

# Harmful effects of sediment-induced turbidity on juvenile fish in estuaries

M. L. Lowe<sup>1,2,\*</sup>, M. A. Morrison<sup>1</sup>, R. B. Taylor<sup>2</sup>

<sup>1</sup>National Institute of Water and Atmospheric Research, Private Bag 99940, Auckland, New Zealand

<sup>2</sup>Leigh Marine Laboratory, Institute of Marine Science, University of Auckland, PO Box 349, Warkworth 0941, New Zealand

**ABSTRACT:** Estuaries are important nursery habitats for juvenile fishes of many species, but are under increasing pressure from anthropogenic stressors. We examined the impacts of suspended sediments/turbidity on the foraging success and health of juvenile snapper *Pagrus auratus* F. Sparidae, which are abundant in many northern New Zealand estuaries and comprise a major coastal fishery as adults. In the laboratory, short-term exposure to turbidity (range <10 to 160 NTU) reduced foraging success of juvenile snapper, while month-long exposure caused higher rates of gill ventilation, gill deformation (epithelial hyperplasia, fusion of the lamellae), weight loss and mortality. In 7 northern New Zealand estuaries with varying catchment land uses, total suspended sediments were negatively correlated with capture rates of juvenile snapper and the condition of individuals, and positively correlated with rates of gill deformation and gill parasite loads. Fish diets changed from zooplankton to benthic prey as turbidity increased. Collectively, our results indicate that elevated turbidity levels have strong negative effects on the health and abundance of juvenile snapper. These turbidity impacts compound the negative effects of sediments on important biogenic fish nursery habitats such as subtidal seagrass beds, likely reducing subsequent recruitment into the fishable adult stock. Management of exploited fish species that have an estuarine life stage must consider the effects of both fishing and sediment runoff from the catchment.

**KEY WORDS:** Foraging success · Condition · New Zealand snapper · Nursery habitat · *Pagrus auratus* · Sedimentation · Suspended sediments · Water quality

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## INTRODUCTION

Estuaries comprise the first and often ultimate receiving environment for contaminants generated on the land, and are amongst the most degraded aquatic habitats worldwide (Syvitski et al. 2005). While high suspended sediment loads are a natural feature of many estuarine waters, the rate and extent of terrestrial sediment delivery has accelerated through human activities (Ellis et al. 2004, Thrush et al. 2004). Increased sedimentation into the coastal zone alters ecological functioning in numerous ways. Deposition of fine sediments (silt and clay) can abrade, clog and smother benthic organisms (Norkko et al. 2002,

Lohrer et al. 2004), while suspended sediments increase turbidity, reducing visibility and depth of the photic zone and leading to declines in primary producers with resultant impacts on both pelagic and benthic prey (e.g. Berry et al. 2003, Thrush et al. 2004, Morrison et al. 2009).

Estuaries provide crucial nursery habitats for many species of fish, including the juveniles of recreationally and commercially important species (e.g. Beck et al. 2001, Orth et al. 2006, Lowe 2013, Morrison et al. 2014). These small fish are potentially vulnerable to the effects of increased sedimentation because the biogenic habitats (e.g. seagrass meadows) that many rely on for food and shelter are themselves suscepti-

ble to sedimentation (Duarte 2002, Green & Short 2003, Grech et al. 2012). Suspended sediment/turbidity can also affect fish more directly, by reducing both reactive distance and foraging efficiency (e.g. Vinyard & O'Brien 1976, Rowe & Dean 1998, Manning 2013), particularly for planktonic prey (Wenger et al. 2012, Johansen & Jones 2013), and by damaging their gills, sometimes fatally (e.g. Sherk et al. 1975, Au et al. 2004, Wong et al. 2013). Suspended sediments can also modify other ecological processes including prolonging larval development and the ability to select habitat at settlement (e.g. Wenger et al. 2011, 2014).

Most of our knowledge of the effects of suspended sediments on fish is based on studies of (1) freshwater species (e.g. Newcombe & MacDonald 1991) rather than estuarine species, the latter of which may be accustomed to much higher sediment loads, (2) acute effects measured in the lab, rather than chronic effects as experienced by fish under natural field conditions (Au et al. 2004), and (3) fish >1 yr old, with limited study on larvae/juveniles, particularly for estuarine fishes (but see Partridge & Michael 2010). Early life stages are thought to be more susceptible than older individuals to elevated levels of suspended sediments (Sigler et al. 1984, Wilber & Clarke 2001), which is of particular concern given that events occurring in the first few months of life can strongly influence subsequent performance (Houde 1987, Sogard 1992, Francis 1994, Manderson et al. 2002, Sutherland & Meyer 2007).

We determined the effects of suspended sediments/turbidity on the health and foraging success of juvenile snapper *Pagrus auratus* F. Sparidae. Snapper form the basis of a major coastal fishery in warm temperate northern New Zealand (Francis 1994), and estuaries are an important nursery for juveniles, particularly when they contain biogenic habitats such as seagrass beds (Lowe 2013, Morrison et al. 2014, Parsons et al. 2014). New Zealand estuaries have catchments ranging from near pristine to heavily modified, with corresponding variations in estuarine sediment input and turbidity (Swales et al. 2002, Morrison et al. 2009), which potentially affects the value of these estuaries as nurseries. Short-term impacts of turbidity on snapper health and foraging success were measured by experimentally manipulating turbidity levels ranging from very low to those occurring in the field after heavy rainfall. To provide context for the results of these lab experiments, we also quantified the abundance, diet and health of juvenile snapper in 7 estuaries varying greatly in turbidity.

## MATERIALS AND METHODS

Lab experiments were conducted from May to September 2004 at the University of Auckland's Leigh Marine Laboratory. The field survey was done from May to July 2006.

### Laboratory experiments

Juvenile snapper *Pagrus auratus* of 55 to 90 mm fork length were captured from the Rangaunu and Mahurangi Harbours using beach seines and baited opera traps (Morrison et al. 2002, Morrison & Carbones 2006), respectively. Fish were initially held in 500 l flow-through tanks, and fed daily with diced mussels and shrimp pellets. After 2 wk, fish were transferred to the experimental tanks and fed mysid shrimps *Tenagomysis* sp. daily to acclimate to the experimental conditions.

Preliminary observations revealed that aggression increased when fish in a tank differed in length by >15 mm. To counteract this, fish were allocated to tanks so that their lengths within any given tank differed by no more than 10 mm. Tanks were then randomly assigned to treatments. The overall length range of fish in the short-term experiments on foraging success (run earlier in the season) ranged from 55 to 74 mm, with an average ( $\pm$ SE) length of  $66 \pm 2$  mm. For the long-term experiment on fish health (run later in the season), overall fish lengths ranged from 55 to 90 mm, with an average of  $72 \pm 2$  mm. There was no significant difference in average initial fish length between turbidity treatments for either the short ( $F = 1.35$ ,  $p = 0.269$ ) or long-term experiments ( $F = 1.35$ ,  $p = 0.289$ ).

Mysids were used for the feeding experiments as they are a common prey item for juvenile snapper (Usmar 2012). The mysids were collected from the nearby Whangateau Harbour, held in a flow-through glass aquarium ( $0.6 \times 0.35 \times 0.3$  m), and fed daily with frozen brine shrimp pellets and organic detritus off shells collected from the harbour.

Experiments were conducted in 15 rectangular plastic aquaria ( $0.5 \times 0.26 \times 0.28$  m) located in a building with a translucent roof, which allowed a natural photoperiod. Aquaria had 10 mm mesh lids to prevent fish escaping. All experiments were conducted during late afternoon (15:00 to 17:00 h) to standardise for diel activity patterns in snapper or mysids (e.g. Minello et al. 1987, Macia et al. 2003). In all experiments, there were 3 aquaria per turbidity level and 3 fish per aquarium. Each aquarium was a

closed system containing a submersible pump (Hi-tech 3500,  $3.6 \text{ l min}^{-1}$ ) and aerator.

A total of 5 turbidity levels were used in the experiments:  $\leq 10$  (control), 20, 40, 80 and 160 nephelometric turbidity units (NTU), encompassing the natural range experienced by juvenile snapper in the nearby Mahurangi Harbour over an 11 mo period (Oldman & Swales 1999). During the experiments, turbidity was measured with a Hach 2100 portable turbidity sensor. Due to natural variation in incoming ambient seawater, the control tank was defined as  $\leq 10$  NTU, as per Cyrus & Blaber (1987a,b). Turbidity was produced by means of fine surficial estuarine sediments ( $< 63 \mu\text{m}$ ) collected from the subtidal zone of the Mahurangi Estuary. Replicate samples of sediment were collected to encompass potential spatial variability in distributions of grain sizes. The sediment was initially mixed with surface seawater, and allowed to settle for 1 h. The container was then stirred and left for 6 min, before the top 10 cm of water and suspended sediment was siphoned off and poured through a  $63 \mu\text{m}$  sieve. This process was repeated until enough stock solution was obtained. Sediment was collected twice a week and kept in a well-aerated container. Turbidity levels were maintained in the tanks over time by the addition of varying amounts of stock solution to the ambient incoming seawater, until the intended turbidity levels (i.e. 20, 40, 80 or 160 NTU) were achieved. To maintain water depth, a matching volume of water was removed after the addition of stock solution. To avoid any substrate effects for the short- and long-term turbidity experiments, tank bottoms were left clear without any sediment cover.

The effect of turbidity on foraging success was determined as follows. Juvenile snapper were acclimated to experimental tanks for 2 h and starved for 24 h prior to each trial. The water pump was switched off 2 h prior to commencement of the short-term feeding experiments. At the start of the experiments, 60 mysids were released into the opposing end of each tank, and were separated from the fish by a Perspex divider for 30 min to acclimate, after which time the divider was removed and fish left to feed. After 30 min of feeding, fish were removed, the tank was completely drained through a  $250 \mu\text{m}$  sieve, and the remaining mysids counted. All mysids missing from the tanks were considered to have been eaten. Preliminary trials revealed that all shrimps were recovered from tanks containing no fish. Turbidity levels were re-measured at the conclusion of the feeding trials ( $\sim 3$  h after adding the sediment). There was a negligible change in the lower turbidity treatments

(20, 40 and 80 NTU), but turbidity in the 160 NTU treatments declined by up to 10% in some trials.

To determine the effect of suspended sediment/turbidity on health, fish were maintained in aquaria for 1 mo. Fish and treatments were randomly assigned to individual tanks. The weight and length of each fish were measured at the start and end of the experiment. Sediments were kept in suspension by circulating the water within each tank through 3 independently-tipping 200 ml cups mounted above the tank (adapted from Barr 2007). This provided intermittent turbulence to the bottom of the tank. Seawater was delivered via adjustable flow nozzles above the plastic cups. The cups were equipped with 13 mm plastic sleeves (to act as a bearings) inserted through a pivot point, slightly forward of the geometric center of gravity in the side of the cup. The 3 cups were positioned equidistantly, on a 10 mm galvanized pipe above the tank. This pipe acted as a pivot, allowing the cups to tip when full and then return to an upright position (see Fig. 3.3a in Lowe 2013).

Each day, a 1 l jug of water was scooped from each tank and poured back into it in order to re-suspend a small amount of sediment that settled along the rear wall of the tank where the turbulence from the tipping cups did not reach. Turbidity levels were measured each day from random locations within the tank and additional sediment from the stock solution was added as required to maintain turbidity levels within 20% of the target level. Approximately 25% of the tank water was changed daily to allow addition of stock solution and reduce waste build-up. Water and sediment were completely replaced every 7 d. Fish were removed and placed into holding tanks of ambient seawater for  $\sim 10$  min while tanks, pumps and connecting pipes were cleaned.

Initially, fish were placed into the experimental tanks for 48 h with ambient seawater. The tipping cups were then turned on and off over a period of 48 h to allow the fish to acclimate to the apparatus and ensure that they were feeding. Sediment was added gradually over 48 h for the higher NTU treatments. Tipping cups were switched off daily (between 15:00 and 17:00 h) for 20 min after fish were provided  $\sim 120$  live mysids  $\text{tank}^{-1}$ . Data on daily ambient seawater temperatures were taken from the Leigh Marine Laboratory's Climate Data Archives (data not shown). During the final week of the experiment, the ventilation rate of the fish (6 fish  $\text{treatment}^{-1}$ ) was assessed and expressed as the rate of gill operculum opening per 15 s. The short time frame was necessary to accommodate the reduced visibility of fish in the higher turbidity

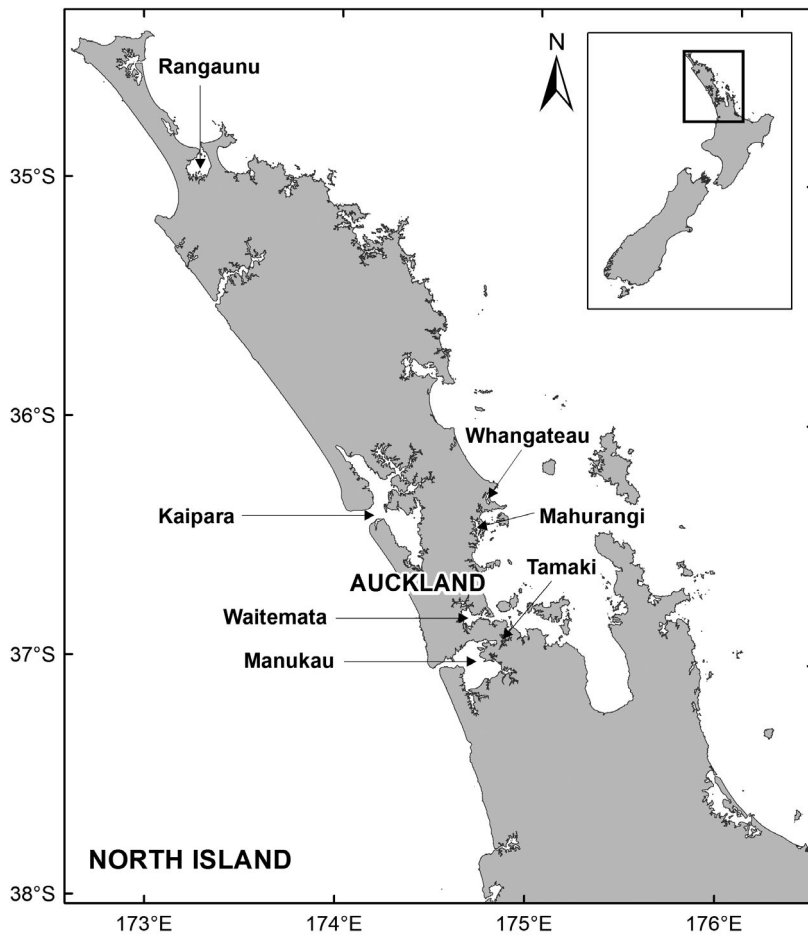


Fig. 1. Estuaries sampled for juvenile snapper *Pagrus auratus* in northern New Zealand

treatments moving away from the forward wall of the tank.

Upon termination of the trial, all surviving fish were euthanized by the iki-jime method (Robb & Kestin 2002), and the first gill arch on the right side of each fish was removed and immediately fixed in Bouin's fluid for 24 h, then transferred to 70% ethanol. Samples were then dehydrated in graded ethanol concentrations and embedded in paraffin wax. Sagittal sections (4 to 7  $\mu\text{m}$  thick) were cut and mounted on glass slides. Sections were de-paraffinized in xylene, hydrated in ethanol and stained with hematoxylin-eosin. The gill epithelia were examined for evidence of any pathological changes that may have compromised respiratory function. This included epithelial hyperplasia of the pillar system (increased proliferation of cells, particularly at the base and tips of the lamellae), fusion of the secondary lamellae and the presence of parasites. Counts of the number of affected lamellae were quantified along 3

randomly chosen entire gill filaments. Parasites were also identified and counted on the 3 randomly selected gill filaments. Where fusion of lamellae had occurred, the number of individual lamellae that were joined together was counted as in Tricklebank (1997).

### Field survey

We attempted to collect at least 10 juvenile snapper (fork length 50 to 100 mm) from each of 7 northern North Island estuaries covering a spectrum of environmental degradation (sedimentation and associated water turbidity; Fig. 1). Fish were caught using opera traps, which were baited with pilchards and set over sandy mud between 14:00 and 18:00 h NZST on an incoming tide. Fish were euthanized by the iki-jime method and the first right gill arch removed and processed as described for the laboratory experiments. Fish were then placed into an ice slurry for transportation back to the lab.

At each site, a 0.25 m diameter black-and-white Secchi disc was used to measure water clarity. A Horiba U10 multi probe was used to measure turbidity (in NTUs), temperature, salinity, pH and dissolved oxygen at ~1 m above the sea

floor. Absence of a power source in the field precluded using the more accurate Hach Turbidimeter for measurements of turbidity as in the laboratory experiments. To quantify the concentration of total suspended sediments (TSS), a single 1 l water sample was collected from ~1 m above the seafloor at each estuary, using a Van Dorn sampler. Water samples were later filtered through acid-washed, dried and pre-weighed 0.45  $\mu\text{m}$  polycarbonate membranes using plastic, acid-washed, vacuum filtration equipment. After filtration, the membrane was dried to a constant weight at 60°C and re-weighed.

To quantify among-estuary differences in fish growth and nutritional condition, a condition index (CI) was calculated. In the laboratory, the fork length of each snapper was measured ( $\pm 1$  mm) and its total wet weight determined ( $\pm 0.001$  g). The liver and digestive system (i.e. stomach and intestine) were removed and weighed separately, and subtracted from the total weight to yield a carcass weight (CW). A rel-

ative CI was calculated for each individual as per Francis (1997), as the measured CW divided by the expected carcass weight (ECW) for an 'average' fish of the same length. The ECW was generated using the power curve  $ECW = aLength^b$ , where  $a$  and  $b$  were estimated from a linear regression of  $\log_{10}CW$  on  $\log_{10}Length$  for fish from all sites. Sites were pooled after initial testing to ensure they had homogeneous regression slopes (Francis 1997).

Foreguts were then preserved in 10% formalin and the contents identified to the lowest possible taxonomic level under a dissecting microscope. To estimate biomass, animals were allocated to log size-classes by eye using a graticule in the microscope and a reference collection consisting of a mixture of species retained by different-sized sieve meshes following Edgar (1994). The equation of Edgar (1990) was then used to estimate individual body mass from sieve mesh size. Ash-free dry weight values utilized for estimation of plankton biomass were calculated from Newcombe (2009). Prey were classified as benthic (amphipods, shrimps, isopods, decapods, bivalves, polychaetes and mysids) or pelagic (zooplankton, gastropod veligers) and biomasses were summed across these 2 categories. Mysids were considered benthic in estuaries with TSS/turbidity values above  $20 \text{ mg l}^{-1}$  or 10 NTU respectively, due to their propensity to school within clear/shallow waters and move independently close to the seafloor in turbid waters (Mauchline 1980).

### Analysis

Ordinary least squares regression was used to model fish health, foraging success and abundance as a function of turbidity (Cottingham et al. 2005), using Sigma Plot 11 run on treatment or estuary means. Results were considered significant at  $p < 0.05$ . Fish that died during the experiments were excluded from analyses.

## RESULTS

### Laboratory experiments

The proportion of mysids eaten by juvenile snapper during feeding trials decreased significantly as turbidity increased ( $p < 0.002$ ), with only 8% of mysids consumed in aquaria with turbidities equating to storm conditions (160 NTU) compared to 77% consumed in the control ( $\leq 10$  NTU; Fig. 2A).

During the month-long experiment, fish maintained at all turbidity levels lost weight on average, but the weight loss was significantly higher at turbidities  $\geq 40$  NTU ( $p = 0.02$ ; Fig. 2B). Weight loss for the  $\leq 10$  and 20 NTU treatments averaged ~7%, doubling to ~14% for the higher treatments.

Gills of healthy fish typically have equally spaced secondary lamellae, and intact cellular layers with no signs of fusion between the lamellae (Tricklebank 1997). In this experiment, clear histopathological changes in gill epithelia occurred with increasing turbidity levels (Figs. 2C–E & 3). The occurrence of epithelial hyperplasia (causing dilation of the lamellae) increased significantly from 20 filament<sup>-1</sup> in the control ( $\leq 10$  NTU) to a peak of 60 at 80 NTU ( $p = 0.01$ ; Fig. 2C). Similarly, the incidence of lamellar fusion increased significantly as turbidity increased, from 10 filament<sup>-1</sup> at  $\leq 10$  NTU to 27 filament<sup>-1</sup> at 160 NTU ( $p = 0.003$ ; Fig. 2D). However, there was no evidence of mechanical abrasion or lodging of sediments into gill epithelia of the lamellae (M. L. Lowe pers. obs.). Epitheliocystis, a bacterial condition affecting the gills of fish, also increased significantly with higher turbidities ( $p = 0.02$ ; Fig. 2E). In total, 88% of the fish in the 160 NTU tanks and 67% of fish in the 80 NTU tanks developed lesions, but only 0.25% in the 20 NTU treatment. No infection was present in the control ( $\leq 10$  NTU). Similarly, rates of infection per gill filament ranged from a peak of  $3.0 \pm 1.18 \text{ fish}^{-1}$  for 160 NTU to  $0.25 \pm 0.16 \text{ fish}^{-1}$  for the 20 NTU treatments.

Quantitative behavioural observations of fish at the higher turbidity levels (80 and 160 NTU) were difficult due to poor visibility. However, the behaviour of the fish that could be observed revealed increased gill flaring, coughing and gulping at the surface along with decreased activity levels, particularly for the 80 and 160 NTU treatments. Fish in these treatments tended to settle on the bottom, were lethargic and interacted little with other fish. Increased ventilation rates occurred at the higher turbidity levels, although marginally insignificant ( $p = 0.07$ ; Fig. 2F). These ranged from  $80 \pm 5.98 \text{ min}^{-1}$  for  $\leq 10$  NTU to  $110 \pm 1.36 \text{ min}^{-1}$  at 160 NTU.

Only 5 fish died during the experiment. In the lower turbidity treatments ( $\leq 10$  and 20 NTU), 2 died within the first week of the trial, associated with aggressive attacks by other individuals. In the higher turbidity treatments (80 and 160 NTU), 3 died in the final days of the experiment. These fish were extremely thin and lethargic, having lost ~25% body weight, and were very pale with evidence of fin rot from fungal infections.



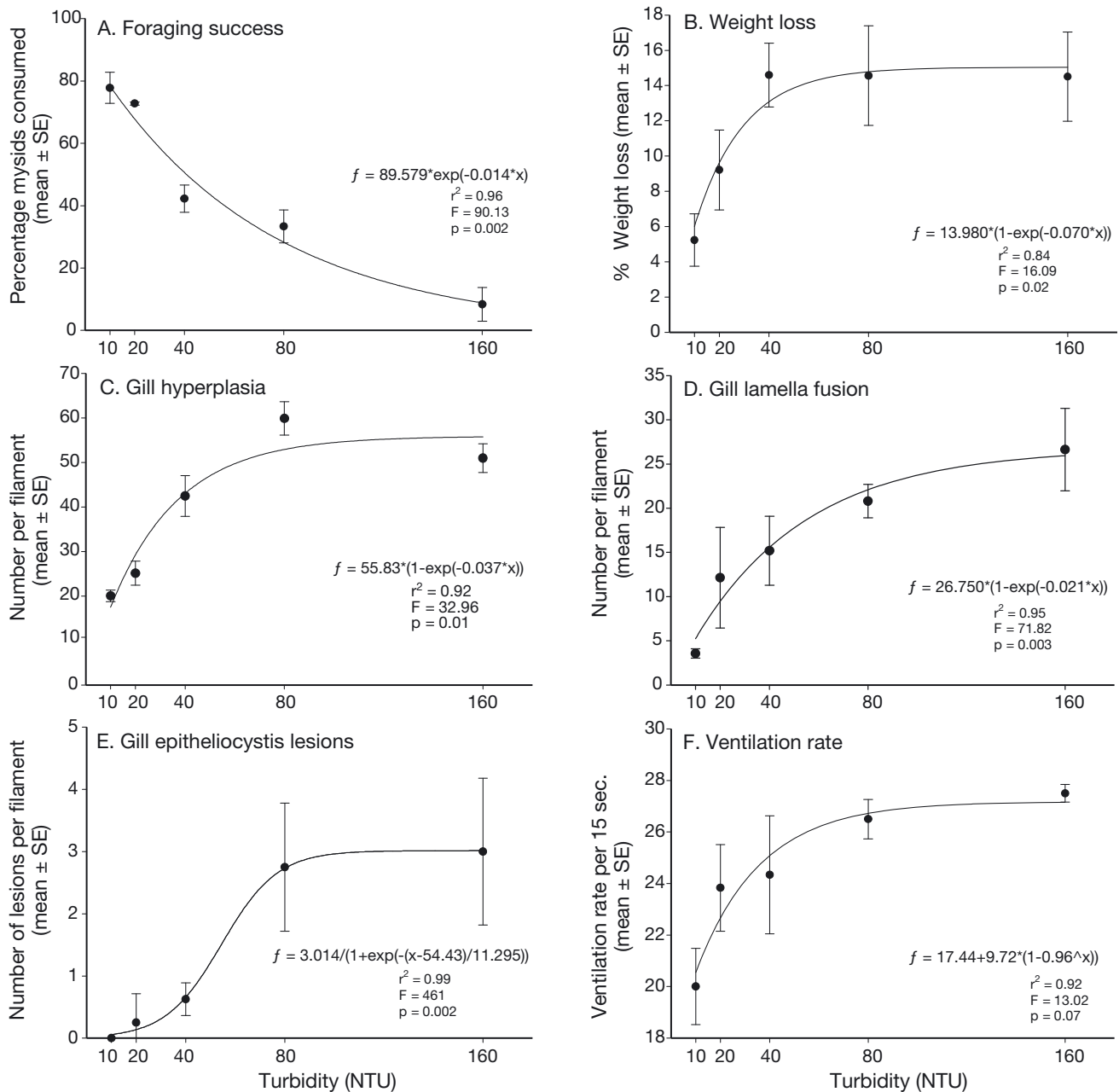


Fig. 2. Effects of turbidity on juvenile snapper *Pagrus auratus*, in experiments lasting (A) 30 min and (B–F) 30 d

### Field survey

The 7 surveyed estuaries had TSS/turbidities ranging from 4 mg l<sup>-1</sup> (~1 to 2 NTU) in the Rangaunu estuary, a pristine isolated area with a relatively undisturbed catchment, to 37 mg l<sup>-1</sup> (17 NTU) in the Waitemata, which has a highly modified catchment that includes a large part of New Zealand's largest city Auckland (Fig. 4). Measures of turbidity (i.e. NTU), Secchi depth and TSS levels were highly cor-

related (TSS–NTU:  $r^2 = 0.93$ ; TSS–Secchi:  $r^2 = 0.96$ ; NTU–Secchi:  $r^2 = 0.86$ ), so TSS values are presented along with NTU values to facilitate comparison with the experimental results. Other measured environmental variables differed little among estuaries, with temperature ranging from 14 to 17°C, pH from 7.9 to 8.3, salinity from 29.4 to 35.6‰ and dissolved oxygen from 7.5 to 10.5 mg l<sup>-1</sup>.

The capture rate of juveniles decreased significantly as TSS/turbidity increased ( $p = 0.002$ ; Fig. 4A),

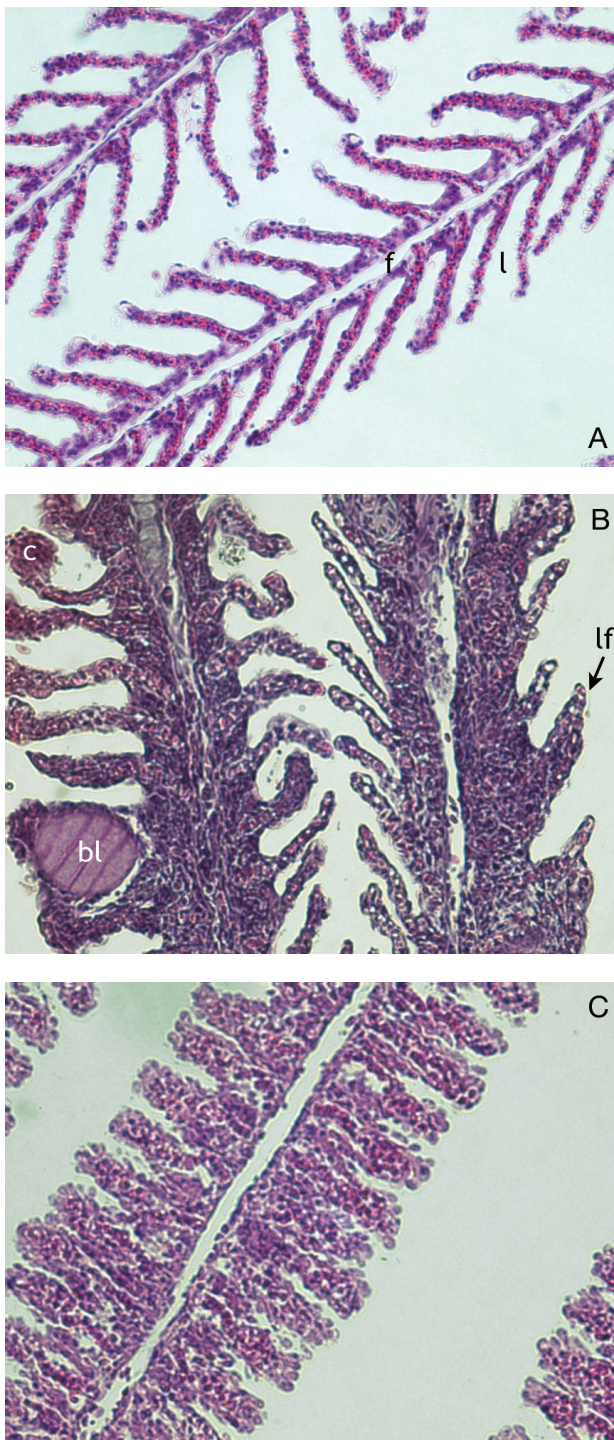


Fig. 3. Gills of juvenile snapper *Pagrus auratus* subject to different turbidities. (A) Normal gill filament (f) in fish from low-turbidity Rangaunu Harbour. l: lamellae. (B) Hyperplasia (cell proliferation) of gill filaments especially at the base and tips (clubbing; c) of the lamellae, and lamellar fusion (lf) in fish maintained in highly turbid (160 NTU) water in an aquarium for 30 d. Note also the bacterial lesion (bl). (C) Hypertrophy (thickening) and shortening of a gill filament in fish from the moderately turbid Manukau Harbour

with only 3 individuals collected from the most turbid estuary, Waitemata Harbour. The average length of juvenile snapper was not significantly correlated with turbidity ( $r^2 = 0.04$ ; data not shown). A significant negative relationship was found between the CI of fish and increasing TSS/turbidity ( $p = 0.01$ ), with fish from Rangaunu Harbour weighing 20% more for their length than fish from the highly turbid Waitemata Harbour (Fig. 4B). The rate of gill epithelial hyperplasia (causing dilation of the lamellae) was lowest in fish from the 2 least turbid estuaries (12 to 20 filament<sup>-1</sup>) and was significantly higher in the 5 more turbid estuaries (30 to 45 filament<sup>-1</sup>) ( $p = 0.03$ ; Fig. 4C). Similarly, the rate of lamellar fusion per filament increased significantly with TSS/turbidity, from 1.4 filament<sup>-1</sup> in the least turbid estuary to 23.7 in the most turbid ( $p = 0.03$ ; Fig. 4D). Other histopathological lesions included hypertrophy (swelling) and shortening of the lamellae (Fig. 3). The occurrence of affected lamellae was proportionately greater in the southern harbours, particularly for Mahurangi (85%) and Manukau (80%). Conversely, this condition was largely absent from the northernmost harbours, Rangaunu (0%) and Whangateau (2%). Between these, Kaipara recorded 20% and Waitemata and Tamaki recorded 30% respectively. Lesions caused by epitheliocystis, a bacterial condition affecting fish gills, were present on the gills of fish from 4 sites, but the abundance of lesions showed no clear relationship with turbidity ( $p = 0.14$ ; Fig. 4E). The Manukau had the highest proportion of affected fish (67%), followed by Mahurangi (38%), Waitemata (33%) and Tamaki (14%).

Mysid and caridean shrimps, and copepods dominated the diets of fish in all 7 estuaries. The relative contribution of the 2 prey categories (benthic, pelagic) varied significantly with increasing suspended sediments ( $p = 0.01$ ; Fig. 4F). In estuaries with relatively clear water (Rangaunu, Mahurangi, Whangateau, Tamaki), pelagic prey dominated (98% total numbers), especially calanoid copepods such as *Paracalanus indicus* and the cladoceran *Penilia avirostris*, while fish in the more turbid estuaries (Manukau, Kaipara, Waitemata) exclusively ate benthic prey comprising mysid and caridean shrimps, including *Tenagomysis* sp. and juvenile *Palaemon affinis*. Modest numbers of benthic copepods *Hemicyclops* sp. and amphipods were also consumed. In addition, the average size of prey items increased with elevated turbidity levels: 91% of the pelagic prey was  $\leq 0.71$  mm, while  $\sim 70\%$  of benthic prey items consumed were  $\geq 0.71$  mm.

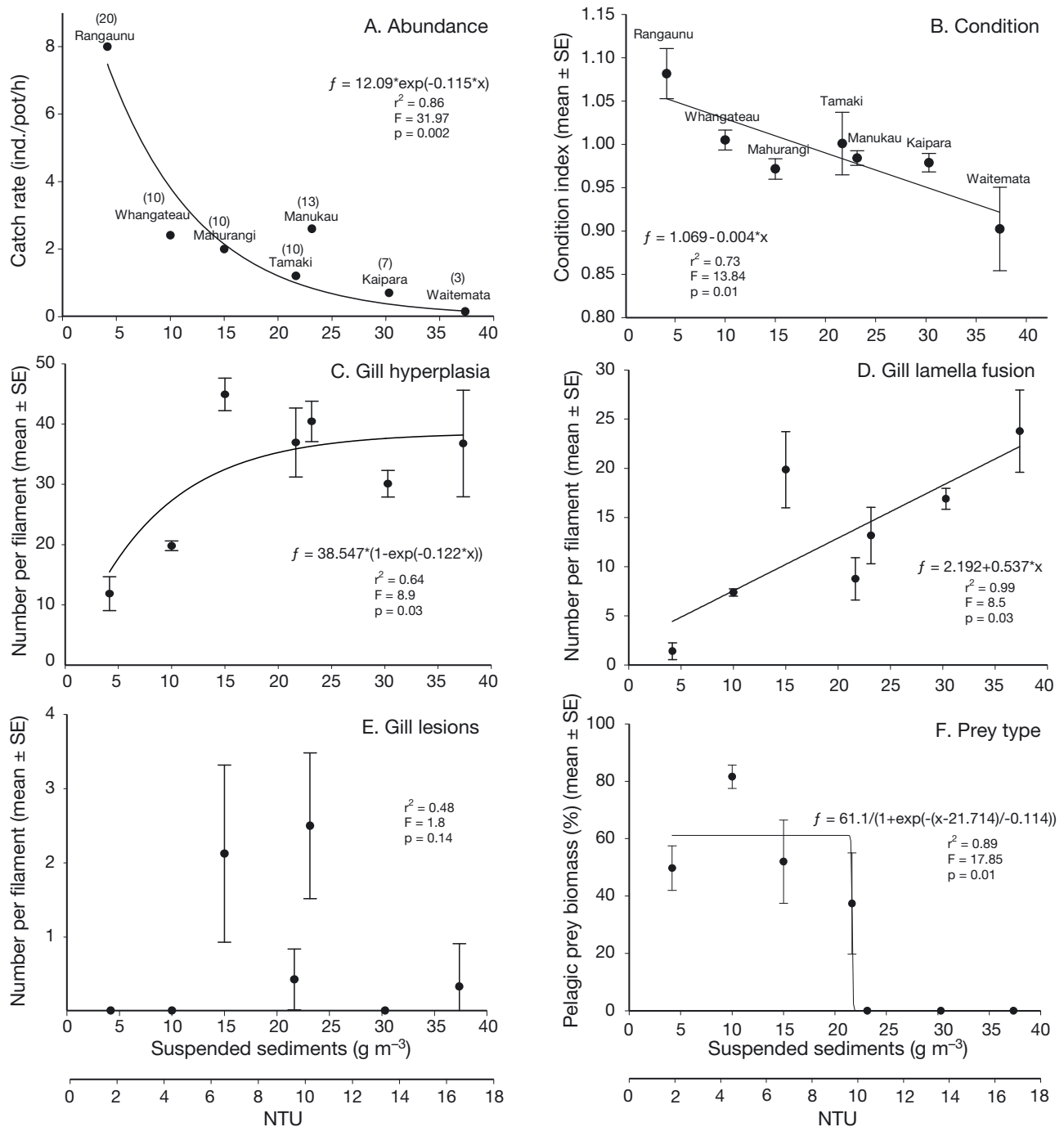


Fig. 4. Relationships between the turbidity of 7 estuaries in northern New Zealand and the abundance, health, and diet of juvenile snapper *Pagrus auratus*. Numbers in parentheses in (A) represent sample sizes. Data are plotted against measured total suspended sediment (TSS) values, which provide higher accuracy. Nephelometric turbidity unit (NTU) values are plotted as an extra axis to facilitate comparison to experimental results, based on the strong correlation between NTU and TSS from the field data ( $r^2 = 0.93$ )

## DISCUSSION

This study clearly demonstrates the impacts of increasing sedimentation/turbidity on juvenile snapper, with foraging success declining markedly following

exposure to short-term (30 min) turbidity pulses. Chronic exposure (30 d) at turbidity levels equating to storm conditions caused acute effects on fish growth and health, including increased weight loss, mortality, presence of gill lesions and behaviours associated



with hypoxia (i.e. gulping at the surface, lethargy and increased ventilation). A survey of 7 northern estuaries revealed a decline in abundance and health (i.e. significantly lower CI and higher incidence of gill lesions) in the more impacted estuaries characterized by increasing sedimentation, lower water clarities and increasing urbanisation. This is consistent with direct, chronic effects of sediments on fish.

### Impacts of turbidity on feeding

Turbid water can reduce the feeding ability of visual predators such as juvenile fish, by reducing both reactive distance and foraging efficiency (Kerr 1995, Bash et al. 2001). The results from our survey are consistent with prior research (e.g. Vinyard & O'Brien 1976, Rowe & Dean 1998, Wenger et al. 2012, Manning 2013), with foraging success of juvenile snapper on mysids declining by 44 to 89% under experimental exposure to short-term pulses of turbidity  $\geq 40$  NTU. Even greater reductions in foraging success could be expected if our experiment had used smaller, highly mobile planktonic prey, which are a major dietary item for newly-settled snapper (Usmar 2009, Johansen & Jones 2013). This may be of particular concern given the increasing frequency of sudden downpours due to climate change, and the short retention times of flood waters in catchments (Willis et al. 2007).

In the field, the diet of juvenile snapper changed abruptly from highly mobile pelagic prey (zooplankton) to larger, slower-moving benthic prey (mysids, shrimps and amphipods) when TSS/turbidity exceeded  $23 \text{ mg l}^{-1}$  ( $\sim 12$  to  $17$  NTU). The extent to which this diet difference was due to differences in prey abundance or to turbidity levels in the estuaries is not known since prey densities were not sampled concurrently. Simultaneous spatiotemporal assessments of prey abundance merits further study. However, assuming both prey types were present at all field sites, this suggests a change in foraging tactics — which has been documented in other studies, often with a 'turbidity' threshold of around 40 NTU, above which declining vision results in more opportunistic, ambush-type predation (Hecht & van der Lingen 1992, Macia et al. 2003, De Robertis et al. 2003, Helenius et al. 2013). Strategy shifts of this nature are likely cost-effective given the higher energetic costs associated with increased foraging time, attendant declines in attack success (Johansen & Jones 2013), and increased exposure to predation (Meager et al. 2005, Engström-Öst & Mattila 2008).

A forced shift from small zooplankton to large benthic prey is likely to be detrimental to juvenile snapper. Newly-settled fish are limited in their ability to eat larger prey due to their small gape size and limited ability to digest larger prey because of their poorly differentiated guts (Morrison 1990, Gillanders 1997, Sudo & Azeta 2001). Juvenile fish prefer smaller, easily digestible prey such as copepods (Mills et al. 1984, Lankford & Targett 1997, Gning et al. 2010) because of their high caloric and protein content, which facilitates rapid growth to minimize size-dependent predation risk (Volk et al. 1984). However, increased TSS can also negatively affect the abundance, nutritional value and composition of zooplankton and other prey species (Forbes et al. 1981, David et al. 2005). Thus, increasing TSS/turbidity may reduce the condition and growth of juvenile snapper by reducing their ability to visually choose zooplankton at optimal prey sizes, and/or by reducing zooplankton densities. Accordingly, both the magnitude and timing of sediment pulses may have profound and long-term effects on survival and recruitment of larval or juvenile fish, particularly where diet is restricted due to ontogeny (Partridge & Michael 2010, Murphy et al. 2012, Manning 2013, Wenger et al. 2014).

### Impacts of turbidity on health

There are significant thresholds of susceptibility to suspended sediment/turbidity in many sensitive species and life stages (e.g. Sigler et al. 1984, Wilber & Clarke 2001, Partridge & Michael 2010). The results from this study have revealed a more gradual response to increased TSS/turbidity, with significantly higher rates of sublethal stress above 40 NTU for chronic exposure (30 d) in the laboratory. In the lab experiments, weight loss reached a threshold response ( $\sim 14\%$ ) at 40 NTU, levelling off for the 80 and 160 NTU treatments. Nominal weight losses recorded for the control group ( $\leq 10$  NTU) could be due to stress from confinement and handling and/or ambient sea surface temperatures being the lowest on record for 38 yr during this trial, which was conducted during autumn and winter (Leigh Marine Laboratory's Climate Data Archives, data not shown). Growth rates for 0+ age juvenile snapper are correlated with temperature, and slow dramatically over the first winter (Francis 1994). Our results are consistent with prior research of freshwater fish showing reduced growth rates, with escalation of ill effects following chronic exposure to increasing sus-

pended sediments (e.g. Sherk et al. 1974, 1975, Newcombe & Jensen 1996, Manning 2013), and suggest that suspended sediment concentration alone is a poor indicator. Rather, effective predictive modelling requires both concentration and exposure duration (Newcombe & MacDonald 1991).

Juvenile snapper showed a progressive increase in the incidence of damage to gill lamellae with increasing turbidity in long-term exposure trials. Gill pathological symptoms observed included epithelial hyperplasia (thickening) of the pillar system and fusion of the secondary lamellae. These changes represent sublethal rather than acute effects, and can be considered a defence mechanism by increasing the distance across which irritants must diffuse to reach the bloodstream (Mallat 1985). However, cell proliferation of epithelial tissue, and eventual loss of surface by clubbing and fusing of lamellae can impair respiration and ammonia excretion, leading to respiratory stress and ammonia intoxication (Goldes et al. 1988, Kerr 1995, Bergstedt & Bergersen 1997, Au et al. 2004). Bacterial infection (i.e. epitheliocystis) of the gills was also recorded. Epitheliocystis affects the epithelial and chloride cells of gills and cause hypertrophy (swelling of individual cells), which can result in respiratory distress (Meijer et al. 2006). The increase in ventilation rates, increased gill flaring, coughing and gulping at the surface, in addition to reduced activity levels recorded in the higher turbidity treatments, are all indicative of anoxia, which is consistent with published information (Berg & Northcote 1985, McLeay et al. 1987, Servizi & Martens 1992). Paler coloration, fin rot and early mortality observed in 17% of fish from the 80 and 160 NTU treatments agrees with prior research, which collectively suggests that exposure to elevated sediment levels decreases tolerance rates to disease and time to death (e.g. Appleby & Scarrett 1989, Redding et al. 1987, see review in Newcombe & Jensen 1996).

However, with laboratory-based studies, experimental artefacts are unavoidable since factors such as prey availability and intra- and interspecific encounters are controlled (Bash et al. 2001). In addition, spatial and temporal factors such as distribution, abundance or availability of suitable habitat, time of year, frequency, duration and magnitude of prior storm events (with associated increase in current velocities) may be more deleterious in the field (Bruton 1985). In these experiments, fish were exposed to sediment unlikely to be contaminated. However, in coastal environments suspended sediments frequently serve as a sink for contaminants (Hack et al. 2007). These chemicals can cause sub-

lethal stress and, in combination with suspended sediments, may have important interactive effects on marine fishes (Au et al. 2004). Therefore laboratory results may be conservative in their estimation of suspended sediment impacts under field conditions.

In sum, these experiments demonstrate that although turbidity levels in the range investigated here (10 to 160 NTU) are unlikely to cause immediate high mortality in juvenile snapper, prolonged exposure to turbidities  $\geq 40$  NTU can result in adverse growth and developmental effects from (1) reduced prey capture success due to impaired vision and/or (b) increased metabolic costs from physiological stress (e.g. respiratory distress/disease). Hence, if exposure continues in the long-term, reduced energy acquisition could be reflected by lower CIs of fish from turbid estuaries relative to those with higher water clarity, and/or by reduced fish abundance in highly turbid estuaries (Hecht & van der Lingen 1992, Au et al. 2004).

Our field survey of 7 northern estuaries similarly revealed significant differences among the biological variables measured for juvenile snapper, particularly for TSS above  $23 \text{ mg l}^{-1}$  ( $\sim 12$  to 17 NTU). The onset of negative health impacts occurred at lower levels in the field than in the laboratory trials (i.e.  $\geq 40$  NTU in the lab). However, given the variety of environmental parameters fish are exposed to in the field (e.g. particle size distribution and angularity, pollutants, prey availability and cumulative storm events) it is not unexpected to find substantial variation between the two (Appleby & Scarrett 1989).

Juvenile snapper had significantly lower CIs (an effective proxy for growth rate and nutritional status) in the more impacted estuaries characterized by increasing sedimentation, concomitant with lower water clarities and increasing urbanisation (e.g. Waitemata, Manukau), whilst fish from Rangaunu Harbour, the most 'pristine' estuary, had the highest average CIs.

Higher levels of gill deformation (hyperplasia/fusion) and parasite loads were also recorded, particularly for Manukau and Mahurangi Harbours. Moreover, observed gill lesions also included shortening and hypertrophy (swelling of epithelial cells) of secondary lamellae, which showed close similarity to lesions brought about by elevated levels of other environmental pollutants such as zinc (Bhagwant & Elahee 2002), nickel (Al-Attar 2007), nitrogen (Schlacher et al. 2007) and phosphate (Omeregie et al. 2009). The markedly elevated histopathological alterations recorded for both the Manukau (80%) and Mahurangi (85%) harbours may well be a result of other environmental pollutants acting alone or

synergistically with TSS. Manukau Harbour, located next to New Zealand's largest city, has historically had high levels of copper (Cu), lead (Pb), zinc (Zn) and polycyclic aromatic hydrocarbons (PAHs) in its sediments (Hack et al. 2007, Mutoro 2001), whilst both harbours have elevated nitrate and phosphate levels (Scarsbrook 2008). However, determining cause and effect relationships between specific chemicals and their effects on juvenile fish is particularly difficult given the myriad of contaminants and their potential synergisms (Tricklebank 1997 and references therein, Wong et al. 2013). In addition, higher levels of both TSS and pollutants have been shown to predispose fish to opportunistic infections (Redding et al. 1987, Goldes et al. 1988). Nowak & LaPatra (2006) found more frequent and severe epitheliocystis in fish exposed to sewage. This agrees with findings in this study, with the highest rates of infection being recorded for the Manukau and Mahurangi Harbours respectively.

Whether the gills of estuarine juvenile fish can recover from these histopathological changes is unknown. However, studies on freshwater species (e.g. Fukuda 1983, Goldes et al. 1988) showed virtual complete recovery from severe reactive hyperplasia in less than 1 mo when the stimulus was removed and adequate water quality was available. Nonetheless, for estuarine fish, exposure to elevated levels of TSS can occur for extended periods during frequent storms, and the effects could be cumulative with ongoing sediment pulses. Our results suggest that physiological stress in fishes in response to increased TSS/turbidity can decrease immunological competence and growth, and agree with prior studies demonstrating negative relationships between fish condition and water clarity in the field (e.g. Amara et al. 2007, Courrat et al. 2009, Zingel & Paaver 2010).

## CONCLUSIONS

This is the first study to demonstrate that increased suspended sediment levels in temperate estuarine fish nurseries can have direct negative effects on individual fish. Lower growth and nutritional status of juvenile snapper may potentially lead to increased vulnerability to predation, physiological stress and disease, in addition to lower overwinter survival and subsequent recruitment to commercially exploitable stocks (Francis 1994, Adams et al. 2003, Amara et al. 2007, Manning et al. 2014). Further research on the effects of TSS/turbidity on larvae and juveniles <50 mm fork length, and tracking the development

of fish from a known range of sedimented estuaries utilizing otolith chemistry to estimate daily/annual growth as fish recruit to offshore fisheries would enhance our understanding of these processes to better inform managers of acceptable NTU thresholds.

The findings from this study support the increasing levels of concern being shown over escalating diffuse source inputs of suspended sediments into rivers, estuaries and the coastal zone. The direct effects of suspended sediments on fish health, physiology and behaviour, as well as the associated loss of seagrasses and other organisms that form important nursery habitats, along with reduced numbers of available prey for juvenile fish, collectively pose a significant and increasing threat to estuarine and coastal fish populations. This highlights the need for management to encompass both marine environments and terrestrial catchments for the effective protection and sustainability of juvenile fish nurseries.

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