Reduced growth, body condition and foot length of the bivalve *Austrovenus stutchburyi* in response to parasite infection

Sorrel A. O’Connell-Milne a,b,⁎, Robert Poulin b, Candida Savage a,c, William Rayment a

a Department of Marine Science, University of Otago, PO Box 56, Dunedin, New Zealand
b Department of Zoology, University of Otago, PO Box 56, Dunedin, New Zealand
c Department of Biological Sciences, University of Cape Town, Cape Town, South Africa

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**A B S T R A C T**

Parasites often have direct impacts on their host physiology or function, which can in turn have indirect effects on interspecific interactions and ecosystem structure. The present study investigates the effect of trematode parasite infection on the ecologically and commercially important venerid clam *Austrovenus stutchburyi*. Although the indirect impacts of clam infection on the broader benthic community, mediated by impaired burrowing of parasitized clams, have been well documented before, the more direct impacts on the clam itself remain poorly studied. The consequence of parasite infection on clam growth rate, mortality, body condition and foot length was quantified in a three-month laboratory experiment, in which juvenile clams were infected with varying levels of the echinostome trematode *Curtuteria australis*. Although mortality was unaffected by parasite infection, greater numbers of parasites deleteriously affected the growth rate, body condition and foot length of clams. This may result in delayed maturity and a lower filtration rate for infected individuals, as well as a reduced ability to bury into the sediment. Consequently, increased parasite infection not only has subsequent broader impacts on the surrounding ecological community, but can also affect the clam host directly and lower its value as a harvested species.

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1. Introduction

Parasites have various impacts on their hosts and the surrounding ecosystem. Generally, parasites have a deleterious effect on the well-being of their host, sometimes resulting in mortality of the host (Fredensborg et al., 2004; Robar et al., 2010). More commonly, parasites negatively affect the growth, reproductive fitness, nutrition, energetic budget and/or behaviour of their host (Poulin, 1994; Poulin and Thomas, 1999, Lafferty et al., 2008, Robar et al., 2011). In addition, changes in the environment, including climate change or invasion of a new species, may increase parasite abundance (Harvell et al., 2002). Subsequently, a previously harmless parasitic organism may negatively affect the host at increased levels of infection (Harvell et al., 1999). In addition to the direct impacts of a parasite on its host, there are often broader effects of the infection, including changes in host population dynamics, the diversity of animal communities and the structure of food webs (Mouritsen and Poulin, 2002, 2005, Lafferty, 2008).

The impact of a parasite on its host is linked to the role the host plays in the parasite's life cycle. In marine systems, parasitic worms generally have multiple-stage life cycles (Kearn, 1998). For trematodes (parasitic flukes) in particular, hosts may serve as either definitive hosts, harbouring the adult stage of the parasite, or intermediate hosts, generally harbouring the asexually multiplying or encysting stages of the parasite (Kearn, 1998). In the first intermediate host, which is usually a gastropod, a trematode parasite often destroys the host gonads and reroutes host energy towards its own asexual reproduction. In contrast, trematodes generally utilise their second intermediate host to facilitate transmission to the final host through predation; they typically encyst in a dormant state within muscle or other tissues in the second intermediate host, causing relatively little damage to key host organs (Combes, 1991, Poulin, 1994, Lafferty, 1995).

Many trematodes use bivalves as a second intermediate host, as their sedentary filter feeding behaviour makes them easy to exploit. Bivalves readily acquire parasites from the water column through the inhalant water current, and trematode infections can have negative consequences for the bivalve hosts. For instance, in European cockles (*Cerastoderma edule*), moderate to high trematode infections have been found to reduce body condition when clams experience reduced availability of food, and increase mortality at low oxygen levels (Wegeberg and Jensen, 1999, 2003, Desclaux et al., 2004).

In New Zealand, the clam *Austrovenus stutchburyi* (family Veneridae) is the second intermediate host for the echinostome parasites *Acanthoparyphium* spp. and *Curtuteria australis* (Allison, 1979, Martorelli et al., 2006), which are phylogenetically related to those
infecting the European cockle. These echinostomes encyst in the foot of the clam as metacercariae, where they remain inactive in a state of hypobiosis (Chappell, 1993). Acanthoparyphium spp. and C. australis are very closely related phylogenetically and are considered ecologically equivalent (Babirat et al., 2004). Both taxa contain cryptic species complexes with numerous genetically distinct but undescribed species (Leung et al., 2009a).

A. stutchburyi is a common species in soft-sediment estuarine communities in New Zealand (Pridmore et al., 1990), often accounting for most of the macrofauna in intertidal sand banks, with densities up to 4500 individuals per square metre (Stephenson and Chanley, 1979, Ministry of Fisheries, 2014). It has economic value as a commercially and recreationally harvested species and is of cultural importance to Māori. A. stutchburyi is an autogenic ecosystem engineer, with its shell providing an important hard substrate for attachment of invertebrates and macroalgae in soft sediment environments (sensu Jones et al., 1997, Thomas et al., 1998). In addition, A. stutchburyi is a bioturbator, increasing oxygen concentrations in surface sediment and acting as an allogenic engineer (Flach, 1996, Jones et al., 1997). It also provides essential ecosystem services and biogeochemical functions, by filtering suspended particles from the water column and enhancing water clarity (Dame, 1993) and increasing organic matter content in sediments through the biodeposition of pseudo-faeces (Newell, 2004). However, infection by echinostome trematodes has been shown to impair the burrowing and bioturbating abilities of the clams, with indirect consequences for the sediment characteristics and the diversity of benthic macroinvertebrates settling on mudflats (Mouritsen and Poulin, 2002, 2005, Lafferty, 2008). Commercial harvesting of A. stutchburyi in New Zealand reduces the biomass of shellfish within harvested beds (Stewart, 2008, Kainamu, 2010). Recent research has shown that this reduced biomass results in the remaining clams in commercially harvested areas having a higher average parasite infection level than clams in comparable unharvested areas (O’Connell-Milne, 2015). Although the indirect, community-level impacts of trematode infections are well-studied (Mouritsen and Poulin, 2002, 2005, Lafferty, 2008), the more direct effects on the general health and growth of clams remain unknown. If these effects are comparable to those seen in the European cockle (Wegeberg and Jensen, 1999, 2003, Desclaux et al., 2004), parasitism could represent an important factor for the quality of commercially harvested New Zealand clams.

The current study investigated the effect of echinostome parasites on the mortality, shell growth, foot length and body condition of the clam A. stutchburyi. Assessment of the foot length was included since the foot is instrumental in burrowing and is the only site in the clam’s body where echinostome parasites accumulate. A laboratory experiment was conducted to quantify the effect of various levels of parasite infection on A. stutchburyi using juvenile clams that were experimentally infected with the echinostome C. australis under controlled conditions.

2. Material and methods

2.1. Field collections

Two hundred juvenile A. stutchburyi (average length 13.2 mm (S.D. = 0.6)) were collected in November 2014 by hand from the south side of Quarantine Island in Otago Harbour, New Zealand, and transported to the laboratory. As the number of pre-existing echinostome metacercariae affect the establishment success of new metacercariae (Leung et al., 2010), juvenile clams were used here as they have high growth rates and harbour low numbers of metacercariae (McKinnon, 1996).

In addition, approximately 1500 whelks (Cominella glandiformis) were collected by hand in Lower Portobello Bay in Otago Harbour and transported to the laboratory. Whelks serve as the first intermediate host of our focal parasite C. australis, from which the free-swimming cercariae are released before they infect clams.

2.2. Clam baseline information

A subset of 70 juvenile clams was dissected and their parasites enumerated to establish the baseline infection level of the cohort. The foot of each clam was removed by cutting along the narrow bridge between the gonad and foot base, as described in Leung and Poulin (2011), mounted on a glass slide and flattened beneath a cover slip (Leung et al., 2010). Encysted metacercariae within the foot were counted under a dissection microscope. No distinction was made between C. australis and Acanthoparyphium spp. metacercariae, as they perform similar roles within the clam and because molecular markers are necessary to distinguish between these two taxa (Babirat et al., 2004).

2.3. Experimental setup

Clams were selected at random to create 12 groups of 10 individuals each. The length of each clam shell was measured to the nearest 0.1 mm from umbo to shell edge using digital callipers. Clams were individually tagged to allow identification. Each group was randomly allocated to one of four treatments (i.e. three groups of 10 clams per treatment), using a random number generator. The four treatments consisted of a control (not exposed to cercariae), a low infection (10 cercariae/clam), a medium infection (50 cercariae/clam), and a heavy infection (100 cercariae/clam) as in Otago Harbour, juvenile clams may have parasite infections greater than 50 (pers. obs. S. O’Connell-Milne). As approximately 60% of available cercariae were successful in infecting the clams in previous studies (de Montaudouin et al., 1998), the number of parasites needed to infect each treatment group (i) was calculated using the following equation:

\[ i = \frac{I_0}{4} \div 0.6 \]

where the final infection level for each treatment (I_i) was divided into four weekly infection events and divided by the predicted infection success rate (60%). Clams were subjected to this dose of cercariae (I_i) once a week for four consecutive weeks, which allowed parasite numbers to accumulate gradually as is typical of in situ infections (Leung et al., 2009b). Therefore, to achieve low, medium and high infection levels clams were individually exposed to 5, 21, and 42 cercariae respectively for each infection event.

Twelve replicate 151 tanks were established containing sand (sieved with 2 mm box sieve) to a depth of 20 mm and a flow-through water system. Air stones maintained adequate aeration and gentle water movement in each tank. Each replicate group was kept in a separate tank to avoid pseudo-replication. Clams were rotated clockwise to a new tank each week, to ensure there was no ‘tank effect’.

Phytoplankton bag cultures were created of the microalgae Isochrysis galbana, Tetraselmis chui, Skeletonema costatum and Pavlova lutheri. These phytoplankton species are common in the study area and were chosen as their nutritional content and size make them suitable feed for juvenile bivalves. Algal cultures were grown in a constant light environment for ten days and harvested in the log phase of growth as feed for the clams.

Juvenile clams were fed an excess of mixed algal culture, made up of equal quantities of I. galbana, T. chui, S. costatum and P. lutheri. A tidal feeding cycle was maintained with 500 ml of the mixed algae feed delivered to each tank at regular 12.4 h intervals with a peristaltic pump (Masterflex L/S multichannel model 07534–08). Pulses of food in the laboratory simulated the tidal flux of phytoplankton in situ to replicate the tidal rhythm and maintain the clams’ endogenous circatidal rhythm (Williams and Pilditch, 1997). Simulating the natural rhythmity of clams ensures that clams all fed at the same time and had equal
chances of acquiring parasites through the inhalant siphon at each infection event.

2.4. Screening first intermediate hosts for parasites

To identify infected whelks, C. australis cercariae emergence was stimulated by incubating the whelks individually in cylindrical containers (40 mm diameter) in the laboratory at 25 °C under constant light for 30 min, following the methods of Fredensborg et al. (2005). The individual containers were then examined under a dissection microscope for cercariae. The whelks producing C. australis were maintained in the laboratory for future use. C. australis was used for experimental infections rather than Acanthocheilonema spp. (which uses a different snail species as first intermediate host) because of their greater cercarial output per whelk and the ease in which cercariae can be induced to emerge from the whelks (Leung et al., 2010). Clams were infected with a single parasite species to ensure the mass of the encysting parasites could be calculated, which was necessary to correct body condition estimates for each clam (see below).

For each experimental infection, a subset of 15 whelks was haphazardly selected from those maintained in the laboratory and cercariae emergence was induced as above. After initial emergence, cercariae display energetic swimming, are positively phototactic and negatively geotactic, which is thought to increase dispersion (Leung, 2008). Three hours after the 'dispersal phase', the larvae become positively geotactic to aid uptake through the clam's filter feeding siphons (Leung et al., 2010). At this time, cercariae were collected with a 200 ml pipette and added to the experimental beakers at the required cercarial density.

2.5. Experimental infection of clams with C. australis

For infection, clams were held individually in cylindrical containers (30 mm diameter) with the posterior end of each clam pushed into the sediment (sieved sand, 20 mm deep) to facilitate burrowing. The clam was covered with 20 mm of sand-filtered seawater. After 60 min of acclimatisation, C. australis cercariae were pipetted into the water. Clams were left immersed for 12 h at 20 °C to allow sufficient time for filtration of the water and uptake of the cercariae (Wegeberg and Jensen, 2003).

2.6. Assessing the effect of infection

Approximately three months (106 days) after the start of the experimental infections, the shell length of each clam was measured with digital callipers. The daily, length-specific growth rate of clams was calculated using the following equation:

\[ G = \frac{(L_f - L_i)}{t} \]

where the daily length-specific growth rate (G) was the difference between the length of clams at the start of the experiment (L_i) and the end of the experiment (L_f), divided by the duration of the experiment in days (t) (Kautsky, 1982).

The foot of the clam was dissected as described above and measured to the nearest 0.1 mm with digital callipers after straightening on a flat surface (Thomas and Poulin, 1998). The foot was then mounted on a glass slide and encysted echinostomes were counted. Clam soft tissue was removed from the shell and, together with the foot, oven-dried at 60 °C for 24 h. The tissue was weighed to the nearest 0.001 g to determine the clam soft-tissue dry weight (STDW).

As C. australis metacercariae occupy a round cyst with an average diameter of 0.023 cm and vary little in body size, the volume of an individual parasite was calculated based on the formula for a sphere. The mean body volume of an encysted parasite equalled 0.0007 cm³, which converts to a mass of 0.0007 g, assuming that parasite density equals that of water (Santoro et al., 2013). The total mass of parasites per clam was the product of the weight of an individual cyst, multiplied by the number of cysts per clam. The soft tissue dry weight of the clam excluding parasites (STDWep) was calculated by deducting the total mass of parasites from the wet weight of the clam. A conversion factor of 8.7% was used to calculate the wet weight of an individual clam from STDW (Ricciardi and Bourget, 1998). The wet weight of the clam without parasites was then converted back to STDW to give STDWep.

The residual index was used to calculate the body condition of each clam (Jakob et al., 1996). Clam body mass (STDW) was regressed on clam shell length, after the data was natural log-transformed to meet the assumptions of regression. The residual distances of individual points from the regression line indicate whether the individual's body condition was above or below that predicted for its length (Cone, 1989). In addition, the body condition excluding parasite mass was also computed for each clam by regressing STDWep on the clam's shell length. This was done because at high infection levels, especially in the case of small juvenile shellfish, 'raw' body condition may be overestimated unless the mass of parasites is taken into account (Lagrué and Poulin, 2015). Body condition, STDW and STDWep could be calculated for only 95 of the 116 clams due to contamination by shell fragments or loss of some samples.

2.7. Statistical analysis

Due to highly variable and overlapping numbers of parasites acquired within and among treatments, regression analysis was considered the most appropriate statistical method to assess the effect of increasing levels of parasite infection on growth rate, body condition and foot length of juvenile clams. A logistic regression on binary data (dead or alive) was used to test the effect of number of parasites per clam on survival. Linear regression was used to test the effect of parasite infections on shell growth rate, with significance set at 5% confidence level (α = 0.05). Inclusion of control (uninfected) clams skewed the distribution of the data (with an excess of zero values) and violated the assumptions of regression analysis; however, excluding them had no effect on the results, therefore they are included here in both figures and analyses. All statistical analyses were carried out using the software package RStudio 0.98.1103 (R Development Core Team, 2014).

3. Results

The baseline infection level of trematodes in the juvenile clams collected for the laboratory experiment was very low at 0.1 metacercaria per clam, with only 8.6% of individuals infected at the start of the experiment. All (100%) clams exposed to experimental infections were successfully infected. After experimental infection, clams in low, medium and high treatments had an average infection intensity of 13.9 (S.E. = 0.8), 67 (S.E. = 4.4), and 167.2 (S.E. = 14.2) metacercariae per clam, respectively. The control group maintained an average of 0.03 metacercariae per clam. The survival rate of clams during the three-month experiment was high, with only 3.33% mortality. The number of parasites acquired did not influence mortality rate of clams in this experiment (logistic regression analysis, P > 0.9).

After three months, the shell growth rate of clams was negatively affected by higher infections of parasites (Fig. 1; regression analysis, r² = 0.05; F_1.114 = 6.1; P = 0.015). During the 106-day experiment, clams from the control group increased in mean shell length from 13.1 mm (S.D. = 0.7) to 15.8 mm (S.D. = 1.2), corresponding to a specific growth rate of 0.026 mm d⁻¹. Clams with a low infection increased in mean shell length from 13.3 mm (S.D. = 0.6) to 15.5 mm (S.D. = 0.7), corresponding to a specific growth rate of 0.021 mm d⁻¹. Clams with a medium infection increased in mean shell length from 13.1 mm
S.D. = 0.5) to 14.1 mm (S.D. = 0.6), corresponding to a specific growth rate of 0.009 mm d\(^{-1}\). Finally, individuals in the high infection group showed an average increase in shell length from 13.3 mm (S.D. = 0.7) to 15.3 mm (S.D. = 1), corresponding to a specific growth rate of 0.019 mm d\(^{-1}\).

Once the parasite mass was removed from the biomass of each clam, there was a negative correlation between parasite infection intensity and clam soft tissue dry weight (Fig. 2; regression analysis, \(r^2 = 0.209\); \(F_{1, 93} = 24.6\); \(P < 0.001\)). There was no correlation between parasite infection intensity and the ‘raw’ body condition of clams (regression analysis, \(r^2 = 0.013\); \(F_{1, 93} = 1.21\); \(P = 0.27\)). However, after correcting for parasite mass (mean = 10.5%, ranging from 0.3 to an unusually high 62.8% of host mass), the adjusted body condition index decreased significantly with increasing parasite intensity (Fig. 3; regression analysis, \(r^2 = 0.219\); \(F_{1, 93} = 26.08\); \(P = 0.001\)).

Clam foot length varied among the treatment groups. As the foot length of the clams in the control group were shorter than those of clams in the group subjected to a low level of infection, a regression of foot length against parasite numbers across all available clams did not show a clear trend. However, when the control group was excluded, there was a negative correlation between foot length and parasite infection intensity (Fig. 4, regression analysis \(r^2 = 0.096\); \(F_{1, 84} = 8.9\); \(P = 0.004\)).

### 4. Discussion

Echinostome infections in A. stutchburyi have a wide range of indirect effects both on the host and their surroundings. As infection levels increase, the burrowing behaviour of A. stutchburyi is affected (Thomas and Poulin, 1998, Mouritsen, 2002, Tompkins et al., 2004, Leung and Poulin, 2011), which indirectly alters the influence of this abundant bivalve on the ecosystem (Mouritsen and Poulin, 2002). Direct impacts of the parasites on the host, such as effects on shell or soft-tissue growth, remain poorly understood, however. Despite the metabolic dormancy of echinostomes within the foot of a clam, at high infection levels the effect of echinostome cysts on the host become more obvious. The present study demonstrated this and supports findings of other studies on phylogenetically related foot-encysting parasites in the European cockle, C. edule (Wegeberg and Jensen, 2003). The shell growth rate, body condition and foot length of A. stutchburyi are deleteriously affected with increasing infections. Encysted metacercariae not only reduce the foot functionality of A. stutchburyi (Thomas et al., 1998, Thomas and Poulin, 1998), but the process of cercarial penetration also causes tissue damage and stress to the clam (Jensen et al., 1999), resulting in immunosuppressant effects (Paul-Pont et al., 2010). In addition to the reduced shell growth rate observed here, this immunosuppression could make the clam more susceptible to further parasitic infection as well as vulnerable to hypoxic conditions (Wegeberg and Jensen, 1999).

High levels of echinostome cysts increase the energetic demand on the hosts as shown by the reduction in shell growth rate as parasite...
infections increased. The shell growth rate of the control group was almost double that of heavily-infected clams. Moreover, since the rate of shell growth in the control group was comparable to that of similar-sized clams in situ (unpublished data, S. O’Connell-Milne), clams in the experiment were adequately nourished and accordingly any negative impacts associated with parasites were not artefacts of insufficient food.

There was a decrease in the soft tissue dry weight and body condition of A. stutchburyi as parasite infections increased in the experiment. If the body condition of a bivalve is reduced, its filtration rate is negatively affected (Riisgard, 2001, Mouritsen et al., 2003). Accordingly, its ability to assimilate food and provide the ecosystem service of filtering estuaria waters is also limited. Trematode infections reduce filter feeding activity of bivalves (Stier et al., 2015) and may also affect the body condition of clams in situ, especially during periods of low food availability, for example in winter. The additional stress from starvation may cause heavily parasitised individuals to lose body condition faster and have a slower recovery rate, as seen in C. edule (Wegeberg and Jensen, 2003). Given the economic importance of A. stutchburyi, any increase in the abundance of these common parasites could have serious impacts on the quality of clams harvested for commercial sales and exportation.

There was also a negative correlation between foot length and increasing parasite infection in this experiment, as in previous studies (Thomas and Poulin, 1998, Mouritsen, 2002). By contrast, at the lowest level of infection, there was an apparent increase in foot length of the clams in the present study: foot length was a little greater in clams exposed to low infection doses than in control clams. This may have been due to the low number of cercariae penetrating the foot causing minimal damage and stimulating compensatory tissue regeneration. It is known that clams have fast regenerative rates for their foot tissue as they are at risk of foot cropping by small fish (Mouritsen and Poulin, 2003). However, at high levels of infection, the parasites may simply overwhelm the clam’s ability to regenerate damaged foot tissue. As cercariae penetrate the epidermis of the foot to encyst, the ensuing holes enable fluid to exude from the clam’s tissue (Lauckner, 1983). The clam host also produces a tissue layer to surround the metacercariae (Jensen et al., 1999), requiring the clam to expend energy for wound healing. The movement of haemolymph and contraction of longitudinal muscles within the clam’s foot appear to be limited by the presence of metacercariae cysts (Mouritsen, 2002). As a result, as infection intensity increases, not only is foot size compromised, but also the clam’s ability to burrow into the sediment (Thomas and Poulin, 1998, Mouritsen, 2002).

Although parasite infection affects the growth rate, body condition and foot length of juvenile clams, it only explains between 7 and 22% of the variation, therefore other factors must also affect these dependant variables. Possible causes for the remaining variation may be inherent differences among clams in feeding rates (Iglesias et al., 1992), food conversion efficiency (Laing and Millican, 1991) and growth rates (de Montaudouin et al., 2012).

Clam mortality was not affected by echinostome infections in the current study. Similarly, when experimentally infected with a phylogenetically similar echinostome parasite, mortality of C. edule was minimal (Wegeberg and Jensen, 2003). Clams in the present study acquired more parasites than anticipated. This may have been due to the predictions of infection success being based only on a single dose of cercariae from a study on a different trematode-bivalve system (de Montaudouin et al., 1998), whereas multiple infection events were utilised in the current study. As pre-existing echinostome metacercariae in a clam have a positive effect on the establishment success of new metacercariae (Leung et al., 2010), this may have increased the infection success of each subsequent infection event. When large clams (30 mm shell length) were exposed to cercariae in two separate doses, the number of successful parasites per clam increased almost 4% in the second infection event (Leung et al., 2010). As Otago Harbour in Dunedin is identified as having the highest abundance of metacercariae per clam within New Zealand (Studer et al., 2013) and greater than 50 metacercariae per juvenile clam have been observed (pers. obs. S. O’Connell-Milne), these greater than anticipated infection levels may still be relevant to the local environment.

In addition to the direct effects of the parasites on the foot tissue of A. stutchburyi, parasite infections may cause deleterious behavioural changes which impact on the surrounding ecosystem (Mouritsen and Poulin, 2002). Higher parasite infections can reduce the mobility of clams within the sediment and prevent burrowing due to reduced foot functionality (Lauckner, 1984, Thomas and Poulin, 1998, Mouritsen and Poulin, 2002, 2005, Mouritsen, 2002). Clams stranded on the sediment surface have increased chances of thermal stress (Thomas and Poulin, 1998) and risk of avian predation increases seven-fold (Thomas et al., 1998, Thomas and Poulin, 1998). Additionally, as parasite infections reduce the ability of the foot to contract, its tip is more likely to be cropped by fish (Mouritsen, 2002, Mouritsen and Poulin, 2003). Once highly parasitised clams are stranded at the surface, they increase the heterogeneity of surface structures (Mouritsen and Poulin, 2002). Surfaced clam shells provide substrate for attachment or shelter for epifauna and cause the density and diversity of benthic organisms to increase (Thomas et al., 1998, Mouritsen and Poulin, 2005). In addition to limiting vertical movement, echinostomes also reduce horizontal movement of surfaced clams via crawling (Mouritsen, 2002). Because bioturbation of the top layer of sediment by clams depresses the density of co-occurring infauna (Flach, 1996, Whittatch et al., 1997), the reduced movement of clams results in increased abundance of infauna (Mouritsen and Poulin, 2002). As infauna increase in number, so too will their predators (Reise, 1985). The result is greater local biodiversity, which may increase the resilience of the ecosystem to certain changes such as local extinctions or invasions (Mouritsen and Poulin, 2002).

In summary, infection by the echinostome parasite C. australis affects A. stutchburyi deleteriously, with shell growth rates and body condition reduced as parasite infections increase. Also, past a certain threshold number of parasites, increasing intensity of infection reduces the length of the foot, which potentially compromises foot functionality. An increase in parasite infection may result in changes to the clams’ age at reproduction, body condition and behaviour, as well as have broader impacts on the ecological community and the commercial value of this harvested species.

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