Evaluation of the therapeutic effect of potassium permanganate at early stages of an experimental acute infection of *Flavobacterium columnare* in channel catfish, *Ictalurus punctatus* (Rafinesque)

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**Abstract**

The efficacy of potassium permanganate (KMnO₄) against the early stages of an experimental acute infection of *Flavobacterium columnare* in channel catfish, *Ictalurus punctatus*, was evaluated. Fish were experimentally challenged by waterborne exposure for 2 h to *F. columnare* after cutaneous abrasion, and treated with KMnO₄ at 2.0 mg L⁻¹ above the KMnO₄ demand at 0, 1, 2 or 4 h postchallenge for 24 h. Challenged non-treated fish acted as a positive control and non-challenged non-treated fish acted as a negative control. Fish challenged and treated with KMnO₄ at 0, 1, 2 or 4 h postchallenge had mortalities of 26%, 63%, 64% and 83% respectively. The mortality of challenged fish treated with KMnO₄ at 0 h postchallenge (26%) was significantly less than the positive control (77%). The mortalities of challenged fish treated with KMnO₄ at 0 h postchallenge (26%) was significantly less than the positive control (77%). 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The results suggest that KMnO₄ has a clear therapeutic value in early stages of columnaris infection but limited therapeutic value once the infection has progressed.

**Keywords:** potassium permanganate, columnaris, *Flavobacterium columnare*, channel catfish, *Ictalurus punctatus*

**Introduction**

Columnaris disease, caused by *Flavobacterium columnare*, exists worldwide and affects a wide variety of freshwater fish; no wild or cultured species, including ornamental fish, are known to be totally resistant to columnaris (Plumb 1999). Columnaris and enteric septicaemia of catfish are the two most important diseases of channel catfish, *Ictalurus punctatus*, the most widely cultured fish in the United States, causing high mortality among infected fish (Wagner, Wise, Khoo & Terhune 2002).

*Flavobacterium columnare* causes a combination of external and systemic infections (Hawke & Thune 1992). The bacterium can be a primary pathogen, but more commonly, it is a secondary pathogen that affects hosts predisposed to stress or trauma. The environment and the condition of the fish are important in determining the rate and severity of infection. Channel catfish are susceptible to columnaris at temperatures from 15 to 30°C and young fish are more severely affected than adults (Plumb 1999). *Flavobacterium columnare* attacks the fins, skin and/or gills of fish. Clinical signs of columnaris include frayed necrotic fins with greyish to white margins and depigmented and necrotic skin. The gill lesions have white to brown necrotic areas and viscera exhibit little or no pathology, even when the infection is systemic (Plumb 1999).

In natural columnaris infections, individual fish in a population are likely to exhibit columnaris at different severity levels and pathogenesis stages. The infections can be classified as follows: (1) chronic with low severity and lingering mortality; (2) acute with severe and a rapid onset of mortality; or (3) subacute with an infection severity and mortality between acute and chronic levels (Plumb 1999).
Potassium permanganate (KMnO₄) is an effective parasiticide and bactericide and kills skin and gill pathogens via its strong oxidizing properties (Noga 1996). The permanganate ion (MnO₄⁻) is responsible for the strong oxidative property and imparts a light pink or purple coloration to water; MnO₄⁻ is reduced to MnO₂ as it oxidizes pathogens and causes their destruction. The reduced ion is relatively non-toxic, colourless, insoluble and biologically unavailable (Boyd 1979; Lasier,Winger & Bogenrieder 2000). As KMnO₄ reacts with organic matter, the amount of KMnO₄ required for effective treatment is higher in organically enriched water (Tucker & Boyd 1977).

The efficacy of KMnO₄ against acute columnaris has been examined in fathead minnows, *Pimephales promelas* and channel catfish (*Ictalurus punctatus*; Jee & Plumb 1981; Thomas-Jinu & Goodwin 2004; Darwish, Mitchell & Hobbs 2008; Darwish, Mitchell & Straus 2009). The studies of Jee and Plumb (1981) and Thomas-Jinu and Goodwin (2004) demonstrated a mortality reduction in columnaris-challenged fish treated with KMnO₄ in a static system but it is unclear whether the efficacy was due to a reduction in the bacterial count in the water and on the host, which prevented further infection, or due to a therapeutic effect of KMnO₄ on infected fish. In *in vitro* studies found that KMnO₄ at 2.0 mg L⁻¹ for 8 h produced a 70% reduction in the colony-forming unit (CFU) count of *F. columnare* (Darwish et al. 2008) while *in vivo* studies demonstrated that KMnO₄ has a prophylactic effect (Darwish et al. 2009). In a flow-through system, KMnO₄ was not efficacious against an experimental acute or subacute infection of columnaris (Darwish et al. 2008, 2009); however, the KMnO₄ treatment intervention was at or after 22 h postchallenge in these studies. It is unclear whether treatments with KMnO₄ at stages earlier than 22 h postchallenge will significantly reduce the mortality of columnaris infection. The objective of this study was to assess the efficacy of KMnO₄ against early stages of an experimental acute columnaris infection by treating challenged fish (waterborne exposure of fish to the bacteria for 2 h after cutaneous abrasion) at 0, 1, 2 and 4 h postchallenge for 24 h.

**Materials and methods**

**Bacteria**

*Flavobacterium columnare* (isolate LV359-01, provided by Andrew Goodwin, University of Arkansas at Pine Bluff (Pine Bluff, AR, USA) was cultured on Hsu-Shotts medium at 25°C. The isolate was presumptively identified using the biochemical method of Griffin (1992), definitively identified using the polymerase chain reaction method of Darwish, Ismaiel, Newton and Tang (2004) and genotyped (genotype II) according to Darwish and Ismaiel (2005).

**Experimental design**

Channel catfish fingerlings weighing 17.1 ± 0.8 g [mean ± standard error of the mean (SE)] were obtained from Harry K. Dupree Stuttgart National Aquaculture Research Center (Stuttgart, AR, USA). Using a randomization table generated by the MINITAB STATISTICAL SOFTWARE® (version 13; MINITAB, State College, PA, USA), groups of 10 fish were randomly assigned to 30 continuously aerated flow-through tanks containing 60 L of well water; two groups were allocated to each tank to yield a total of 20 fish. The flow rate was set at approximately 1.5–1.8 L min⁻¹ and the temperature was maintained at 23.4 ± 1.4 °C. Fish were acclimated to the experimental conditions for 7 days, during which they were fed commercial channel catfish feed (Arkat Feed, Dumas, AR, USA). At the end of the acclimation period and before the challenge, all fish were cutaneously abraded according to Darwish et al. (2009). Briefly, fish were scrubbed five times with a dishwashing scrub pad. The abraded area was the left lateral surface of the fish from the caudal end of the adipose fin to the base of the caudal fin. All tanks were randomly assigned to one of six treatment groups (five tanks per treatment) as follows: (1) fish group challenged, cutaneously abraded and waterborne exposed to *F. columnare* and not treated with KMnO₄ (positive control); (2) non-challenged non-treated group (negative control); and (3) four treatment groups of challenged fish treated with KMnO₄ at 0, 1, 2 or 4 h postchallenge.

Dissolved oxygen (> 5.5 mg L⁻¹) and total ammonia nitrogen (< 0.3 mg L⁻¹) were measured in five tanks daily (HACH DR/2010, HACH Chemical, Loveland, CO, USA). Five different tanks were sampled each day so that all 30 tanks were tested within 6 days, and this protocol was repeated throughout the experiment.

**Bacterial challenge protocol**

The bacterial isolate was stored at −80°C in Hsu-Shotts broth (with 25% glycerin added). Before use,
the bacterium was thawed and plated on Orda’s medium (Anacker & Ordal 1959). An isolated colony was used to inoculate 5 mL of start-up medium [F. columnare growth medium broth (FCGM; Farmer 2004)] and incubated for 24 h at 28 °C. The start-up medium was then used to inoculate 1 L of FCGM broth, which was incubated for 24 h at 28 °C with orbital shaking of 200 revolutions per minute (New Brunswick Scientific, Edison, NJ, USA). The purity of the culture was confirmed by streaking an Orda’s medium plate. The bacterial challenge was initiated 1 h after the cutaneous abrasion by placing the fish from each tank in a bucket containing 8 L of continuously aerated water and adding 87 mL of the FCGM broth with an optical density of 0.95 (measured at 550 nm wavelength). Colony-forming units in the FCGM broth were used to extrapolate the buckets’ bacterial concentration as 2.9 × 10⁸ CFU mL⁻¹ (Herigstad, Hamilton & Heersink 2001). After 2 h of the bacterial challenge, the fish were removed from the challenge buckets and returned to their respective tanks. The non-challenged negative controls were similarly handled but not exposed to F. columnare.

Dead and moribund fish were necropsied and bacterial isolation was attempted from skin lesions and the kidney on plates of selective Orda’s medium (Hawke & Thune 1992). At the conclusion of the experiment, 20 days postchallenge, four fish from each treated tank after the water flow was turned off and at the conclusion of 24-h treatment, normal water flow was restored.

To verify the KMnO₄ dosing in treated tanks, water samples were immediately collected from treated and non-treated tanks, preserved by adding HNO₃ to achieve 1% (v/v) and analysed for Mn with an inductively coupled optical emission spectrometer (Perkin-Elmer Optima 2000DV, Norwalk, CT, USA). The detection limit of the Mn method was 0.003 mg L⁻¹ (APHA 1998).

In the tanks, the KMnO₄ concentrations upon dosing with the nominal rate of 2.5 mg L⁻¹ at 0, 1, 2 and 4 h postchallenge were 2.27 ± 0.21, 2.51 ± 0.04, 2.48 ± 0.07 and 2.46 ± 0.06 mg L⁻¹ respectively.

Statistical analysis
At the conclusion of the experiment, the survival percentages within tanks were arcsine transformed, and using the MINITAB STATISTICAL SOFTWARE* (version 13), the transformed data were subjected to one-way analysis of variance and the differences among treatment means were determined using Tukey’s test (Zar 1974; Sokal & Rohlf 1995). Treatment effects were considered significant at P ≤ 0.05.

The GRAPHPAD PRISM SOFTWARE* (version 4; GraphPad Software, San Diego, CA, USA) was used to conduct the following survival analysis: (1) Kaplan–Meier method to calculate the probability of survival at each day and (2) log-rank tests to compare the survival curves (Motulsky 1995). A Bonferroni’s correction of P/n, where P ≤ 0.05 and n is the number of pair-wise comparisons, was used in order to account for increasing Type I error with the number of comparisons made (Motulsky 1995).

KMnO₄ treatments
The KMnO₄ demand (PPD) of five random tanks was determined (Boyd 1979) and the average was calculated before treatment. The KMnO₄ dose was calculated as the average PPD plus 2 mg L⁻¹ (Plumb 1999); the average PPD of the test waters was 0.5 mg L⁻¹. Four of the six treatments were treated with KMnO₄ as described previously and the positive and negative controls were not treated. A stock solution of KMnO₄ was prepared by dissolving 1 g of KMnO₄ in 1 L of reagent-grade water (18.2 MΩ cm⁻¹). A 150 mL aliquot of the stock solution was added to each treated tank after the water flow was turned off and at the conclusion of 24-h treatment, normal water flow was restored.

Results
Fish challenged and treated with KMnO₄ at 0 h post-challenge had 26% mortality, which was significantly different (P ≤ 0.05) from challenged fish not treated with KMnO₄ (77%). Challenged fish treated with KMnO₄ at 1 and 2 h postchallenge had mortality rates of 63% and 64% respectively; these mortalities were not statistically significant (P ≤ 0.05) from the challenged non-treated fish (Table 1). According to the log-rank comparisons, the fish challenged and treated with KMnO₄ at 0 h postchallenge had a survival curve significantly different (P ≤ 0.05) from the non-challenged fish and the challenged non-treated fish (Table 2). The survival curve of the challenged non-treated fish was not significantly different from challenged fish treated at 1 and 2 h postchallenge but was significantly different (P ≤ 0.05) from the
The non-challenged negative control tanks appeared normal with no notable clinical signs, and only two fish died during the study. In moribund fish, the area of the skin scrubbed before the bacterial challenge developed a focal ulcerative necrotizing dermatitis surrounded by cutaneous depigmentation. As the infection progressed, challenged fish had discrete and diffuse multifocal skin depigmentation lesions, often encompassing most of the body and the muscles exhibited severe necrotizing myositis. The fins were frayed and necrotic. The buccal mucosa was yellow tinged. The gills had multifocal necrotizing bronchitis with haemorrhages and yellowish mucoid material. Moribund fish were lethargic. No visceral gross pathology was notable.

Clinical signs and gross pathology

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Bacterial isolation from fish

*Flavobacterium columnare* isolations were made externally and internally 61.6% of the time and only externally 23.5% of the time; 85.1% of dead and moribund fish cultured positive. The methods described earlier confirmed the identity and the genotype of the isolates to be *F. columnare* genotype II. No *F. columnare* were isolated from the skin or the kidneys of the two negative control (non-challenged) fish that died during the experiment or from sacrificed fish at the conclusion of the experiment.

Discussion

Potassium permanganate has therapeutic value in the early stages of columnaris infection. In the present study, fish challenged with bacteria and immediately treated with KMnO₄ at 0 h postchallenge displayed significantly less mortality than the challenged non-treated fish. The rate of death in the...
challenged non-treated fish was 3.6 times higher than the challenged fish treated at 0 h postchallenge. This study clearly demonstrates the beneficial effect of KMnO4 at early stages of columnaris infection but limited efficacy as columnaris infections become more advanced.

The intricacies of the experiments used to evaluate the efficacy of therapeutants are critical for the understanding of their benefits and limitations. The studies of Jee and Plumb (1981) and Thomas-Jinu and Goodwin (2004) used a static system (bacteria were not flushed out and fish were exposed to a high CFU count for an extended period) to test the efficacy of KMnO4 against columnaris infection. Jee and Plumb (1981) found that KMnO4 treatment at 4 mg L\(^{-1}\) above the 15 min PPD reduced columnaris mortalities in fathead minnows; they reported 62–65% mortality in the treated fish compared with 86–100% mortality in untreated fish. Thomas-Jinu and Goodwin (2004) reported similar results in channel catfish where the therapeutic dose (calculated according to Tucker 1989) reduced columnaris mortalities from 100% to 69%. Both studies demonstrated a beneficial effect but no distinction was made between the therapeutic and the prophylactic effect of KMnO4. Darwish et al. (2009) demonstrated a prophylactic effect of KMnO4 in which fish were challenged and simultaneously treated. In a flow-through system (\(F.\) columnare would be flushed and fish would not be exposed to large numbers of the bacteria for an extended period), KMnO4 had a limited therapeutic effect against experimental acute and subacute infections of columnaris (Darwish et al. 2008, 2009). These treatments were initiated about 22 h postchallenge and it was suggested that treatments at earlier stages of the infection might prove more useful.

The current study evaluated the usefulness of KMnO4 treatment at very early stages of columnaris infection and demonstrated a statistically significant reduction in the mortality of fish treated at 0 h postchallenge. But as the infection progressed, at 1, 2 and 4 h postchallenge, KMnO4 had a limited therapeutic effect, with a trend towards a higher mortality level (not significantly different from the challenged non-treated fish). The survival curve analysis showed that the challenged non-treated fish were significantly different from the challenged fish treated at 4 h postchallenge and the mortality rate of the treated fish was significantly accelerated. The acceleration of the mortality in advanced infections without affecting the final mortality is similar to the report of Darwish et al. (2008).

The confirmed efficacy of KMnO4 at early stages of columnaris infection could be attributed to its ability to reduce both the number of bacteria in the water and on the fish surface; bacterial attachment and colonization on the host are the first steps for the establishment of the infection. Darwish et al. (2008) demonstrated 70% in vitro reduction in the bacterial CFU count of \(F.\) columnare treated with 2 mg L\(^{-1}\) KMnO4. As the infection progressed, the beneficial effect of KMnO4 diminished partly due to: (1) KMnO4 being an external treatment, and the bacteria are known to infiltrate the skin layers of the fish, to lodge between necrotic tissue in the gills, and to become systemic, virtually rendering the bacteria inaccessible to the oxidative affect of KMnO4 (Darwish et al. 2008); and (2) the 2 mg L\(^{-1}\) KMnO4 dose only reduced the CFU count of bacteria but did not eliminate it (Jee & Plumb 1981; Darwish et al. 2008).

Disease models like the one used in the current experiment are a useful research tool, but simulating a

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**Figure 1** Survival curves of *Flavobacterium columnare*-challenged channel catfish showing the daily survival percentage. Fish were cutaneously abraded and divided into five treatments: challenged by waterborne exposure to *F. columnare* and not treated with potassium permanganate (KMnO4) [(positive control (PC)]; challenged and treated at 0, 1, 2 or 4 h postchallenge for 24 h with KMnO4 at a dose of 2.5 mg L\(^{-1}\) (0, 1, 2 and 4 h respectively); fish not challenged and not treated [negative control (NC)]. Each treatment group had 100 fish equally divided among five tanks.
natural infection will always remain a challenge due to the sheer complexity of the factors involved in the natural process. In a natural infection, individual fish are likely to exhibit colunmaris in different stages of the pathogenesis and some individuals may not be infected. Applying KMnO4 against colunmaris will have an unmistakable benefit in curbing colunmaris infections (Jee & Plumb 1981; Thomas-Jinu & Goodwin 2004; Darwish et al. 2009). The present study and the studies of Jee and Plumb (1981) and Darwish et al. (2008, 2009) demonstrate the benefits and limitations of KMnO4 against F. columnare infection. In an infected population with bacterial shedding in the water, KMnO4 oxidizing power will protect individual fish not yet infected by reducing the bacterial CFU count in the water column. In individual fish at an early stage of disease development, KMnO4 would reduce bacterial attachment and colonization on the fish surface. The current results demonstrate the importance of early detection and treatment intervention as an effective management tool. In subacutely infected fish, KMnO4 may not reduce the mortality partly because the bacteria might be systemic and the bacterial reduction on the fish surface could be insignificant to the infection (Darwish et al. 2009). In acutely infected fish, KMnO4 might accelerate the mortality, as demonstrated in the current study and the study of Darwish et al. (2008).

The current study did not explore the efficacy of increasing the KMnO4 dose above 2 mg L$^{-1}$. The dose used in the current study was similar to the one recommended by Plumb (1999). The KMnO4 dose used in the present study was shown to reduce the CFU of F. columnare when applied for 8 h but would not eliminate the bacteria (Darwish et al. 2008). It would be desirable to use KMnO4 at a dose that will inhibit bacterial growth, the minimum inhibitory concentration (MIC). A colunmaris infection treated 1 day postchallenge with diquat at the MIC to F. columnare (5 mg L$^{-1}$) produced a significant reduction in mortality, but treatments below the MIC (2.5 mg L$^{-1}$) did not significantly reduce mortality (Darwish & Mitchell 2009). A 10 mg L$^{-1}$ treatment of KMnO4 for 8 h completely inhibited the growth of the F. columnare isolate used in the current challenge (Darwish et al. 2008), but application of 10 mg L$^{-1}$ treatment for 8 h will likely be toxic to the fish (Darwish, Griffin, Straus & Mitchell 2002). To find the most efficacious application of KMnO4, future research would have to explore the KMnO4 concentration and duration with the most inhibition to bacterial growth and the least adverse affect on the fish.

As colunmaris is typically a secondary infection that occurs with other bacterial and parasitic pathogens (Plumb 1999), applying KMnO4 in cases of mixed infections can have a beneficial effect. This is because the oxidative power of KMnO4 is indiscriminate and can help eliminate or reduce other external pathogens contributing to the overall disease condition of the fish (Noga 1996). Field studies with detailed descriptions of fish disease conditions will be essential to substantiate the overall beneficial effect of KMnO4.

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