

Review Article

Streptococcosis in Tilapia (*Oreochromis niloticus*): A Review

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ABSTRACT

Tilapia (*Oreochromis niloticus*) is a hardy, most cultured freshwater fish in the world. It has been contributing to the world aquaculture since the ancient Egyptian days and remains a major freshwater fish species to be cultured. Although tilapias are more resistant to unfavourable water quality than other freshwater fish, tilapias have been reported to succumb to infection by *Streptococcus*, which was first observed among the populations of rainbow trout (*Oncorhynchus mykiss*) farmed in the Shizouka Prefecture in Japan in April 1957. Since then, the disease that is also known as ‘pop eye’ has been reported in many other fish species throughout the world, contributing to an annual loss of approximately USD 150 million. Affected tilapia shows loss of appetite, spine displacement, haemorrhages in the eye, corneal opacity, haemorrhages at the base of the fins and in the opercula. The most prominent signs are uni- or bi-lateral exophthalmia (also known as “pop-eye”), distended abdomen and erratic swimming. Control is mainly through implementing some preventive measure and antibiotic therapy, while vaccination is generally not effective in preventing *Streptococcus* outbreaks in tilapias.

Keywords: *Streptococcus*, tilapia, infection

INTRODUCTION

The tilapias are freshwater fish that belong to the family Cichlidae, and they are exclusively associated with Africa and Middle East (Trewaves, 1983). The Nile tilapia (*Oreochromis niloticus*) is one of the first fish species to be cultured in the world. Illustrations from Egyptian tombs suggested that the Nile tilapias had been cultured more than 4,000 years ago, i.e. about 1000 years before carp was introduced into China (Balarin & Hatton, 1979). Tilapias have been called the “Saint Peter’s fish” in reference to biblical passages about the fish being fed to the multitudes (Popma & Masser, 1999). Pillay (1990) reported that tilapias were introduced

into many tropical, sub-tropical and temperate regions of the world during the second half of the 20th century.

In the world where captured wild fisheries are becoming increasingly depleted, tilapias offer a possibility of commercialization because of their superior culture adaptability. According to FAO (2004), tilapias (*Oreochromis* sp.) are among the most cultured fish worldwide. In fact, the production of tilapias made the fish one of the most important species for the 21st century aquaculture (Fitzsimmons, 2000) which also rose commercially in more than 100 countries (Shelton & Popma, 2006).

The estimated global tilapia production for the year 2000 was 1.5 million metric tonnes

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(MT) compared to merely 28,260 metric tonnes in 1970. Similarly, the value of the farmed tilapias also increased from about USD 154 million in 1984 to USD 1,800.7 million in 2002 (FAO, 2004). China alone produced 706,585 MT of farmed tilapia (representing 50% of the total world production), followed by Egypt (167.7 MT), the Philippines (122.4 MT), Indonesia (109.8 MT), Thailand (100.6 MT), Taiwan (85.1 MT), Brazil (42 MT), Colombia (24 MT), Malaysia (20.8 MT), and Laos (20.8 MT). The farmed tilapias had exceeded 2 MMT in 2004 worldwide (FAO, 2004; El-Sayed, 2006).

Tilapias have good characteristics for farming and are now so domesticated that this fish species has earned the title “aquatic chicken”. Moreover, tilapias are fast-growing with firm, white flesh, and able to survive in poor water conditions, eat a wide range of food types, breed easily with no need for special hatchery technology (Nandlal & Pickering, 2004), as well as feed at the base of the aquatic food web (Beveridge & Baird, 1998). Tilapias are tough and can tolerate a wide range of environmental conditions; therefore, little environmental modification with low technology system is needed for culturing tilapias (Pullin & Lowe-McConnell, 1982; Welcomme, 1988; Beveridge & McAndrew, 1998; Nandlal & Pickering, 2004).

Initially, tilapias were considered to be more resistant to bacterial, parasitic, fungal, and viral diseases compared to other species of cultured fish. In more recent times, however, tilapias have been found to be susceptible to both bacterial and parasitic diseases. Common tilapia pathogens include *Streptococcus* sp., *Flavobacterium columnare*, *Aeromonas hydrophila*, *Edwardsiella tarda*, *Ichthyophthirius multifiliis*, *Tricodhina* sp., and *Gyrodactylus niloticus* (Klesius *et al.*, 2008). It is important to note that streptococcal infections have become a major problem in tilapias farming and contributed to severe economic losses (Shoemaker & Klesius, 1997). *Streptococcus iniae* and *Streptococcus agalactiae* are the major bacterial species that affect the production of tilapias in the world (Evan *et al.*, 2006a).

The aims of this paper are to review the current information on streptococcosis, including epidemiology, main water quality that contributes to the disease, mode of transmission, pathogenesis, as well as disease diagnosis, and control measures in farmed tilapias.

STREPTOCOCCUS AND STREPTOCOCCOSIS

Streptococcosis is a disease that develops following the infection by *Streptococcus* sp. They are spherical or ovoid in shape and 0.5-2.0 μm in diameter. They occur in pairs or chains when grown in liquid media, are non-motile, non spore-forming and Gram-positive. A major identification feature of *Streptococcus* is that it is Gram-positive that appears purple/blue when stained using a procedure called a Gram stain. On the contrary, most of the common disease-causing bacteria of fish are Gram-negative and appear pink with Gram stain (Yanong & Floyd, 2002). It is facultatively anaerobic, requiring nutritionally rich media for growth and commonly attacks red blood cell to produce greenish discoloration (α -hemolysis) or complete clearing (β -hemolysis) on blood agar. In addition, it is also a type of bacteria that are fermentative in metabolism, producing mainly lactic acid, but without gas and catalase negative (Holt *et al.*, 1994).

In fish, it was initially described among the populations of rainbow trout (*Oncorhynchus mykiss*) farmed in the Shizouka Prefecture in Japan in April 1957 (Hoshina *et al.*, 1958). After that, Robinson & Meyer (1966) reported two epizootics, both involving infections of golden shiner (*Notemigonus crysoleucas*) with *Streptococcus*. Meanwhile, Plumb *et al.* (1974) isolated *Streptococcus* sp. from over 50% of the diseased fish during an epizootic in the estuarine bays along the Florida, Alabama, and the Gulf Coast of Mexico in the United States in 1972. In fish, *Streptococcus* spp. has been reported to cause considerable morbidity and mortality worldwide. Estimated losses were around USD 150 million annually in 2000 and these further increased to USD 250 million annually in 2008 (Klesius *et al.*, 2000; Klesius *et al.*, 2008).

Streptococcus iniae was first isolated from a skin lesion of a captive Amazon River fish, *Inia geoffrensis* (Pier & Madin, 1976). Since then, the bacterium has been reported in many species of fresh, estuarine and marine fish species from 15 countries in 6 continents, including Africa, Asia, Australia, Europe, as well as North and South Africa. The susceptible fish include ayu (Kitao *et al.*, 1981), barramundi (Bromage *et al.*, 1999), coho salmon (Eldar *et al.*, 1995a), European seabass (Zlotkin *et al.*, 1998), grey mullet (Eldar *et al.*, 1995a), grouper (Kvitt & Colorni, 2004), rainbow trout (Eldar *et al.*, 1994), red drum (Shen *et al.*, 2005), snapper (Ferguson *et al.*, 2000), silver bream (Bromage & Owen, 2002), tilapia (Klesius *et al.*, 2006a), and yellowtail (Kaige *et al.*, 1984).

Group B *Streptococcus agalactiae*, another emerging fish pathogen, has been shown to cause significant morbidity and mortality among a variety of freshwater and saltwater fish species throughout the world (Robinson & Meyer, 1966; Plumb *et al.*, 1974; Evans *et al.*, 2002). *Streptococcus agalactiae* was first reported in the captive freshwater shiners in 1966 (Robinson & Meyer, 1966). Recently, this particular bacterium has been reported in fish from 7 countries in 3 continents, namely the United States (North America), Israel, Japan, Kuwait and Thailand (Asia), Honduras (Central America), and Brazil (South America). This pathogen has also been isolated from 17 fish species including rainbow trout, seabream, tilapia, yellowtail, *catfish* sp., croaker, killfish, *menhaden* spp., *mullet* spp. and silver pomfret (Wilkinson *et al.*, 1973; Plumb *et al.*, 1974; Rasheed & Plumb, 1984; Elliot *et al.*, 1990; Baya *et al.*, 1990; Eldar *et al.*, 1995a; Vandamme *et al.*, 1997; Evans *et al.*, 2002; Duremdez *et al.*, 2004; Suanyuk *et al.*, 2005; Salvador *et al.*, 2005; Evans *et al.*, 2006a; Kim *et al.*, 2007; Garcia *et al.*, 2008).

Streptococcus spp. is considered a diverse group of bacteria that possess the capacity to infect a wide range of hosts. Among other, *S. iniae* has been isolated from humans with bacteraemia, cellulitis, meningitis, and osteomyelitis (Facklam *et al.*, 2005). The source of human infections has been associated with

the preparation of *S. iniae* infected tilapias for cooking (Lehane & Rawlin, 2000). *S. agalactiae* is the causative agent of neonatal meningitis, sepsis and pneumonia in human (Baker, 1980). It has been isolated from chickens, cattle, camels, dogs, bottlenose dolphins, horses, emerald monitors, cats, fish, frogs, hamsters, humans, mice, monkeys, and nutria (Wilkinson *et al.*, 1973; Elliott *et al.*, 1990; Evans *et al.*, 2002; Zappulli *et al.*, 2005).

Transmission

Many studies have been carried out to reveal the transmissions of *Streptococcus* sp. According to Nguyen *et al.* (2002), the newly introduced fish is the most important factor that introduced *S. iniae* and *S. agalactiae* into the farm. The bacteria are excreted in the faeces of infected fishes, survive in the water and be infectious to other healthy fish (Nguyen *et al.*, 2002). Besides, using the infected thrashed fish as feed is believed to be responsible for the outbreaks of streptococcosis among flounder in Korea (Kim *et al.*, 2007). Similarly, an experimental study revealed that cohabitation of dead or infected fish with healthy fish resulted in the infection of the healthy fish. Meanwhile, the horizontal transmission of the pathogens between fish is believed to be the most common mechanism of dissemination. A study by Xu *et al.* (2007) showed that the infection by this particular pathogen could occur through wounds and abrasions of the skin. This mechanism usually involved in fish that were cultured in high densities. Furthermore, the transmission of *Streptococcus* between different species of wild and cultured fish, within the same aquatic environment, is likely to occur (Evans *et al.*, 2002). This is because wild fish and fish cultured nearby have been found to be infected with the same *S. iniae* strains in Israel (Colorni *et al.*, 2002). Similarly, Bromage & Owen (2002) reported that the fish cohabiting barramundi pens had the same *S. iniae* strains as the barramundi. In addition, the transmission among the species of reef fish has also been reported in the Caribbean (Ferguson *et al.*, 2000).

Pathogenesis

Infection by *Streptococcus* leads to various clinical signs, which include haemorrhages at the gill plate, loss of appetite, spine displacement, haemorrhages in the eye, corneal opacity, and haemorrhages at the base of the fins and in the opercula. The most prominent signs are uni- or bi-lateral exophthalmia, also known as “pop-eye”, and distended abdomen. The post-mortem examinations of the affected fish revealed the presence of blood-tinged fluid in the body cavity, enlarged and reddened spleen, pale but enlarged liver, as well as inflammations around the heart and kidney. Meanwhile, hemorrhagic lesions were observed on the skin (Bullock, 1981; Yanong & Floyd, 2002; Salvador *et al.*, 2005). Other clinical signs include darkening of the skin and erratic swimming, which is either spiralling or spinning just below the surface of water. In some cases, however, the affected fish showed no obvious clinical signs before death and the mortality is mainly due to septicaemia and infection of the brain and nervous system (Barham *et al.*, 1979; Yanong & Floyd, 2002).

Buchanan *et al.* (2005) identified enzyme phosphoglucomutase as the virulence factor for *S. iniae*. This enzyme inter-converts glucose-6-phosphate and glucose-1-phosphate which play important role in the production of *S. iniae* polysaccharide capsules. Unlike *S. iniae*, the regulatory proteins and enzymes associated with cell surface metabolism have been revealed as the virulence factors for *S. agalactiae*. Therefore, the removal of the genes that are involved in these functions can reduce the virulence. Fuller *et al.* (2002) found that the virulence factor could also be caused by the gene that is associated with β -hemolysis. However, additional research should be carried out to identify and characterize the genes and the virulence factors that regulate their expression.

FACTORS CONTRIBUTING TO THE DEVELOPMENT OF STREPTOCOCCOSIS

The presence of the pathogen in the environment of the fish is inadequate to cause a disease

outbreak. Other factors usually come into play, such that either the pathogen has an advantage over the host or the immune system of the host is compromised in some ways, increasing its susceptibility to the pathogen. This phenomenon is often precipitated by “stress” (Yanong & Floyd, 2002). Therefore, stress often plays a significant role in the outbreaks of infectious disease in fish populations. Some stressors that have been associated with the Streptococcal outbreaks include high and low water temperatures, high salinity and alkalinity (pH>8), low dissolved oxygen concentration, poor water quality (such as high ammonia or nitrite concentrations), high stocking densities, as well as harvesting and handling effects (Chang & Plumb, 1996; Bunch & Bejereno, 1997; Bowser *et al.*, 1998; Yanong & Floyd, 2002).

Meanwhile, water quality parameters can contribute to the development of disease. It is a well-known fact about the intolerance of tilapias to low temperatures, which is a serious constraint for commercial culture in temperate regions (Chervinski, 1982; Cnaani *et al.*, 2000). Reproduction of tilapia is best in water temperatures above 27°C, but it does not occur when water temperature is below 20°C. In subtropical regions with a cool season, the numbers of fry produced are decreased when daily water temperature averages less than 24°C. It was concluded that the optimal water temperature for the growth of tilapias is between 29°C and 31°C (Popma & Masser, 1999), but a water temperature of $\geq 31^\circ\text{C}$ predisposes tilapias to the outbreaks of *Streptococcus agalactiae* infection (Evans *et al.*, 2006a; Amal *et al.*, 2008).

Oxygen is the first limiting factor for growth and well-being of fish. Fish require oxygen for respiration, which physiologists express as the mg of oxygen consumed per kilogram of fish per hour (mgO₂/kg/h). Although tilapia can survive acute low DO concentrations of less than 0.3 mg/L for several hours, tilapia ponds should be managed to maintain the DO concentrations above 1 mg/L. Metabolism, growth, and disease resistance are depressed when DO falls below this level for a prolonged period (Popma & Masser, 1999), predisposing tilapias to streptococcosis.

Moreover, it is a well-known fact that increasing water temperature will reduce the rate of DO in the water. The high water temperature also leads to increased respiration rate and oxygen consumption by tilapias because of the high metabolism rate. This further increases the demand for oxygen by tissues. Therefore, dissolved oxygen concentration greater than 5 ppm is required for a good growth of tilapias (Swann, 1992; El-Sayed, 2006).

Other than water temperature and dissolved oxygen, massive mortality of tilapia occurs within a few days when fish are suddenly transferred into water without ionized ammonia concentration greater than 2 mg/L. Meanwhile, a prolonged exposure for several weeks to un-ionized ammonia concentration greater than 1 mg/L in water with low dissolved oxygen predisposes tilapias to diseases including streptococcosis. In fact, the prolonged exposures to 0.2 mg/L of un-ionized ammonia concentration are found to be detrimental to fish (Popma & Masser, 1999). Ahmed *et al.* (1992) have found that Nile tilapias exposed to ammonia had lower number of red blood cells leading to haemolytic anaemia and significant reduction in blood oxygen content, which enhances ammonia toxicity.

Nitrate is relatively non-toxic to tilapias. However, a prolonged exposure to elevated levels of nitrate may decrease the immune response and induce mortality (Plumb, 1997). Inversely, nitrite is highly toxic to tilapias because it disturbs the physiological function of the fish and leads to growth retardation (El-Sayed, 2006). Nitrite may enter the bloodstream passively as nitrous acid and freely diffuses across the gill membranes of the fish. After entering the bloodstream, nitrite oxidizes the iron in the haemoglobin molecule from ferrous state (Fe^{2+}) to ferric state (Fe^{3+}) and the resulting product is called methemoglobin. Since methemoglobin is incapable of reversibly binding with oxygen, exposures to nitrite can cause considerable respiratory distress because of the loss in blood oxygen-carrying capacity (Boyd & Tucker, 1998).

In general, tilapias can survive in pH ranging from 5 to 10, but they do best in a pH range of 6 to 9 (Popma & Masser, 1999). On the contrary, low water pH leads to behavioural changes, damage of the gill epithelial cells, reduction in the efficiency of the nitrogenous excretion and increased mortality. Wangead *et al.* (1998) reported that fingerlings and adult tilapias exposed to pH 2-3 showed rapid swimming and opercula movement, surfacing and gulping of air, as well as lack of body position and mass mortality within 1-3 days. A study by Chen *et al.* (2001), on the other hand, showed that tilapias exposed to high water pH for 7 days decreased ammonia excretion, but increased urea nitrogen excretion. Bonga *et al.* (1987) revealed that slow acclimatization of tilapias to low or high pH levels might enable the fish to withstand long-term exposures to the acidic or alkaline water. Thus, farmers should be aware of the sudden change in water pH to prevent stress on their cultured tilapias that may lead to disease outbreaks (Bonga *et al.*, 1987).

Diagnosis

The presence of typical clinical signs and demonstration of Gram-positive cocci from the brains, kidneys, eye or other internal organs constitutes a presumptive diagnosis of streptococcosis. The causative bacteria are best detected in the brains of diseased fish (Sugiyama & Kusuda, 1981). Streptococcal infection should be highly suspected if the affected fish exhibit abnormal swimming behaviour, pop-eye, haemorrhages, and rapid severe mortalities, while Gram-positive cocci are found in brain, kidney, and/or other organs. A confirmed diagnosis requires culture of internal organs, specifically the brain and kidney, followed by identification of the bacterium (Yanong & Floyd, 2002).

To recover the streptococci is apparently straightforward; bovine blood tryptose agar (Naude, 1975; Roode, 1977; Boomker *et al.*, 1979), brain heart infusion agar (BHIA) (Minami *et al.*, 1979, Ugajin, 1981), Todd-Hewitt broth,

nutrient agar supplemented with rabbit blood (Kitao *et al.*, 1981) are suitable media for culture. Inoculated media should be incubated at 22-37°C for up to 48 hours before the “dull grey” colonies of approximately 1-2 mm in diameter develop. This pathogen is easily grown on BHIA (Plumb *et al.*, 1974). Beside that, it also can grow on trypticase soy agar supplemented with 0.5% glucose, Todd-Hewitt broth agar (THBA), and horse blood agar (Kitao, 1982).

A single colony from pure culture should be Gram-positive cocci, oxidase, and catalase negative and either non-hemolytic or β -hemolytic on agar plate. The carbohydrate group antigen test should be also among the first presumptive test performed. The only group B streptococcal species is *S. agalactiae*. In contrast, *S. iniae* does not have a carbohydrate group antigen. If the streptococci hydrolyze starch, it is also presumptive test for *S. iniae* (Evans *et al.*, 2004). Meanwhile, the biochemical and other identification tests have been fully described elsewhere (Shoemaker *et al.*, 2001; Evans *et al.*, 2002).

Rapid kits such as API 20E, API Rapid Strep 32, and API CH50 could not be used to identify *S. iniae* because this particular bacterium is not included in the database system. However, these rapid kits can be used for the identification of *S. agalactiae* and other *Streptococcus* spp. (Evans *et al.*, 2006a). Jayarao *et al.* (1991) compared the identification systems between Vitek-Gram positive and API Rapid Strep 32 system and found that 93% of *S. agalactiae* isolates could be identified using both the kit systems. A comparison between API Rapid Strep 32 System and Biology system using Gram-positive plates revealed that both the systems produced 100% identification of the *S. agalactiae* isolates (Evans *et al.*, 2006b). However, the Biology system using Gram-positive plates was able to correctly identify approximately 70% of *S. iniae* (Roach *et al.*, 2006).

Molecular diagnosis using the PCR technique is useful to identify streptococcus. Many of the PCR techniques make use of the 16S rRNA gene as the molecular marker for the identification of *S. iniae* (Zlotkin *et al.*,

1998). Besides, a PCR technique using 16S-23S ribosomal DNA intergenic spacers was found to be useful for the identification of *S. agalactiae* from fish (Berridge *et al.*, 2001). However, the results of the PCR assay should be supported by presumptive techniques to ensure the accuracy of the detection.

Klesius *et al.* (2006a) developed an indirect fluorescent antibody technique (IFAT) based on a highly specific monoclonal antibody for a rapid detection of *S. iniae*. The olfactory epithelium of naturally infected tilapias was demonstrated to be a reliable, sensitive and non-lethal sample site for the detection and identification of *S. iniae*.

Controls

Chemotherapy

Several drugs have been tested for the treatment of streptococcosis. Among other, Darwish & Griffin (2002) found that oxytetracycline was effective in controlling *S. iniae* in blue tilapias (*O. aureus*). Oxytetracycline was incorporated into the feed at 0, 25, 50, 75, and 100 mg/kg body weight. The 75 and 100 mg doses significantly increased the survival of the infected fish from 7% to 85 and 98%, respectively.

Some reports concluded that erythromycin is effective against streptococcal infections in cultured yellowtails (Shiomitsu *et al.*, 1980) and rainbow trout (Kitao *et al.*, 1979) at doses of 25-50 mg/kg/day for 4 to 7 days. Doxycycline, oxytetracycline, kitasamycin, oleandomycin, josamycin, and lincomycin have also been used to control streptococcosis in the cultured yellowtail in Japan (Kitao *et al.*, 1979). Doxycycline, at 20 mg/kg/day for an undetermined duration, has also been advocated (Nakamura, 1982). Similarly, a novel fisheries therapeutant, i.e. sodium nifurstyrenate, dosed at 50 mg/kg body weight of fish/day for three to five days has been proven to be successful in treating streptococcosis when incorporated with feed (Kashiwagi *et al.*, 1977).

Meanwhile, streptococcal infections respond to antibiotic therapy, but the disease cannot be legally controlled with antibiotics all the way to the market because the withdrawal period for all

effective antibiotics is longer than it takes for the streptococcal infection to return. Furthermore, it is only a matter of time before *Streptococcus* develops resistance to the antibiotics. In fact, streptococcal strains at several facilities have already developed resistance to some antibiotics (Darwish & Hobbs, 2005). Therefore, antibiotic treatment is generally ineffective and the need of proper vaccine has become a must (Klesius *et al.*, 2000).

Preventive measures

If causative streptococci are present in the mud and water throughout the aquatic environment, avoidance is not an easy or practical means of disease prevention. However, purchasing specific pathogen-free stock, quarantining new arrival fish stock, reducing overcrowding, avoiding overfeeding, keeping separate water supplies for culture systems, minimizing unnecessary handling or transportation, removing dying and dead fish frequently, feeding pathogen free ration, and keeping excellent sanitary conditions will reduce the risks of disease outbreak (Inglis *et al.*, 1993; Klesius *et al.*, 2008). Furthermore, periodic cleaning and disinfections of all the production units and equipment should be done to decrease the transmission of pathogens. Maintaining good water quality in the systems is also necessary (Klesius *et al.*, 2008).

Management control by 'break-cycle' has been suggested (Amal *et al.*, 2008). April-June is a critical period in tilapia culture because of the high water temperature, while fish that weigh 150-300 gram are in critical condition. Huge and slow flow water bodies are critical situations for development streptococcosis in Malaysia. Farmers are advised to manage the cultured fish so that harvesting of adult fish of more than 200 gram can be done before the coming critical period of April-June and to ensure that only fish of less than 100 gram are available in the cage at the critical months of April-June. Nevertheless, farmers who still keep fish of 150-300 gram during the critical period of April-June are advised to reduce overcrowding by re-distributing the fish in cages.

Vaccines

A vaccine is a preventive tool used in a health management strategy for controlling infectious disease (Klesius *et al.*, 2006a). In aquaculture, the development and use of vaccines are now making rapid progress to achieve their full potential as effective disease prevention tools. The objective of vaccination is to provide a strong immune response to an administered antigen that is able to produce acquired long-term protection against a pathogen. Killed and modified live vaccines have been developed for use in aquaculture. The type of immunity needed, antibody or cell mediated, against a particular pathogen are among the deciding factors in the development of a vaccine. Killed vaccines are usually administered by intraperitoneal (IP) or intramuscular injection (IM) of individual fish. Injection is the least cost effective in terms of labour and time. Meanwhile, killed vaccines are considered safer than the modified live vaccines, which may revert to virulence. Consequently, future trends may include oral delivery of vaccine, immersion delivery of killed vaccine, development of additional modified live vaccines and multivalent vaccines and improved vaccine adjuvants and immunostimulant. Vaccines prevent disease and mortality, but they may not completely eliminate streptococci in surviving fish (Klesius *et al.*, 2008).

The first killed vaccine was developed to prevent losses in trout due to *S. iniae* infection in Israel (Eldar *et al.*, 1997). The mortality of rainbow trout intraperitoneally (IP) immunized with formalin killed *S. iniae* vaccine was 5%, whereas in non-immunized rainbow trout, the mortality exceeded 50% in the field trial. Whole cell and bacterial protein vaccines were produced against *S. agalactiae* (Eldar *et al.*, 1995b). A non-autogenous killed *S. iniae* vaccine supplemented with its extracellular products (ECP) was found to be effective in tilapia (Klesius *et al.*, 1999). The mortality was reduced by 91.3% in tilapia immunized IP with this vaccine at 30 days post-experimental challenge with *S. iniae*. The molecular weight of the extracellular product was greater than 2kD. The relative percent survival was 95%

in 25 gram tilapia and 84.2% to 94.7% in 100 gram tilapia. Besides that, western blot analysis revealed predominant 54 and 55 kDa antigens in the extracellular products (ECP) of *S. agalactiae* (Pasnik *et al.*, 2005). The results of the study provided a correlation between protection and antibody production against ECP and for the importance of the 55 kDa antigen for vaccine efficacy against *S. agalactiae*.

CONCLUSION

In conclusion, *Streptococcus* spp. (specifically *S. agalactiae* and *S. iniae*) are very pathogenic as they can affect many fish species in the world. In particular, Streptococcosis has been reported to occur in fresh, marine and brackish water fish; thus, it has caused millions economic losses of aquaculture in the world. Tilapias have become a perfect host for *Streptococcus* infection. Tilapia farmers should be advised and educated on a proper management of tilapias so as to prevent the outbreak and spread of the disease. In addition, water quality parameter plays an important role in tilapias farming. In more specific, an optimum water quality parameter should be maintained to prevent "stress" in fish which can lead to outbreaks of disease. The diagnosis of diseased and carrier fish can be made by using rapid and accurate immunological and molecular techniques. Although chemotherapy was not really suggested, good management practice and vaccination could be parts of the plan to prevent and control streptococcosis.

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