Coral Reefs (2021) 40:1361–1367
https://doi.org/10.1007/s00338-021-02113-x

NOTE

Exposure to degraded coral habitat depresses oxygen uptake rate during exercise of a juvenile reef fish

Adam T. Downie[1] · Caroline M. Phelps[2] · Rhondda Jones[3] · Jodie L. Rummer[1,4] · Douglas P. Chivers[5] · Maud C. O. Ferrari[6] · Mark I. McCormick[4]

Received: 26 October 2020 / Accepted: 12 May 2021 / Published online: 29 May 2021

Abstract Coral reef ecosystems are currently under unprecedented stress due to anthropogenic induced climate change. Such stress causes coral habitats to degrade, which has been found to negatively impact the behaviour of some reef fishes. However, it is unknown whether the same chemical stresses from degraded habitats that impacts fish behaviour also impacts energy supporting swimming performance traits of fishes during the pelagic-to-reef life-history bottleneck. Here, we exposed newly settled juvenile Ambon damselfishes (*Pomacentrus amboinensis*) to either water that had passed over healthy or degraded coral for 24 h. Fishes were then swum at an ecologically relevant swimming speed for 200 min, and oxygen uptake rates were measured periodically. In general, fish swimming in water from degraded coral depressed oxygen uptake rates by 21%, which suggests that degraded habitats can have strong effects on fish physiology during this ecologically-critical time window.

Keywords Climate change · Swimming performance · Active metabolic rate · Metamorphosis · Early life history · Endurance swimming · Anthropogenic stress

Topic Editor Alastair Harborne

Supplementary Information The online version contains supplementary material available at https://doi.org/10.1007/s00338-021-02113-x.

✉ Adam T. Downie
adam.downie@my.jcu.edu.au

[1] Australian Research Council Center of Excellence for Coral Reef Studies, James Cook University, Townsville, QLD, Australia

[2] Oregon State University, Corvallis, OR, USA

[3] Division of Tropical Health and Medicine, James Cook University, Townsville, QLD, Australia

[4] College of Science and Engineering, James Cook University, Townsville, QLD, Australia

[5] Department of Biology, University of Saskatchewan, Saskatoon, SK, Canada

[6] Department of Biomedical Sciences, WCVM, University of Saskatchewan, Saskatoon, SK, Canada

Introduction

Hard corals, the architects of the world's coral reefs, are under increasing threat from anthropogenic stress (Hughes et al. 2018a). Warming ocean temperatures and ocean acidification can lead to the symbiotic zooxanthellae expelling themselves from corals, a process known as bleaching, which may lead to whole colony death (Hughes et al. 2018b). Like many coral reef ecosystems, the Great Barrier Reef has been recently impacted by several major bleaching events (e.g., 2016, 2017, 2019) resulting in mass loss of reef-building corals (Hughes et al. 2018b). Once hard corals die, their skeletons break apart and are rapidly colonised by algae, cyanobacteria, and sessile invertebrates. This process of degradation has widespread ripple effects onto other reef fauna, including fishes.

Coral reef fishes are not only susceptible to direct changes in their physiology from climate change (e.g., elevated oxygen uptake rates from ocean warming; Rummer and Munday 2017), but also indirect stressors caused by the degradation of their coral reef habitats. For example, standard metabolic rate (SMR), which represents an organism's metabolic costs for basic maintenance, is elevated by 8% in anemonefishes living in bleached anemones after two weeks of exposure (Norin et al. 2018), but SMR

decreased after two months of exposure to a bleached anemone (Cortese et al. 2020). Furthermore, there has been recent evidence suggesting that degraded coral habitats negatively impact sensory behaviours involved in the predator–prey interactions of coral reef fishes (Ferrari et al. 2017). Indeed, many fishes lose their ability to respond to chemical sources of information as a result of the changing odour-scape. Much of this research has investigated newly-settled juvenile reef fishes, as they are extremely vulnerable to predation and face many challenges upon finding a new reef home. Moreover, survival of this life stage can be a critically important driver for population demography.

The pelagic-to-reef transition period is a critical stage in reef fish life history. After a pelagic developmental period, larval reef fishes navigate to a patch of reef where they can experience intense and selective mortality-pressures, mainly due to predation (Gagliano et al. 2007). Indeed, upwards of 60% of new recruits succumb to predation within the first couple of days (Almany and Webster 2006). Considered a bottleneck of reef fish population structure (Leis and McCormick 2002), larvae must make a rapid transition (< 24 h) to the reef, and this is marked by physiological and morphological changes, known as metamorphosis (McCormick et al. 2002). Strong swimming performance is required to facilitate this transition, as hydrodynamic processes between pelagic and reef environments are dramatic in terms of flow velocity and tidal energy (Fulton et al. 2005). However, chemical stresses in aquatic systems have been shown to change swimming performance in larval fishes (Downie et al. 2020), and overall, it is currently unknown as to whether the chemical stresses associated with coral degradation, which have been shown to alter behaviour and SMR in reef fishes, may have a direct impact on the oxygen uptake rates that supports swimming during the pelagic-to-reef transition period. Given that the first 24 h are critical for fish survival on reefs, the aim of this study was to determine whether water from degraded coral habitats detrimentally alters the metabolic rate that supports swimming of newly-settled coral reef fishes. We hypothesize that metabolic demands would increase in fishes swum in water from degraded coral habitats, as acute exposure to stressors generally elevate energy demands.

Methods

Study site and species

Collection of fish and coral and experimentation occurred from November to December 2018 at Lizard Island Research Station on the northern Great Barrier Reef, Australia (14°40′S, 145°28′E). Settlement-stage larval Ambon damselfish, *Pomacentrus amboinensis,* were collected at night away from the fringing reef using light traps (8–16 m depth). Traps were deployed in the afternoon (at approximately 17:00) and subsequently collected the following morning (at approximately 06:00) to capture metamorphosing juveniles. Once sorted to species, captured individuals were immediately but randomly placed in 20L flow-through seawater tanks (n = 2 fish per tank; 2 replicate tanks per treatment group) at the research station (natural photoperiod, water temperature = 28 °C). Seawater flowing into the replicate tanks came from separate, aerated header tanks that contained either live coral or dead-degraded coral (*Pocillopora damicornis*) collected from the fringing reef (dimensions per colony: ~ 200 mm length × 200 mm width × 150 mm height). This hard coral is a common component of coral colonies on reefs where larval Ambon damselfishes settle and metamorphose into juveniles (McCormick et al. 2010; McCormick et al. 2019a). Newly settled juvenile fishes were maintained in these water sources (e.g., live or degraded coral habitat) for 24 h. Fishes were fed *Artemia* spp. nauplii upon arrival to the laboratory, but food was withheld for 12 h prior to experimentation to minimise the impacts of standard dynamic action on respiration rates (Rummer et al. 2016). Three endurance tests could be performed per day, and treatments were randomly selected (e.g., 1 degraded and 2 healthy treatment tests). At the end of the 24 h exposure period, remaining individuals in the replicate tanks (e.g., those not randomly selected for experimentation) were replaced by newly captured fish (to reduce tank effects) until the achieved sample size per treatment group was met (n = 10 per treatment). All ethics to capture, maintain, and experiment upon these animals were approved by James Cook University's Animal Ethics Committee (ethics approval number A2425).

Larval swimming respirometer

Fish were swum using a glass, Blazka-style swimming respirometer (volume (V) = 125 ml; length (L) = 14.5 cm; diameter (ϕ) = 2.7 cm), which provides a simultaneous estimate of oxygen consumption rates ($\dot{M}O_2$) while an individual swims at any given speed (Supplementary Materials Fig. S1). The swimming respirometer was calibrated prior to experimentation using a high-speed camera and passive particles. To reduce the volume of the respirometer, thus providing a more accurate $\dot{M}O_2$ measurement for the size of animals swum, an insert (ϕ = 2.6 cm, L = 5.5 cm) was placed in the respirometer to create the working section (Supplementary Materials Fig. S2). The working section of the respirometer (V = 50 ml, L = 6.5 cm, ϕ = 2.7 cm) fit a smaller

chamber, where fish swum (volume with swimming chamber = 40 ml) (Supplementary Materials Fig. S1). This smaller chamber (V = 5.1 ml, L = 4.5 cm, ϕ = 1.2 cm) was fitted with a flow straightener (made from capillary tubes of ϕ = 1.1 mm, L = 40 mm) to mitigate micro-turbulent flow and a downstream mesh barrier to prevent the fish from being sucked into the propeller (mesh ϕ = 0.415 mm). The swimming chamber was large enough for a fish to swim in any direction comfortably (i.e., thus mitigating stress of enclosure) and prevented blocking effects (< 5%), which would alter the flow within the chamber. An external flush pump was used to deliver clean seawater (temperature (T) = 28 °C; pressure (P) = ~ 1 bar; salinity (S) = 33, dissolved oxygen (DO) = ~ 6.1 mg ml^{-1}) to the system in between measurement periods (see *Experimental protocol* for details). Seawater pumped into the respirometer originated from a header tank containing either live or degraded coral, respective of the fish's treatment. Oxygen (mg O_2 ml^{-1}) and T (°C) were simultaneously measured using respective probes (oxygen probe: OXROB3 Robust Oxygen Probe, PyroScience, Achen Germany; T sensor: TSUB36 Shielded submersible temperature sensor, Pyroscience, Achen, Germany) (Supplemental Materials Fig. S1). Oxygen probes were calibrated to 100% air saturation using fully aerated seawater (T = 28 °C, S = 33, P = 1 bar, DO = 6.1 mg O_2 ml^{-1}) and to 0% oxygen saturation (T = 28 °C, S = 33, P = 1 bar, DO = 0 mg O_2 ml^{-1}) using sodium sulphite (Na_2SO_3; UNIVAR Analytical Reagent, Ajax Finechem, New South Wales, Australia). Oxygen and temperature probes were connected to a Firesting oxygen meter (4-channel optical oxygen meter, Pyroscience, Achen Germany), which constantly measured both of these variables throughout each experiment (i.e., a reading for temperature and oxygen was provided every second). An outer temperature jacket (V = 85 ml, L = 6.5 cm) maintained water temperature at experimental conditions (T = 28 °C), even at high water velocities. Prior to any swimming experiment, all components of the swimming respirometer and header tank were washed using a 10% bleach solution to eliminate microbial activity from the system.

Experimental protocol

Fish from both treatments (n = 10 per treatment) were tested using the same experimental protocol and swum individually using the same swim respirometer. Fish remained under their respective treatment water conditions for 24 h. Prior to each individual experiment, background respiration (i.e., microbial oxygen consumption) was measured for 10 min. To mitigate microbial respiration, seawater used for all experiments was ultra-violet filtered (Blagdon Pro 24 W ultra-violet clarifier, Dreative Pumps, South Australia, Australia), keeping background respiration below 2% (Rummer et al. 2016). Then, individual fish were randomly selected from one of the replicate tanks (degraded or healthy coral), placed in the swimming chamber, and allowed to habituate for 1 h at a velocity of 1 body length (BL) s^{-1}. Following this period, fish underwent a constant velocity test at an ecologically relevant flow velocity (10 cm s^{-1}; Johansen 2014) mimicking their depth at settlement (McCormick and Weaver 2012). Oxygen uptake ($\dot{M}O_2$; mg O_2 s^{-1}) was simultaneously measured throughout the swimming test; intermittent flow respirometry included repeated cycles consisting of a 20-min measurement period followed by a 3-min flush period to replenish levels to 100% air saturation (live or degraded coral; T = 28 °C, DO = 6.1 mg L^{-1}) (Rummer et al. 2016). Seawater pumped into the respirometer came from a header tank containing either live or degraded coral, respective of the fish's treatment. Oxygen levels within the swimming chamber were not allowed to drop below 90% air saturation to prevent oxygen uptake of the fish being influenced by hypoxia (Rummer et al. 2016). The test ended if either: (1) the fish was impinged on the downstream barrier (fatigue), or (2) the fish swam for 200 min, at which it is assumed the fish could swim at that speed indefinitely (Brett 1967). Fish were then removed from the chamber, euthanized in an ice bath, and subsequently weighed (blotted wet mass, g) and measured (total length, TL, mm). A final reading for microbial respiration was done for 10 min after the fish was removed from the respirometer. Text files were imported from the Firesting and analysed in LabChart (ver. 8, AD Instruments, New South Wales, Australia). The oxygen uptake rate ($\dot{M}O_2$) at each 20-min interval was calculated as:

$$\dot{M}O_2\left(\mathrm{mg}O_2 kg^{-1}h^{-1}\right) = S \cdot V_{\mathrm{resp}} M^{-}$$

where S is slope of the linear regression during the measurement period (mg O_2 s^{-1}), V_{resp} is the volume of the respirometer (minus the fish), and M is the mass of the fish (kg) (Rummer et al. 2016). Background respiration was subtracted from each value of $\dot{M}O_2$.

Statistical analyses

All statistical analyses were performed in *R* (ver 4.0.2). Data and residuals were checked for normality using Shapiro–Wilk's tests and diagnostic plots, and subsequently scale transformed for statistical analysis. We found there was quite a bit of instability in the oxygen uptake rates of individual fish within the first 100 min of the test, leading to convergence problems with more complex models (i.e., complex models could not be fitted). As such,

we considered the first 100 min as an adjustment period for the fish to habituate to the experimental setting and flow speeds, and therefore we only used data from 120 to 200 min in formal analyses. Linear mixed-effects models (lme4 package) were used to analyze differences in $\dot{M}O_2$ between treatments during the experiment. Oxygen uptake ($\dot{M}O_2$) acted as a response variable. The most complex model tested was a random slopes model using elapsed time during the experiment (min), habitat (live or degraded coral) and the interaction between the two as fixed effects, with Fish ID nested within Tank as random effects, because there were multiple measurements per individual fish and several different tanks. Model selection was based on lowest second-order Akaike information criterion (AIC) using the 'MuMIn' package (Table 1). The final model remained a random slopes model allowing the oxygen consumption of each fish to have a different relationship with time. Tank was removed as a random effect as it did not improve the model (Table 1), and the only fixed effect which improved the model was habitat. A one-way analysis of variance was used to determine differences in oxygen uptake rates between habitats, with the nature of differences further examined using Tukey's post-hoc tests ($\alpha = 0.05$).

Results and discussion

Coral reef degradation has been shown to alter community structure of reef fishes (Jones et al. 2004), and results from our study are the first to show coral degradation may also metabolically impact newly-settled reef fishes during activity. Oxygen uptake rates while fish were swimming at an ecologically relevant flow speed were, on average, 21% lower if fish were exposed to degraded coral (mean $\dot{M}O_2$ ± s.e. = 1314.9 ± 32.4 mg O_2 kg^{-1} h^{-1}) when compared to those exposed to live coral (mean $\dot{M}O_2$ ± s.e. = 1664.5 ± 81.4 mg O2 kg^{-1} h^{-1}; $p = 0.0391$; Fig. 1, Table 2). However, all fish were able to complete the 200-min endurance test, regardless of treatment. Considering the major physiological changes associated with rapid metamorphosis (e.g., Nilsson et al. 2007; Leis et al. 2011), a depression in oxygen uptake rates during this period may constrain available energy required to support processes characterising successful recruitment, such as swimming to find an ideal patch of reef, competing for a shelter, growth, and avoiding predators. Depressed oxygen uptake rates during exercise may be partly responsible for the high mortality rates of juvenile *P. amboinensis* when recruiting to degraded coral reefs, which has been observed in the field (Lönnstedt et al. 2013). This finding contradicted our initial hypothesis, in which we predicted oxygen uptake rates would increase, and this may be due to damage to the cardio-respiratory system from microbes and algae from degraded habitats.

Generally, acute exposure to chemical stressors causes immediate changes to an animal's physiology as a compensatory mechanism to cope with tissue damage and stress (Beyers et al. 1999). McCormick et al. (2017) found that dead-degraded coral reefs are populated with diatoms (e.g., *Pseudo-nitzschia sp.*), cyanobacteria (e.g., *Okeania sp.*), and algae (e.g., *Galaxauria robusta*) that inhibit the

Table 1 AIC scores from linear mixed effects models explaining the changes in oxygen uptake rates ($\dot{M}O_2$) during exercise of juvenile ambon damselfish (*Pomacentrus amboinensis*) exposed to degraded or healthy coral habitat for 24 h. Best model (i.e., the model with the lowest AIC score, and therefore used for statistical analysis) is in bold, and differences in AIC scores (ΔAIC) represents differences between the best model and the other candidate models. Description of variables given below table

Model	AIC	ΔAIC
Scale(MO2) ~ Habitat + (1|Tank/fish)	63.8	19
Scale(MO2) ~ Habitat + (Time|fish), control = lmer(optimizer = "bobyqa")	**44.8**	**0**
Scale(MO2) ~ Habitat + (Time|tank/fish), control = lmer(optimizer = "bobyqa")	50.2	5.4
Scale(MO2) ~ Habitat + Time + (1|Tank/Fish)	74.9	30.1
Scale(MO2) ~ Habitat + Time + (Time|tank/fish)	63.2	18.4
Scale(MO2) ~ Habitat*Time + (1|Tank/fish)	88	43.2
Scale(MO2) ~ Habitat*Time + (Time|tank/fish), control = lmer(optimizer = "bobyqa")	74.9	30.1
Scale(MO2) ~ Habitat*poly(Time, 2, raw = TRUE) + (Time|Tank/Fish), control = lmer(optimizer = "bobyqa")	114.9	70.1

MO2 = measure of oxygen uptake rate ($\dot{M}O_2$; mg O_2 kg^{-1} h^{-1})

Habitat = Experimental treatment; fish were exposed to either healthy or degraded coral for 24 h

Time = Elapsed experimental time. $\dot{M}O_2$ was measured in 20 min intervals over the 200 min experiment. However, due to convergence, we compare MO2$\dot{M}O_2$ in 20 min intervals from timepoint 120–200 min

Fish = Fish ID. Multiple measurements of $\dot{M}O_2$ (i.e., every 20 min) were made for each individual fish over the 200 min experiment

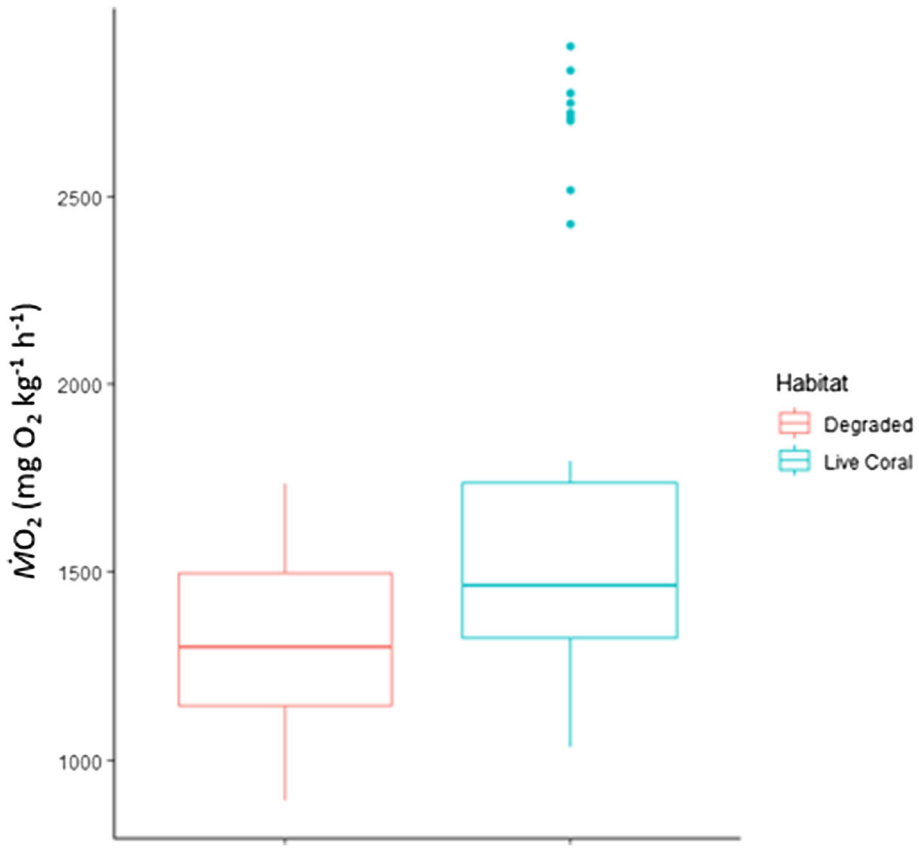

Fig. 1 Mean oxygen uptake rate ($\dot{M}O_2$; mg O2 kg^{-1} h^{-1}) measurements of juvenile Ambon damselfish (*Pomacentrus amboinensis*) swum at a constant speed (10 cm s^{-1}) over a 200-min endurance test (10 measures of $\dot{M}O_2$ per fish; ± s.e.), exposed to water from healthy (blue, $n = 10$) or dead-degraded (pink; $n = 10$) coral for 24 h. Boxplots represent 25–75% interquartile range

Table 2 One-way analysis of variance summary results of the best model explaining the changes in oxygen uptake rates ($\dot{M}O_2$) during exercise of juvenile ambon damselfish (*Pomacentrus amboinensis*) exposed to degraded or healthy coral habitat for 24 h

	Chi-sq	df	*p*
Habitat	4.26	1	0.0391

perception of chemical alarm cues in reef fishes. *Pseudo-nitzschia* and *Okeania* species produce harmful toxins and secondary metabolites that have potential negative influences on aquatic organisms (Trainer et al. 2012; Sneed et al. 2017). While direct impacts of these specific organisms on physiological systems of fishes are unknown (e.g., $\dot{M}O_2$), several studies have noted metabolic depression in fishes exposed to similar, chemically-active plankton. *Chattonella marina*, a eukaryotic alga responsible for mass fish deaths along the coasts of Japan and China, depresses arterial partial pressure of oxygen, lowers gill ventilation and heart rates, and causes direct damage to gill epithelia of yellowtail kingfish (*Seriola quinqueradiata*) (Ishimatsu et al. 1997; Hishida 1999; Lee et al. 2003). Similarly, *Prymnesium parvum*, a toxic algal species responsible for global fish kills, depresses routine oxygen uptake rates in European plaice (*Pleuronectes platessa*) (Bergsson et al. 2019) and SMR in rainbow trout (*Onchorynchus mykiss*) (Svendsen et al. 2018). These studies noted that the plankton directly damaged the gill structure that would influence cardio-respiratory performance. While not quantified in the current study, gill-related damage by planktonic organisms has the potential to be the mechanism underpinning decreases in $\dot{M}O_2$, as shown in fishes exposed to degraded habitats, and this would be an area of particular interest for future studies.

Our study demonstrated that exposure to degraded coral habitat directly impacts whole-animal physiology during exercise ($\dot{M}O_2$) for fish around the time of settlement. This pelagic-to-reef transition is a critical developmental milestone in reef fish life history and is characterised by a rapid change in metabolic and sensory physiology (McCormick 1993; Nilsson et al. 2007) and concomitantly high predation-related mortality (Almany and Webster 2006). Given the severe habitat degradation experienced by coral reefs, newly-settled reef fishes may have to physiologically adjust their energy budgets in order to swim in poor quality water. Exposing fishes for longer time periods (e.g., > 24 h) may elicit a more chronic metabolic response, as found for fish in other degraded habitats (e.g., Beyers et al. 1999; Goto and Wallace 2010). If the chemical components from

degraded water has long-lasting effects, this should tax a fish's energy budgets, which could result in less energy for growth and, in turn, detrimentally affect survival trajectories, as early-life mortality is growth-dependent (Hoey and McCormick 2004, Gagliano et al. 2007). Future studies should examine longer (i.e., weeks to months) exposure periods to establish whether reef fishes possess the capacity to allocate energy to tolerate the stresses of living in a degraded reef ecosystem.

The Great Barrier Reef and coral reefs worldwide are suffering from anthropogenic disturbances, and as a result are less resilient to stress and have a lower recovery potential (Dietzel et al 2020). This will have large-ripple effects on the species that call reefs home. The metabolic depression evidenced here may have lethal effects as suggested, but could also dramatically impact recruitment success of reef fishes, which would further decrease future population size, genetic variability, and overall resilience to habitat change.

Acknowledgements We would like to thank the staff at Lizard Island Research Station for technical support, students for helping to sort fish, W. Morris and R. Warburton for construction of the swim respirometer, I. Bouyoucos for advice on statistical analyses, and anonymous reviewers for helpful feedback.

Author contributions Conceptualization: ATD, MIM, DPC, MCOF; Experimentation: ATD, CMP; Data Analysis: RJ, ATD; Writing: ATD with substantial input from MIM, DPC, MCOF, RJ, CMP, JLR; Funding: MIM, JLR.

Declarations

Conflict of interest On behalf of all authors, the corresponding author deems no conflict of interest.

References

Almany GR, Webster MS (2006) The predation gauntlet: Early post-settlement mortality in reef fishes. Coral Reefs 25:19–22

Bergsson H, Andersen NR, Svendsen MBS, Hansen PJ, Steffensen JF (2019) Respiratory physiology of European plaice (Pleuronectes platessa) exposed to Prymnesium parvum. Fishes 4:11–19

Beyers DW, Rice JA, Clements WH, Henry CJ (1999) Estimating physiological cost of chemical exposure: Integrating energetics and stress to quantify toxic effects in fish. Can J Fish Aquat Sci 56:814–822

Brett JR (1967) Swimming Performance of Sockeye Salmon (Oncorhynchus nerka) in relation to Fatigue Time and Temperature. J Fish Res Board Canada 24:1731–1741

Dietzel A, Bode M, Connolly SR, Hughes TP (2020) Long-term shifts in the colony size structure of coral populations along the Great Barrier Reef. Proc Royal Soc B. https://doi.org/10.1098/rspb.2020.1432

Downie AT, Illing B, Faria AM, Rummer JL (2020) Swimming performance of marine fish larvae: review of a universal trait under ecological and environmental pressure. Rev Fish Biol Fish 30:93–108

Cortese D, Norin T, Beldade R, Crespel A, Killen SS, Mills SC (2020) Physiological and behavioural effects of anemone bleaching on symbiont anemonefish in the wild. Fun Ecol. https://doi.org/10.1111/1365-2435.13729

Ferrari MCO, McCormick MI, Allan BJM, Chivers DP (2017) Not equal in the face of habitat change: Closely related fishes differ in their ability to use predation-related information in degraded coral. Proc R Soc B Biol Sci. https://doi.org/10.1098/rspb.2016.2758

Fulton C, Bellwood D, Wainwright P (2005) Wave energy and swimming performance shape coral reef fish assemblages. Proc R Soc B Biol Sci 272:827–832

Gagliano M, McCormick MI, Meekan MG (2007) Survival against the odds: Ontogenetic changes in selective pressure mediate growth-mortality trade-offs in a marine fish. Proc R Soc B Biol Sci 274:1575–1582

Goto D, Wallace WG (2010) Bioenergetic responses of a benthic forage fish (fundulus heteroclitus) to habitat degradation and altered prey community in polluted salt marshes. Can J Fish Aquat Sci 67:1566–1584

Hishida Y (1999) Effect of environmental hyperoxia on respiration of yellowtail exposed to chattonella marina. Fish Sci 65:84–90

Hoey AS, McCormick MI (2004) Selective predation for low body condition at the larval-juvenile transition of a coral reef fish. Oecologia 139(1):23–9

Hughes TP, Anderson KD, Connolly SR, Heron SF, Kerry JT, Lough JM, Baird AH, Baum JK, Berumen ML, Bridge TC, Claar DC, Eakin CM, Gilmour JP, Graham NAJ, Harrison H, Hobbs JPA, Hoey AS, Hoogenboom M, Lowe RJ, McCulloch MT, Pandolfi JM, Pratchett M, Schoepf V, Torda G, Wilson SK (2018) Spatial and temporal patterns of mass bleaching of corals in the Anthropocene. Science 359:80–83

Hughes TP, Kerry JT, Baird AH, Connolly SR, Dietzel A, Eakin CM, Heron SF, Hoey AS, Hoogenboom MO, Liu G, McWilliam MJ, Pears RJ, Pratchett MS, Skirving WJ, Stella JS, Torda G (2018) Global warming transforms coral reef assemblages. Nature 556:492–496

Ishimatsu A, Maruta H, Oda T, Ozaki M (1997) A Comparison of Physiological Responses in Yellowtail to Fatal Environmental Hypoxia and Exposure to Chattonella marina. Fish Sci 63:557–562

Johansen JL (2014) Quantifying Water Flow within Aquatic Ecosystems Using Load Cell Sensors: A Profile of Currents Experienced by Coral Reef Organisms around Lizard Island, Great Barrier Reef. Australia Plos One 9:e83240

Jones GP, McCormick MI, Srinivasan M, Eagle JV (2004) Coral decline threatens fish biodiversity in marine reserves. Proc Natl Acad Sci U S A 101:8251–8253

Lee KS, Ishimatsu A, Sakaguchi H, Oda T (2003) Cardiac output during exposure to Chattonella marina and environmental hypoxia in yellowtail (Seriola quinqueradiata). Mar Biol 142:391–397

Leis JM, McCormick MI (2002) The biology, behavior, and ecology of the pelagic, larval stage of coral reef fishes. In: Sale PF (ed) Coral reef fishes: dynamics and diversity in a complex ecosystem. Elsevier, Amsterdam, pp 171–199

Leis JM, Hay AC, Gaither MR (2011) Swimming ability and its rapid decrease at settlement in wrasse larvae (Teleostei: Labridae). Mar Biol 158:1239–1246

Lönnstedt OM, Mccormick MI, Chivers DP (2013) Degraded Environments Alter Prey Risk Assessment. Ecol Evol 3:38–47

McCormick MI (1993) Development and changes at settlement in the barbel structure of the reef fish, Upeneus tragula (Mullidae). Environ Biol Fishes 37:269–282

McCormick MI, Moore JA, Munday PL (2010) Influence of habitat degradation on fish replenishment. Coral Reefs 29(3):537–546

McCormick MI, Barry RP, Allan BJM (2017) Algae associated with coral degradation affects risk assessment in coral reef fishes. Sci Rep 7:1–12

McCormick MI, Fakan EP, Palacios MM (2019) Habitat degradation and predators have independent trait-mediated effects on prey. Sci Rep. https://doi.org/10.1038/s41598-019-51798-2

McCormick MI, Weaver CJ (2012) It Pays to Be Pushy: Intracohort Interference Competition between Two Reef Fishes. PLoS ONE 7:e42590

McCormick MI, Makey L, Dufour V (2002) Comparative study of metamorphosis in tropical reef fishes. Mar Biol 141:841–853

Nilsson GE, Östlund-Nilsson S, Penfold R, Grutter AS (2007) From record performance to hypoxia tolerance: respiratory transition in damselfish larvae settling on a coral reef. Proc R Soc B Biol Sci 274:79–85

Norin T, Mills SC, Crespel A, Cortese D, Killen SS, Beldade R (2018) Anemone bleaching increases the metabolic demands of symbiont anemonefish. Proc R Soc B Biol Sci 285:20180282

Rummer JL, Binning SA, Roche DG, Johansen JL (2016) Methods matter: Considering locomotory mode and respirometry technique when estimating metabolic rates of fishes. Conserv Physiol. https://doi.org/10.1093/conphys/cow008

Rummer JL, Munday PL (2017) Climate change and the evolution of reef fishes: past and future. Fish Fish 18:22–39

Sneed JM, Meickle T, Engene N, Reed S, Gunasekera S, Paul VJ (2017) Bloom dynamics and chemical defenses of benthic cyanobacteria in the Indian River Lagoon, Florida. Harmful Algae 69:75–82

Svendsen MBS, Andersen NR, Hansen PJ, Steffensen JF (2018) Effects of harmful algal blooms on fish: Insights from Prymnesium parvum. Fishes 3:1–12

Trainer VL, Bates SS, Lundholm N, Thessen AE, Cochlan WP, Adams NG, Trick CG (2012) Pseudo-nitzschia physiological ecology, phylogeny, toxicity, monitoring and impacts on ecosystem health. Harmful Algae 14:271–300