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## Assessing barotrauma among angled snapper (*Pagrus auratus*) and the utility of release methods

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#### ABSTRACT

Australian recreational and commercial fishers catch snapper (Pagrus auratus) from down to  $\sim$ 120 m, often evoking barotrauma. Owing to minimum legal sizes and quotas or non-consumptive fishing practices, some afflicted fish are released, raising concerns over the potential for unaccounted fishing mortality. Two experiments were completed to quantify the (1) clinical signs of barotrauma among fish angled from 6 to 120 m (experiment 1) and (2) mortality (over 3 d) of fish with barotrauma that were released following either no treatment, venting (e.g. needle) or recompression (e.g. release weight) (experiment 2). In experiment 1, barotrauma was evident in some fish angled from 11 m and all from >20 m. Fish were considered to have barotrauma if they had a prolapsed cloaca combined with a distended coelomic cavity and/or gastric herniation into the buccal cavity. Despite similar clinical signs among fish in experiment 2, none died, however, the associated trauma raises welfare concerns. Both recompression and venting offered benefits over no treatment by returning fish to depth quickly and releasing gases, respectively. Nevertheless, the release method should be dictated by the clinical signs of barotrauma and extrinsic conditions, including the competency of the fisher (e.g. for venting), presence of predators (e.g. for recompression), and exposure to sun or warm water (e.g. no treatment). More selective gears, spatial and temporal closures, or possibly replacing minimum legal sizes with quotas in deep water only could also reduce the frequency of barotrauma among released P. auratus and therefore, negative impacts on stocks.

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#### 1. Introduction

Snapper (*Pagrus auratus*) is widely distributed throughout the Indo-Pacific (Paulin, 1990). In the southern hemisphere, individuals mostly occur in temperate and subtropical coastal waters off New Zealand and southern Australia; with the latter populations in depths <200 m across their distribution from Hinchinbrook Island in Queensland (QLD) to Barrow Island in Western Australia (Kailola et al., 1993).

The Australian stocks of *P. auratus* are extensively exploited by both recreational and commercial fishers, typically to depths <120 m. Following a peak in exploitation late last century, commercial landings (primarily from traps and hook-and-line) have declined in most states, and stocks in QLD and New South Wales (NSW) are considered to be experiencing growth-overfishing (Kailola et al., 1993; Allen et al., 2006). Associated concerns with

population sustainability led to temporal closures in QLD and improvements to trap selectivity in NSW. However, 30–50% of the total annual NSW commercial catch (currently 250 t) is still released (Stewart and Ferrell, 2002, 2003; Stewart, 2008). *P. auratus* is also recreationally important, with an estimated total harvest (primarily from angling) of almost 1.3 million individuals nationally (over 12 months in 2000/2001, Henry and Lyle, 2003). A further 2.5 million *P. auratus* were estimated to have been released (Henry and Lyle, 2003); not only in response to minimum (28–40 cm total length, TL) and maximum legal sizes (40–70 cm TL) and daily personal quotas (1–10 fish), but also as a voluntary conservation measure, reflecting the increasing popularity of fishing for sport rather than food.

The large numbers of commercially and recreationally discarded *P. auratus* warrant detailed assessments of (1) the degree of associated unaccounted fishing mortality, and where appropriate, (2) mitigation strategies. These issues have been investigated during previous studies on *P. auratus*; mostly for recreational fisheries in shallow (<10 m) water (Broadhurst et al., 2005, 2012; Grixti et al., 2010). For example, in two embayments, total post-release

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mortality after angling was estimated to be  $\sim$ 8–33%, with most deaths attributed to cumulative technical (e.g. hook type) and environmental (e.g. warm water temperatures) impacts (Broadhurst et al., 2005) or particularly detrimental treatments like hook ingestion and subsequent removal (Grixti et al., 2010; Broadhurst et al., 2012). Simple changes to angling and onboard handling techniques, including actively fishing the line, using carbon-steel hooks (that oxidise rapidly if ingested), immediately releasing fish (especially those exposed to warm water), and cutting the line on ingested hooks, were all shown to be potentially appropriate strategies for reducing impacts (McGrath et al., 2011; Broadhurst et al., 2012).

Although most of the commercial and  $\sim 30\%$  of the recreational effort occurs in deep water (>10 m), the associated fate of released *P. auratus* has not been studied in great detail (Stewart and Ferrell, 2002, 2003; Henry and Lyle, 2003). One concern associated with catching deep-water fish is barotrauma caused by decompression. Fish would need to be slowly retrieved to the surface over several days for gas to secrete without associated impacts (Pribyl et al., 2009). The occurrence and severity of barotrauma are often species specific (Pribyl et al., 2011), but the condition can manifest as >70 different injuries from the overexpansion of the swim bladder alone (Rummer and Bennett, 2005). Common internal and external signs include a distended coelomic cavity, stomach eversion, prolapsed cloaca, exophthalmia, corneal or subcutaneous gas bubbles, organ torsion, swim-bladder rupture and haemorrhaging (Rummer and Bennett, 2005; Jarvis and Lowe, 2008).

Two relevant studies have quantified barotrauma effects for *P. auratus*. Stewart (2008) and Lenanton et al. (2009), found that, like other sparids (e.g. Rudershausen et al., 2007; Overton et al., 2008), capture depth explained much of the variation in short-term mortalities (up to 55% over a period of less than four days) for trap- and hook-caught individuals. However, both studies involved individuals being released into small cages submerged to the capture depths, thereby spatially restricting movement and potentially affecting recovery (Hannah et al., 2012). An alternative 'confinement' method is to release fish into 'bathy-cages' or 'socks' (Brown et al., 2010; Roach et al., 2011) that encompass more of the water column, thereby allowing fish to better orientate vertically or horizontally after release; which for many species may be important for survival and recovery (Nichol and Chilton, 2006).

Many other studies have investigated ways to alleviate barotrauma in fish, although data regarding specific protocols are lacking. Most studies have assessed venting, which involves puncturing the swim bladder through the body wall (typically with a hypodermic needle) so that expanded gases can escape (Sumpton et al., 2010; Roach et al., 2011). Alternatively, fish have been left untreated and immediately released, or recompressed by returning them to depth with a weighted line (Brown et al., 2010; Hochhalter and Reed, 2011; Roach et al., 2011). In terms of welfare, both recompression and especially venting are potentially more contentious than releasing fish untreated. Further, the choice of such proactive methods requires some degree of certainty that the clinical signs of barotrauma are sufficient to warrant their implementation. However, like for many other species that experience barotrauma, data supporting definitive clinical diagnosis are lacking for *P. auratus*.

The aims of this study were to first assess the prevalence of clinical signs of barotrauma and associated lesions and immediate mortality among *P. auratus* conventionally angled off south-eastern Australia, and identify the shallowest depth at which fish are inflicted (experiment 1). Using this information, the second aim was to retrieve *P. auratus* from two depths by rapid decompression (using cages) and then release them into bathy cages to more closely investigate any negative responses after three handling treatments (experiment 2).

#### 2. Materials and methods

### 2.1. Experiment 1: identifying the clinical signs of barotrauma during angling

#### 2.1.1. Experiment 1a: catch-and-release tournament

Data on the condition of *P. auratus* were collected from 111 anglers on 54 boats during one afternoon and two 6-h morning sessions in an established tournament at Coffs Harbour, NSW (30°18′S 153°09′E) in June 2009. All anglers used one rod, rigged with an artificial lure, handled their fish as normal, photographed them and measured their TL to the nearest 1 mm before recording the following data: approximate capture depth; whether the fish had a distended coelomic cavity; the release method (i.e. no treatment, venting or recompression – see Section 2.2.1 for more details); and swimming behaviour for fish released at the surface (i.e. fish either swam away (1) immediately or (2) after 10–40 s, or (3) did not swim away). These latter data were used to make inferences about postrelease mortality based on the methods developed by Patterson et al. (2000).

#### 2.1.2. Experiment 1b: angling by researchers

Between November 2009 and January 2010, four researchers targeted *P. auratus* in 15–120 m off Coffs Harbour using rods equipped with paternoster rigs and baited with Australian sardine (*Sardinops sagax*). The capture depth (m) and rate of ascent (ms<sup>-1</sup>) were recorded for all fish. After landing, fish were visually assessed for external clinical signs of barotrauma, including: a distended coelomic cavity; partial gastric herniation into the buccal cavity or complete herniation out of the mouth; exophthalmia; prolapsed cloaca; and ocular or subcutaneous gas bubbles. Within 2 min of landing, all specimens were euthanized in 100 mg l<sup>-1</sup> of ethyl-p-amino benzoate (benzocaine) in seawater (Barker et al., 2009), and then measured (TL), dissected and examined for internal barotrauma, including haemorrhages in the liver, kidney and coelomic cavity, organ displacement or a ruptured swim bladder.

### 2.2. Experiment 2: fate of Pagrus auratus after barotrauma and three release methods

The second experiment was done in Port Jackson, Sydney, NSW (33°47′S 151°13′E) over 12 d in November 2009 using two 62 000-l cylindrical bathy cages (2.5 m  $\times$  20 m) and fifty-six 110-l cylindrical cages (see Roach et al., 2011). All cages were anchored in a protected bay (>25 m depth), termed the 'monitoring site'. The 110-l cages were attached in pairs to 20-m retrieval lines (twisted polyethylene – PE, 8-mm diameter – Ø) set 10 m apart on a 600-m buoyed line (twisted PE Ø 12-mm) anchored at 100-m intervals. The cages were designed (and validated by Roach et al., 2011) to orientate horizontally in the water column during suspension. A 7-m vessel equipped with a motorised line-hauler (3.7 kW) was configured to retrieve each pair of cages at 0.9 m s $^{-1}$ ; a rate of ascent chosen based on data collected during experiment 1b (see Section 3).

*P. auratus* were originally trapped (gear described by Stewart, 2008) in  $\sim$ 12–25 m, and retrieved ( $\sim$ 0.35 m s<sup>-1</sup>; a rate normally used by trap fishers) off Coffs Harbour between July and September, 2009. All fish (n=200; 21–42 cm TL) were then immediately assessed and only those without external signs of barotrauma were placed into 150-l live-wells with flow-through water ( $101 \, \mathrm{min}^{-1}$ ) for transport to shore where they were anaesthetised using 25 mg l<sup>-1</sup> of Aqui-S® (Barker et al., 2009), and then into 380-l tanks (stocking density of <15 kg fish m<sup>-3</sup>) containing seawater at ambient temperature ( $\sim$ 18–20 °C). The tanks were transferred to the National Marine Science Centre (NMSC) where fish were removed, reassessed for external signs of barotrauma and other unrelated physical damage (i.e. fin, scale loss and lesions) due to their capture

and handling before they were evenly distributed among three aerated 4000-l holding tanks containing oxytetracycline at a rate of  $100 \,\mathrm{mg}\,\mathrm{l}^{-1}$  for  $3\,\mathrm{d}$  (to try and prevent infections). Each tank was subsequently provided with seawater at a constant flow  $(5\,\mathrm{l}\,\mathrm{min}^{-1})$  and the fish were fed a diet of school prawns (*Metapenaeus macleayi*) at a rate of 1% biomass per day and monitored for mortalities.

Twenty-five days after the last fish was trapped and transported to the NMSC, three from each tank (n=9) were euthanized using benzocaine and any internal clinical signs of barotrauma (described above) incurred during their collection were quantified using the necropsy examination procedures described by Barker et al. (2009). Three weeks before starting experiment 2, the remaining fish were anaesthetised with Aqui-S® and transferred as above to the Cronulla Fisheries Research Centre, Sydney, NSW (CFRC,  $34^{\circ}04'S$   $151^{\circ}09'E$ ) where they were evenly distributed between two aerated and flow-through 5000-l holding tanks, and monitored and fed daily.

### 2.2.1. Experiment 2a: post-release mortality and external clinical signs of barotrauma

On the first day of the experiment, 56 fish were transported (as above) to the monitoring site and individually placed into the 110-1 cages. All 110-1 cages were secured in pairs along the line (at 28 'positions'). Fourteen 110-l cages (at seven positions) were kept at 5 m for use as controls and the remaining 42 were lowered as above to 20 m (at 21 positions), so that along the line these groups were separated as 11 pairs at 20 m, followed by three at 5 m, 10 at 20 m and four at 5 m. All cages were left at their specified depth for 24 h; a period chosen from a pilot study which showed that it was sufficient for fish to incur similar clinical signs of barotrauma as those collected from 20 m during experiment 1. After 24 h, each pair of cages was retrieved using the line-hauler across the same distance; the 20-m cages were hauled vertically (inducing barotrauma in the confined fish), while the 5-m cages were hauled diagonally. It was hypothesised that hauling diagonally would control for the effects of hauling fish in their cages (i.e. similar haul times; Roach et al.,

Immediately after retrieval, the caged fish were assessed for external signs of barotrauma as per experiment 1b. The buoyancy (upright = normal, or sideways/inverted = excess) and swimming position (defined as swimming at either the bottom = normal, or clearly compromised at the surface = affected) were also noted, before each fish was removed and measured (TL). All fish were then fin clipped (either left or right pectoral or bottom or top caudal fins) for identification as controls (from 5 m) and three treatment groups (all from 20 m – see below).

During assessment and fin clipping, 14 of the 20-m P. auratus comprising the first treatment group (termed 'vented' fish) had their swim bladder punctured through the body wall at the vertical intersection of the fifth dorsal spine and the top of the pectoral fin with a 16-gauge hypodermic needle. These and all other control and treatment fish were then placed into 70-l PVC live-wells  $(0.7 \,\mathrm{m} \times 0.4 \,\mathrm{m} \times 0.4 \,\mathrm{m})$  and reassessed for buoyancy and swimming position, before they were released. The controls, vented fish and 14 of the 20-m fish comprising the second treatment group (termed 'no treatment') were released at the surface in each bathy-cage  $(n=7 \text{ cage}^{-1} \text{ for each treatment})$ . The remaining 14 fish in the third treatment group (termed 'recompressed') were returned to 20 m inside the bathy-cages ( $n = 7 \text{ cage}^{-1}$ ) by attaching a barbless hook (made from 2-mm wire and a 0.6 kg lead weight) with 20 m of line (twisted PE  $\varnothing$  2-mm) through the membrane of their lower jaw. The rope was pulled to release the hook. Where fish were visible during release, they were classified according to the swimming behavioural index described for experiment 1a.

Both bathy cages were monitored daily at the surface over 3 d for any mortalities. A Greenspan data logger was used to record water temperature (°C) and salinity (psu) adjacent to each bathy-cage, while dissolved oxygen (mg l $^{-1}$ ) was measured with an Horiba U 10 meter (Horiba Ltd.; Kyoto, Japan). At the end of monitoring, the bathy cages were retrieved and all fish were removed. Except for any physical damage (i.e. at the venting and release-weight puncture sites), it was not possible to assess the condition of fish because of the potential for confounding, repeat effects of barotrauma during cage retrieval (i.e. among those individuals that were located >15 m). All fish were released back into the wild. The entire methodology above was then repeated the following week, providing a total of 28 fish for each of the treatment and control groups.

#### 2.2.2. Experiment 2b: internal clinical signs of barotrauma

In addition to the above work, 20 P. auratus were transferred from the CFRC to Port Jackson and individually placed into 110-l cages. Five of these fish were assessed for barotrauma before they were euthanized. The remaining 15 fish were deployed to 5 m (n=5 controls) or 20 m (n=10 treatments) for 24 h as above. All cages were then retrieved before each fish was assessed for clinical evidence of barotrauma and also their buoyancy and swimming position. Five treatment fish from 20 m were vented after their initial assessment and then placed into 70-l PVC live-wells for 2 min. These vented fish and all other individuals were then reassessed for buoyancy and swimming position. After assessment, all fish were immediately euthanized. The dead fish were measured (TL) and dissected to identify any internal evidence of barotrauma, as described for experiment 1b.

#### 2.3. Statistical analyses

The effects of depth, TL, rate of ascent and treatment on clinical signs of barotrauma in experiments 1 and 2 were assessed using generalised linear mixed models (GLMMs) that were fitted via penalized quasi-likelihood. *p*-Values for the fixed factors of interest were derived via the asymptotic distribution of either: (1) a pseudo *F*-to-enter based on a Wald value from the GLMM; or where the random terms had small variance component estimates (and the data were sparse), (2) the change in deviance from a generalised linear model obtained by their exclusion. All analyses were completed in ASReml-R (R Development Core Team, 2008; Butler et al., 2009).

#### 3. Results

#### 3.1. Identifying the clinical signs of barotrauma during angling

#### 3.1.1. Experiment 1a: catch-and-release tournament

A total of 315 P. auratus (mean TL  $\pm$  SD of 51.6  $\pm$  14.4 cm; range of 26.5–95.5 cm) were caught from 6.0 to  $60.5 \, \text{m}$  (mean  $\pm \, \text{SE}$  of  $19.7 \pm 0.4 \,\mathrm{m}$ ) during the tournament. According to the anglers, 61 fish had a distended coelomic cavity (and therefore were assumed to have barotrauma); of which 26 were just vented, seven were just lowered with a release weight (recompressed), seven were subjected to both treatments, and 21 were released at the surface without any treatment. An additional 17 fish were lowered with a release weight even though they had no distended coelomic cavity. The shallowest depth at which clinical signs of barotrauma occurred was 11 m. Of 266 fish that were assessed at the surface for their release condition (the remaining fish were either lowered with a release weight or not assessed), all but one (78 cm TL) swam away immediately. This latter fish was angled from 20 m and vented before it floated for 10-40 s after release (before swimming away) which, based on the index proposed by Patterson et al. (2000), suggests that it would have died eventually; providing a predicted mortality of 0.4%.

To explain the variation in the presence of a distended coelomic cavity, a GLMM comprising the random effects of angler, fishing

Table 1

Experiment 1b: summary of p(Wald) statistics and regression coefficients for the fixed effect of capture depth  $(15-120\,\mathrm{m})$  in generalised linear mixed models (GLMMs) applied to explain variation in the occurrence of six clinical signs of barotrauma in Pagrus auratus during sampling by researchers offshore from Coffs Harbour. Because no fish had exophthalmia, kidney haemorrhage or ocular or subcutaneous gas bubbles, and all had a distended coelomic cavity and a prolapsed cloaca, these responses were not considered. Total length of fish and their ascent rate were included as fixed co-variates in all GLMMs, but were not significant [p(Wald) > 0.05].

	p(Wald)	Estimate	SE	Z ratio
Clinical signs of barotrauma				
Ruptured swim bladder	0.04	0.582	0.283	2.06
Gastric herniation into buccal cavity	0.26	0.016	0.014	1.14
Gastric herniation out of mouth	0.50	-0.015	0.022	-0.68
Coelomic cavity haemorrhage	0.56	0.007	0.012	0.59
Organ displacement	0.57	-0.008	0.014	-0.57
Hepatic haemorrhage	0.63	0.008	0.017	0.49

session and their interaction, and the fixed effects of TL of fish and depth of capture was fitted to the data. Both fixed effects showed a significant positive correlation with distension [p(Wald) < 0.05].

#### 3.1.2. Experiment 1b: Pagrus auratus angled by researchers

Thirty-two *P. auratus*  $(33.1 \pm 3.3 \text{ cm TL}; \text{ range of } 24.5-43.0 \text{ cm TL})$  were angled from 15 to 120 m by researchers. Irrespective of their depth, none of the fish had ocular or subcutaneous gas bubbles, exophthalmia or kidney haemorrhages, while all had a distended coelomic cavity and a prolapsed cloaca. Many fish suffered a ruptured swim bladder (84.4%), with associated 0.1-3.0-cm lesions, organ displacement (65.6%), hepatic (84.4%) and peritoneal cavity haemorrhages (40.6%), and gastric herniation extending into the buccal cavity (56.0%) or, less frequently, out of the mouth (9.4%).

Of these clinical signs of barotrauma, only six varied among depths for all fish and were subsequently included as response variables in GLMMs that comprised anglers as a random effect, TL and rate of fish ascent as fixed co-variates and the capture depth as the fixed factor of interest (Table 1). Only the presence of a ruptured swim bladder was significantly affected by depth [p(Wald) < 0.05; Table 1]. Specifically, only one of five fish caught from 15 m, compared to 95% of fish (n=27) retrieved from  $\geq 20$  m had a ruptured swim bladder. Neither TL, nor the rate of ascent  $(0.9 \pm 0.3 \, \text{m s}^{-1})$ , significantly affected the clinical signs of barotrauma among fish [p(Wald) > 0.05; Table 1].

### 3.2. Experiment 2: fate of P. auratus after barotrauma and three release methods

Of the 200 fish that were trapped off Coffs Harbour and released into tanks at the NMSC, 24% (n=48) were initially classified as being in poor condition (with fin and scale loss and multiple lesions attributed to contact with the traps and/or onboard housing) and subsequently died (96% in 3 d and all within 5 d). All mortalities were autopsied, and death was attributed to infections arising from repeated handling and confinement. All other fish survived. None of the randomly sampled survivors had any external signs of barotrauma or other damage. Water conditions in Port Jackson remained similar within and among days, with means ( $\pm$  SD) of  $21.8 \pm 1.1$  °C for temperature;  $8.5 \pm 0.3$  mg l<sup>-1</sup> for dissolved oxygen; and  $36.8 \pm 0.2$  psu for salinity. Air temperature during daylight hours varied slightly between days ( $23.7 \pm 3.2$  °C).

### 3.2.1. Experiment 2a: post release mortality and external clinical signs of barotrauma

All 112 *P. auratus*  $(27.9\pm3.5\,\mathrm{cm}$  TL; range of  $21.6-40.2\,\mathrm{cm})$  deployed in 110-1 cages were retrieved with similar rates of ascent  $(0.9\pm0.04\,\mathrm{m\,s^{-1}})$  and exposed to similar surface conditions for the same duration (<60 s). None of the controls (hauled from 5 m) had

#### Table 2

Experiment 2a: percentage of *Pagrus auratus* sustaining relevant external clinical signs of barotrauma immediately after they were hauled from 5 m (controls; n = 28) and 20 m (treatments; n = 84) and associated p-values derived from generalised linear mixed models (GLMMs) assessing for equality of propensity. Total length was included in all GLMMs, but was not significant [p(deviance) > 0.05]. Owing to sparse data, GLMMs did not converge (–) for distended coelomic cavity or prolapsed cloaca, but the effect of depth was clearly significant for these two variables.

	5 m	20 m	p(deviance)
Clinical signs of barotrauma (% frequency)			
Gastric herniation into buccal cavity	0.0	76.2	< 0.0001
Gastric herniation out of mouth	0.0	5.9	0.0731
Distended coelomic cavity	0.0	100	-
Prolapsed cloaca	0.0	99.0	-
Excess buoyancy	0.0	6.6	0.0034

external clinical signs of barotrauma (Table 2). In contrast, all of the fish hauled from 20 m had a distended coelomic cavity, all but one had a prolapsed cloaca, and most (76.2%) had their stomach distended into the buccal cavity (Table 2). Only a few fish (16.6%) had excess buoyancy, while none had ocular or subcutaneous gas bubbles, and only one had exophthalmia (in one eye).

Variation in clinical signs observed among immediately hauled fish, and then again just prior to their release (and after the relevant group were vented) was assessed using GLMMs. Random terms included cages, the position of each pair, their depth cluster (on the line), and their deployment day. The fixed factors comprised TL and the hauling depth for clinical signs of barotrauma recorded immediately post hauling. For the subsequent reassessment of fish after the relevant group was vented, depth was replaced by a factor with three levels: (1) controls from 5 m, and (2) non-vented and (3) vented fish from 20 m.

Three of the external clinical signs of barotrauma were non-informative (exophthalmia, and ocular or subcutaneous gas bubbles), but the others provided significant evidence of impacts among fish hauled from  $20 \,\mathrm{m}$  [p(deviance) < 0.01; Table 2]. Venting the 20-m fish had positive effects, with no fish having excess buoyancy and only 3.6% fish having compromised swimming, respectively [p(deviance) < 0.001]. All untreated 20-m fish had compromised swimming while 23.2% had excess buoyancy. The TL of fish had no significant effect on any of their clinical signs of barotrauma [p(deviance) > 0.05; Table 2].

Immediately after release into the bathy-cages, all of the vented and control fish rapidly swam down, while most of those that were left untreated did so more slowly. Two of these latter fish remained inverted for 10–20 s at the surface before swimming away. One of the recompressed fish returned to the surface after 20 s and then spent 30 min inverted before swimming away. Despite these observed impacts, no *P. auratus* died, and only small lacerations from the needles or release hooks were evident after 3 d. However, at the end of the monitoring period, all fish (including those originally retrieved from 5 m) had a prolapsed cloaca, which may have been sustained during bathy-cage retrieval or, for the 20-m fish, the trauma that occurred during 110-l cage retrieval.

#### 3.2.2. Experiment 2b: internal clinical signs of barotrauma

Of the 20 *P. auratus* ( $29.1\pm4.6\,\mathrm{cm}$  TL; range of  $22.9-41.2\,\mathrm{cm}$ ) that were assessed and then euthanased, only the 10 hauled from 20 m were clinically affected, with all having a prolapsed cloaca, distended coelomic cavity, ruptured swim bladder and organ displacement (Table 3). In most ruptured swim bladders, there was a unilateral linear longitudinal tear measuring  $1-4\,\mathrm{cm}$ , which was often associated with the peritoneal attachments. No fish had gastric herniation out their mouth or ocular or subcutaneous gas bubbles, exophthalmia or renal haemorrhages, but most had gastric herniation into the buccal cavity and evidence of hepatic haemorrhage (Table 3).

Table 3

Experiment 2b: percentage of *Pagrus auratus* presenting relevant internal and external clinical signs of barotrauma at the surface immediately prior to deployment (0 m; n=5) in cages, and then after hauling from 5 m (controls; n=5) and 20 m (treatments; n=10). The associated p-values were derived from generalised linear mixed models (GLMMs) assessing for equality of propensity. Total length was included in all GLMMs, but was not significant [p(deviance)>0.05]. Owing to sparse data, GLMMs did not converge (–) for prolapsed cloaca, distended coelomic cavity, ruptured swim bladder and organ displacement, but the effect of depth was clearly significant for these variables.

	0 m	5 m	20 m	p(deviance)
Clinical signs of barotrauma (% frequency)				
Gastric herniation into buccal cavity	0	0	70	0.0001
Prolapsed cloaca	0	0	100	_
Distended coelomic cavity	0	0	100	_
Ruptured swim bladder	0	0	100	-
Organ displacement	0	0	100	-
Hepatic haemorrhage	0	0	70	0.0004
Coelomic cavity haemorrhage	0	0	90	< 0.0001
Excess buoyancy	0	0	40	0.0366

GLMMs comprising the position of the paired cages as a random term and the TL of fish and their deployment depth as fixed factors confirmed the significance of the above results [p(deviance) < 0.04; Table 3]. There was also a significant impact of depth on gastric herniation within the buccal cavity, peritoneal and hepatic haemorrhages and excess buoyancy; all of which appeared in 20-m fish, but not those sampled prior to caging, or the controls hauled from 5 m [p(deviance) < 0.05; Table 3]. For the 20-m fish, venting significantly improved their buoyancy and swimming ability to the same level as those fish prior to caging or after they were hauled from 5 m [p(deviance) < 0.05). Total length had no impact on any clinical signs of barotrauma [p(deviance) > 0.05; Table 3].

#### 4. Discussion

Like many other species retrieved from >10 m, *P. auratus* incur barotrauma (Rummer and Bennett, 2005; Campbell et al., 2010). But unlike previous studies on this species (Stewart, 2008; Lenanton et al., 2009), there was minimal variation in the clinical signs of barotrauma among the depths examined (6–120 m), and few associated predicted mortalities in experiment 1. Similarly, a comparable severity of barotrauma among fish in experiment 2 did not cause any mortality over 3 d. These results suggest that in the absence of other deleterious factors, including deep hooking (Broadhurst et al., 2005, 2012; Grixti et al., 2010; McGrath et al., 2011) or perhaps the confounding effects of confining fish in cages at restricted depths after release (Stewart, 2008; Lenanton et al., 2009), *P. auratus* can recover from barotrauma; at least over the short term (and under the environmental conditions assessed).

Such apparent resilience can be explained by considering the extent of physical trauma imposed, and the likely associated physiological impacts (Rummer and Bennett, 2005; Jarvis and Lowe, 2008; Phelan, 2008). The clinical signs of barotrauma among P. auratus were relatively mild, but consistent with those noted in many other species and some were probably reversible (Rummer and Bennett, 2005; St John and Syers, 2005). Typically, in severely affected fish, elevated intracoelomic pressure due to over distension of the swim bladder would markedly reduce cardiac function by impeding venous blood return. This impact, along with physical obstruction of water flow over the gills caused by the displaced stomach filling the buccal cavity, can result in hypoxia, and ultimately death if it is prolonged (Phelan, 2008). However P. auratus may be more tolerant of such impacts than other species; perhaps due to their multiple haemoglobin isomorphs (Stephens et al., 2002).

Notwithstanding the apparent broad resilience of *P. auratus*, some of the observed clinical signs could still have longer-term

negative consequences, and possibly mortalities (Rummer and Bennett, 2005; Gravel and Cooke, 2008). For example, trauma to the stomach may lead to ischaemic necrosis of the gastric mucosa, or disruption of the epithelial barrier and consequent bacterial translocation and septicaemia which, for fish with moderate and severe damage, could cause death (Phelan, 2008). Similarly, a prolapsed cloaca could predispose a fish to intussusceptions, obstruction or inflammation and secondary bacterial infection. Rupture of the swim bladder could also result in bacterial infection and peritonitis, as well as more immediate problems with buoyancy control (Rummer and Bennett, 2005). This may result in fish being less efficient at foraging and susceptible to predation and sun damage (Gitschlag and Renaud, 1994). Furthermore, all fish with ruptured swim bladders would presumably expend greater energy regulating their buoyancy through swimming, and could impact nutritional reserves depending on the period required for the swim bladder to repair.

The temporal regeneration of the swim bladder for *P. auratus* remains unknown, but the sub-sample of trapped fish at the beginning of experiment 2 had no evidence of ruptures 28 d after capture. Other species have been observed to repair their swim bladders, but with some temporal variability, including as quickly as one day (e.g. Pacific cod *Gadus macrocephalus*, Nichol and Chilton, 2006; and largemouth black bass *Micropterus salmoides* Shasteen and Sheehan, 1997), or up to two-weeks (rainbow trout, *Oncorhynchus mykiss*, Bellgraph et al., 2008), three-weeks (rockfish, *Sebastes melanops* and *Sebastes nebulosus*, Parker et al., 2006) or even eightweeks (burbot, *Lota lota*, Bruesewitz et al., 1993) after release.

Other barotrauma clinical signs of concern included hepatic haemorrhages, which would have caused fractures to Glisson's capsule owing to the increased portal venous and/or intracoelomic pressure. For the survivors of such lesions, any consequences are probably limited to short-term welfare issues, given the large functional reserve and regenerative capacities of the liver. However, in severe cases hepatic dysfunction would cause death. Organ displacements are more likely to have adverse immediate and long-term consequences (ultimately death) because these can disrupt blood supply to visceral organs and or flow of ingesta through the gastrointestinal tract (Rummer and Bennett, 2005; Phelan, 2008).

The potential for negative welfare issues associated with gross physical disruptions, such as organ displacement and intracoelomic pressure caused by barotrauma, warrant their decisive resolution. Based on the results observed here, it is apparent that all three release methods eventually facilitate this endpoint; albeit at different rates. The implications of such temporal variation among release methods might provide some basis for their prioritization by recreational and commercial fishers. For example, venting P. auratus immediately alleviated their intracoelomic pressure, so that they regained buoyancy control and orientation and, after release, were able to swim away much faster than untreated fish. In the wild, fish that quickly return to their habitat could have a lower probability of other unaccounted mortality or sublethal impacts, including predation (Keniry et al., 1996; Overton et al., 2008), oxygen demand (Marty et al., 1995), energy expenditure (Strand et al., 2005), stress from warm surface water temperatures (Shasteen and Sheehan, 1997), or sun exposure (Keniry et al., 1996), or even being struck by boats (Gravel and Cooke, 2008).

The potential for such outcomes might support venting over untreated release. But in addition to causing an additional lesion and localised trauma (and possibly mortality through incorrect technique; Rummer and Bennett, 2005; Jarvis and Lowe, 2008), venting would not remove gas throughout all soft tissues and organs, leaving the individual with only partial resolution which could have at least some longer-term (unknown) consequences. Recompression would be quickly achieved by forcing the fish back

to depth using the weighted line, which may be sufficient to recommend this release method over the others, although one consideration is the potential for predation (especially from sharks) attracted to a struggling or disorientated fish. Additional research is required to quantify this component of unaccounted fishing mortality, which could be substantial in some areas (Danylchuk et al., 2007).

While there are several environmental and biological factors that might affect an angler's decision about which release method to use, a common prerequisite is some definitive diagnosis of actual barotrauma, since imposing proactive release methods to nonafflicted fish will cause unnecessary injury. The need for such data, especially for fish caught across the depths where signs of barotrauma become evident ( $\sim$ 10–20 m), is supported by the results from experiment 1a (catch-and-release tournament), during which most fish were probably misdiagnosed as not having barotrauma, and were subsequently released without any treatment, while others were unnecessarily vented and then recompressed. Based on the results here, in addition to the obvious gross abnormalities (e.g. distended coelomic cavity and gastric herniation into the buccal cavity), barotrauma in P. auratus might simply be identified by the presence of a prolapsed cloaca. Diagnosis could be confirmed by placing fish into a water container to assess excess buoyancy and orientation, however, it would seem pertinent to release individuals as quickly as possible (Jarvis and Lowe, 2008). Anglers also need to be better informed about the lack of any relationship between speed of ascent and barotrauma in P. auratus. It is often assumed among fishers that slowly retrieved fish are less impacted, yet this study and others have shown that this is not the case (Rummer and Bennett, 2005; Stewart, 2008).

Although this study has identified protocols for identifying barotrauma in P. auratus and demonstrated apparent homogenous short-term impacts of the available release methods, the results are nevertheless limited to the experimental design and the tempo-spatial sampling. Specifically, while there were no impacts of TL on barotrauma among fish during experiment 2, we assessed discrete sizes ( $\sim$ 22–42 cm TL). Total length was identified as a significant co-variate affecting the anglers' perceptions of a distended coelomic cavity, although, this may have been partially confounded by reproductive condition. For example, while none of the fish collected by researchers in either experiment were reproductively active, some anglers may have mistaken a distended coelomic cavity for developing gonads among fish. Further work is required to assess any trauma associated with fish that are caught during their spawning season (typically October-March and May-August for southern and northern distributions, respectively; Kailola et al., 1993), when gonads occupy a large portion of the coelomic cavity and could possibly be expelled and/or damaged (Roach et al., 2011). Also, while water temperatures in this study were representative of those most commonly experienced, some P. auratus at their northern distribution occupy warmer water during summer (~25–30 °C), which could exacerbate physiological responses to barotrauma due to exposure to a greater range of temperatures during ascent (Diamond and Campbell, 2009). Telemetry would facilitate field-based assessments of such broader impacts, and also help to ascertain whether barotrauma influences the longer-term ability of *P. auratus* to survive, forage and evade predation.

Despite the need for ongoing research, there are interim protocols that could be implemented to limit the number of fish exposed to barotrauma. Relevant strategies might include (1) using gears (i.e. larger hooks and baits or escape panels in traps) that select mostly legal-size fish, (2) spatial and temporal closures to coincide with large abundances of susceptible fish (e.g. juveniles or spawning aggregations), or possibly (3) replacing minimum legal lengths with quotas only in deep water. Combined with selecting an appropriate release method, these options will help to minimise negative impacts on *P. auratus* and ultimately benefit the sustainability of stocks.

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#### **Further reading**

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