Ecophysiology of Brown Ring Disease in the Manila clam Ruditapes philippinarum, experimental and modelling approaches

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Summary

Ecophysiology of Brown Ring Disease in the Manila clam *Ruditapes philippinarum*, experimental and modelling approaches

Brown Ring Disease (BRD) in the Manila clam, *Ruditapes philippinarum*, is a disease caused by the bacterium *Vibrio tapetis*. This pathology was first observed in Northern Brittany (France) in 1987. The pathogenic agent enters in the extrapallial cavity (*i.e.* the space between mantle and shell), disrupts the normal production of periostracal lamina and causes an anomalous deposition of periostracum on the inner shell of the clams. Infected clams exhibit a characteristic brown deposit on the peripheral inner surface of the valves that gave the disease its name. This disease can be associated with mass mortalities impacting clam aquaculture. The objective of this thesis, based on both modelling and experimental approaches, was to provide further insight on the linkages between the host physiology and the development of BRD, with a special emphasis on the host energy balance.

The first part of this thesis describes the development of an interaction model between the host, the pathogen and the environment. Chapter 2 describes the development of an energy balance model based on the "scope for growth" concept for the Manila clam. This model allowed to describe variations in growth, condition index and reproduction of the Manila clam under forcing of trophic resource and temperature. This work emphasized the difficulty in estimating food availability for such an infaunal bivalve and suggested that chlorophyll *a* in the water column was not a good estimator for the trophic resource for the Manila clam. Contribution of microphytobenthos and detritic matter may also be taken into account. This work also emphasized the difficulty in validating such a model when asynchronous spawning events occur in

the studied population.

The interaction model between the host, the pathogen and the environment presented in chapter 3 attempts to link the development of the disease to the energy balance of the host. The host's portion of the model is based on the model developed in chapter 2. The defence system against the pathogen is both controlled by the condition index of the host and the temperature. Individuals that differ phenotypically were simulated by varying parameters values implicated in functions susceptible to play a critical role in disease development. This phenotypic variability allowed to provide a potential explanation for the observed variability in disease development.

The second part of the manuscript deals with observations of BRD in the field. Chapter 4 shows the results of a field monitoring of hemocyte parameters of the Manila clam, environmental factors (temperature, trophic resource and salinity) and disease (BRD and perkinsosis) development. This study showed the high degree of variability of hemocyte parameters and demonstrated that temperature and clam length explain the greater part of the recorded variability. During this survey, BRD prevalence and intensity, evaluated on the basis of symptom development, were low and it was not possible to find any significant relationship between any of the measured parameters and BRD in the clams. On the basis of this results, chapter 6 discusses the model assumptions. These results do not confirm that BRD development can be explained by the energy balance of the host.

By using data sets from the study presented in chapter 4 and additional data sets, chapter 5 presents a simple hypothesis for the first step of infection. We show that: (1) prevalence is correlated to clam size, (2) prevalence is correlated to the abundance of large particles in the sediment and (3) that a shell breakage is a potential portal for pathogen entry. From these observations, this study hypothesizes that the main factor controlling the infection process may be a mechanical disruption of the periostracal lamina or shell edge by large sediment particles, thus opening a portal of entry for *V. tapetis*. This hypothesis suggests that (1) clam handling in aquaculture beds may favour BRD development and (2) variations in the initial injury of the periostracal lamina or shell edge could explain part of the observed variability in disease development.

The third part of the manuscript deals with the impact of BRD development on the energy budget of the Manila clam. Experimental results presented in chapter 7 indicate that severely infected clams are subject to a higher weight loss than uninfected ones, indicating that BRD affects the energy budget. Measurements showed that the clearance rate of severely diseased clams was sig-

nificantly decreased by both a decrease in filtration capacity and a reduction of the time spent on filtration activity. Thus one primary way of modification of the energy balance is a decrease in the food intake. Data in the literature suggested that a second way of could be an increase in the maintenance costs due to energy needed for immune response and repair of lesions induces by the disease.

DEB theory provides a mechanistic framework to study mass and energy balances in living systems and describes the energy flow through organisms from assimilation to allocation for growth, reproduction development and maintenance. A model based on this theory was developed in chapter 8 to discern the effect of disease development on maintenance. A starvation experiment presented showed that in highly infected clams weight loss was higher than in uninfected one. This allowed to confirm that the energy balance was modified by the disease independently of the effect on filtration activity. Subsequently we could show that the disease could be associated to an increase in maintenance costs. Coupling the DEB model simulations and starvation observations provides a quantitative and dynamic evaluation of the effect of BRD on maintenance costs and indicated that BRD development could be associated with an important increase in the maintenance cost. This demonstrates that DEB theory can provide a powerful tool to study the effect of disease/parasites on the energy budget of the host. Further development of the model is needed to describe the relative contribution of the two ways for degradation of the energy balance and to assess the effect of the environment on the whole system.