REVIEW ARTICLE

Relationship Between the Productivity Losses of Tilapia and Aeromonas Veronii Infection

Rasha M. Reda*, Abdelhakeem El-Murr, Yasser Abd Elhakim and Wessam El-Shahat
Fish Diseases and Management Department, Faculty of Veterinary Medicine, Zagazig University, 44511 Zagazig, Sharkia, Egypt.

Article History: Received: 21/03/2021 Received in revised form: 11/04/2021 Accepted: 17/05/2021

Abstract

Nile tilapia (Oreochromis niloticus) is considered one of the top cultured fish in Egypt and the second globally. The high demand for animal protein was associated with intensive fish culturing, which resulted in the emergence of disease outbreaks causing significant losses in tilapia aquaculture among the recent years. With special reference to bacterial outbreaks, Aeromonas veronii (A. veronii) is a recently reported bacterial pathogen affecting farmed tilapia and other fish species worldwide. A. veronii has been notified as a significant threat on Nile tilapia populations resulting in heavy mortality and was recorded to have a wide range of antibiotic resistance. Subsequently, recent publications focused on immunostimulant alternatives. The previous science data on summer tilapia mortality phenomena were outlined in this review article with special reference to A. veronii as an important pathogen. Clinical and postmortem symptoms, histopathological lesions, genetic diversity, and associations with others submitted to GenBank, diagnosis, control steps, and significance to public health were highlighted.

Keywords: Tilapia, Aeromonas veronii, Summer Mortality.

Introduction

Aquaculture showed rapid growth in the last 30 years with the main role in maintenance of fish health and performance [1]. Globally, Egypt is ranked the ninth in aquaculture and from the largest fish producers in Africa as where aquaculture supplies many benefits in social and economic aspect [2, 3]. Kafr El-Sheikh, Behera, Damietta, Sharkia, and Fayoum considered the largest producer governorates share by more than 80% of the national aquaculture production [4].

Nile tilapia (Oreochromis niloticus) considered the second most valuable fish after carp and the most important cultured fresh water fish [5]. Nile tilapia has many advantages more than other fishes summarized in the following: (i) adapt easily to different environmental conditions, (ii) less susceptibility to diseases, (iii) eat wide range of food types, (iv) reared easily with other fish species with high stocking density, and (v) breed easily with no need for special hatchery techniques[5, 6]. Therefore, tilapia rearing has been moving towards intensification and development to meet the large demand [7]. This intensification with low health management care cause stress leads to spread of bacterial, viral, fungal, and parasitic diseases, which will be resulted in high mortalities and economic losses [8].

In the last years, the Egyptian tilapia fish farms are suffering from unknown cause mortalities that lead to great economic losses during summer [9]. Bacterial diseases are considered the most serious causes and one of the significant factors leading to huge annual losses in the fish culture industry that was estimated by billions of US dollars [10, 11]. There are two main categories of bacterial diseases, which reported as a pathogenic for fishes. The first

*Corresponding author e-mail: rashareda55@yahoo.com, Fish Diseases and Management Department, Faculty of Veterinary Medicine, Zagazig University, 44511 Zagazig, Sharkia, Egypt.
category belongs to Gram-negative bacteria such as Aeromonas, Flavobacterium, Francisella, Pseudomonas, Edwardsiella, Vibrio, Yersinia, while the second one belongs to Gram-positive bacteria such as Streptococcus, Lactococcus, Clostridium perfringens and Staphylococcus aureus [10]. Despite the accusation of many of these species of bacteria in diseases of tilapia and other types of fish but, to the fact that the largest part of the cases that were recorded even in fish or human was caused by Aeromonas spp. [12].

From the most recorded motile aeromonads that causing 80 -100% mortality in fish farms are Aeromonas hydrophila, A. caviae, A. sobria, A. jandei, and A. veronii [13-17]. The ability to adapt to various trophic conditions and the ability to survive inside biofilms on different surfaces are one of the key factors for the proliferation of aeromonads in the aquatic environment [18, 19]. It is worth noting that Aeromonads is an opportunistic bacterium that attacks fiercely when there is any imbalance in the aquatic environment, such as high density or a change in water parameters and high temperatures[20, 21]. Aeromonas veronii infection is a hemorrhagic septicemia disease that is accused for massive tilapia fish mortalities in hot seasons in Egypt, Saudi Arabia, Thailand, India, and Malaysia [17, 22-25]. The pathogenicity of A. veronii is related to virulence genes and extracellular enzymes [26].

This review summarizes the currently available scientific data on tilapia industry in Egypt, tilapia summer mortality syndrome with reference to: (i) the most important etiological agents for summer mortality and their phenotypic and genetic characters, especially A.veronii, (ii) clinical, postmortem signs, and histopathological finding, (iii) Molecular diagnosis, and (iv) economic and zoonotic importance for this syndrome.

**Tilapia industry in Egypt**

Aquaculture was first introduced to the African countries in the 20th century, where its aquaculture production is ~2.7% with large-scale production were recorded in Egypt, Nigeria, Uganda, and Ghana [27-29]. Egypt is ranked 9th in fish farming production globally and 1st among African nations [30]. The aquaculture industry in Egypt plays a crucial role in the country's economy, offering food security and work opportunities [31].

Nile tilapia is the major farmed fish, where Egypt is the second largest producer in the world after China [32]. Tilapia is categorized as one of the most popular fish species that is intensively cultured all over the world. Tilapia ranked the second most common farmed fish globally next to carps [33]. Globally, Nile tilapia, commonly known as the aquatic chicken, is one of the fastest growing aquaculture industries and the major cultured freshwater fish species [34]. The international tilapia industry was accounted to be 4.5 million tons in 2014 and is expected to reach to 7.3 million tons by 2030, supplying an inexpensive protein source in developing countries [29]. Tilapia fish represents 65.15% of the Egyptian fish production [4].

In the future, the tilapia industry is projected to increase due to its potential for disease resistance, resilience to diverse environmental conditions, its fast growth rate even at intensive culture, in addition, it is also an affordable source of protein [5]. Most of the coastal lagoons are located in the lower Delta, so utmost of tilapia fish farms development are concentrated in main four north governors Kafr El Sheikh, Port Said, Sharkia, and Beheira [4]. There are few studies that investigate the relation between fish farm development and low-income consumers demand to fish [35-37]. Furthermore, Murphy et al. [35] recorded the relation between Egypt’s farmed tilapia markets, diversity and the different grades of product sold from where the prices according to size, quality, location, and market.

**Tilapia summer mortality syndrome**

Worldwide, fish diseases are among the most significant challenging obstacle in
Outbreaks of mortality in cultured Nile tilapia can occur by certain bacteria or co-infection. Several studies recorded outbreaks threatening tilapia industry by certain bacteria, such as Aeromonas species[17, 22, 43], Streptococcus species[44-46], Francisella species [47, 48], Edwardsiella species[49]. Other summer tilapia outbreaks were resulted from co-infections. Co-infections are known by the multiple interaction between several pathogens and the implications for host [50]. On the base of the primary invader pathogens, it will be determined the degree of host susceptibility to the secondary infection, also determine the course, severity, and incubation period of the infection [51]. Taking in consideration stressors such as water temperature, limited dissolved oxygen, and high culturing densities have a significant role in the enhancement of fish susceptibility to pathogen invasion [41,42]. From the reported summer tilapia outbreaks, which were resulted from co-infections: Aeromonas in co-infection with other pathogens such as Gyrodactylus cichlidarum [41], Tilapia Lake Virus [13, 25] and Streptococcus agalactiae [52] or co-infection between Streptococcus agalactiae and Francisella noatunensis [53]. Also, several pathogens such as Citrobacter freundii, Proteus vulgaris and Pseudomonas fluorescence in mixed infection with A. veronii from several fish farm in Egypt were reported [24]. In Thailand, co-infection of multiple pathogens (Flavobacterium columnare, Streptococcus agalactiae, Vibrio cholera, Plesiomonas shigelloides, and Irido virus) with A. veronii was reported in Nile tilapia fingerlings outbreaks in August 2014 [54]. Moreover, A. jandaei and A. veronii were recovered from Nile tilapia juveniles with eye exophthalmia and turbidity with septicemic lesions in Thailand [17]. Another investigation in Egypt during September 2015 described the mixed infection between A. veronii, A. hydrophila, A. ichthiosmia, A. jandaei, A. enteropelogenes, and Tilapia Lake Virus showing symptoms of skin hemorrhagic lesions, loss of scales, ulcers,

Bacterial agents implicated in summer mortality syndrome of tilapia with special reference to A. veronii
and dark pigmentation [40]. In Thailand and Vietnam 2012 A. caviae, A. veronii, and A. jandaei were isolated from diseased Nile tilapia exhibited signs of ascites, protruded eyes, and hemorrhagic lesions [55].

*Aeromonas* species as *A. hydrophila*, *A. veronii*, and *A. jandaei* are considered the major accused for increased mortality among wild and cultured fishes, resulting in massive economic losses [12]. *A. veronii* has recently been monitored as the common accused pathogen in outbreaks globally occurring in fish characterized by ulcerative syndrome in different fish species [22, 56, 57]. *A. veronii* is an opportunistic pathogen that may attack fish farms as a secondary infection or in immune disturbance cases as a result of stressor exposure [26, 57].

**Susceptible species and life stages**

*A. veronii* have been isolated from different species of fish for different life stages worldwide. Among the cases in which the infection by *A. veronii* was recorded around the world as follow: in China, the infection was recorded in channel catfish, long snout catfish, Siberian sturgeon, and loach fish [56, 58-61]; in India, the infection cases was reported in Oscar and fresh water ornamental fish [62, 63]. The *A. veronii* infection was recorded also in zebra fish in Korea [64]; guppy in Israel [65]. With regard to *A. veronii* infections in tilapia fish and its different species, cases have been recorded in Egypt [24, 66, 67], India [22], Saudi Arabia [23], Thailand [17, 54, 55], and in Malaysia [25].

With regard to infection and the different life stages, the previous studies have been documented *A. veronii* infection at the different age stages such as fry of grass carp [68], fingerling of tilapia [22, 24, 54], juvenile [25] and adult stage [24, 66, 67].

**Seasonal prevalence**

Recently, Nile tilapia has been suffered from phenomena of massive mortality outbreaks mainly in summer season. In 2015, about 37% of the Egyptian tilapia fish farms have faced unexplained summer mortality with an average 9.2%, which have great economic losses reached to US$100 million [9]. Youssuf *et al.* [24] documented 50:80% mortality outbreaks in Egyptian farmed Nile tilapia from 13 farms in different localities from April to October 2018. In the Eastern Province of Saudi Arabia, *A. veronii* were isolated from 31.07% of the examined fish and causes mortalities in reared Nile tilapia in the duration from January to December 2015 [23]. Eissa *et al.* [67] has recorded the higher prevalence of *A. veronii* during summer months (86.25%) in cultured Nile Tilapia in El-Sharkia Governorate, Egypt. In contrast, Hassan *et al.* [23] has reported *A. veronii* infection overall the year with higher incidence in winter months (45.71%) than summer ones (25.71%). In agreement with this result, a study was performed from January to March 2018 confirmed the higher incidence of *A. veronii* in Nile tilapia showing signs resemble streptococcal infection [22]. In Malaysia, *A. veronii* was successfully isolated from 50% of moribund red hybrid tilapia in May 2017, which suffering from massive mortalities [25].

**Clinical signs and diagnosis**

**Clinical symptoms and postmortem lesions**

*A. veronii* is one of the fish septicemic diseases that contribute to high mortality rates and significant economic losses for the aquaculture industry. The naturally infected fish by *A. veronii* were externally characterized by sluggish movement, loss of appetite, loss of scales, dark skin pigmentation, corneal opacity, uni or bilateral exophthalmia, skin ulcers, and erythema [22, 66]. Some cases were characterized by hemorrhagic lesions throughout the body surface, opercular cover and fins base, anal opening protrusion with ascites as shown in Figure 1 [23, 25, 67]. Internally, the infected fish showed enlarged, congested, and hemorrhagic internal organs especially the hemopoietic organs (liver, spleen and kidney), enteritis [23, 55, 66] with enlarged gall bladder in some cases [25] (Figure 1).
Experimentally infected Nile tilapia with 9 X 10^8 A. veronii shown 80% cumulative mortality, skin black discoloration, loss of appetite with internal hemorrhage and hepatic congestion [66]. Some cases shown hemorrhagic internal organs, especially liver, kidney, and intestinal inflammation with mortalities up to 100% within 24 h after artificial infection accompanied by a stressor such as transport-induced stress [17, 54]. Bilateral exophthalmia was observed in 70% of Nile tilapia experimentally infected intra-peritoneally by 3.2 x 10^7 colony forming unit (CFU)/fish with 100% cumulative mortality [22]. Other cases were reported to have signs and postmortem lesions in the experimentally infected fish more severe than the naturally infected, which characterized by external inflammation at the site of injection and hemorrhage on the skin and scales pocket with internal enteritis [23]. Besides, other signs were recorded such as abdominal dropsy, hemorrhagic ulcers on the body, reddening at anal orifice and fine bases, unilateral or bilateral pop eye, fin rot, and internal hemorrhagic fluids with congested organs [24] (Figure 2).

Figure 1: Oreochromis niloticus naturally infected with Aeromonas veronii showing skin darkness and abdominal dropsy (A) [23], bilateral exophthalmia (B) [22], hemorrhagic ulcer (C) [67] and ulcerations varied in their degrees (red arrows) (D) [23], enlarged liver and gall bladder and hemorrhages on liver (E) [22].
Figure 2: *Oreochromis niloticus* experimentally infected with *Aeromonas veronii* showing hemorrhage at basal fins (A) [54], generalized external hemorrhage on fish body skin erosions (B), reddening and inflammatory reaction at the site of injection (C) [23], exophthalmia (unilateral and bilateral) (D) [24], yellow liquid accumulation in the swollen intestine (arrows) (E) [17], and hemorrhage in the liver (F) [54].

**Phenotypic properties of Aeromonas veronii**

*A. veronii* has been described as Gram-negative, rod shaped, mesophilic, motile, and facultative anaerobic bacteria [69], that normally inhabit aquatic environment, sewage, food, and soil [70]. *A. veronii* colony in tryptic soy agar appeared as circular colony, which its diameter average from 0.9–1.1 mm, slightly elevated, shiny, and creamy to yellow color [23, 24]. While *A. veronii* grown as yellow colonies on Rimler-Shotts (RS) medium, and small, round, and dark green with dark center colonies on Aeromonas base media and not able to ferment lactose on MacConkey's agar as described by Abd El Latif et al. [66]. In conventional biochemical tests, *A. veronii* was indole, Voges-Proskauer, ornithine decarboxylase, citrate, β-galactosidase, gelatin-positive [17, 24, 66] and shows resistance to Vibrio-static reagent O/129 (150 µg ml⁻¹) according to Hassan et al. [23].

Several investigations spotted the light on the virulence factors, which responsible for *A. veronii* pathogenicity such as caseinase, gelatinase, lipase-enterotoxins, alt, act, hemolysin, serine protease, aero, floR, sul1, qacED1, qnrS, and aada1[59, 60, 65, 66]. *A. veronii* also characterized by its ability to multiply in a broad range of temperatures and salinity, to auto-aggregation and biofilm formation [65, 71].

**Genetic diversity of the Egyptian A. veronii strains and those from other countries**

Recently, *A. veronii* sequences from isolates originating from Egypt, China, Bangladesh, Eastern Province (KSA), India, Israel, Malaysia, Thailand are accessible in the GenBank database (https://www.ncbi.nlm.nih.gov/genbank/). The reported sequences identity among isolates originating from different continents has been recorded in Table 1. The basic local
alignment search tool (BLAST) analysis of the 16S rRNA gene sequence of *A. veronii* isolates from Egypt or other countries showed identity % ranged from 97 to 100%. Dong *et al.* [17] who stated that genetic characterization of *A. veronii* isolated from Nile tilapia in Thailand are like that recovered from Nile tilapia in Malaysia. While *A. veronii* that isolated from Malaysian hybrid red tilapia by Amal *et al.* [25] are closely related to *A. veronii* recovered from water of loach (*Paramisgurnus dabryanus*) culture in China.

Table 1. Overview of available *Aeromonas veronii* sequences in GenBank and the percentage nucleotide identity for sequences originating from Egypt and other countries.

<table>
<thead>
<tr>
<th>Fish species (country)</th>
<th>Genbank accession number</th>
<th>Identity %</th>
<th>References</th>
</tr>
</thead>
<tbody>
<tr>
<td>Nile tilapia (Egypt)</td>
<td>MK584926</td>
<td>100</td>
<td>[24]</td>
</tr>
<tr>
<td>Nile tilapia (India)</td>
<td>MH998019</td>
<td>100</td>
<td>[22]</td>
</tr>
<tr>
<td>Red hybrid tilapia (Malaysia)</td>
<td>MG283140</td>
<td>98</td>
<td>[25]</td>
</tr>
<tr>
<td>Nile tilapia (Thailand)</td>
<td>KP899499 - KP899504</td>
<td>99.9</td>
<td>[54]</td>
</tr>
<tr>
<td>Nile tilapia (Thailand)</td>
<td>KX714288</td>
<td>99.9</td>
<td>[17]</td>
</tr>
<tr>
<td>Nile tilapia (Thailand)</td>
<td>KU975017, KU975018, KU975021, KU975022</td>
<td>99.8</td>
<td>[55]</td>
</tr>
<tr>
<td>Longsnout catfish (China)</td>
<td>HQ434550, HQ540319 and HQ540320</td>
<td>97 - 100</td>
<td>[56]</td>
</tr>
<tr>
<td>Channel catfish (China)</td>
<td>GQ180116, GQ280902, KF761317 and KF761318</td>
<td></td>
<td>[58]</td>
</tr>
<tr>
<td>Guppy (Israel)</td>
<td>MF276645</td>
<td>99</td>
<td>[65]</td>
</tr>
</tbody>
</table>

**Histopathology**

Histopathological examination of moribund fish infected by *A. veronii* was reported in several papers from different organs including brain, spleen, liver, kidney, muscles, eye, gills, skin, and intestine (Figure 3). Liver of fish infected with *A. veronii* showed blood congestion and sever tissue necrosis and liver vessels were surrounded by hemosiderin, massive fatty changes with swollen hepatocyte [22, 66]. On other cases, hepatocyte and vacular degeneration with congested sinusoid were reported [23, 72]. Kidney of fish infected by *A. veronii* showed glomerulus congestion with moderate degenerative alterations in renal tubules combined with absence of nuclei and pyknosis, while in other cases necrotized renal tubules and glomerular destructions were recorded [22, 66, 72]. Other studies recorded only mild vacular degeneration in epithelial lining of some renal tubules [23]. Spleen was congested and hyperemic with marked degenerative alterations [17]. Spleen hemopoietic tissue depletion has been widespread in some cases [23]. Sometimes the spleen may show focal hemorrhages with proliferating lymphocytes [25]. The intestine showed epithelium necrosis, sloughing and hemorrhage [17]. Inflammatory exudates between secondary lamellae are seen in the gill section [22]. Regarding the brain, the main observed histopathological alteration is focal inflammation in the leptomeninges and congestion of blood vessels [25]. Whereas, Raj *et al.* [22] described sensory retina separation from epithelium retinal pigment.
Figure 3: Histological changes of Oreochromis niloticus tissues infected with A. veronii. Liver: showing severe blood congestion (A) (H&E, 100 µm) [17], B: vacuolar degeneration of the hepatocytes (v) (H&E) [23], C: showing vacuolar degeneration (*) and necrosis (n) around the area of hepatopancreas (H&E) [23], D: severe fatty changes (black arrows) with hepatocyte necrosis (line arrow) along with sinusoidal congestion (arrowheads) (H&E, 100 µm) [22], and E: focal area of hepatocellular swelling and presence of syncytial hepatitis cells (arrows) (H&E, 200 µm) [25]. Kidney: showing interstitial hemorrhage (F) (arrow) and melanophores aggregation (m) (H&E) [23], G: vacuolar degeneration of the epithelium lining renal tubules (head of arrow), hemorrhage (arrow), thickening of glomerular capillaries’ basement membrane (tg) and hyaline droplets (hd) in the lumen of some renal tubules (H&E) [23], H: multiple Gram-negative bacilli (gnb) in the renal tissue (Stained with Gram stain) [23], and K: mild degenerative changes along with loss of nuclei (black arrows) and pyknosis (line arrows) (H&E, 100 µm) [22]. Eye (L) showing detachment of sensory retina (black arrow) from retinal pigment epithelium (red arrow) (H&E, 200 µm) [22]. Spleen (M) exhibited hyperemia and hemorrhage (H&E, 50 µm) [17], focal hemorrhages with proliferating lymphocytes (H&E, 100 µm) (N) [25]. Intestine (P) showed epithelial cell damage and sloughing into gut lumen (H&E, 200 µm) [17]. Gills (S) exhibiting inflammatory exudate between secondary lamellae (arrows) (H&E, 100 µm) [22]. Brain (X) showing severe blood congestion occurred in the brain (H&E, 200 µm, respectively) [17].

**Disease control measures**

The unaware application of antibiotics as a magic tool for disease control in aquaculture sector led to the appearance of antimicrobial resistance phenomena, which subsequently threaten the public health. Therefore, the recent research pays their interest to the environmentally friendly alternatives to antibiotics for the microbial infection control such as natural herbs, probiotics, immunostimulants and others [73].

There were numerous studies for treatment of A. veronii infection in tilapia fish species (Table 2). From these, the study of Elabd et al. [74] who recorded Astragalus membranaceus nanoparticles (ANP) at 2% kg⁻¹ diet had significantly enhanced lysozyme and nitric oxide (NO) activities, as well as improved superoxide dismutase (SOD), catalase (CAT), and glutathione peroxidase (GPx) activities, interleukin 1, beta (IL-1β) genes expression after 30 days of feeding. Abdelghany et al. [75] testified the vital effects of dietary microalgae, Nannochloropsis oculata (NP) at a concentration of 5% medicated feed on immunity enhancement and disease resistance of Nile tilapia against A. veronii. Sewaka et al. [76] studied the efficacy of synbiotics medicated diets which resulted in improved growth.
performance, biochemical, and immunological parameters mainly against A. veronii in juvenile red tilapia. Also, Yilmaz [78] reported that caffeic acid especially at 5 g kg\(^{-1}\) has increased Nile tilapia resistance against A. veronii as a result of the significantly increased phagocytic index, potential killing activity, respiratory burst activity, serum myeloperoxidase activity and serum CAT activity. Furthermore, increased levels of immune expression [HSP70, IL-1\(\beta\), TNF-\(\alpha\), CC-chemokine (CC1), IL-8, toll-like receptor 7 (tlr-7), IFN-\(\gamma\) and IgM] and antioxidant related genes [SOD, CAT and GPx] were observed. Crude glucan (Cr-glucan) is one of the alternatives used also, which gave good results in increasing the resistance of tilapia fish against A. veronii as documented by Chirapongsatonkul et al. [79].

Aeromonas veronii was treated in other fish species using various medication trials. An Indian research was done in Xiphophorus hellerii using silver (AgNPs) and zinc oxide (ZnONPs) nanoparticles at 1 mgL\(^{-1}\) concentration for evaluation their effectiveness against A. veronii. They showed 83.3% and 100% survivability in infected fish, respectively [80]. A novel antagonistic bacteria named Streptomyces flavotricini X101 promoting growth rate and survivability of grass carp after a month feeding at concentration of 900 \(\mu\)gmL\(^{-1}\)[81]. An investigation was done using selenium reported that 1 and 2 mgkg\(^{-1}\) concentration have ability to enhanced growth and immunological performance, increase resistance against A. veronii and limitation of multiple stresses in P. hypophthalmus [82]. Sewaka et al. [76] recorded the therapeutic effect of Lactobacillus rhamnosus GG supplemented diets against A. veronii in juvenile red tilapia. Vaccination is regarded as an important alternative strategy for combating A. veronii, as shown by numerous studies. From these vaccines, live attenuated vaccine \(\Delta\)hisJ [83], recombinant Lactobacillus casei expressing OmpAl [84] and recombinant Lactobacillus casei expressing flab [85].

Zoonotic importance

Latest literature confirmed that four species of Aeromonas (A. veronii, A. hydrophila, A. caviae and A. dhakensis) were responsible for 95.4% of human clinical cases and A. veronii alone sharing with 21.54% of total Aeromonas infections [86]. The highest incidence was recorded in children, elderly people suffering from hematologic malignancies or hepatobiliary disease and immunocompromised individuals [70, 87, 88]. According to Janda et al. [89] A. veronii was the most famous pathogen suspected of being the pathogen among the 7 Aeromonas species recognized by human infection. A. veronii adding to the critical value of public health as previously stated in many articles [70, 90, 91]. Several investigations recorded the significant impact of A. veronii on human health resulting in signs such as septicemia, kidney disorders, pulmonary disorders, diarrhea, and gastroenteritis [92-95]. Additionally, Ko et al. [96] recorded a case exhibiting variety of clinical symptoms, including gastroenteritis, pneumonia, infection of the hepatobiliary tract, infection in the skin and soft tissue, empyema, septic inflammation in joints, osteomyelitis, meningitis, endocarditis, and bacteremia. Furthermore, another non-traumatic case of old woman suffering from progressive pain with right shoulder joint immobility was recorded in china was found to be due to A. veronii infection[97]. Similarly; Roberts et al. [98] had recorded case report in an immunocompetent 81-year-old man developed septic arthritis and bacteraemia with A. veronii. In Bangladesh, Kühn et al. [99] reported a case suffering from cellulitis, peritonitis, meningitis and respiratory tract disorders due to A. veronii infection. Joseph et al. [100] documented an exudate formed after aquatic contact under the left eye of a 10-year-old male from previously infected wounds, which was cultured and revealed two different Aeromonas spp. (A. jandaei and A. veronii). McKenzie et al. [101] reported a
remarkable and un-common case of *A. veronii* septicemia, secondary to a drilling

Table 2. Alternative approaches to antibiotics for treatment and control of *A. veronii* infection in Nile tilapia and other fish species

<table>
<thead>
<tr>
<th>Treatment</th>
<th>Fish Species</th>
<th>Dose and route of administration</th>
<th>Duration</th>
<th>Immune-Stimulatory Role</th>
<th>Reference</th>
</tr>
</thead>
<tbody>
<tr>
<td><em>Astragalus membranaceus</em> nanoparticles (ANP)</td>
<td>Nile tilapia</td>
<td>1, and 2%kg⁻¹ diet</td>
<td>30 days</td>
<td>Significantly enhanced lysozyme activity and NO activities, as well as improved SOD, CAT, and GPX, IL-1β genes.</td>
<td>[74]</td>
</tr>
<tr>
<td><em>Nannochloropsis oculata</em> (NP)</td>
<td>Nile tilapia</td>
<td>5, 10, and 15% in diet</td>
<td>60 days</td>
<td>Significantly improved serum lysozyme activity, NO, and NBT levels with significant upregulation of cytokines (IL-1β, IL-8, IFN-γ, TGF-β and TNF-α).</td>
<td>[75]</td>
</tr>
<tr>
<td>Black mulberry (Morus nigra)</td>
<td>Nile tilapia</td>
<td>0.75%, 1.5%, 2.0%, and 3.0% in diet</td>
<td>60 days</td>
<td>Increased activities of serum lysozyme, MPO, SOD and CAT, and increased the expression levels of immune-related genes [IL-1β, TNF-α, IgM, IFN-γ and HSP70] and antioxidant-related genes (SOD, CAT, and GPX).</td>
<td>[77]</td>
</tr>
<tr>
<td>Caffeic acid</td>
<td>Nile tilapia</td>
<td>1, 5, 10 g kg⁻¹ in diet</td>
<td>60 days</td>
<td>Caffeic acid especially at 5 g kg⁻¹ significantly increased phagocytic index, potential killing activity, respiratory burst activity, serum MPO activity and serum CAT activity. Furthermore, increased levels of immune expression [HSP70, IL-1β, TNF-α, CC1, IL-8tr-7,IFN-γ and IgM] and antioxidant related genes [SOD, CAT and GPx].</td>
<td>[78]</td>
</tr>
<tr>
<td>Crude glucan (Cr-glucan) and commercial β-glucan from <em>Saccharomyces cerevisiae</em> (Yb-glucan)</td>
<td>Nile tilapia</td>
<td>10 μg fish⁻¹ by intraperitoneal injection</td>
<td>24h</td>
<td>Cr-glucan developed the immunity that protect Nile tilapia from <em>A. veronii</em> infection</td>
<td>[79]</td>
</tr>
<tr>
<td>Jerusalem artichoke (JA) and <em>Lactobacillus rhamnosus</em> GG (LGG)</td>
<td>Red tilapia</td>
<td>10.0 JAg kg⁻¹ diet, 1⁰⁶ CFU g⁻¹ LGG diet and 10.0 JA g kg⁻¹ +1⁰⁸ CFU g⁻¹ LGG diet.</td>
<td>30 days</td>
<td>JA and LGG enhanced gut mucosal immunity and lysozyme activity against <em>A. veronii</em> challenge in red tilapia</td>
<td>[76]</td>
</tr>
<tr>
<td>Silver (AgNPs) and zinc oxide (ZnONPs) nanoparticles</td>
<td><em>Xiphophorus hellerii</em></td>
<td>0.5, 1.5 and 10 mgL⁻¹ intramuscular and bath dip</td>
<td>48h and 96h</td>
<td>enhance the survival rate of 83.3% in AgNPs and 100% in 1 mgL⁻¹ of ZnONPs</td>
<td>[80]</td>
</tr>
<tr>
<td>Selenium</td>
<td><em>Pangasianodon hypophthalmus</em></td>
<td>1 and 2 mgkg⁻¹ diet</td>
<td>60 days</td>
<td>Increased anti-oxidative status in different tissues, and immunological status.</td>
<td>[82]</td>
</tr>
<tr>
<td>Recombinant <em>Lactobacillus casei</em></td>
<td>Common carp</td>
<td>1⁰⁶ CFUg⁻¹ diet“Oral vaccination”</td>
<td>66 Days</td>
<td>The recombinant vaccine candidate stimulated high serum or skin mucus specific antibody titters and induced a higher lysozyme, ACP. SOD activity with upregulation to IL-10, IL-β, IFN-γ, TNF-α genes expression.</td>
<td>[84]</td>
</tr>
<tr>
<td>Recombinant <em>Lactobacillus casei</em></td>
<td>Common carp</td>
<td>2 x 10⁶ cells in diet</td>
<td>58 days</td>
<td>The recombinant <em>L. casei</em> was effectively induce humoral immunity, increase the serum immunological index, leukocytes phagocytosis percentage with significant increase of IL-10, IL-β, IFN-γ and TNF-α genes expression.</td>
<td>[85]</td>
</tr>
</tbody>
</table>

NO=nitric oxide, SOD=superoxide dismutase, CAT= catalase, GPX= glutathione peroxidase, MPO= myeloperoxidase, IL-1β=interleukin 1, beta, TNF-α=tumor necrosis factor alpha, IFN-γ=interferon gamma, NBT= nitroblue tetrazolium, IL-8=interleukin 8, TGF-β=transforming growth factor-beta, IgM=immunoglobulin M, HSP70=heat shock protein 70,tlr-7=toll-like receptor 7, CC1=CC-chemokine,ACP=acid phosphatase activity, AgNPs =Silver, and ZnONPs=zinc oxide nanoparticles, JA=Jerusalem artichoke, LGG=*Lactobacillus rhamnosus* GG
injury in an immunocompetent 29-year-old man without any significant past medical history. In Taiwan, *A. hydrophila* and *A. veronii* were implicated in a case suffering from empyema with fever, dyspnea, and pain in the chest [102]. *A. veronii* have been confirmed to have a significant impact on consumer health as foodborne pathogens [100, 103-105].

**Conclusion**

*A. veronii* is an important fish pathogen causing mass mortality of Nile tilapia during the summer months. For the control of this disease, the determination of the virulence genes associated with disease outbreaks and successful therapeutic compounds are necessary. It's important to find out therapeutic alternatives for minimizing the risk of antibiotics use and the antimicrobial resistance.

**Acknowledgment**

We would like to thank all staff members of Fish Diseases and Management Department, Faculty of Veterinary Medicine, Zagazig University.

**Conflict of interest**

The authors declare that there is no conflict of interest.

**References**


[40] Nicholson, P.; Fathi, M.; Fischer, A.; Mohan, C.; Schieck, E.; Mishra, N.;


الملخص العربي

رشا محمد رضا * ، عبد الحكيم المر ، ياسر عبد الحكيم ووسام الشحات
قسم أمراض ورعاية الأسماك، كلية الطب البيطري، جامعة الزقازيق، الزقازيق، الشرقية، مصر.

يعتبر البلطي النيلي من أفضل الأسماك المستزرعة في مصر والثاني على مستوى العالم. تفاقم ارتفاع الطلب على البروتين الحيواني مع الاستزراع المكثف للأسماك مما أدى إلى انتشار الأمراض التي تسببت في خسائر كبيرة في تربية الأحياء المائية للبلطي خلال السنوات الأخيرة. مع إشارة خاصة إلى تشذب الأمراض البكتيرية، فإن الأوبيومس فيروني هو أحد مسببات الأمراض البكتيرية التي تم الإبلاغ عنها مؤخرًا والذي يؤثر على البلطي المستزرع وأنواع الأسماك الأخرى في جميع أنحاء العالم. في الآونة الأخيرة، تم الإبلاغ عن الأوبيومس فيروني كتهديد كبير على تجمعات البلطي النيلي مما أدى إلى حالات نفوق عالية. ولقد تم الإشارة إلى تشذب الأمراض البكتيرية عامة، والأوبيومس فيروني خاصة كأحد مسببات الأمراض البكتيرية التي تسجلها مؤخرًا والتي تؤثر على البلطي المستزرع وأنواع الأسماك الأخرى في جميع أنحاء العالم. إن الأوبيومس فيروني يمثل تهديد كبير على البلطي النيلي والذي يؤدي إلى نفوق شديد بالإضافة إلى مقاومته الواسعة للمضادات الحيوية. وعليه فقد ركزت الدراسات الحديثة على استخدام رافعات المناعة كبدائل.

الملخص تلخص هذه المقالة مجموعة من البيانات العلمية السابقة عن ظواهر النفوق الصيفي للبلطي مع إشارة خاصة إلى الأوبيومس فيروني كمسبب مهم مع تسلط الضوء على علاماتها السريرية وعلامات ما بعد الوفاة، التغيرات النسيجية المرضية، والتنوع الجيني والعلاقة مع العوامل الأخرى التي تم تحملها على الجين بنك، والتشخيص، وتداريب الاحتكام، والآثار الاقتصادي لذلك وأهميته على الصحة العامة.