Gill lesions are the main cause of death in yellowfin seabream (*Acanthopagrus latus*) following infection with *Amyloodinium ocellatum*

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Abstract

Amyloodiniosis, caused by the ectoparasite *Amyloodinium ocellatum*, affects the healthy development of mariculture. This study used a local infection method to identify the pathogenic target organ responsible for the death of infected fish. Comparing the relationship between the abundance of trophonts in gills and skin with the mortality of infected fish using local infection showed that severe gill infections cause the mortality of infected fish.

Local infection

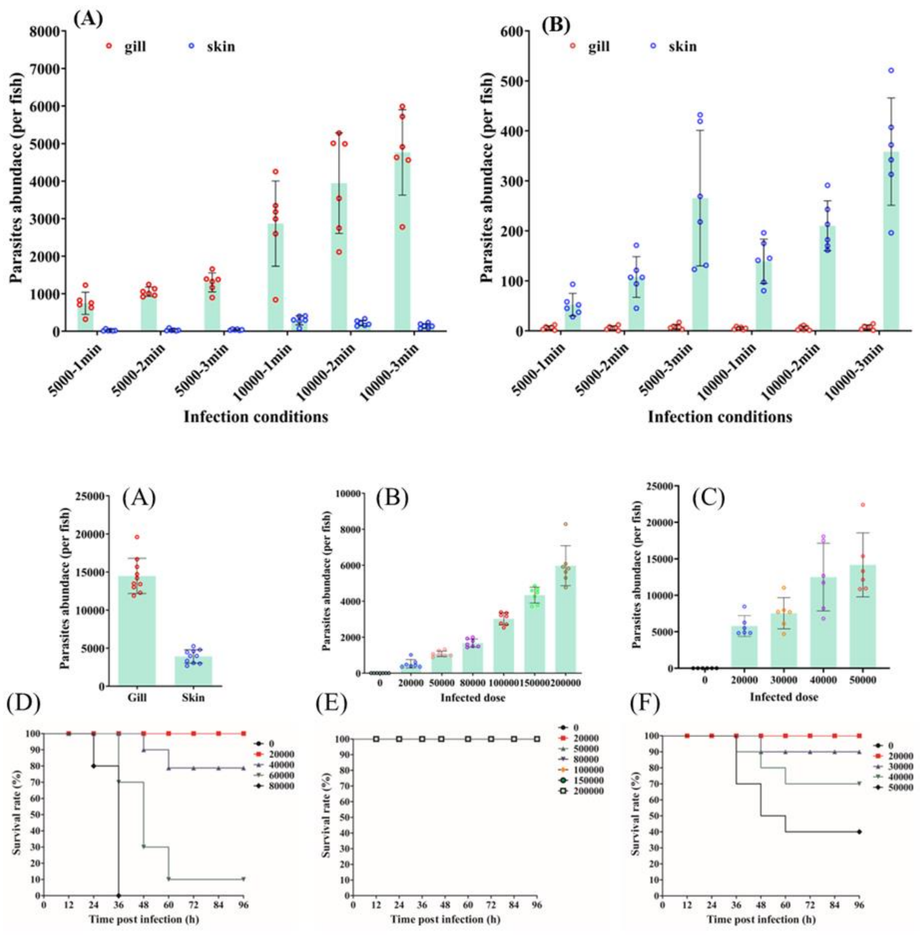


Fig. 2. Survival rate and parasite abundance of *Acanthopagrus latus* after infection with *Amyloodinium ocellatum*. (A, D) Total infection group: A, the parasite abundance in the gill and skin of *A. latus* after infection with *A. ocellatum* at 60,000 dinospores per fish; B, the survival rate of *A. latus* post *A. ocellatum* total infection; (B, E) Skin local infection group: B, the parasite abundance in the skin at different infection doses of dinospores; E, the survival rate of *A. latus* post *A. ocellatum* skin local infection; (C, F) Gill local infection group: C, the parasite abundance in the gill at different infection doses of dinospores; F, the survival rate of *A. latus* post *A. ocellatum* gill local infection.

Developmental rate

Fig.1. Parasite abundance of infected fish. (A) Gill and skin parasite abundance using gill local infection methods; (B) Gill and skin parasite abundance using skin local infection methods. Date was presented as mean ± SD (n= 6).

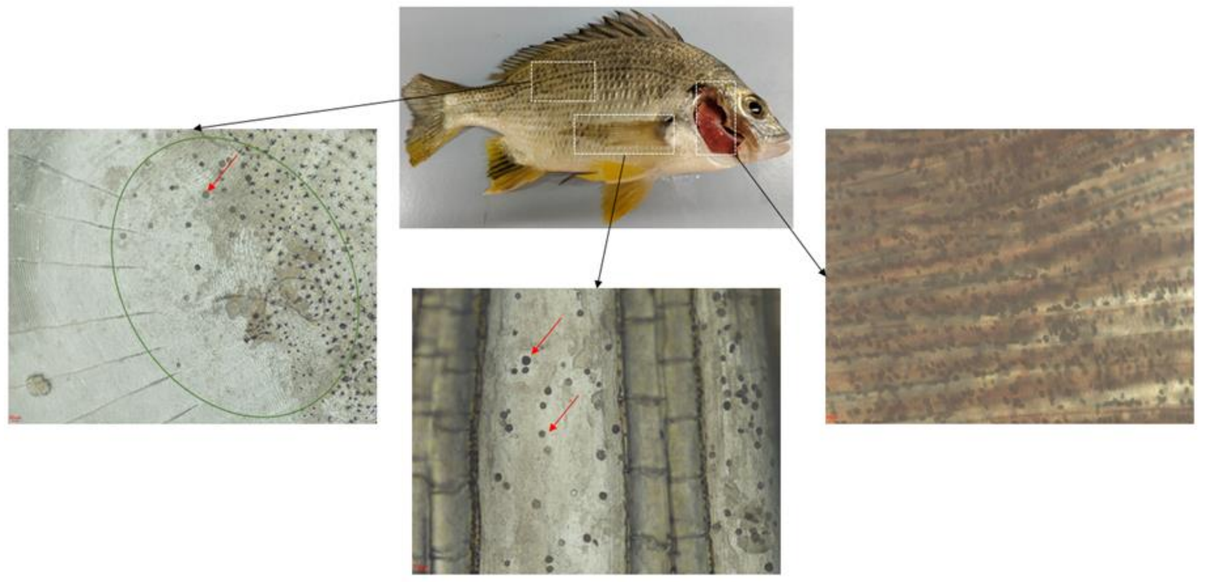


Fig. 3. Trophonts parasitizing on gills, fins and skin after infection with *Amyloodinium ocellatum*. For skin infections, trophonts were parasitized on the scales of infected fish.

Fig. 4. Diameter of trophonts parasitizing on different organs. Date was presented as mean ± SD (n= 20).

Histopathology

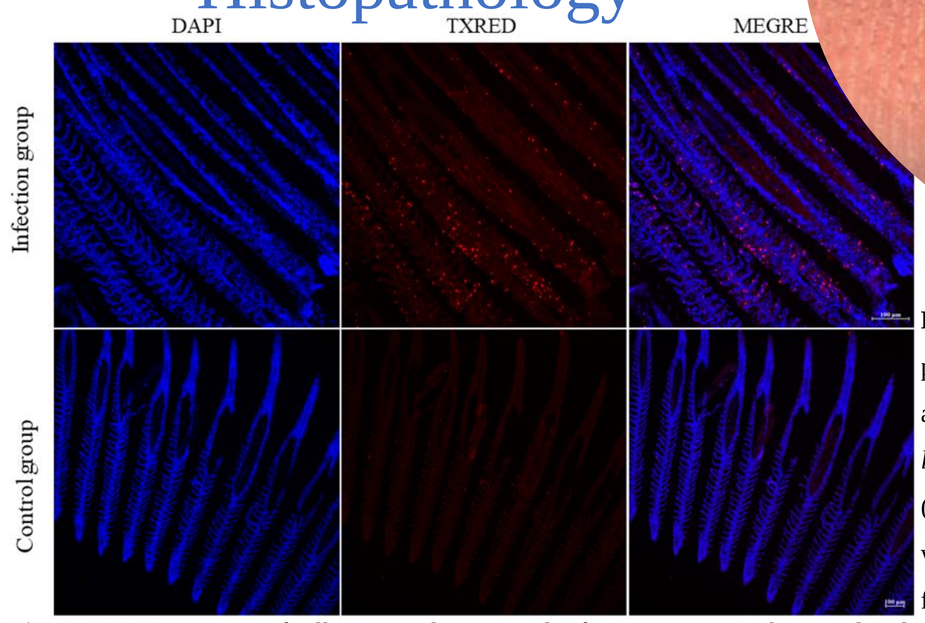


Fig. 6. TUNEL staining of gill in control group and infection group. Both normal and apoptotic cells nuclei can be stained with blue fluorescence, and only apoptotic cells DNA can be stained with red fluorescence.

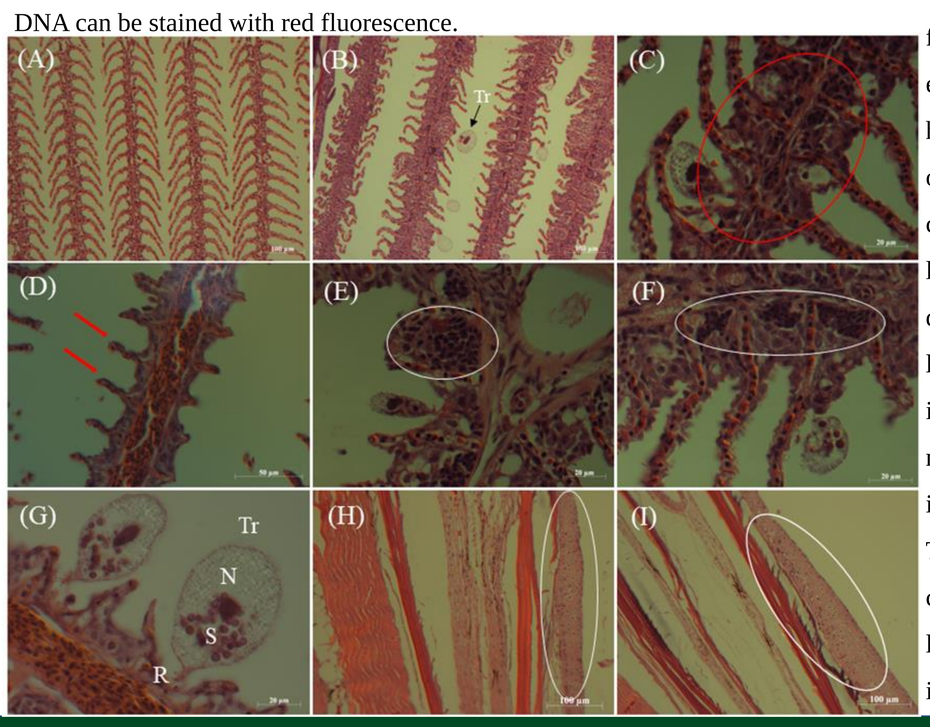
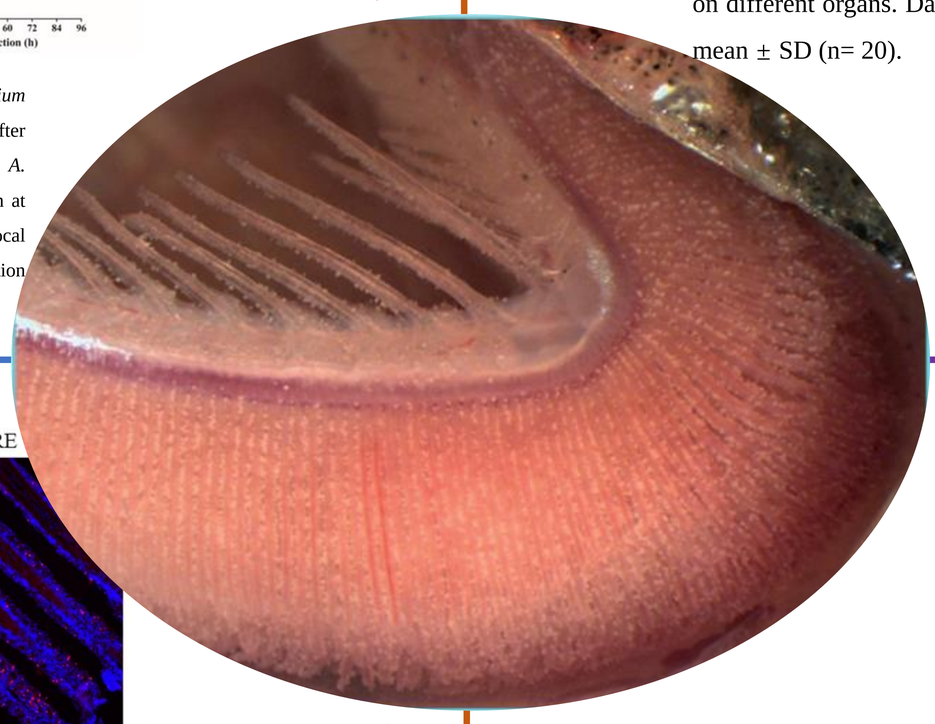


Fig. 5. Histological pathological changes in the gill and skin of *Acanthopagrus latus* with HE-stained sections. (A) The gill of the control fish: with well-arranged gill filaments and tightly packed epithelial cells; (B-G) The gill of the *A.ocellatum* infected fish: structurally disordered, epithelial cell degeneration and hyperplasia leading to fusion of adjacent lamellae (red circle), detachment of the lamellar epithelium and disappearance of the secondary lamellae (red arrow), inflammatory foci with massive lymphocytic infiltration (white circle); (H) The different layer skin the control fish; (I) The different layer skin of the *A.ocellatum* infected fish.



Microbial community

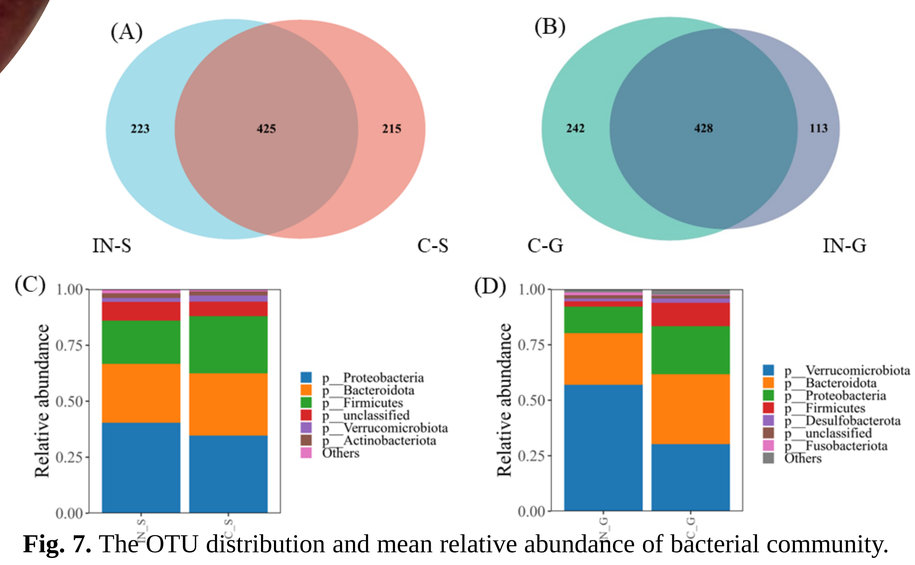
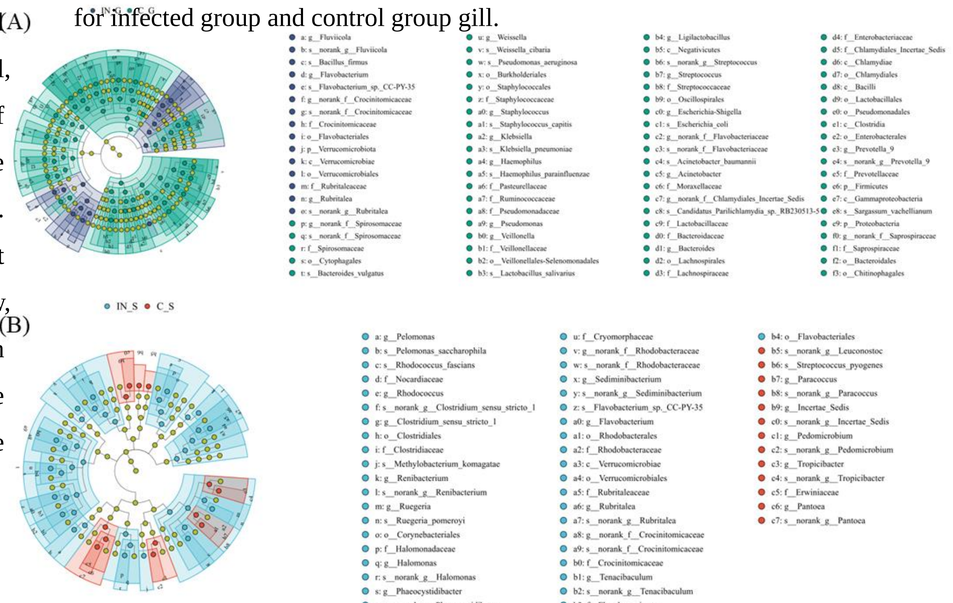


Fig. 7. The OTU distribution and mean relative abundance of bacterial community. (A) Venn diagram of skin microbiota OTU distribution; (B) Venn diagram of gill microbiota OTU distribution; (C) Relative abundance at the phylum level for infected group and control group skin; (D) Relative abundance at the phylum level for infected group and control group gill.

Fig. 8. Species-level variation of *Acanthopagrus latus* gill and skin microbiota present in infected and control groups. Circles radiating outwards from the figure represent the taxonomic level from phylum to genus, each small circle at a different taxonomic level represents a taxon at that level, and the size of the diameter of the small circle represents the size of the relative abundance. Species with no significant differences are colored yellow, and biomarkers with significant differences are coloured according to the group annotation.



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