduced and/or alkaline (e.g., sodium bicarbonate) could be supplemented in water to enhance fish resistance. During the first PAA application, a safe approach is to add aliquots of PAA lower than desired to allow the fish to acclimatise. This also provides a chance for fish farmers to observe the fish's response and verify their understanding of the hydrodynamics and distribution of disinfection intensity in their systems. DPD-based chlor-tester can be used to estimate the approximate PAA concentration onsite, but the test residues should be properly disposed. A blank water sample should be measured prior to PAA application to exclude interference from other oxidants in water. The monitoring of other water quality parameters, in particular the pH (and nitrogenous compounds in RAS), is important to ensure fish safety (and the functionality of biofilter). Despite stress adaptation of fish to repeated PAA applications at safe concentrations, the accumulated oxidative stress from chronic exposures to PAA may cause mild histological and physiological changes. These changes are considered reversable when the oxidative stressor is withdrawn. From this aspect, periodic and semi-continuous application modes instead of full-continuous application mode allow fish to recover from potential oxidative damages during the intervals. Because the PAA toxicity is strongly affected by physiochemical water parameters, it is impossible to predict an ideal PAA concentration for each condition. To solve this problem, we propose a universal control of redox potential to ensure antimicrobial efficacy and simultaneously avoid overdosing of PAA. Regardless of variations in water parameters, a redox potential value of about 300 mV seems to be fish-safe and effectively antibacterial based on limited published studies. More studies are needed to verify the compatibility of this value and especially its usability in production practice. At fish-safe concentrations, PAA mainly impairs the reproductivity of microorganisms instead of realising true inactivation. The resuscitation of survived microorganisms may promote the development oxidation resistance over generations. To avoid this risk, a complete microbial inactivation during the production intervals is strongly recommended.

AUTHOR CONTRIBUTIONS

Dibo Liu: Conceptualization; funding acquisition; project administration; writing – original draft; writing – review and editing. **David L. Straus:** Writing – original draft; conceptualization; writing – review and editing. **Lars-Flemming Pedersen:** Conceptualization; writing – review and editing. **Christopher Good:** Conceptualization; writing – review and editing; funding acquisition. **Carlo C. Lazado:** Conceptualization; writing – review and editing; funding acquisition. **Thomas Meinelt:** Conceptualization; writing – original draft; project administration; funding acquisition; writing – review and editing.

ACKNOWLEDGEMENTS

This review was supported by the German Government's Special Purpose Fund held at Landwirtschaftliche Rentenbank (fund no. 887139). C.L. would like to thank the Norwegian Seafood Research Fund (ref. 901472, 901826) and the Research Council of Norway (ref. 237856).

C.G. would like to acknowledge the US Department of Agriculture—Agricultural Research Service (ref. 59-8082-0-001). Special thanks to Dr. Chiqian Zhang from University of Missouri for help with redox potential explanations and calculations for E' and E° values in Table 3, and to Dr. Tero Luukkonen from University of Oulu for help with understanding the principals of his work and directing us to the work of Dr. Zhang. All authors would like to thank the anonymous reviewers for the improvement of the manuscript. Mention of trade names or commercial products in this article is solely for the purpose of providing specific information and does not imply recommendation or endorsement by any of the authors affiliations.

DATA AVAILABILITY STATEMENT

Data sharing not applicable to this article as no datasets were generated or analysed during the current study.

ORCID

Dibo Liu https://orcid.org/0000-0002-7348-3635

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SUPPORTING INFORMATION

Additional supporting information can be found online in the Supporting Information section at the end of this article.

How to cite this article: Liu D, Straus DL, Pedersen L-F, Good C, Lazado CC, Meinelt T. Towards sustainable water disinfection with peracetic acid in aquaculture: A review. *Rev Aquac*. 2024;1-26. doi:10.1111/raq.12915