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Ectoparasites increase swimming costs in a coral reef fish

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Ectoparasites can reduce individual fitness by negatively affecting behavioural, morphological and physiological traits. In fishes, there are potential costs if ectoparasites decrease streamlining, thereby directly compromising swimming performance. Few studies have examined the effects of ectoparasites on fish swimming performance and none distinguish between energetic costs imposed by changes in streamlining and effects on host physiology. The bridled monocle bream (Scolopsis bilineatus) is parasitized by an isopod (Anilocra nemipteri), which attaches above the eye. We show that parasitized fish have higher standard metabolic rates (SMRs), poorer aerobic capacities and lower maximum swimming speeds than non-parasitized fish. Adding a model parasite did not affect SMR, but reduced maximum swimming speed and elevated oxygen consumption rates at high speeds to levels observed in naturally parasitized fish. This demonstrates that ectoparasites create drag effects that are important at high speeds. The higher SMR of naturally parasitized fish does, however, reveal an effect of parasitism on host physiology. This effect was easily reversed: fish whose parasite was removed 24 h earlier did not differ from unparasitized fish in any performance metrics. In sum, the main cost of this ectoparasite is probably its direct effect on streamlining, reducing swimming performance at high speeds.

1. Introduction

Ectoparasites can substantially affect hosts by impacting physiological, behavioural and morphological traits, and damaging the host’s integument [1–3]. In fishes, ectoparasites pose additional challenges because streamlining is important to reduce the costs of locomotion [4]. Changes to fish morphology created by ectoparasites potentially reduce streamlining and increase friction drag along the fish’s body, which may considerably reduce host performance [3,5]. Although the consequences of carrying ectoparasites can be high, few studies have examined the effects of ectoparasitism on the swimming performance and energetics of infected fishes [3,5,6]. Crucially, there have been no attempts to separate out costs owing to the hydrodynamic effects of reduced streamlining from the effects of parasites on host physiology.

Cymothoid isopods are ectoparasitic crustaceans that infect fishes throughout the tropics [1]. These abundant and relatively large (4.2–23.0 mm) parasites either attach themselves to a fixed location on their host, or move around freely on the host’s body [1]. On coral reefs, these isopods parasitize several species including the bridled monocle bream (Scolopsis bilineatus), with approximately 4 per cent of the population infected by Anilocra nemipteri at some locations on the Great Barrier Reef (S. A. Binning 2012, unpublished data). This isopod can typically grow to over 15 per cent of its host’s total length. It attaches itself firmly above the eye of the fish, and has the potential to reduce host swimming performance.

Here, we measured the effects of A. nemipteri on the swimming performance and energetics of the bridled monocle bream, S. bilineatus. To separate the physiological
and hydrodynamic effects of this ectoparasite, we evaluated aerobic swimming performance and swimming speed in fish that were (i) parasitized, (ii) unparasitized, (iii) parasitized but had the parasite experimentally removed, and (iv) unparasitized but had a model parasite experimentally added. We compared (i) and (iii) to test for physiological effects of the parasite on host performance as well as (iii) and (iv) to test for hydrodynamic effects of parasitism. We compared (i) and (ii) to quantify the net effect of parasitism on hosts. We predicted that the negative effects of parasitism on the swimming performance of *S. bilineatus* are mainly owing to physiological effects at slow swimming speeds, but that hydrodynamic effects become important at high swimming speeds.

2. Material and methods

(a) Experimental swimming and respirometry trials
Adult *S. bilineatus* were collected using ultrafine barrier nets and hand nets between February and March 2012 from reefs surrounding Lizard Island, northern Great Barrier Reef, Australia (14°40′S; 145°28′E). Fish were transported live in buckets to the aquarium facilities at the Lizard Island Research Station within 2 h of capture. Eighteen unparasitized (total length $L_T = 13.27 \pm 0.17$ mm; mass $= 38.2 \pm 0.8$ g; means $\pm$ s.e.) and 20 parasitized ($L_T = 12.66 \pm 0.18$ mm; mass $= 34.8 \pm 0.9$ g; means $\pm$ s.e.) fish were divided into four treatment groups: unparasitized (eight fish), parasitized (10 fish), parasite-removed (10 fish) and model-parasite-added (10 fish); electronic supplementary material, figure S1). Parasites were removed using forceps 24 h before the start of swim trials. The average length, width and mass of the isopods were used to mould model parasites from Instamorph polyester thermoplastic ($L_T = 2.48 \pm 0.06$ mm; body width $W_B = 0.99 \pm 0.03$ mm; mass $= 0.6 \pm 0.0$ g; means $\pm$ s.e.). Model parasites were attached with EA Cyberbond 2610 instant adhesive. Swimming trials were carried out in an 11.9 l Loligo swimming respirometer at a constant temperature of $28 \pm 0.1$ °C. We measured oxygen consumption rate ($M_O_2$) as a function of swimming speed ($U$), following a standard $U_{crit}$ protocol [7]. Trials were stopped when fish could no longer swim unassisted or were forced to rest against the back grid of the flow chamber ($U_{crit}$) for greater than 5 s (see the electronic supplementary material, materials and methods).

(b) Oxygen consumption curves and aerobic scope
We used an exponential function to describe the relationship between $M_O_2$ and $U$ for each treatment group [7,8]

$$M_O_2 = a10^{bU},$$

which in its log-transformed linear form becomes

$$\log M_O_2 = \log a + bU,$$

where $a$ is the estimated $M_O_2$ at zero speed (SMR) and $b$ is the slope of the semi-logarithmic regression. Maximum metabolic rate (MMR) was measured at $U_{crit}$. We calculated the factorial aerobic scope (AS) as the ratio of MMR to SMR.

(c) Statistical analyses
We used one-way ANOVAs with Tukey HSD post hoc tests to examine differences in swimming ($U_{crit}$) and metabolic performance (SMR, MMR, AS) among treatments. Factorial AS was log$_{10}$ transformed to meet the assumptions of the model. The linear forms of the oxygen consumption rate curves were used to test for differences in the relationship between fish swimming speed ($U$) and oxygen consumption rate (log $M_O_2$) among treatments using a linear mixed effects model (LMM; lme function). All analyses were performed in R v. 2.11.1 [10]. Data are deposited in the Dryad Repository: http://dx.doi.org/10.5061/dryad.r73v3.

3. Results
Parasitized fish had higher SMR ($F_{3,34} = 7.152, p < 0.001$) and lower AS ($F_{3,34} = 8.897, p = 0.001$) than fish from the other three treatments (Tukey’s HSD, $p < 0.01$ for all contrasts; figure 1). MMR did not differ among treatment groups ($F_{3,34} = 0.992, p = 0.408$), with differences in AS resulting from an increased SMR in parasitized fish. Both parasitized fish and fish with a model-parasite-added swim slower than unparasitized and parasite-removed individuals ($F_{3,34} = 4.922, p < 0.01$; Tukey’s HSD, $p < 0.01$ for all contrasts; figure 1). Parasitized individuals consumed oxygen at a consistently higher rate than individuals in other treatments (LMM intercept: $F_{3,34} = 4.20, p = 0.013$; figure 2). There was no difference in the rate of oxygen consumption at any swimming speed between parasite-removed and unparasitized individuals (intercept estimate $= −0.0014, 95% CI = 0.0733$ to $−0.0760, p = 0.97$; slope estimate $= 0.0060, 95% CI = 0.0218$ to $−0.0099, p = 0.46$). However, the costs of swimming increased at higher speeds in fish with a model-parasite-added (LMM slope: $F_{3,234} = 4.68, p < 0.01$; figure 2).

Figure 1. Bar plots with average values ($\pm$ s.e.) for parasitized (black), unparasitized (white), parasite-removed (dark grey) and model-parasite-added (light grey) fish for (a) standard metabolic rates (SMRs), (b) factorial aerobic scope and (c) experimental ($U_{crit}$) swimming speeds ($U$). Different letters (a, b) indicate significant differences between treatment groups ($p < 0.01$).
4. Discussion

The ectoparasitic isopod *Anilocra nemipteri* probably increases friction drag along the fish’s body surface. Although this effect is non-lethal, the consequences of a reduced maximum swimming speed and lower AS, as well as a higher SMR and a greater overall oxygen consumption rate are potentially significant for individual fitness and population demographics.

Hydrodynamic effects of the parasite were more pronounced at speeds above approximately 2.5 body lengths s\(^{-1}\). At lower speeds, drag effects appeared to be minimal. Parasitized fish, however, continued to consume an average of 24 per cent more oxygen on average when at rest than fish in the other three treatments. Ostlund-Nilsson *et al.* [5] found similar increases in the resting metabolic rate of the cardinalfish, *Cheilodipterus quinquelineatus*, parasitized by a congeneric isopod, *Anilocra apogonae*. The authors attributed this increase in energetic cost to the destabilizing effect of the asymetrically attached parasite rather than to any physiological effects of parasitism. Indeed, they found that parasitized fishes used their pectoral fins more while at rest, presumably in order to maintain stability [5]. Our results suggest otherwise, because SMR did not increase when a model parasite was added, and we did not observe any change in pectoral fin beat frequency when fish were at rest (S. A. Binning 2012, unpublished data). Physiological effects owing to parasitism are probably responsible for the elevated SMR observed in our study. Interestingly, these physiological effects appear to be rapidly reversed. Fish that had their parasite removed 24 h earlier performed as well as unparasitized individuals across all performance measures. There appears to be no detectable long-term physiological damage from this ectoparasitic isopod once removed.

High-speed swimming is required during anaerobic burst events such as predator escapes, and for sustained aerobic swimming such as during severe weather events. In these situations, parasitized individuals will be strongly disadvantaged as their maximum swimming speed is significantly reduced. Parasitized fish also had a reduced capacity for aerobic activity, which compromises their ability to engage in multiple energy-expending activities at one time. Although burst swimming involves anaerobic metabolic pathways, these activities are negatively affected by a reduced aerobic capacity: bursting incurs an oxygen debt that must be repaid at the expense of other fitness-enhancing activities. Additionally, because water flow velocity varies dramatically across sites at Lizard Island [11], an inability to cope with high water flows could severely limit the range of habitats that parasitized fish can exploit. Furthermore, the metabolic rates of parasitized individuals were higher at all swimming speeds suggesting that energy requirements of parasitized fish exceed that of uninfected fish. As a result, parasitized individuals either need to spend more time foraging, putting them at greater risk of predation, or may suffer from reduced growth and/or reproduction [1,12,13].

Although parasites are known drivers of morphological change in hosts, many of these changes are the result of underlying physiological modifications [14,15]. Here, we show that parasites can alter the hydrodynamic profile of hosts with measurable consequences on swimming performance in the absence of any physiological effects. By interfering with streamlining via increased friction drag, large ectoparasites potentially compromise important activities such as sustained swimming, habitat use, foraging and predator evasion.

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