



Integrated developmental atlas and molecular network analysis: mechanisms of tolerance threshold collapse during the blastula-gastrula mortality window in *Trachinotus blochii* embryos



Chaoyue Deng^a, Huan Liang^a, Xinxin Wang^a, Huapeng Chen^a, Wenyan Lin^a, Changlin Chen^a, Shanshan Fu^a, Fuli He^a, Junlong Sun^a, Jian Luo^{a,b}, Feibiao Song^{a,*}

^aSchool of Marine Biology and Fisheries, Hainan Aquaculture Breeding Engineering Research Center, Hainan University, Haikou 570228, China

^b Hainan seed industry Laboratory, Sanya 572024, China.

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Introduction

- *Trachinotus blochii* aquaculture faces low hatchability (<70%) due to high embryonic mortality.
- Transcriptomic mechanisms during critical embryonic development remain largely unexplored in this species.
- This study employs RNA-Seq to compare normal and arrested embryos, aiming to identify key dysregulated pathways.

Methods

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- Three females and three males were selected for artificial fertilization.
- Collect embryos at various stages and during mortality phases.
- cDNA library sequencing and data analysis.
- Cluster analysis and functional analysis.
- PPI network analysis and qPCR validation.

Result

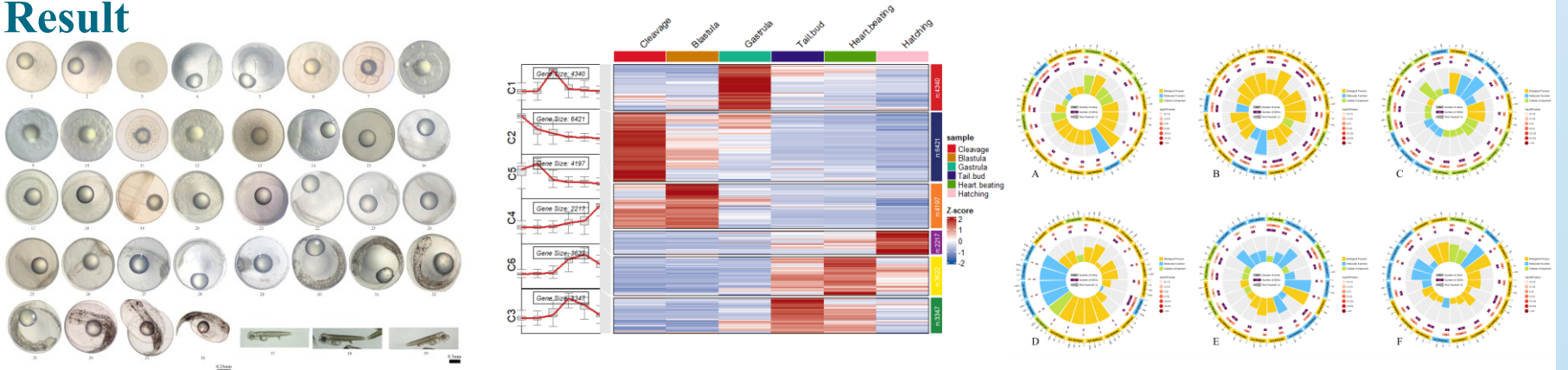


Fig. 1. Embryonic development of the *T. blochii*. Fig. 2. Time-series heatmap of gene expression dynamics. Fig. 3. GO enrichment results of genes across clusters.

Embryonic Developmental Characteristics of *T. blochii*

Result

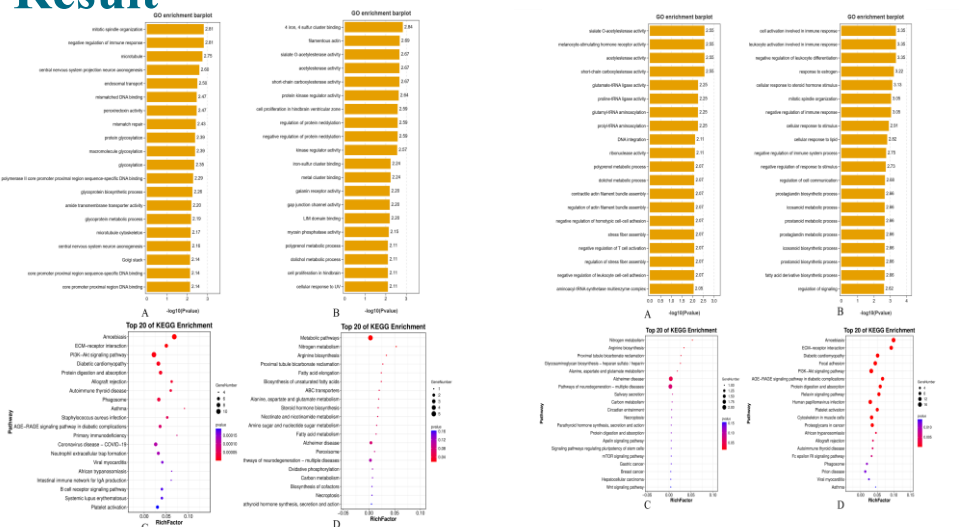


Fig. 4. PPI network analysis of DEGs between blastula-stage lethal and normal embryos.

Fig. 5. PPI network analysis of DEGs between gastrula-stage lethal and normal embryos.

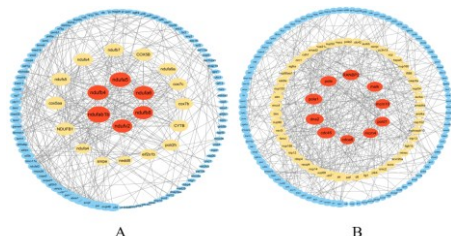


Fig. 6. PPI network analysis of DEGs between blastula-stage lethal and normal embryos.

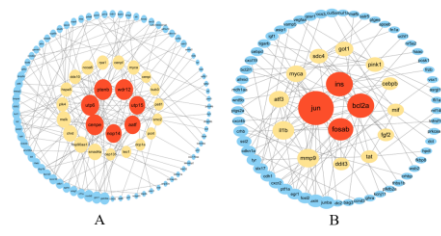


Fig. 7. PPI network analysis of DEGs between gastrula-stage lethal and normal embryos.

Mechanisms of Embryonic Mortality in *T. blochii*

Conclusion

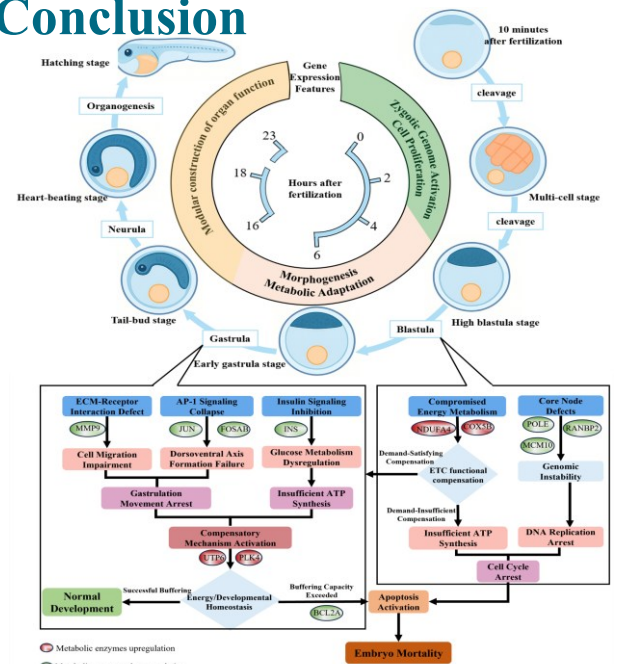


Fig. 8. Schematic Diagram of Embryonic Development and Mortality.

- ◆ Blastula-stage: Driven by disrupted ECM-receptor interactions and PI3K-Akt signaling, causing proliferation-apoptosis imbalance.
- ◆ Gastrula-stage: Triggered by AP-1 pathway hyperactivation inducing intolerable inflammatory responses that override compensatory mechanisms.