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Comparative Bacteriological, Molecular, and Pathological Studies on *Streptococcus agalactiae*, *Psychrobacter*, and *Enterococcus* Bacteria Co-Infecting Cultured *Oreochromis niloticus* and *Mugil cephalus*

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Abstract

INFECTIOUS diseases, especially bacterial diseases, are regarded as one of the primary problems in aquaculture systems, resulting in significant economic losses and impeding aquaculture's sustainability. Different fish species exhibit varying degrees of susceptibility to bacterial pathogens due to differences in immune competence, anatomical barriers, and genetic resistance. Subsequently, the purpose of this study is to assess the severity of the pathological alterations caused by bacterial infection in *Oreochromis niloticus* (*O. niloticus*) and *Mugil cephalus* (*M. cephalus*). Equal numbers (305) from each species of *O. niloticus* and *M. cephalus* were sampled from private farms in Egypt and examined for any gross lesions. From the examined fishes, 19 and 11 that showed the most severe gross changes were collected from *O. niloticus* and *M. cephalus*, respectively. Postmortem examination and internal organs were examined for both bacteriological and histopathological analysis. *Streptococcus agalactiae* (*S. agalactiae*) was the most predominant pathogen isolated from both diseased *O. niloticus* (42.1%) and *M. cephalus* (45.4%), followed by *Psychrobacter spp.* Other pathogens such as *Enterococcus faecalis* (*E. faecalis*) and *Enterococcus faecium* (*E. faecium*) were isolated at lower frequencies. This highlights *S. agalactiae* as a key infectious agent in both cultured fish species. The examined *O. niloticus* and *M. cephalus* exhibited numerous pathological alterations. *O. niloticus* and *M. cephalus* differ significantly in the severity of their lesions. These variations might be attributed to host-specific and pathogen-related factors, such as anatomical and physiological differences, innate immune competence, and bacterial virulence expression variation in different hosts, which need further investigations.

Keywords: *O. niloticus*; *M. cephalus*; *S. agalactiae*; *Psychrobacter spp.*; *E. faecalis*; *E. faecium.*

Introduction

Fish is one of the most popular foods in the world, and it continues to gain popularity because many people consider it a wholesome and nourishing part of their diet. Over the next ten years, fish consumption is predicted to increase even more [1]. Aquaculture is growing globally and is regarded as the food production industry with the most rapid growth rate. *Oreochromis niloticus* (*O. niloticus*) is currently the second most farmed freshwater fish worldwide after carp and the most extensively cultivated freshwater fish species in Egypt [2, 3]. Over the past decade, *Mugil cephalus* (*M. cephalus*) has emerged as a significant aquaculture commodity and holds considerable economic value in Egypt [4].

Egypt is regarded as a major agricultural region utilizing drained and recycled water in aquaculture. Consequently, numerous atypical diseases caused by

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bacterial infections are identified in these aquaculture systems [5]. In fact, there are ongoing governmental initiatives to enhance and sustain the quality of freshwater bodies in Egypt; however, the utilization of poultry and livestock manure as fertilizer by certain fish farmers in their aquaculture ponds [6] and anthropogenic pollution [7] may be introducing novel bacterial isolates into the country's aquaculture system. The expansion of aquaculture and intensive fish farming has resulted in various challenges, increasing stressors, and a heightened risk of different fish disorders [8, 9]. Infectious diseases are regarded as one of the primary issues in fish farms, resulting in significant economic losses [10]. Particularly, bacterial diseases are a serious problem in fish farming, leading to significant economic damage and hindering aquaculture sustainability around the world [1].

Host species (species variation) can affect the severity of bacterial infections and their associated pathological lesions in freshwater fishes. Different fish species exhibit varying degrees of susceptibility to bacterial pathogens due to differences in immune competence, anatomical barriers, and genetic resistance [11]. Additionally, variations in gill and skin morphology among species can influence bacterial adherence, invasion, and further modulation of infection outcomes [12]. For instance, there was a variation between farmed *O. niloticus* and African catfish infection severity when they were coinfected with *Aeromonas spp.*, *Enterococcus faecalis (E. faecalis)*, and *Vibrio alginolyticus* [13]. Another example is the variation in severity of infection between *O. niloticus* and European sea bass *(Dicentrarchus labrax)* when they were infected with *Vibrio anguillarum* and *Streptococcus iniae* [14].

The aquaculture industry is seriously threatened by several bacterial pathogens; one of them is *Streptococcus agalactiae* (*S. agalactiae*), a gram-positive bacterium that causes meningoencephalitis and septicemia in both freshwater and saltwater fish species [15]. *S. agalactiae* infection reveals a variety of clinical signs in tilapia, mostly loss of appetite [16], lethargy and dorsal rigidity due to lesions in the central nervous system [17], nervous manifestations [18], erratic swimming (spiraling or spinning), unilateral or bilateral exophthalmia (pop-eye), corneal opacity, eye hemorrhages, and hemorrhages at the base of the fins and opercula. Moreover, skin darkening, an expanded belly, anomalies of the spine, and body curvature have all been reported [19]. On the other hand, *M. cephalus* has shown several characteristic symptoms of a streptococcal infection, unpredictable swimming behavior, opacity, intraocular and peri-orbital hemorrhage, and exophthalmia. In addition, reddening and bleeding were frequently observed in the musculoskeletal and integumental systems. Both inside the skull and on the exterior of the body, hemorrhagic regions were seen, especially near the mouth, snout, operculum, and fins [20].

One of the major fish infections that has a significant influence on aquaculture techniques globally is *Enterococcus species*, which causes economically significant losses in both freshwater and marine farmed fish and is considered as an indication of fecal pollution of the aquatic environment [21]. *O. niloticus* is one of the most vulnerable freshwater species to *Enterococcus species* infection [22]. It is also regarded as one of the most significant infectious diseases affecting *M. cephalus* [23]. Septicemic signs, lethargy, anorexia, exophthalmia, abdominal distention, and skin and fin base bleeding are among the clinical symptoms observed in infected fish [22, 24]. There are also indications of a septicemic eye lesion, such as exophthalmia and unilateral or bilateral eye opacity, and skin abnormalities, such as dark coloring, bleeding, ulceration, or disconnected scales, have been reported. In addition, ascites may also result from this infection [22, 25].

Another important salt-tolerant bacteria is the *Psychrobacter species*, which are gram-negative and cold-adapted [26]. Lethargy and slow movement, body and operculum hemorrhages and ulcers, sporadic scale loss, and fin congestion and rot, particularly on the tail fin, were among the clinical symptoms displayed by the marine fishes infected with *Psychrobacter glacincola* (*P. glacincola*). Additionally, the postmortem examination revealed congestion in the kidneys, spleen, and liver [27]. Unfortunately, there is limited literature about *Psychrobacter spp*. infection in *O. niloticus* and *M. cephalus*.

Therefore, the aim of this study was to identify the most common bacterial pathogens affecting *O. niloticus* and *M. cephalus* in some fish farms in Egypt, in addition to comparing the severity of pathological changes associated with each bacterial infection, thereby providing valuable information regarding species-specific variations in disease susceptibility. Such comparative analysis can serve as a guiding framework for developing targeted prevention and control strategies for bacterial infections in freshwater fish species. The implementation of these species-specific management approaches is expected to impact Egyptian aquaculture systems positively and subsequently contribute to the overall economic stability of the regional fish farming industry.

Material and Methods

Ethical of approval

All applicable national and institutional guidelines for the care and use of animals were followed. The protocol of this study was revised in detail and approved by the Institutional Animal Care

and Use Committee, Faculty of Veterinary Medicine, Cairo University, Egypt (Vet. CU. IACUC, Accession No. Vet CU25122023886). All methods are adhered to ARRIVE guidelines (https://arriveguidelines.org) for the reporting of animal experiments.

Fish Sampling and Collection:

Three hundred and five *O. niloticus*, weighing 110 ± 5 gm, were examined for any gross abnormalities. Of these fish, nineteen *O. niloticus* showed the most severe gross changes were sampled for further analysis. Additionally, three hundred and five *M. cephalus*, weighing 250 ± 5 gm, were examined, and eleven fish showing the most severe gross abnormalities were collected. This purposive sampling method was chosen to ensure that samples were representative of the pathological condition being investigated [28]. The collected fishes were from private farms in Shader Azzam, Port Said Governorate, Egypt. The farms operated under a polyculture system, with a combined stocking density of approximately 3–4 fish/m^3, *comprising O. niloticus* and *M. cephalus* at a ratio of roughly 4:1. The fishes were transported in an icebox to the wet lab of the Fish Diseases Department, Animal Health Research Institute, Dokki, Egypt. All fishes were grossly examined for detection of any lesions according to the methods described by [29]. For demonstration of the internal abnormalities, the postmortem examination was performed on all moribund fishes according to [30]. The fishes were examined in a sterile manner using a three-line incision for *O. niloticus* and *M. cephalus* [31]. The skin was sterilized and treated with 70% ethyl alcohol. The incision was made using sharp, pointed scissors, which were inserted close to the anus so that an intra-abdominal point remained fixed, while the cut was made in close contact with the ventral side to avoid internal tissue damage. The abdominal wall was removed, and the internal organs were exposed and then examined for both bacteriological and histopathological analysis.

Bacteriological examination:

Isolation of bacterial isolates

Loopfuls were taken aseptically from lesions in the liver, kidneys, brain, and spleen. We immediately streaked the samples into Trypticase Soy Agar plates and subsequently incubated them at 37°C for 24 hours. Bacterial isolation was performed according to [29].

Identification of bacterial isolates

All bacterial isolates were identified by studying colony growth characteristics. Smears were prepared from the colonies, stained with Gram's stain, and examined microscopically to demonstrate the morphology, arrangement, and staining reaction of the microorganisms. The bacterial isolates were identified according to schemes of biochemical reactions, such as catalase and oxidase tests, provided in Bergey's Manual of Systematic Bacteriology [32].

Molecular characterization of isolated bacteria

The identification of the isolates was confirmed by a tool of identification such as conventional polymerase chain reaction (PCR). Both collected gram-negative and positive isolates were subjected to 16S rRNA (using the universal primer pair) and further sequencing.

DNA Extraction

The isolates were inoculated into Brain Heart Infusion Broth (BHIB) and incubated for 24 hours. According to the manufacturer's instructions, DNA was extracted using the PathoGene-spin™ DNA Extraction Kit.

Genotypic confirmation by 16S rRNA

PCR was carried out using oligonucleotide primers for the general 16srRNA BSF8/27 (5-AGAGTTTGATCCTGGCTCAG-3) and BSR1541/20 (5-AAGGAGGTGATCCAGCCGCA-3) [33]. The PCR protocol was as follows: an initial denaturation at 95°C for 5 min, followed by 35 cycles at 94°C for 2 min; 35 cycles of 94°C for 30 s, 55°C for 30 s, and 72°C for 30 s; and 72°C for 5 min using the Applied Biosystems Veriti Dx 96-well thermocycler (Life Technologies). The primer used in the amplification study was obtained from [33].

Sequencing

PCR products of 16S rRNA were purified using a QIAquick PCR product extraction kit. (Qiagen, Valencia). BigDye Terminator V3.1 Cycle Sequencing Kit (Perkinelmer) was used for the sequence reaction, and then it was purified using a Centrisep spin column. DNA sequences were obtained by an Applied Biosystems 3130 genetic analyzer (HITACHI, Japan), and a BLAST® analysis (Basic Local Alignment Search Tool) [34] was initially performed to establish sequence identity to GenBank accessions. The phylogenetic tree was created by MegAlign. Phylogenetic analysis was done using neighbor joining in MEGA X: Molecular Evolutionary Genetics Analysis across computing platforms [35].

Pathological examination:

Tissue specimens from the spleen, kidneys, liver, intestine, gills, skin, and muscle of the same apparently diseased fishes used in the bacteriological examination were freshly sampled immediately and immersed in 10% neutral buffered formalin (10% NBF) for 24 hours to ensure proper fixation. The tissue samples then were trimmed, washed, and dehydrated in ascending grades of ethyl alcohol, cleared in xylol, and embedded in paraffin. Paraffin

sections of 3-5 µm thickness were prepared and stained with Harris hematoxylin and eosin stain [36]. Histoarchitecture and pathological changes observed in all examined sections were described and photographed using the Olympus CellSens dimension software of an Olympus DP27 camera connected to an Olympus BX43 light microscope.

Lesion scoring:

All the detected histological alterations in the H&E-stained sections obtained from the liver, kidneys, gills, spleen, and intestines of the four fish per fish species were scored by calculating the percentage of the lesion frequencies per ten distinct randomly chosen high-power microscopic fields per organ per fish (40 fields/organ/species) using the following six-grade score system: - = absence of the lesion in all fish of the fish species, + = 1–10%, ++ = 11–25%, +++ = 26–50%, ++++ = 51–75%, and +++++ = over 75% [37].

Statistical analysis

To evaluate the importance of the mean difference between the frequencies of the recorded histopathological alterations of *O. niloticus* and *M. cephalus*, data were analyzed using a T-test using SPSS version 27.0.1 software (IBM, USA). A P-value under 0.05 was determined to signify statistical significance [38].

Results

Bacteriological findings

Bacteriological isolation and identification

The microbiological examination revealed that the pure colonies were acquired from Trypticase Soy Agar (TSA) after 24 hours at 37°C. Culture revealed a pinpoint-to-minute circular white appearance of *S. agalactiae* colonies, while *Enterococcus faecium* (*E. faecium*) appeared as small brownish circular colonies. Furthermore, colonies of *E. faecalis* appeared as small white translucent colonies. All previous isolates were oxidase and catalase negative. When stained by Gram stain, it appeared as gram-positive, non-motile cocci. Furthermore, *Psychrobacter spp.* were acquired from Trypticase Soy Agar (TSA) after 24 hours at 37°C and appeared as white opaque colonies with a smooth surface. Isolates of *Psychrobacter spp.* were oxidase and catalase weak positive and appeared as gram-negative, non-motile coccobacilli when stained by Gram stain. The phenotypic and biochemical identification of isolated bacteria is demonstrated in Table (1).

Molecular characterization of isolated bacteria

The isolates gave a specific band at 1500 bp. The sequence of the 16S rRNA gene from this study includes *S. agalactiae* with accession numbers on Gene Bank PP837574.1, PQ860928.1, and PQ860931.1; *Psychrobacter spp.* with accession numbers on Gene Bank PP837575.1, PP837576.1, and PQ860929.1; *E. faecalis* with accession number on Gene Bank PQ860927.1; and *E. faecium* with accession number on Gene Bank PQ860930.1. The results of molecular identification are briefly summarized in Table (2) and Fig. (1).

Prevalence of the isolated bacteria

The overall prevalence of fish showing most severe gross abnormalities was 6.2% (19/305) in the examined *O. niloticus* and 3.6% (11/305) *in M. cephalus*. From these clinically affected fish, bacterial isolates were recovered. From the nineteen *O. niloticus*, *S. agalactiae* was isolated from eight fish (42.1%), *Psychrobacter spp.* was isolated from six fish (31.6%), *E. faecalis* was isolated from three fish (15.8%), and *E. faecium* was isolated from two fish (10.5%). Regarding *M. cephalus*, out of the eleven fish, *S. agalactiae* was isolated from five fish (45.4%), *Psychrobacter spp*. was isolated from three fish (27.3%), *E. faecalis* was isolated from two fish (18.2%), and *E. faecium* was isolated from only one fish (9.1%). The co-infecting bacterial species were not consistently present among all affected fish; for example, two bacterial species were isolated from certain fish, while others contained three distinct pathogens, highlighting the variability of polymicrobial infections within the sampled fish.

Pathological findings

Gross pathology of affected fish

Gross examination of the collected *O. niloticus* revealed numerous pathological alterations, including hemorrhage of the operculum near the mouth, loss of scales near the eye, and erosion of skin at the abdominal side (Fig. 2A). Moreover, ulceration of the dorsal caudal part of the body and near the caudal fin, a very protruded vent with darkness (Fig. 2B), deformity of the lateral line (Fig. 2C) and congestion all over the body were also observed (Fig. 2D). Furthermore, the necropsy findings included hepatomegaly, which appeared congested with focal necrosis at the caudal part (Fig. 2E) and pale at different parts, with a friable appearance of hepatic tissue (Fig. 2F) and greenish areas at the head of the liver were also seen. Furthermore, turbidity in the air sacs was clearly observed (Fig. 2G). In addition, enlargement and congestion of the gall bladder (Fig. 2H) and the intestinal blood vessels and gills were also observed (Fig. 2I).

On the other hand, the gross examination of *M. cephalus* showed corneal opacity of the right eye (Fig. 3A), hemorrhage of the operculum (Fig. 3B), congestion of the abdominal part (Fig. 3C), and redness of the upper and lower jaws (Fig. 3D). Moreover, the postmortem examination of *M. cephalus* revealed adhesion of the internal organs (Fig. 3E), a friable and marbled liver, and severely

congested gills and intestine, in addition to an enlarged spleen (Fig. 3F). All alterations observed in *O. niloticus* and *M. cephalus* are consistent with an acute form of bacterial septicaemia.

Histopathological findings

Hepatopancreas

Microscopical examination of *O. niloticus* hepatic tissue sections stained with H&E revealed congestion of hepatic blood vessels, vacuolar degeneration of hepatocytes, and eosinophilic granular cell infiltration (Fig. 4A). In addition, thrombosis of hepatic veins (Fig. 4B) and congested hepatic sinusoids were also seen (Fig. 4C). Moreover, inflammatory cell infiltration was also noticed (Fig. 4D). Furthermore, the disorganization of hepatic tissue architecture resulted from hydropic degeneration of hepatocytes, which exerted pressure on the surrounding hepatocytes and led to their atrophy (Fig. 4E). Hepatocytes also showed pyknosis and necrosis (Fig. 4F). Histological examination of the hepatopancreas revealed congestion of blood vessels, pyknosis of some hepatopancreatic cells, perivascular aggregation of melanophores, and activation of sinusoidal Von Kupffer cells (Fig. 4G&H).

The recorded microscopical alterations in the *M. cephalus* hepatic tissue stained with H&E were subcapsular hemorrhage (Fig. 4I), intrahepatic hemorrhage, focal aggregation of inflammatory cells and vacuolar degeneration of hepatocytes (Fig. 4J), and severe congestion of hepatic blood vessels (Fig. 4K). In addition, aggregation of melanophores and eosinophilic granular cell infiltration was also detected (Fig. 4L).

Gills

Histopathological findings of gills obtained from *O. niloticus* exhibited congestion of blood vessels (Fig. 5A) and focal and nodular hyperplasia of epithelial cells of the secondary gill lamellae (Fig. 5B&C). Additionally, we observed the fusion of gill lamellae, hyperplasia of mucous cells (Fig. 5D), and hemorrhage (Fig. 5E). Furthermore, hyperplasia of the gill raker (Fig. 5F) and mucous cells (Fig. 5G) was clearly seen after staining with alcian blue stain (Fig. 5H).

Regarding the gills of *M. cephalus*, they showed severe congestion of blood vessels (Fig. 5I), edema (Fig. 5J), and mucous cell hyperplasia of the gill racker (Fig. 5K) and gill lamellae that were obviously seen when stained with alcian blue stain (Fig. 5L).

Spleen

Splenic sections of *O. niloticus* stained with H&E stain revealed subcapsular necrosis, hemorrhage, and severe depletion of lymphoid tissue (Fig. 6A). Moreover, congestion and hemorrhage of the lymphoid tissue were observed all over the splenic tissue with the appearance of melanomacrophage centers devoid of melanin (Fig. 6B&C). Furthermore, there is an increase in the number of intravascular leucocytes (Fig. 6D), edema, perivascular proliferation of melanomacrophage centers, eosinophilic granular cell infiltration, thickening of blood vessel walls with endothelial swelling of splenic blood vessels (Fig. 6E, F, &G), and splenic ellipsoids (Fig. 6H).

On the other hand, splenic tissue of *M. cephalus* showed inflammatory cell infiltration, mainly macrophages and epithelioid cells with necrobiotic changes in some inflammatory cells (Fig. 6I), congestion, lymphoid depletion, and hemorrhage (Fig. 6J&K). Moreover, hemosiderin pigment appeared as deep bluish granules with Prussian blue stain (Fig. 6L).

Renal tissue

Microscopical examination of *O. niloticus* renal tissue exhibited few histopathologic alterations represented by interstitial hemorrhage (Fig. 7A&B). Similarly, the renal tissue of *M. cephalus* revealed congestion of renal blood vessels and pyknosis of nuclei of renal tubules lining epithelium (Fig. 7C&D).

Intestine

Intestinal tissue of *O. niloticus* showed catarrhal enteritis (Fig. 8A&B), desquamation of the lining epithelium, submucosal edema, eosinophilic granular cell infiltration (Fig. 8C), congestion of blood vessels, and hyperplasia of mucous-secreting cells (Fig. 8D, E, &F). When stained with alcian blue, the intestinal villi showed hyperplasia of mucous-secreting cells (Fig. 8G&H). No detectable histological alterations were noticed in the examined intestines of *M. cephalus*.

Lesion scoring

The statistical analysis for the frequencies of the recorded histopathological alterations is represented in Table (3).

Discussion

Numerous factors determine the fish species-specific immunological responses, including, but not limited to, genetics, environment, diet, age, anatomy, and pathogens. Deepening our knowledge regarding these factors will improve productivity, disease resistance, prevention, and control strategies, along with a more profound understanding of the pathophysiology in aquaculture. For these reasons, and despite the limitations of such studies, this study was a survey that seeks to identify the predominant bacterial pathogens impacting *O. niloticus* and *M. cephalus* in certain fish farms in Egypt, while also comparing the severity of pathological alterations linked to each bacterial infection, thus offering

critical insights into species-specific differences in disease susceptibility. We perform this study in the field that mimics the natural infection of fish. In the future, we may conduct further investigations using an experimental infection design.

The current investigation identified eight bacterial isolates co-infecting the two examined species. In the future, we may examine each pathogen individually. The present study demonstrated varying prevalence rates of bacterial pathogens in these fish species. The prevalence rates of *S. agalactiae, Psychrobacter spp*., *E. faecalis, and E. faecium* in *O. niloticus* were 42.1%, 31.6%, 15.8%, and 10.5%, respectively. Meanwhile, the prevalence rates of these bacterial pathogens in *M. cephalus* were 45.4%, 27.3%, 18.2%, and 9.1% for *S. agalactiae, Psychrobacter spp*., *E. faecalis, and E. faecium*, respectively. These results suggest that *S. agalactiae* may serve as one of the predominant pathogenic agents responsible for bacterial diseases in *O. niloticus* and *M. cephalus*, followed *by Psychrobacter spp*., *E. faecalis, and E. faecium* in terms of their relative epidemiological importance. Our findings of *S. agalactiae* in *O. niloticus* were consistent with what was recorded by [39], who found a 44.4% prevalence rate of *S. agalactiae.* In China, *S. agalactiae* has been responsible for over 90% of *O. niloticus* infections since 2009 [40]. Earlier studies reported a lower prevalence of 7% of *S. agalactiae* in *O. niloticus* compared to our findings [41]. For instance, a study conducted in Kafr El-Sheikh, Egypt, detected *S. agalactiae* in *O. niloticus* during summer months with a prevalence of only 13% [42]. Several environmental and husbandry factors can influence the variation in *S. agalactiae* (GBS) prevalence [43]. *O. niloticus* reared under suboptimal conditions are particularly vulnerable to bacterial infections, especially *S. agalactiae*. Key contributing factors include elevated water temperatures, high stocking densities, and poor water quality, often characterized by excessive ammonia levels and reduced dissolved oxygen [44]. The presence of *S. agalactiae* in other fish species raises concerns about its possible occurrence in *M. cephalus* populations, especially in aquaculture settings where disease transmission can occur. Contrary to current knowledge, studies reporting the exact prevalence rate of *S. agalactiae* in *M. cephalus* are scarce. This gap in literature indicates that there should be further investigation into *S. agalactiae* infections in *M. cephalus*. Our results of *Psychrobacter spp*. prevalence rates in *O. niloticus* and *M. cephalus* were higher than those of *P. glacincola* (16.7%, 10%, and 13.3%), which were found in *Lutjanus ehrenbergii, Rhabdosargus haffara, and Cheilinus lunulatus* fish, respectively [27]. To the best of our knowledge, no existing literature was found documenting the prevalence rates of *Psychrobacter spp.* in either *O. niloticus* or *M. cephalus*. These findings of *E. faecalis* were in agreement with that of [45], who found that the prevalence rate in *O. niloticus* (59%) was higher than that recorded in *M. cephalus* (42%) in the same study. Another study was nearly in agreement with [46], who recorded a prevalence rate of *E. faecalis* of 21.25%, and with that of [47], who found a 23.76% prevalence rate of *E. faecalis* in *O. niloticus*. On the contrary, [5] stated 81.82% and 71.11% as prevalence rates of *E. faecalis* in El Fayoum and El Sharkia governorates, respectively. Our findings of *E. faecium* in *O. niloticus* were inconsistent with [48], who found an 11.4% prevalence rate of *E. faecium* in *O. niloticus*, and with [49], who recorded a 15% prevalence rate of *E. faecium* in *O. niloticus.* These variations might be caused by a variety of agricultural activities, including the addition of chicken dung to the sample, wastewater that contains human and animal waste, eating chicken intestines, and using farm water as a primary source of water [50]. Furthermore, the number of isolates examined, environmental variables, and sampling time and period cannot be disregarded as determinants of study variance [51]. However, the aim of this study was to discuss the variation in bacterial infection between the two studied species.

The bacteriological results of our study revealed cultures of pinpoint to minute white circular colonies, small brownish circular colonies, and small white translucent colonies representing *S. agalactiae, E. faecium, and E. faecalis*, respectively, that were oxidase and catalase negative and were gram-positive, non-motile cocci. Furthermore, *Psychrobacter spp.* appeared as white opaque colonies with smooth surfaces, oxidase and catalase weak positive, and gram-negative, non-motile coccobacilli. These results agreed with those reported by [5], and [52], who found pinpoint to minute circular white colonies when *S. agalactiae* was cultured on TSA. They also observed that the pathogen was both gram-positive and oxidase- and catalase-negative. The results of *Psychrobacter spp.* were consistent with [53], who stated that all presumptive *Psychrobacter spp.* colonies cultured on Tryptic Soy Agar supplemented with 1.5% NaCl (TSAS) exhibited creamy-white to opaque morphology. Gram staining revealed gram-negative, non-motile coccobacilli. Biochemical characterization confirmed that all isolated *Psychrobacter* strains were positive for both oxidase and catalase activity. The results of *E. faecalis* were in agreement with [54], who found that *E. faecalis* appeared as pinpoint translucent to large white colonies on TSA and also as gram-positive cocci with spherical to ovoid morphology, typically arranged in pairs or forming short to long chains. Biochemical testing demonstrated that all isolates were negative for both oxidase and catalase activity. The findings of *E. faecium* were consistent with that of [21], who stated that the bacterial isolates appeared as non-motile, gram-positive cocci,

predominantly arranged in pairs with occasional formation of short chains. Biochemical analysis confirmed their oxidase- and catalase-negative phenotypes.

From a pathological perspective, our gross examination declared that *O. niloticus* showed numerous pathological alterations, including hemorrhage at the operculum near the mouth, loss of scales near the eyes and erosion of the skin at the abdominal side, ulceration in the dorsal caudal part of the body and near the caudal fin, a very protruding vent with darkness, deformity of the lateral line, and congestion all over the body. On the other hand, *M. cephalus* showed corneal opacity of the right eye, hemorrhage at the operculum, congestion of the abdominal part, and redness of the upper and lower jaws. Furthermore, the necropsy findings of *O. niloticus* included an elongated and enlarged liver, which appeared congested with focal necrosis at the caudal part and pale at its different parts. Moreover, the hepatic tissue appeared friable with greenish areas at the head of the liver. The examination also revealed turbidity in the air sacs, congestion and enlargement of the gall bladder, congested intestinal blood vessels, and congested gills. In addition, the postmortem examination of *M. cephalus* revealed adhesion of the internal organs, a friable and marbled liver, severely congested gills and intestine, and an enlarged spleen.

Our findings of *O. niloticus* gross examination were in agreement with [48], who reported loss of scales, ulceration, skin discoloration, and hemorrhages on all the body surfaces, in addition to skeletal deformity in fish infected with *S. agalactiae, E. faecium*, and *E. faecalis.* Our results were also consistent with those observed by [52], who recorded hemorrhages on different body parts and ulceration on the body surface of the *O. niloticus* infected by *S. agalactiae*, and with [19], and [55], who noted hemorrhages at the base of the fins and opercula, skin darkening, and body curvature. [54] also found hemorrhages and skin darkness in the *O. niloticus* infected by *S. agalactiae* and *E. faecalis*. In addition, the results were inconsistent with [5], who stated that the *O. niloticus* infected with *E. faecalis* showed signs of septicemia, which included hemorrhages, congested body fins, scale loss, and ulceration of the dorsal body. On the other hand, the findings of *M. cephalus* gross examination were inconsistent with [56], and [45], who found petechial hemorrhages scattered on different parts of the body and darkness of the skin, and with [20], who reported body curvature and hemorrhage at different parts of the body, including the operculum, caused by *S. agalactiae*. Furthermore, the gross examinations agreed with [27], who found ulcers, hemorrhages on the body and on the operculum, loss of scales, and congested fins of some Red Sea fishes infected with *P. glacincola*. Another study indicated that when *O. niloticus and M. cephalus*, which had a mixed infection with *Yersinia ruckeri* and *Psychrobacter spp*., were clinically examined, the results showed dark skin, hemorrhages in various body parts, such as the tail, fin rot, and base of fins, abdominal distension, ulcers, and sloughing of fish scales [53]. Our study postmortem examination of *O. niloticus* was nearly similar to [16], [57], [55], [42], [5], [58], and [52], who found enlarged pale or hemorrhagic liver, enlarged and congested spleen, and engorged enlarged gallbladder in the infected fish with *S. agalactiae*, and to [48], [59], and [54], who recorded hepatomegaly, splenomegaly, and congestion of all the internal organs due to *S. agalactiae, E. faecalis*, and *E. faecium* infection. On the other hand, our gross examination of *M. cephalus* was inconsistent with [45], who found enlargement and congestion in gills, liver, and spleen in the infected fish with *E. faecalis*. Furthermore, the study recorded by [4], showed hemorrhages and congested liver in fish infected with *S. agalactiae* and *E. faecalis*. Moreover, gross examination of some Red Sea fishes infected with *P. glacincola* revealed congested liver, spleen, and kidneys [27].

Our histopathological findings regarding the hepatic tissue of *O. niloticus* included congestion of hepatic blood vessels, vacuolar degeneration, pyknosis and necrosis of hepatocytes, eosinophilic granular cell infiltration, thrombosis of hepatic veins, congested hepatic sinusoids, inflammatory cell infiltration, and disorganization of the hepatic tissue architecture. This disorganization resulted from hydropic degeneration of hepatocytes that exerted pressure on the surrounding hepatocytes, leading to their atrophy. In addition, histological examination of the hepatopancreas revealed congestion of blood vessels, pyknosis of some hepatopancreatic cells, perivascular aggregation of melanophores, and activation of sinusoidal Von Kupffer cells. On the other hand, our recorded microscopical alterations of the *M. cephalus* hepatic tissue were subcapsular hemorrhage, intrahepatic hemorrhage, focal aggregation of inflammatory cells, vacuolar degeneration of hepatocytes, severe congestion of hepatic blood vessels, aggregation of melanophores, and eosinophilic granular cell infiltration. Our findings were supported by the findings of [5], which included vacuolar degeneration of hepatocytes, melanin deposition predominantly within the hepatopancreas, and congestion of hepatoportal vasculature within the *O. niloticus* infected with *S. agalactiae* and *E. faecalis*, and with the observations of [60], [42], and [58], who found that fish infected by *S. agalactiae* had shown thrombosis of portal blood vessels, congested blood vessels, vacuolar degenerated hepatocytes, and inflammatory cell infiltration. In addition, [58], reported necrobiotic change in the hepatocytes of the hepatopancreas, vacuolar degenerated hepatocytes, hemorrhage in the hepatic tissue, and inflammatory cell infiltration in

the fish infected with *S. agalactiae*. Moreover, [61], [22], and [54] recorded vacuolar degeneration of hepatocytes and necrosis of hepatic tissue in fish infected with *S. agalactiae* and *E. faecalis*. Furthermore, [25], and [21], found degenerated hepatocytes and focal necrosis in the hepatic tissue of fish infected with *E. faecium.* Moreover, our histopathological findings regarding the gills of *O. niloticus* revealed congestion of blood vessels, hemorrhage, focal and nodular hyperplasia of the epithelial cells in the secondary gill lamellae, hyperplasia of mucous cells, and fusion of gill lamellae with the gill raker. Regarding the gills of *M. cephalus*, they showed severe congestion of blood vessels, edema, and mucous cell hyperplasia of gill rakers. These findings were inconsistent with what was reported by [52], who found gill filament hyperemia and hyperplasia in *O. niloticus* infected with *S. agalactiae*. Furthermore, [62], and [45], recorded congested lamellar blood vessels, hyperplasia of secondary lamellae, and edema of fish infected with *E. faecalis.* Moreover, [54], reported fusion, hyperplasia of gill lamellae, and congested lamellar blood vessels in fish infected with *S. agalactiae* and *E. faecalis*.

In addition to that, splenic alterations of *O. niloticus* were subcapsular necrosis, hemorrhage, severe depletion of lymphoid tissue, congestion, hemorrhage, appearance of melanomacrophage centers devoid of melanin, increase in number of intravascular leucocytes, edema, perivascular proliferation of melanomacrophage centers, eosinophilic granular cell infiltration, and thickening of blood vessel walls with endothelial swelling of splenic blood vessels and splenic ellipsoids. On the other hand, the splenic tissue of *M. cephalus* showed inflammatory cell infiltration, mainly macrophages and epithelioid cells, with necrobiotic changes in some inflammatory cells, congestion, lymphoid depletion, hemorrhage, and hemosiderin pigment deposition. Our findings were in agreement with those reported by [5], which included hemorrhage surrounding melanomacrophage centers, along with melanophore aggregation localized around blood vessels, and hemosiderin pigment deposition throughout the splenic parenchyma, accompanied by noticeable lymphocytic depletion in the white pulp regions in the *O. niloticus* infected with *E. faecalis*. Furthermore, [54], and [45], found lymphoid depletion and melanomacrophage center activation and congestion of blood vessels in fish infected with *E. faecalis*. Furthermore, [60], [42], [58], and [52], found lymphoid depletion and melanomacrophage center activation and congestion of blood vessels in fish infected with *E. faecalis*. Furthermore, [48], who recorded activation of melanomacrophage centers, lymphocytic depletion, and dilatation of splenic blood vessels in fish infected with *E. faecium.*

Regarding the microscopical examination of the renal tissue, our findings revealed a few histopathologic alterations represented by interstitial hemorrhage in *O. niloticus*. Similarly, the renal tissue of *M. cephalus* revealed congestion of renal blood vessels and pyknosis of nuclei of renal tubules lining epithelium. These findings were in agreement with [5], who found pyknosis of renal tubular epithelium and interstitial hemorrhage in *O. niloticus* infected with *E. faecalis*. Furthermore, [54] recorded hemorrhage in the renal tissue of fish infected with *S. agalactiae* and *E. faecalis*. Moreover, [55], and [42] recorded interstitial hemorrhage in the renal tissue of red tilapia infected with *S. agalactiae*. Similar results were obtained by [58], and [48], who reported pyknosis of renal tubular epithelium in *O. niloticus* infected with *S. agalactiae*.On the other hand, our findings of the intestinal tissue of *O. niloticus* were catarrhal enteritis, desquamation of the lining epithelium, submucosal edema, eosinophilic granular cell infiltration, congestion of blood vessels, and hyperplasia of mucous-secreting cells. These findings were nearly similar to the results obtained by [48], who reported chronic enteritis in *O. niloticus* infected with *S. agalactiae*, and with the findings of [59], who recorded excessive accumulation of mucous in the intestine of *O. niloticus* infected with *S. agalactiae*.

On the other hand, our findings of the intestinal tissue of *O. niloticus* were catarrhal enteritis, desquamation of the lining epithelium, submucosal edema, eosinophilic granular cell infiltration, congestion of blood vessels, and hyperplasia of mucous-secreting cells. These findings were nearly similar to the results obtained by [52], who reported chronic enteritis in *O. niloticus* infected with *S. agalactiae*, and with the findings of [62], who recorded excessive accumulation of mucous in the intestine of *O. niloticus* infected with *S. agalactiae*.

Virulence factors of *S. agalactiae*, such as capsular polysaccharides (CPSs), which are frequently isolated from a diseased *O. niloticus*, also play a vital role in immune evasion by resisting phagocytosis, which is more efficient in *O. niloticus* than in other species [63, 64]. *E. faecalis* produced virulence factors such as lipoteichoic acid [65], and [66], cytolysin and hemolysin, and surface proteins like the M proteins [24, 61, 67-69]. Cytolysin is a critical virulence factor for *E. faecalis* that induces haemolysis and toxicity in macrophages and leukocytes [70]. Gelatinase facilitates the hydrolysis of gelatin, elastin, collagen, and hemoglobin [71]. while hyaluronidase degrades the mucopolysaccharides in connective tissue [72]. These factors might interact more effectively with receptors or tissues in *O. niloticus* than in *M. cephalus*. Specific virulence factors expressed by the isolated microorganisms may be responsible for the characteristic pathological lesions observed in our

study. The varying severity of clinical manifestations between host species likely stemmed from these pathogenic mechanisms. A significant component of our findings is the recurrent isolation of multiple possible pathogens from a single diseased fish. Co-infections may considerably affect the severity of the infection and host response [73]. These bacteria may also function synergistically, Synergistic effects may arise when the initial pathogen promotes immunosuppression in the host, impairing the immune response to subsequent infections, hence exacerbating infection severity and elevating mortality rates [74]. In our study a primary infection by a pathogen, such as *Enterococcus spp.*, may be induce initial tissue damage and immune suppression by their virulence factors, hence fostering an opportunistic environment for secondary invaders like *S. agalactiae,* and *Psychrobacter spp.* Future research into the dynamics of these polymicrobial infections is necessary to comprehensively understand their impact on disease outbreaks in aquaculture.

Conclusion

The current study revealed a notable difference in lesion severity between *O. niloticus* and *M. cephalus*, with *O. niloticus* exhibiting more pronounced pathological manifestations. This observed variation in disease presentation may be attributed to multiple host-specific and pathogen-related factors, including anatomical and physiological differences between species, variations in innate immune competence, distinct feeding behaviors and nutritional status, and potential differences in bacterial virulence expression across host species. Further studies are needed to explore the epidemiological and correlated pathological conditions of these fish species in different localities in Egypt. Enhancing the management of these bacterial diseases via targeted, species-specific strategies is essential for the economic sustainability of local farms and supports the overarching objectives of sustainable aquaculture and global food security, in accordance with UN Sustainable Development Goals, specifically SDG 2 (Zero Hunger) and SDG 14 (Life Below Water).

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Declaration of Conflict of Interest

The authors declare that they have no conflicts of interest to disclose.

Author Contributions

MTE and MAM: Developed the research concept. **MTE, MAM, and ZSOA:** Examined the histopathological samples. **MTE:** Sampled the fish and followed up on the practical aspect of the research. **JIA:** Performed conventional isolation and identification of the bacteria and provided the pure colonies for further molecular identification. All authors are drafting, revising, and approving the final manuscript.

TABLE 1. Phenotypic and biochemical identification of isolated bacteria

	Gram stain	Motility test	Oxidase	Catalase
S. agalactiae	+ve	Non motile	-ve	-ve
E. faecium	+ve	Non motile	-ve	-ve
E. faecalis	+ve	Non motile	-ve	-ve
Psychrobacter spp.	-ve	Non motile	Weak +ve	Weak +ve

TABLE 2. Demonstrating nucleotide identity between A) *S. agalactiae,* B) *Psychrobacter spp*., C) *E. faecalis, D) E. faecium*, and other GenBank nucleotides related to the same strains

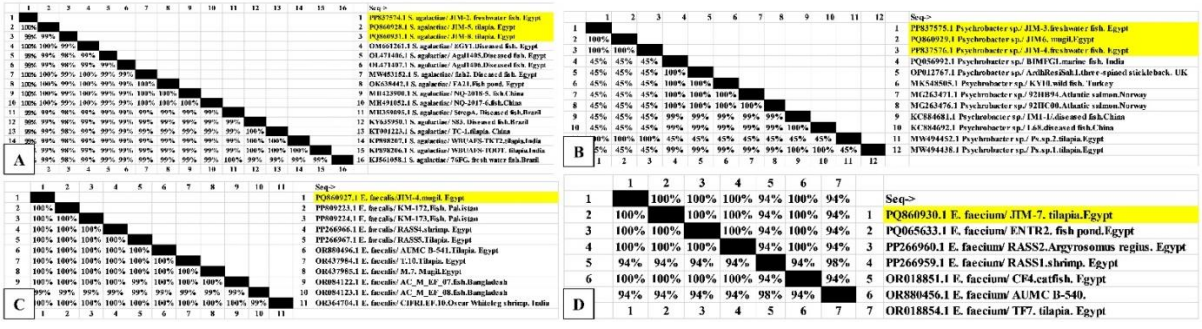

TABLE 3. Statistical analysis for the frequencies of the recorded histopathological alterations in *O. niloticus* and *M. cephalus.*

Organ	Lesion	*O. niloticus*	*M. cephalus*	*O. niloticus*	*M. cephalus*
	Congestion	+++	+	30.0 ± 9.13*	10 ± 4.08
	Hemorrhage	+	+	10 ± 0.00	5 ± 2.89
	Thrombus	+	-	2.5 ± 2.50*	0.0 ± 00
	Lymphocytic infiltration	++	+	15.0 ± 2.89*	5 ± 2.89
	eosinophilic granular cells	+	+	10 ± 0.00	10 ± 0.00
Liver	Hydropic degeneration	++	+	15.0 ± 2.89*	5 ± 2.89
	Vacuolar degeneration	++	+	25.0 ± 5.00*	10 ± 4.08
	Pyknosis	++	+	12.5 ± 2.50	10.0 ± 00
	Necrosis	+	-	7.5 ± 2.50*	0.0 ± 00
	Aggregation of melanophores	+	+	10 ± 4.08	7.5 ± 2.50
	Congestion	++	+	15.0 ± 2.89*	2.5 ± 2.5
	Hemorrhage	+	+	7.5 ± 2.50	7.5 ± 2.5
	Edema	+	-	7.5 ± 2.50*	0 ± 2.5
Spleen	necrosis	+	+	10.0 ± 0.00*	2.5 ± 0.0
	lymphoid depletion	++	+	15.0 ± 2.89*	2.5 ± 2.5
	Activation of MMC	+	-	10.0 ± 4.08*	0 ± 4.0
	Eosinophilic granular cells	+	-	2.5 ± 2.50*	0 ± 2.5
	Pyknosis	-	+	0 ± 0	2.5 ± 2.5
Kidney	Congestion	-	+	0 ± 0	5 ± 2.89
	Interstitial hemorrhage	+	+	2.5 ± 2.5	2.5 ± 2.5
	Hyperplasia	+	-	7.5 ± 2.50*	0 ± 0
	fusion of gill lamellae	+	-	2.5 ± 2.50*	0 ± 0
Gills	mucous cell hyperplasia	+	+	7.5 ± 2.5	5 ± 5
	Hemorrhage	+	-	7.5 ± 2.50*	0 ± 0
	Congestion	+	+	7.5 ± 2.5	12.5 ± 12.5
	Edema	+	+	2.5 ± 2.5	2.5 ± 2.5
	Catarrhal enteritis	+	-	5.0 ± 2.89*	0 ± 0
	Mucous cells hyperplasia	++	-	12.5 ± 2.50*	0 ± 0
Intestine	Congestion	+	+	2.5 ± 2.5	2.5 ± 2.5
	Edema	+	-	2.5 ± 2.50*	0 ± 0
	Eosinophilic granular cells	+	-	2.5 ± 2.50*	0 ± 0

Score system: - = absence of the lesion, + = 1–10% showing the lesion, ++ = 11–25%, +++ = 26–50%, ++++ = 51–75%, +++++ = over 75%

The scores presented represent the overall pathological alterations observed in the entire clinically affected fish from which bacterial isolates were recovered, and are not attributed to a single, specific pathogen.

The values are shown in the mean ± SE. An asterisk (*) indicates a statistically significant difference ($p < 0.05$) between the two fish species for that specific lesion.

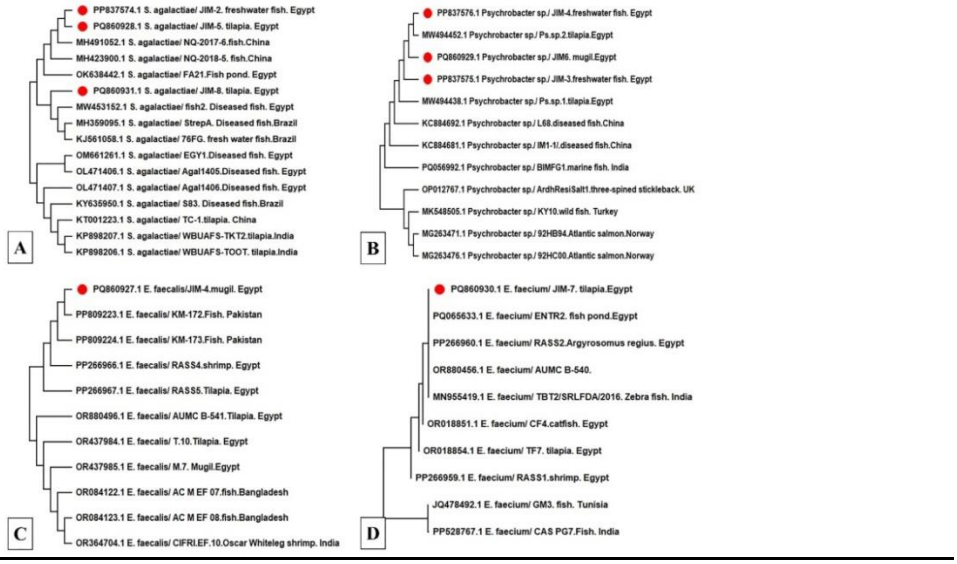

Fig. 1. Shows the phylogenetic tree of the 16s rRNA gene of A) *S. agalactiae,* B) *Psychrobacter spp*., C) *E. faecalis, and D) E. faecium*. Red circles represent isolates of the current study.

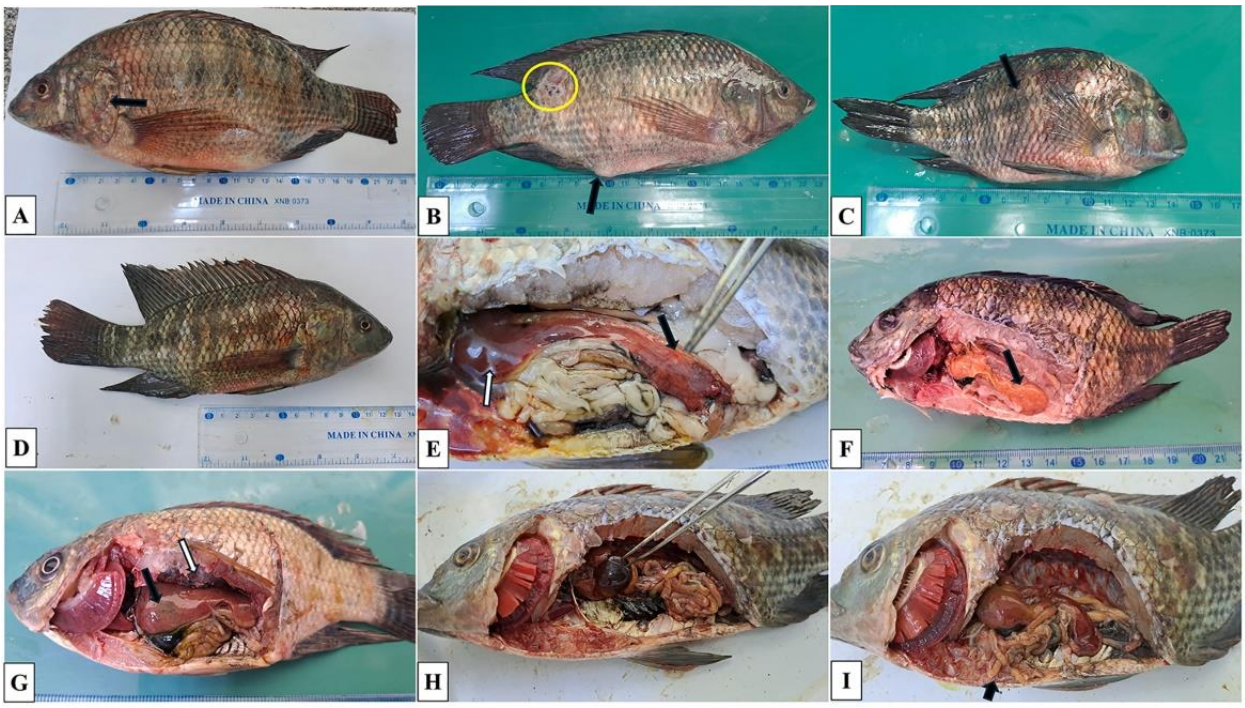

Fig. 2. Representative images of the necropsy findings of the examined *O. niloticus* showing A) Hemorrhage on the operculum (black arrow). B) Ulceration on the dorsal region of the fish (circle) and protruded vent (black arrow). C) Deformity of the lateral line (black arrow). D) Diffuse darkness and congestion in the body. E) Hepatomegaly with congestion (white arrow) and focal necrosis at the caudal part of the liver (black arrow). F) Friable and pale liver (black arrow). G) Inflamed air sacs (white arrow) and greenish discoloration of the liver (black arrow). H) Congested and enlarged gall bladder (forceps). I) Congested intestine (black arrow).

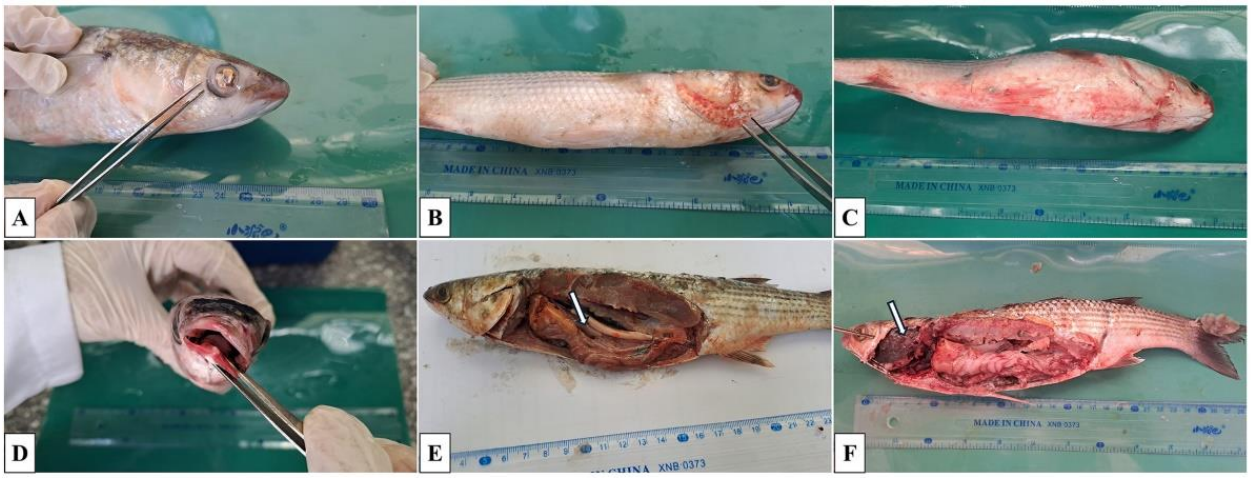

Fig. 3. Representative images of the necropsy findings of the examined *M. cephalus*. A) Corneal opacity of the right eye. B) Hemorrhage of operculum. C) Diffuse congestion in the abdominal part. D) Redness of upper and lower jaws. E) Adhesion of internal organs (white arrow). F) Congested gills (white arrow), friable marbled liver.

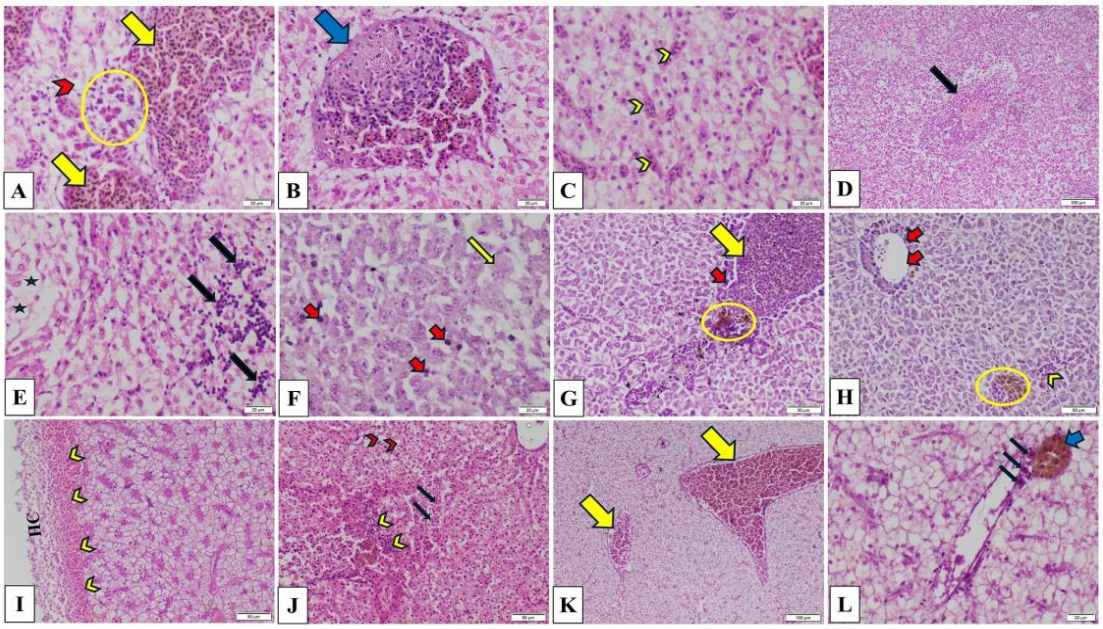

Fig. 4. Photomicrographs of hepatic tissue sections stained with H&E stain. A-H: (Hepatic tissue of *O. niloticus)* showing A) Congested blood vessels (yellow arrow), eosinophilic granular cell infiltration (circle), and vacuolar degeneration of hepatocytes (red arrowhead). B) Thrombus in the hepatic vein (blue arrow). C) Congestion of hepatic sinusoids (yellow arrowheads) D) Inflammatory cell infiltration (black arrow). E) Hydropic degeneration of hepatocytes (asterisk) and lymphocytic infiltration (black arrow). F) Pyknosis (red arrows) and necrosis (yellow arrow) of hepatocytes. G&H: Congested blood vessels (yellow arrow), perivascular aggregation of melanophores (circle), and pyknosis of some pancreatic cells (red arrow). I-L: (Hepatic tissue of *M. cephalus)* showing I) Hemorrhage (yellow arrowheads) under the hepatic capsule (HC). J) Focal aggregation of inflammatory cells (black arrow), vacuolar degeneration of hepatocytes (red arrowhead), and hemorrhage (yellow arrowhead). K) Severe congestion of hepatic blood vessels (yellow arrows). L) Aggregation of melanophores (blue arrow) and infiltration of eosinophilic granular cells (black arrow).

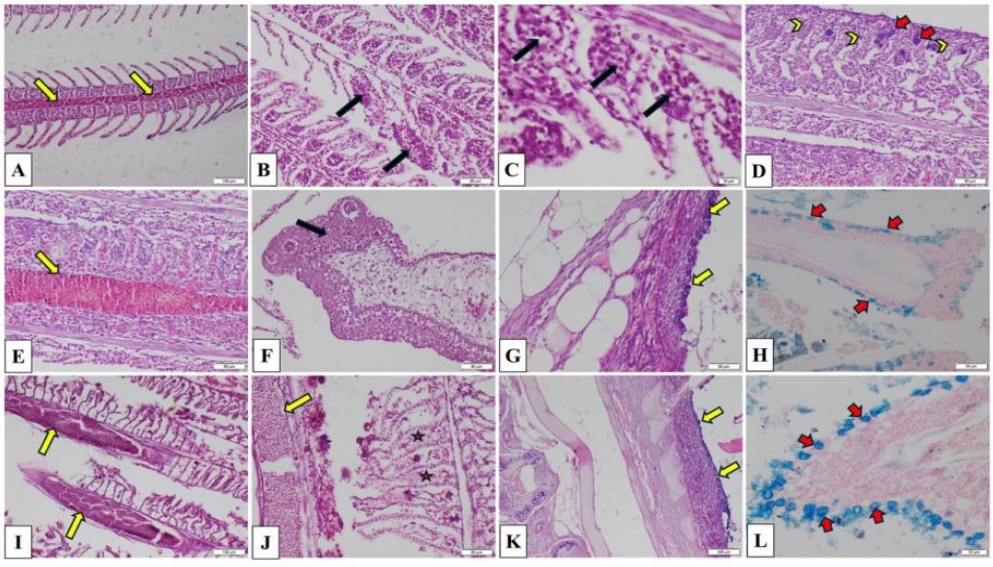

Fig. 5. Photomicrographs of gill sections of *O. niloticus*. A-G: (gill sections stained with H&E stain) showing A) congested blood vessels (yellow arrows). B) Focal hyperplasia of gill lamellae (black arrows). C) Nodular hyperplasia of gill epithelial cells (black arrows) D) Fusion of gill lamellae (yellow arrowheads) and mucous cell hyperplasia (red arrows). E) Hemorrhage (yellow arrow). F) Hyperplasia of gill rakers (black arrow). G) Mucous cell hyperplasia in gill rakers (yellow arrows). H) The gills section of *O. niloticus* stained with Alcian blue stain showing mucous cell hyperplasia of the gill raker (red arrows). I-K: (gill sections of *M. cephalus* stained with H&E stain) showing I) Congestion (yellow arrows). J) Congestion (yellow arrow) and lamellar edema (asterisk). K) Mucous cell hyperplasia of gill racker (yellow arrows). L) The gills section of *M. cephalus* stained with Alcian blue stain showing mucous cell hyperplasia of gill lamellae (red arrows).

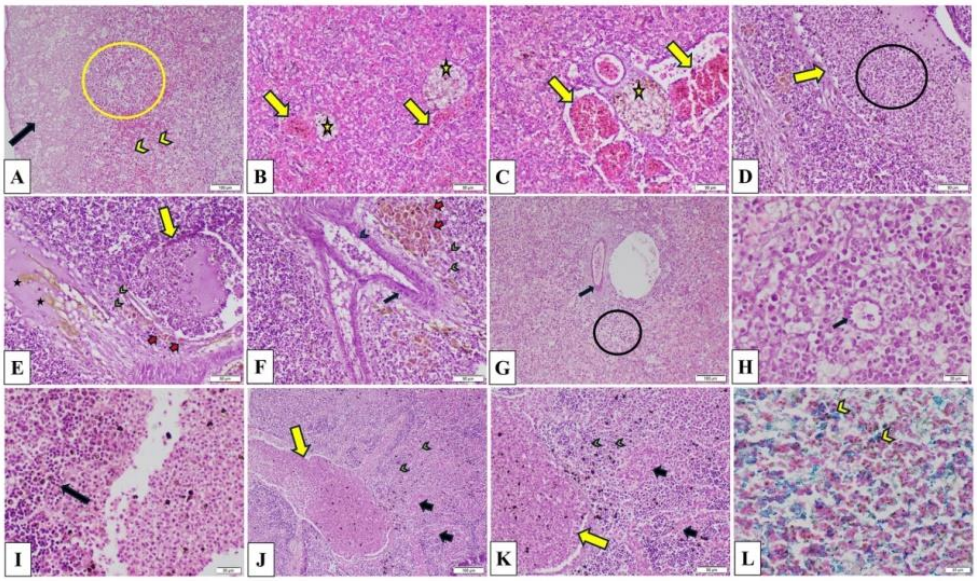

Fig. 6. **Photomicrographs of splenic tissue sections**. A-H: (Splenic tissue of *O. niloticus* stained with H&E stain) showing A) Subcapsular necrosis (black arrow), lymphoid depletion (circle), and hemorrhage (yellow arrowheads). B) Hemorrhage (yellow arrows) and melanomacrophage centers devoid of melanin (yellow asterisks). C) Congestion (yellow arrows) and melanomacrophage centers devoid of melanin (yellow asterisks). D) Congestion (yellow arrow) and increased intravascular leucocytes (black circle). E-G) Congested blood vessel (yellow arrow), edema (black asterisks), perivascular activation of melanomacrophage centers (red arrows), eosinophilic granular cells (yellow arrowheads), endothelial swelling and thickening of blood vessel wall (black arrow), and lymphoid depletion (black circle). H) swollen endothelium of splenic ellipsoid (black arrow). I-K: (Splenic tissue of *M. cephalus* stained with H&E stain) showing I) Inflammatory cells, mainly macrophages and epithelioid cells (black arrow), with necrobiotic changes in some of the inflammatory cells. J&K) Congested blood vessels (yellow arrows), hemorrhage (black arrows), and lymphoid depletion (yellow arrowheads). L) Splenic tissue of *M. cephalus* stained with Prussian blue stain showing hemosiderin pigment (arrowheads).

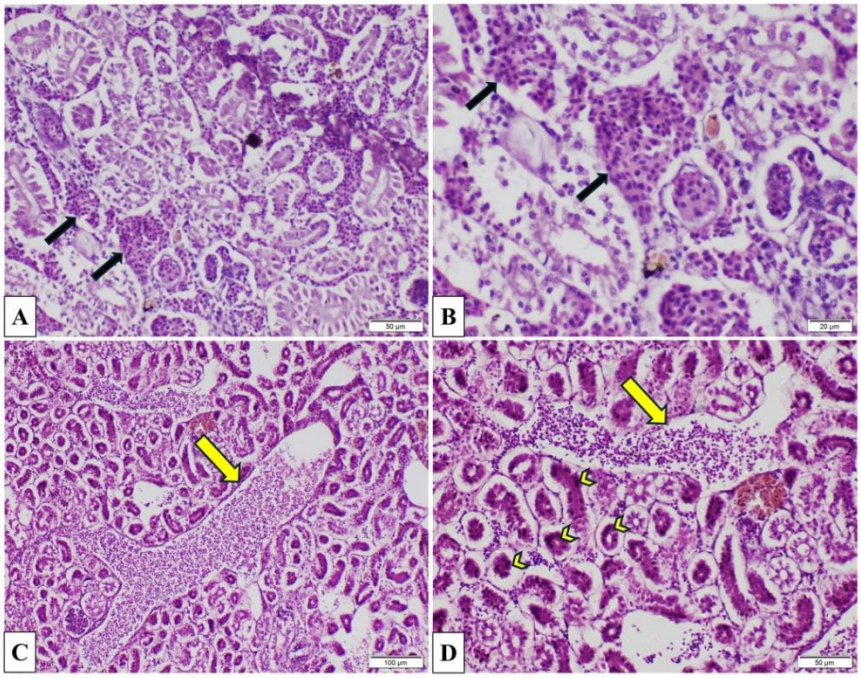

Fig. 7. Photomicrographs of renal tissue sections stained with H&E stain. A&B (renal tissue of *O. niloticus)* showing interstitial hemorrhage (black arrows). C&D (renal tissue of *M. cephalus)* showing congested renal blood vessels (yellow arrows) and pyknosis of nuclei of renal tubular epithelium (yellow arrowheads).

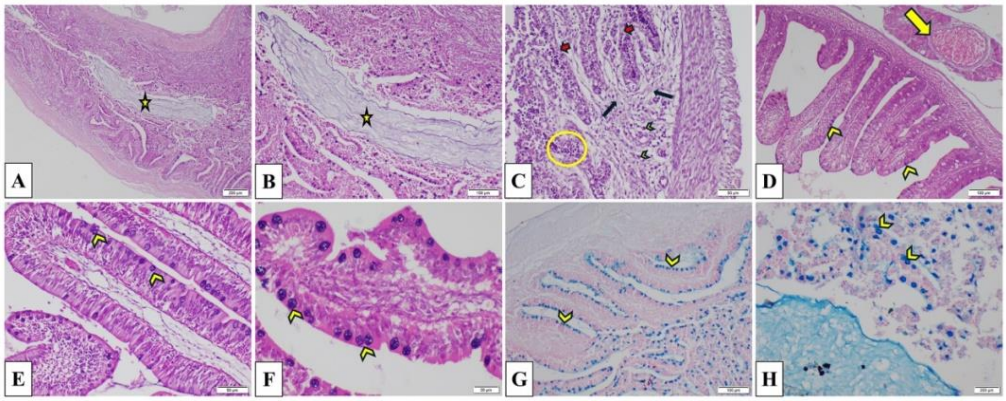

Fig. 8. Photomicrographs of intestinal tissue sections of *O. niloticus*. A-F: (Intestinal tissue stained with H&E stain) showing A&B) Catarrhal enteritis characterized by excess mucous (asterisks). C) Submucosal edema (black arrows), eosinophilic granular cells (yellow arrowheads), inflammatory cell infiltration (yellow circle), and desquamated epithelium (red arrows). D-F) Congested blood vessel (yellow arrow) and mucous-secreting cell hyperplasia (yellow arrowheads). G&H) Intestinal tissue stained with Alcian blue stain showing mucous-secreting cell hyperplasia (yellow arrowheads).

References

1. FAO, The State of World Fisheries and Aquaculture 2020. Sustainability in action, FAO, Rome (2020).
2. Araujo, G.S., Silva, T.F. and Costa, L.S., Fish farming techniques: Current situation and trends, *J. Mar. Sci. Eng.*, **10** (11), 1598 (2022). doi: 10.3390/jmse10111598.
3. LFRDPA, Fish statistics year book, LFRDPA, Cairo (2021).
4. Mahmoud, M.A., El-Mahdy, O.I. and Soliman, H., *Ergasilus extensus* and bacterial co-infection in flathead grey mullet, *Mugil cephalus* (Linnaeus, 1758), are associated with pathological changes and immunological gene expression alterations, *Aquacult. Res.*, **52** (12), 6143–6151 (2021). doi: 10.1111/are.15476.
5. Elgohary, I., El-Sayed, A.F. and El-Mahdy, O.I., Bacteriological, molecular, and pathological studies on the Gram-positive bacteria *Aerococcus viridans* and *Enterococcus faecalis* and their effects on *Oreochromis niloticus* in Egyptian fish farms, *Aquacult. Res.*, **52** (5), 2220–2232 (2021). doi: 10.1111/are.15074.
6. Minich, J.J., El-Gohary, A.E. and Mahmoud, M.A., Microbial effects of livestock manure fertilization on freshwater aquaculture ponds rearing tilapia (*Oreochromis shiranus*) and North African catfish (*Clarias gariepinus*), *MicrobiologyOpen*, **7** (6), e00716 (2018). doi: 10.1002/mbo3.716.
7. Mahmoud, M.A., El-Mahdy, O.I. and Soliman, H., Infectious bacterial pathogens, parasites and pathological correlations of sewage pollution as an important threat to farmed fishes in Egypt, *Environ. Pollut.*, **219**, 939–948 (2016). doi: 10.1016/j.envpol.2016.09.044.
8. Eltholth, M., Tran, T. and Rich, K.M., Characterisation of production, marketing and consumption patterns of farmed tilapia in the Nile Delta of Egypt, *Food Policy*, **51**, 131–143 (2015). doi: 10.1016/j.foodpol.2015.01.002.
9. Reverter, M., Sarter, B. and Caruso, D., Use of plant extracts in fish aquaculture as an alternative to chemotherapy: Current status and future perspectives, *Aquaculture*, **433**, 50–61 (2014). doi: 10.1016/j.aquaculture.2014.05.048.
10. Bulfon, C., Volpatti, D. and Galeotti, M., Current research on the use of plant-derived products in farmed fish, *Aquacult. Res.*, **46** (3), 513–551 (2015). doi: 10.1111/are.12238.
11. Pridgeon, J. and Klesius, P., Major bacterial diseases in aquaculture and their vaccine development, *CABI Rev.*, **7**, 1–16 (2012). doi: 10.1079/pavsnnr20127048.
12. Plumb, J.A. and Hanson, L.A., Health maintenance and principal microbial diseases of cultured fishes, Wiley-Blackwell, Ames, Iowa, USA, 3rd ed. (2010).
13. Abdelsalam, M., El-Mahdy, O.I. and Soliman, H., Coinfections of *Aeromonas spp.*, *Enterococcus faecalis*, and *Vibrio alginolyticus* isolated from farmed Nile tilapia and African catfish in Egypt, with an emphasis on poor water quality, *Microb. Pathog.*, **160**, 105213 (2021). doi: 10.1016/j.micpath.2021.105213.
14. Saleh, A.A., El-Sayed, A.F. and Zahran, E., Expression and Immune Response Profiles in Nile Tilapia (*Oreochromis niloticus*) and European Sea Bass (*Dicentrarchus labrax*) During Pathogen Challenge and Infection, *Int. J. Mol. Sci.*, **25** (23), 12829 (2024).
15. Leal C.A.G., Figueiredo H.C.P. and Oliveira S.D., *Streptococcus agalactiae* Sequence Type 283 in Farmed Fish, Brazil, *Emerg. Infect. Dis.*, **25** (4), 776–779 (2019). doi: 10.3201/eid2504.180543.
16. Yanong R.P. and Francis-Floyd R., *Streptococcal infections* of fish, *Univ. Fla. IFAS Ext.*, 1–5 (2002).

17. Iregui, C.A., Ochoa, S. and Sánchez, J.A., Experimental early pathogenesis of *Streptococcus agalactiae* infection in red tilapia *Oreochromis spp*., *J. Fish Dis.*, **39** (2), 205–215 (2016). doi: 10.1111/jfd.12347.

18. Younes, M.A., El-Mahdy, O.I. and Soliman, H., Genotyping and pathogenicity of *Streptococcus iniae* strains recovered from cultured *Oreochromis niloticus* at Kafr El-Shiekh Governorate, Egypt, Egypt. *J. Aquat. Biol. Fish.*, **23** (3), 467–474 (2019). doi: 10.21608/ejabf.2019.51630.

19. Ali Abuseliana, A.A., Al-Harbi, A.H. and Al-Ghanim, K.A., *Streptococcus agalactiae* the etiological agent of mass mortality in farmed red tilapia (*Oreochromis sp.*), *J. Anim. Vet. Adv.*, **9** (20), 2640–2646 (2010).

20. Evans, J.J., Klesius, P.H. and Shoemaker, C.A., Characterization of *β-haemolytic* Group B *Streptococcus agalactiae* in cultured seabream, *Sparus auratus L.*, and wild mullet, *Liza klunzingeri* (Day), in Kuwait, *J. Fish Dis.*, **25** (9), 505–513 (2002). doi: 10.1046/j.1365-2761.2002.00392.x.

21. El-Nobi, G., El-Mahdy, O.I. and Soliman, H., Isolation, identification, molecular, and histopathological investigations of two pathogenic *Enterococcus species* from tilapia in Egyptian farms, *Slov. Vet. Res.*, **58** (Suppl. 24), 33–43 (2021). doi: 10.26873/svr-1425-2021.

22. Zahran, E., El-Sayed, A.F. and El-Mahdy, O.I., Experimental pathogenesis and host immune responses of *Enterococcus faecalis* infection in Nile tilapia (*Oreochromis niloticus*), *Aquaculture*, **512,** 734319 (2019).

23. Lee, J.L., Chen, S.C. and Lin, C.F., Identification and Differences in Antimicrobial Susceptibility of *Lactococcus garvieae* from Farmed Grey Mullet (*Mugil cephalus*) and Non-Grey Mullet in Southern Taiwan, Preprints, 2020070265 (2020). doi: 10.20944/preprints202007.0265.v1.

24. Martins, M.L., Tavares-Dias, M. and Fujimoto, R.Y., Haematological changes in Nile tilapia experimentally infected with *Enterococcus sp.*, *Braz. J. Biol.*, **68** (3), 657–661 (2008). doi: 10.1590/S1519-69842008000300025.

25. El-Deen, A. and El-Nisr, N., Bacteriological and histopathological studies on Streptococcosis in Nile tilapia, *Oreochromis niloticus*, *Assiut Vet. Med. J.*, **57** (131), 109–129 (2011).

26. Bowman, J.P., The Genus *Psychrobacter*, in The Prokaryotes: *Proteobacteria*: Gamma Subclass, Dworkin M. et al. (Eds.), Springer, New York, NY, 920–930 (2006).

27. El-Sayed, M.R., Soliman, H. and El-Mahdy, O.I., Detection and description of a novel *Psychrobacter glacincola* infection in some Red Sea marine fishes in Hurghada, Egypt, *BMC Vet. Res.*, **19** (1), 23 (2023). doi: 10.1186/s12917-022-03542-8.

28. Thrusfield, M., Veterinary epidemiology, Wiley-Blackwell, 4th ed. (2018).

29. Noga, E.J., Fish disease: diagnosis and treatment, Wiley-Blackwell, Ames, Iowa, USA, 2nd ed. (2010).

30. Meyers, T.R., Standard necropsy procedures for finfish, in NWFHS Laboratory Procedures Manual, *US Fish Wildl. Serv.*, Washington, USA, 64–74 (2009).

31. Fadel, A., El-Mahdy, O.I. and Soliman, H., *Aeromonas veronii* infection in cultured *Oreochromis niloticus*: prevalence, molecular and histopathological characterization correlated to water physicochemical characteristics, with the protective autochthonous probiotic, *Aquacult. Int.*, **33** (4), 298 (2025). doi: 10.1007/s10499-025-01960-7.

32. Holt, J.G., Krieg, N.R. and Sneath, P.H.A., Bergey's Manual of determinative bacteriology, Williams & Wilkins, 9th ed. (1994).

33. Benga, L., Benten, W.P.M. and Liebert, U.G., 16S ribosomal DNA sequence-based identification of bacteria in laboratory rodents: a practical approach in laboratory animal bacteriology diagnostics, *Lab. Anim.*, **48** (4), 305–312 (2014). doi: 10.1177/0023677214538240.

34. Altschul, S.F., Gish, W. and Miller, W., Basic local alignment search tool, *J. Mol. Biol.*, **215** (3), 403–410 (1990). doi: 10.1016/S0022-2836(05)80360-2.

35. Kumar, S., Stecher, G. and Tamura, K., MEGA X: Molecular Evolutionary Genetics Analysis across Computing Platforms, *Mol. Biol. Evol.*, **35** (6), 1547–1549 (2018). doi: 10.1093/molbev/msy096.

36. Bancroft, J.D., Suvarna, S.K. and Layton, C., Theory and Practice of Histological Techniques, Elsevier, 8th ed. (2019).

37. Bernet, D., Schmidt, H. and Meier, W., Histopathology in fish: proposal for a protocol to assess aquatic pollution, *J. Fish Dis.*, **22** (1), 25–34 (1999). doi: 10.1046/j.1365-2761.1999.00134.x.

38. Field, A., Discovering statistics using IBM SPSS statistics, Sage, 5th ed. (2018).

39. Al-Harbi, A.H., Phenotypic and genotypic characterization of *Streptococcus agalactiae* isolated from hybrid tilapia (*Oreochromis niloticus×O. aureus*), *Aquaculture*, **464**, 515–520 (2016). doi: 10.1016/j.aquaculture.2016.07.036.

40. Chen, M., Wang, K.Y. and He, J.Y., Screening vaccine candidate strains against *Streptococcus agalactiae* of tilapia based on PFGE genotype, *Vaccine*, **30** (42), 6088–6092 (2012). doi: 10.1016/j.vaccine.2012.07.044.

41. Alazab, A., Sadat, A. and Younis, G., Prevalence, antimicrobial susceptibility, and genotyping of *Streptococcus agalactiae* in Tilapia fish (*Oreochromis niloticus*) in *Egypt, J. Adv. Vet. Anim. Res.*, **9** (1), 95–103 (2022). doi: 10.5455/javar.2022.i573.

42. Ghetas, H., El-Mahdy, O.I. and Soliman, H., *Streptococcus agalactiae* Isolation and Characterization in Nile Tilapia (*Oreochromis niloticus*) with Histopathological Studies, *J. Curr. Vet. Res.*, **3** (1), 70–79 (2021). doi: 10.21608/jcvr.2021.160254.

43. Amal, M.N.A., Siti-Zahrah, A.F. and Wee, W.Y., Water quality influences the presence of *Streptococcus agalactiae* in cage cultured red hybrid tilapia, *Oreochromis niloticus* × *Oreochromis mossambicus*, *Aquacult. Res.,* **46** (2), 313–323 (2015). doi: 10.1111/are.12180.

44. Adikesavalu, H., Raja, M. and Karunasagar, I., Meningoencephalitis in farmed monosex Nile tilapia (*Oreochromis niloticus L.*) caused by *Streptococcus agalactiae*, *Fish. Aquat. Life,* **25** (3), 187–200 (2017). doi: 10.1515/aopf-2017-0018.

45. Hassan, M.A., El-Mahdy, O.I. and Soliman, H., Isolation and identification of *Enterococcus faecalis* from cultured *Oreochromis niloticus* and *Mugil cephalus* with a special emphasis on a possible integrated control strategy, *Aquacult. Res.,* **53** (16), 5521–5535 (2022). doi: 10.1111/are.16034.

46. Osman, K.M., El-Mahdy, O.I. and Soliman, H., Characterization and susceptibility of *streptococci* and *enterococci* isolated from Nile tilapia (*Oreochromis niloticus*) showing septicaemia in aquaculture and wild sites in Egypt, *BMC Vet. Res.*, **13** (1), 357 (2017). doi: 10.1186/s12917-017-1289-8.

47. Khafagy, A., El-Mahdy, O.I. and Soliman, H., Isolation of *Enterococcus faecalis* from tilapia in Lake Temsah in Ismailia governorate, *Suez Canal Vet. Med. J.,* **14** (2), 45–54 (2009).

48. Mohamed, A., El-Mahdy, O.I. and Soliman, H., Prevalence of Streptococcosis-related Mortalities in Farmed Nile Tilapia (*Oreochromis niloticus*) at Different Life Stages, *Egypt. J. Vet. Sci.,* **54** (5), 949–964 (2023). doi: 10.21608/ejvs.2023.207062.1494.

49. Mohamed Hussein, M.A., El-Mahdy, O.I. and Soliman, H., Prevalence, Antimicrobial Susceptibility, and Virulence Gene Profile of *Enterococcus Species* isolated from Some Farmed Fish Retailed in Zagazig City, Egypt, *Zagazig Vet. J.*, **52** (1), 35–48 (2024). doi: 10.21608/zvjz.2024.262051.1229.

50. Ezzat, M., El-Mahdy, O.I. and Soliman, H., Prevalence and Resistance Pattern of *Enterococcus faecalis* in Cultured *Oreochromis niloticus* at Ismailia Governorate, Egypt, *Suez Canal Vet. Med. J.*, **29** (2), 269–279 (2024). doi: 10.21608/scvmj.2024.367479.

51. Enany, M., El-Mahdy, O.I. and Soliman, H., Occurrence of Multidrug-Resistant *Enterococci* in Fresh Water Fishes, *Suez Canal Vet. Med. J.,* **27** (1), 91–100 (2022). doi: 10.21608/scvmj.2022.249298.

52. Mohamed, R.S., Wassif, I. and Tahoon, E., Histopathological Lesions of Some Microbial Infections in Tilapia Fish at El Salam Canal, Egypt, *Damanhour J. Vet. Sci.,* **9** (1), 23–28 (2022). doi: 10.21608/djvs.2022.164851.1095.

53. El-Sharaby, S.M., El-Mahdy, O.I. and Soliman, H., Phenotypic and Molecular Characterization of *Yersinia ruckeri* and *Psychrobacter Species* Isolated from Farmed Fishes, *J. Curr. Vet. Res.*, **3** (2), 1–7 (2021). doi: 10.21608/jcvr.2021.199408.

54. Abu-Elala, N.M., Abd-Elsalam, R.M. and Younis, N.A., Streptococcosis, Lactococcosis and Enterococcosis are potential threats facing cultured Nile tilapia (*Oreochomis niloticus*) production, *Aquacult. Res.,* **51** (10), 4183–4195 (2020). doi: 10.1111/are.14760.

55. Abdullah, S., Siti-Zahrah, A.F. and Wee, W.Y., Clinicopathological features and immunohistochemical detection of antigens in acute experimental *Streptococcus agalactiae* infection in red tilapia (*Oreochromis spp.*), *Springer Plus*, **2** (1), 286 (2013). doi: 10.1186/2193-1801-2-286.

56. Chen, S., Liaw, L.L. and Chen, S.C., *Lactococcus garvieae*, a cause of disease in grey mullet, *Mugil cephalus L.*, in Taiwan, *J. Fish Dis.*, **25** (12), 727–732 (2002). doi: 10.1046/j.1365-2761.2002.00415.x.

57. Najiah Musa, N.M., Siti-Zahrah, A.F. and Wee, W.Y., Streptococcosis in red hybrid tilapia (*Oreochromis niloticus*) commercial farms in Malaysia, *Aquacult. Res.*, **40** (5), 630–632 (2009). doi: 10.1111/j.1365-2109.2008.02142.x.

58. Sherif, A., El-Mahdy, O.I. and Soliman, H., Occurrence of infectious *Streptococcus agalactiae* in the farmed Nile tilapia, *Egypt. J. Aquat. Biol. Fish.*, **26** (3), 403–432 (2022).

59. Ali, G.I., Abd El-Hady, H.A. and Abou Zeid, M.A., Rapid Detection of *Streptococci* in Cultured Tilapia Fish Using PCR and Chemical Analysis, *World Vet. J.*, **10** (3), 286–296 (2020). doi: 10.36380/scil.2020.wvj37.

60. Laith, A.A., Najiah, M. and Wee, W.Y., Molecular identification and histopathological study of natural *Streptococcus agalactiae* infection in hybrid tilapia (*Oreochromis niloticus*), *Vet. World*, **10** (1), 101–111 (2017). doi: 10.14202/vetworld.2017.101-111.

61. Fawzy, N.M., El-Mahdy, O.I. and Soliman, H., Streptococcosis in tilapia: Clinico-pathological picture of experimentally infected tilapia, *Life Sci. J.*, **11**, 1005–1012 (2014).

62. Aly, S.M., Taha, R. and Dessouki, A.A., Experimental pathological, haematological and biochemical studies on streptococcosis in Nile tilapia (*Oreochromis niloticus*), *Glob. J. Fish. Aquacult. Res.,* **5**, 42–57 (2012).

63. Zhang, D., Wang, K.Y. and He, J.Y., Capsular polysaccharide of *Streptococcus agalactiae* is an essential virulence factor for infection in Nile tilapia (*Oreochromis niloticus Linn.*), *J. Fish Dis.*, **42** (2), 293–302 (2019). doi: 10.1111/jfd.12935.

64. Abdallah, E.S.H., El-Mahdy, O.I. and Soliman, H., *Streptococcus agalactiae* Infection in Nile Tilapia (*Oreochromis niloticus*): A Review, *Biology*, **13** (11), 914 (2024).

65. Papasian, C.J., Deng, J.C. and Standiford, T.J., Anomalous Role of Tumor Necrosis Factor Alpha in Experimental *Enterococcal* Infection, *Infect. Immun.*, **70** (12), 6628–6637 (2002). doi: 10.1128/IAI.70.12.6628-6637.2002.

66. Park, O.-J., Han, S.H. and Lee, S.J., Lipoteichoic acid of *Enterococcus faecalis* induces the expression of chemokines via TLR2 and PAFR signaling

pathways, *J. Leukoc. Biol.*, **94** (6), 1275–1284 (2013). doi: 10.1189/jlb.1012522.

67. Rahman, M., Hossain, M.A. and Rahman, M.S., Molecular Identification of Multiple Antibiotic Resistant Fish Pathogenic *Enterococcus faecalis* and their Control by Medicinal Herbs, *Sci. Rep.,* **7** (1), 3747 (2017). doi: 10.1038/s41598-017-03673-1.
68. El-Refaey, A., Studies on major bacterial diseases affecting fish; Tilapia *Oreochromis niloticus*, Catfish, *Clarias gariepinus* and mullets in Port Said, Egypt with special references to its pathological alterations, *Res. J.*, **5** (2), 5–14 (2013).
69. Abou El-Geit, E., El-Mahdy, O.I. and Soliman, H., Microbial infections among some fishes and crustacean species during blooming phenomenon in Qaroun Lake-Egypt, *Life Sci. J.*, **10** (2), 1217–1224 (2013).
70. De Vuyst, L., Foulquié Moreno, M.R. and Revets, H., Screening for enterocins and detection of hemolysin and vancomycin resistance in *enterococci* of different origins, *Int. J. Food Microbiol.,* **84** (3), 299–318 (2003). doi: 10.1016/S0168-1605(02)00425-7.
71. Archimbaud, C., Forestier, C. and Gavini, F., In vitro adhesive properties and virulence factors of *Enterococcus faecalis* strains, *Res. Microbiol.,* **153** (2), 75–80 (2002). doi: 10.1016/S0923-2508(01)01291-8.
72. Vankerckhoven, V., Van Autgaerden, T. and Vael, C., Development of a Multiplex PCR for the Detection of asa1, gelE, cylA, esp, and hyl Genes in *Enterococci* and Survey for Virulence Determinants among European Hospital Isolates of *Enterococcus faecium*, *J. Clin. Microbiol.,* **42** (10), 4473–4479 (2004). doi: 10.1128/JCM.42.10.4473-4479.2004.
73. Kotob, M.H., Menanteau-Ledouble, S., Kumar, G., Abdelzaher, M. and El-Matbouli, M., The impact of co-infections on fish: a review, *Vet. Res.*, **47**(1), 98 (2016). doi: 10.1186/s13567-016-0383-4.
74. Abdel-Latif, H.M.R., Dawood, M.A.O., Menanteau-Ledouble, S. and El-Matbouli, M., The nature and consequences of co-infections in tilapia: A review, *J. Fish Dis.*, **43**(6), 651–664 (2020). doi: 10.1111/jfd.13164.

دراسات مقارنة بكتريولوجية، وجزيئية، وباثولوجية على بكتيريا المكورة العقدية اللالبنية، والبكتريا المحبة للبرودة، والمكورة معوية البراز والمكورة المعوية البرازية المسببة لعدوى مشتركة في أسماك البلطي النيلي والبوري المستزرعة

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الملخص

تُعتبر الأمراض المعدية، وخاصة الأمراض البكتيرية، إحدى المشكلات الرئيسية في أنظمة الاستزراع المائي، مما يؤدي إلى خسائر اقتصادية كبيرة ويعيق استدامة هذا القطاع. تُظهر أنواع الأسماك المختلفة درجات متفاوتة من القابلية للإصابة بمسببات الأمراض البكتيرية بسبب الاختلافات في الكفاءة المناعية، والحواجز التشريحية، والمقاومة الوراثية. وبناءً على ذلك، تهدف هذه الدراسة إلى تقييم شدة التغيرات المرضية التي تسببها العدوى البكتيرية في أسماك البلطي النيلي والبوري. تم أخذ عينات بأعداد متساوية (305) من كل نوع من أسماك البلطي النيلي والبوري من مزارع خاصة في مصر وفحصها للكشف عن أي آفات ظاهرية. من بين الأسماك التي تم فحصها، تم جمع 19 سمكة من البلطي النيلي و 11 سمكة من البوري، وهي التي أظهرت أشد التغيرات العيانية. تم إجراء فحص ما بعد الموت (الصفة التشريحية)، وفُحصت الأعضاء الداخلية لإجراء التحاليل البكتريولوجية والنسيجية المرضية (الهيستوباثولوجية). بلغ معدل انتشار بكتيريا المُكَوِّرَة العِقْدِيَّة اللاَّلَبَنِيَّة، والبكتيريا المحبة للبرودة، والمكورة معوية البراز والمُكَوِّرَة المِعَوِيَّة البَرازِيَّة نسبة 42.1%، 31.6%، 15.8%، و 10.5% على التوالي في أسماك البلطي النيلي، بينما كانت النسبة في أسماك البوري 45.4%، 27.3%، 18.2%، و 9.1% على التوالي لنفس العزلات. أظهرت أسماك البلطي النيلي والبوري التي تم فحصها العديد من التغيرات المرضية، وقد اختلف النوعان بشكل كبير في شدة هذه الآفات. حيث أظهرت أسماك البلطي النيلي آفات أكثر وضوحًا بعد الموت وتغيرات نسيجية مرضية أكبر. يمكن أن تُعزى هذه الاختلافات إلى عوامل خاصة بالعائل وأخرى مرتبطة بمسبب المرض، مثل الاختلافات التشريحية والفسيولوجية، والكفاءة المناعية الفطرية، وتباين التعبير عن ضراوة البكتيريا في العوائل المختلفة، وهو ما يحتاج إلى مزيد من البحث والدراسة.

الكلمات الدالة: البلطي النيلي، البوري، المكورة العقدية اللالبنية، البكتريا المحبة للبرودة، المكورة معوية البراز، المكورة المعوية البرازية.