

Mortality and blood loss by blue swimmer crabs (*Portunus pelagicus*) after simulated capture and discarding from gillnets

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Two laboratory experiments were carried out to quantify the mortality and physiological responses of juvenile blue swimmer crabs (*Portunus pelagicus*) after simulated gillnet entanglement, air exposure, disentanglement, and discarding. In both experiments, all but control blue swimmer crabs were entangled in 1-m² gillnet panels for 1 h, exposed to air for 2 min, subjected to various treatments of disentanglement ranging between the forceful removal of none, one, two, and four appendages, then “discarded” into individual experimental tanks and monitored for 10 d. In Experiment 1, mortalities were associated with the number of appendages removed and the occurrence of unsealed wounds. In Experiment 2, live blue swimmer crabs were sampled for blood at 2 min and 6, 24, and 72 h post-discarding to test for the effects of disentanglement and appendage removal on total haemocyte counts, clotting times, protein levels (by refractive index), and blood ion concentrations. Compared with blue swimmer crabs that had sealed or no wounds, those with unsealed wounds had lower total haemocyte counts, protein, and calcium concentrations and increased clotting times and magnesium and sodium levels. Induced autotomy, as opposed to the arbitrary, forceful removal of appendages has the potential to minimize the mortality and stress of discarded, juvenile blue swimmer crabs.

Keywords: appendage removal, discard mortality, gillnet fisheries, physiological stress, *Portunus pelagicus*.

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Introduction

Blue swimmer crabs (*Portunus pelagicus*) form the basis of important commercial (including aquaculture) and recreational fisheries throughout their distribution in coastal and estuarine waters of the Indo-Pacific (Stephenson, 1962; Kailola *et al.*, 1993). In New South Wales (NSW), Australia, some 200 t, valued at around AU\$ 1.4 million, are caught in the wild each year by commercial fishers using a range of small-scale gears, including otter trawls, seines, traps, and gillnets. There are no similar data describing NSW recreational catches by weight, although a survey estimated that >0.4 million crabs were retained (mostly from baited tanglenets) during 12 months in 2000/2001 (Henry and Lyle, 2003).

All the fishing gears used to catch blue swimmer crabs in NSW are managed by input controls that include minimum legal mesh sizes. However, none are 100% selective so, in addition to the harvested catches, large numbers of undersized (<6 cm carapace length, CL) crabs are caught and discarded. This is particularly the case with commercial gillnets (80–100 mm mesh) used to target teleosts throughout 85 estuaries in NSW (see Gray, 2002, for a detailed description of gears and methods). Owing to their aggressive foraging behaviour, blue swimmer crabs are strongly

attracted to catches in these gears, and as a result, a wide range of sizes (including juveniles) may become entangled in the nets. No data are available on the total numbers of blue swimmer crabs discarded each year by NSW gillnetters, but short-term fishery-dependent studies have estimated >1000 crabs month⁻¹ in some estuaries (Gray, 2002; Gray *et al.*, 2004).

The potential for wide-scale mortalities to valuable species discarded from gillnets has raised concerns over the sustainability of populations (Gray, 2002). Attempts have been made to address this issue by improving the selectivity of the various configurations for the targeted teleosts and their sizes; mostly through changes to rigging, materials, and mesh sizes (Broadhurst *et al.*, 2003; Gray *et al.*, 2005). Although these changes have reduced significantly the discarding of some juvenile teleosts, the morphology of blue swimmer crabs precludes their defined selection in gillnets so, irrespective of the configurations, large numbers are still caught and discarded (Broadhurst *et al.*, 2003). A more appropriate strategy for reducing the potential for negative impacts associated with these discards may be to promote operational and/or handling methods that maximize post-capture survival (Broadhurst *et al.*, 2006).

No reports have been published in the primary literature to describe the fate of blue swimmer crabs, or the types of injuries sustained, after discarding by gillnetters. However, anecdotal observations during observer-based studies suggest that, as for other crustaceans caught in similar local fisheries (e.g. Kennelly *et al.*, 1990), common injuries include the loss of appendages, particularly the chelipeds (claws). This damage occurs as fishers attempt to quickly disentangle the crabs from the meshes, often by physically removing the chelipeds (to avoid personal injury) while firmly holding the crabs behind the fifth pereopods (paddle), which may also be broken. Some fishers argue that such injuries have minimal long-term impacts, primarily because live and apparently healthy amputees are often observed in the wild (Davis, 1981; Durkin *et al.*, 1984).

In some cases, appendage loss may not result in mortality to juvenile blue swimmer crabs, if this takes place at the fracture plane (termed “autotomy”—Wood and Wood, 1932). These breakages are typical in response to injury, or to evade predation, and are preventive reflexes involving either some or no external force (Wood and Wood, 1932). Nevertheless, some appendage loss may evoke sublethal stress and wounding, and could eventually reduce growth or foraging efficiency (Juanes and Smith, 1995) and/or increase susceptibility to infections and predation (Potter *et al.*, 1991). Any study that seeks to quantify the fate of discards should, therefore, include at least some assessment of the potential for sublethal physiological impacts. Appropriate indices of compromised physiological condition for crustaceans include a number of blood parameters such as total haemocyte counts, clotting times, protein levels (by refractive index), and ionic concentrations (Paterson and Spanoghe, 1997; Jussila *et al.*, 2001). Circulating haemocyte numbers, coagulation, and ionic regulation are also likely to be acutely influenced by excessive blood loss following injury (Soderhall and Cerenius, 1992). Ultimately, adequate understanding of the responses of blue swimmer crabs to catch-and-discarding mechanisms is required to validate existing management regulations involving minimum sizes.

Our first objective in this study was to examine the effects of gillnet entanglement, air exposure, and appendage removal in the laboratory on the short-term mortality and physiological responses of live blue swimmer crabs. Based on this information, our second objective was to suggest subtle changes to handling methods that might maximize survival and reduce negative physiological impacts.

Material and methods

Two experiments were carried out at the National Marine Science Centre (NMSC) in Coffs Harbour, NSW, between November 2006 and July 2007, using 204 blue swimmer crabs, one 2000-l fibreglass holding tank, and 259 small (12 l) polyvinyl chloride experimental tanks. The holding tank was outdoors, but the experimental tanks were in an enclosed room with a regulated photoperiod (12:12 h). All experimental tanks were supplied with flow-through seawater at ambient temperature (17–25°C) and salinity (34–36 psu) at a rate of 0.2 l min⁻¹.

Animal collection and husbandry

In November 2006, 400 farm-reared juvenile blue swimmer crabs (<~2.5 cm CL) were collected using castnets, placed into 12 plastic trays (60 × 35 × 10 cm) stacked inside two 380-l fish transporters (filled with water and aerated with oxygen), and

transferred to the NMSC. Intact crabs with no visible physical damage were selected randomly from the transporters and placed individually into the experimental tanks. The remaining animals were kept in the holding tank.

For 6 months before the first experiment, all blue swimmer crabs were fed school prawns (*Metapenaeus macleayi*) at a rate of ~10% of their body mass per day. Faeces and excess food was siphoned off 12 h after every feeding. To maintain independence among experimental tanks, the siphon hose was rinsed in fresh water after cleaning each tank. Any dead crabs were removed before the tanks were cleaned and restocked with an intact, live crab from the holding tank. Once the crabs in the experimental tanks had reached an average size of ~3 cm CL, they were all subjected to the treatments outlined in the experiments below.

Experiment 1

In all, 120 intermoult blue swimmer crabs were selected randomly and divided into one control and five treatment groups ($n = 20$). The controls were not touched. For all treatment groups, 1-m² panels of 80-mm polyamide mesh (1.5 mm diameter twisted twine) were folded up and placed into each of the experimental tanks. A single dead prawn was also dropped into each bundle of netting to promote the attraction and manual entanglement of the crab. Treated crabs were left entangled for 1 h, before being removed, exposed to air for 2 min, then subjected to one of the following five treatments during disentanglement and before being “discarded” (dropped from heights of 20–50 cm) back into their experimental tanks: T1, no appendages removed; T2, one cheliped removed; T3, one fifth pereopod removed; T4, one cheliped and one fifth pereopod removed; and T5, both chelipeds and both fifth pereopods removed. Appendages were forcibly removed near their bases. The work was divided among four researchers (each handling five replicate crabs for the five treatments).

After simulated capture and discarding, the 20 control and 100 treatment crabs were fed (as above) and monitored daily for their status (dead or live) over 10 d. The following data were recorded for each crab that died or was still alive at the end of the monitoring period: CL and carapace width, CW (both to the nearest 0.1 mm); sex; location of experimental tank in the room (divided into five zones); researcher responsible for the various treatments; and whether or not the wound caused by the removal of an appendage resulted in an opening of the body cavity (missing the entire appendage and coxopodite) uncovered by membranous tissue (termed “unsealed”), or resulted in a break where segments of the leg were still attached to the carapace, forming a tissue-covered bud (termed “sealed”).

Experiment 2

In all, 84 untreated blue swimmer crabs were selected and divided into the control ($n = 12$) and treatment groups (T1: $n = 12$; T2: $n = 14$; T3: $n = 14$; T4: $n = 15$; T5: $n = 17$), which were subjected to the same entanglement and discarding as described above, before being returned to their experimental tanks. After being confined for 2 min or 6, 24, or 72 h, three live intermoult crabs from the control and each treatment group were randomly and destructively sampled for blood.

At each sampling time, the selected crabs were placed on a foam block. Two consecutive blood samples were extracted by inserting hypodermic syringes through the membrane of the basis or coxae of either or both fifth pereopods of each crab. The first syringe

(1 ml, 25-gauge needle) was chilled at 4°C and prefilled with 0.20 ml of anti-coagulant (Jussila *et al.*, 2001). Approximately 0.20 ml of blood was drawn, and the syringe inverted several times to mix the blood with the anti-coagulant. The sample was transferred into an Eppendorf vial and refrigerated at 4°C for up to 5 d, before counts of the total numbers (10^6 cells ml⁻¹) of haemocyte cells were made following the procedures of Jussila *et al.* (2001).

The second syringe (2 ml, 22-gauge needle) was used to remove 1–2 ml of blood, of which 0.05 ml was immediately placed in the centre of a microscope slide. The sample was stirred with the tip of the syringe at ambient air temperature and the clotting time recorded. Samples that did not clot after 90 s were noted. Approximately 0.05 ml of remaining unclotted blood was then placed onto the prism of a clinical refractometer with automatic temperature compensation, and the refractive index was recorded (scale: 1.340–1.360). The remaining sample was immediately frozen at –22°C for up to 26 d. Serum samples were subsequently analysed for calcium and magnesium (mmol l⁻¹) using an Olympus® model AU500 autoanalyser and following the Trace Arsenazo III and Calmagite Kit methods, respectively. Potassium and sodium (mmol l⁻¹) were determined by atomic absorption spectrophotometry using a Varian AA-40 AAS®. Additionally, CL, CW, sex, and the type of wound were recorded for each crab, as described for Experiment 1.

Statistical analyses

The null hypothesis of an equal sex ratio among all experimental crabs was investigated using a Chi-squared goodness-of-fit test. At the end of the monitoring period in Experiment 1, the key parameters were collated as being either categorical (treatment; sex; location of experimental tank in the room; and researcher responsible for the appendage-removal treatment) or continuous (CL and CW). Binomial generalized linear models using a logit link function (McCullagh and Nelder, 1989) were employed to test for the effect of these parameters on the proportion of crabs surviving in each treatment in Experiment 1. A separate logistic regression model was used to test for the effect of wound type (unsealed, sealed, or no wound) on the proportion of survivors, and also for the effect of the treatments on the relative proportions of wound type. For all models, the most significant terms were fitted first to allow for the most precise testing of the more marginal terms. Pairwise analyses using least-square differences (LSDs) were carried out on adjusted means for factors that were shown to be significant after initial accumulated analysis of deviance tests.

Appropriate unbalanced analyses of variance were used to test for the effects of wound type and sampling time (2 min, and 6, 24, and 72 h), and their interaction on total haemocyte counts, clotting times, blood protein levels (refractive index), and concentrations of calcium, magnesium, potassium, and sodium ions sampled from the live crabs in Experiment 2. Magnesium concentrations were log₁₀(*x* + 1)-transformed to stabilize the variance. All the above analyses were performed using GenStat, 9th edn (GenStat, 2007).

Results

Of the 259 blue swimmer crabs originally placed into the experimental tanks, nine died during the 6 months before starting Experiment 1 (4% mortality). There was a significant bias in the ratio of males (*n* = 115) to females (*n* = 76) used in the

experiments ($\chi^2 = 7.96$, $p < 0.05$), but with a mean \pm s.e. CL of 4.36 ± 0.04 cm, all juveniles. Of the treatment crabs, 13 were not sexed, because they either died before sampling or were not ultimately used in the experiment (see below for details in Experiment 2).

Experiment 1

Of the 100 treated blue swimmer crabs, 23% died over the 10-d monitoring period. There were no deaths in the control group. Irrespective of the treatment, most mortality (83%) was within the first 3 d, and except for one T4 crab that died after moulting on the 10th day, all deaths had ceased by the 7th day (Figure 1). Logistic regression of the categorical and continuous parameters on mortality failed to detect any significant interactions ($p > 0.05$). The only significant main effect was the treatment of crabs, with no mortalities among control and T1 individuals, compared with 1, 3, 8, and 11 deaths among those subjected to the T2, T3, T4, and T5 treatments, respectively (deviance ratio: 7.75, $p < 0.01$; Figure 2). Pairwise LSD tests revealed that the

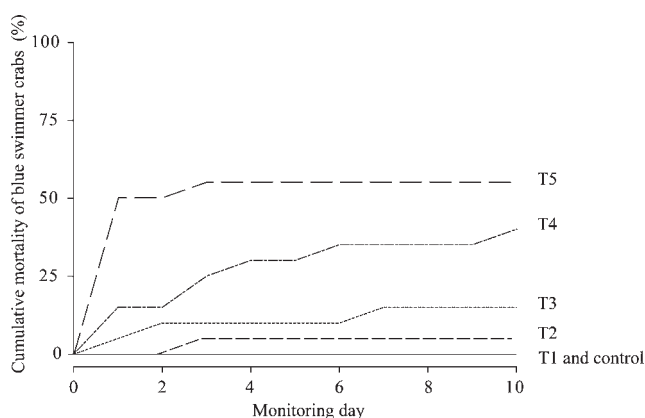


Figure 1. Cumulative percentage mortalities of blue swimmer crabs in each of the treatment groups over the 10-d monitoring period in Experiment 1: control; T1, no removal of appendages; T2, one cheliped removed; T3, one fifth pereopod removed; T4, one cheliped and one fifth pereopod removed; and T5, both chelipeds and both fifth pereopods removed.

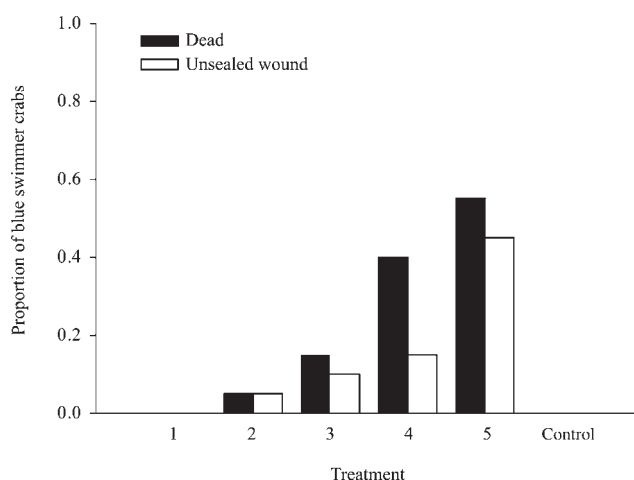


Figure 2. Proportions of dead blue swimmer crabs and those with unsealed wounds in each of the six treatment groups in Experiment 1 (*n* = 20 in each group).

mortalities in T4 and T5 were similar and significantly greater than those in the remaining treatments.

Overall, of 160 wounds caused among the 80 crabs in the four appendage-removal treatments, 16% were unsealed. The numbers of unsealed wounds were 1, 2, 3, and 20 in the T2, T3, T4, and T5 treatments, respectively. Regression analyses of mortalities revealed a significant main effect of the type of wound (unsealed, sealed, or no wound), with 100% mortality among 15 crabs that had at least one unsealed wound. By comparison, there were eight mortalities among 65 crabs with sealed wounds and none among those with no wounds (i.e. 40 crabs in the control and T1 groups; deviance ratio: 34.39, $p < 0.01$). There was also a significant main effect for the treatment of crabs on the type of wound (deviance ratio: 5.01, $p < 0.01$). The proportion of crabs with unsealed wounds increased significantly between T4 and T5, when twice as many appendages were removed ($p < 0.05$; Figure 2).

Experiment 2

Of the 84 treated blue swimmer crabs, eight died before sampling and five were not sampled. These excess (unsampled) crabs were originally treated to account for potential mortalities before sampling. In all, 71 live crabs were successfully sampled for blood across the four periods at 2 min, 6, 24, and 72 h post-selection or post-discarding. One blood sample was contaminated and excluded from the analysis. For all the sampled crabs, of the 92 wounds inflicted, 16% were unsealed ($n = 59$). Specifically, the numbers of unsealed wounds were 1, 1, 4, and 9 in T2, T3, T4, and T5. None of the crabs sampled for blood 24 and 72 h after selection and/or discarding had an unsealed wound. Seven blood samples did not clot within 90 s, with 1 in T3 and 3 each in T4 and T5. Five of these samples were from crabs with unsealed wounds. Further, blood did not clot from these five crabs 6 h post-discarding (Figure 3a).

Irrespective of the sampling time, compared with crabs with sealed or no wounds, those with unsealed wounds had significantly fewer total haemocytes and less protein (refractive index) and calcium, but more sodium in their blood ($p < 0.05$; Table 1; Figure 4). There were significant interactions between wound type and sampling time for the rate of blood clotting and the concentration of magnesium ($p < 0.05$; Table 1). Plots of the relevant means showed that crabs with unsealed wounds had longer blood-clotting times and greater concentrations of magnesium at 6-h post-release than those with sealed or no wounds (Figure 3). Irrespective of their treatment, all crabs showed a significant reduction in potassium concentration over the sampled times ($p < 0.01$; Table 1; Figure 5).

Discussion

Our study provides the first estimates of the mortality of juvenile blue swimmer crabs after entanglement in gillnets, air exposure, appendage removal, and discarding. By quantifying the physiological responses of live crabs, we also obtained important information on some of the sublethal impacts associated with these treatments. Both these outcomes ultimately facilitate the development of post-capture handling procedures that may enhance the survival of blue swimmer crabs.

The observed mortalities in Experiment 1 (0–55%) were positively correlated with the number of removed appendages and the associated wounding and blood loss. Although no other comparable estimates are available for this species after discarding from

gillnets, the results are consistent with the trend observed for local trawl-caught crabs (Wassenberg and Hill, 1989, 1993) and for other brachyurans discarded from a range of different gears (Simonson and Hochberg, 1986; Kennelly *et al.*, 1990; Stevens, 1990; Bergmann and Moore, 2001). For example, during a study on the short-term (< 7 d) mortality of 50 discarded trawl-caught blue swimmer crabs, Wassenberg and Hill (1993) observed multiple appendage loss that contributed towards an overall mortality of 38%, compared with $< 7\%$ for crabs that sustained no obvious injury. It is unclear, however, if blood loss was associated with the observed injuries and whether, as in the present study, this was a key cause of mortality.

More detailed examination of the relationship between appendage loss and mortality was provided for spanner crabs (*Ranina ranina*) by Kennelly *et al.* (1990), tanner crabs (*Chionoecetes bairdi*) by Stevens (1990), and for stone crabs (*Menippe mercenaria*) by Simonson and Hochberg (1986). In those studies, it was suggested that the location and severity of wounding (e.g. wound width) were significant predictors of mortality; typically because of the associated blood loss when an appendage break is not distal to the fracture plane. In contrast, many crabs that shed appendages at the fracture plane (autotomy) are able to reduce wounding, blood loss, and hence mortality (Juanes and Smith, 1995).

Portunids can readily shed their appendages in response to appropriate stimuli (Wood and Wood, 1932; Paterson *et al.*, 2007). At least some autotomy was observed in this study, because 84% of all wounds ($n = 252$) were sealed. In some cases, however, wound sealing may have been at least partially compromised, because the forceful appendage removal precluded a clean break at the fracture plane. This might explain the mortality of seven blue swimmer crabs with sealed wounds during Experiment 1. Moulting exacerbated the cheliped wound of the individual that died on the 10th day (in T4, Experiment 1). Other factors contributing towards mortalities may have included cumulative stressors associated with capture, entanglement, air exposure, and appendage removal. However, the first three factors were unlikely to be particularly severe, because there were no mortalities of any crabs that retained their appendages.

Unsealed wounds and the associated blood loss were the main predictors of immediate deaths (within 24 h). This type of wounding occurred when entire appendages, including the basal segment (coxopodite), were removed from the cephalothorax (causing an immediate exchange of blood with the surrounding seawater), and it increased with the number of appendages removed. These injuries are comparable with a punctured carapace, which nearly always causes immediate mortality among crustaceans (Wassenberg and Hill, 1993; Ferner *et al.*, 2005). One single dead blue swimmer crab with an unsealed wound after 7 d and the rare occurrence of moults (and therefore tissue regeneration) suggest that, for at least some crabs, wounds may not seal over time.

The results from Experiment 2 support the potential for negative effects of unsealed wounds and associated acute blood loss on the physiology of blue swimmer crabs. Inevitably, with the loss of blood, the numbers of haemocyte cells and protein concentration (measured as refractive index) were reduced, and the blood-clotting process was protracted. The same negative relationship between haemocyte cells and blood-clotting times was observed for western rock lobsters (*Panulirus cygnus*) exposed to air (by Fotedar *et al.*, 2001) and exercise (by Jussila *et al.*, 2001). In accord with these studies, our results indicate that haemocyte

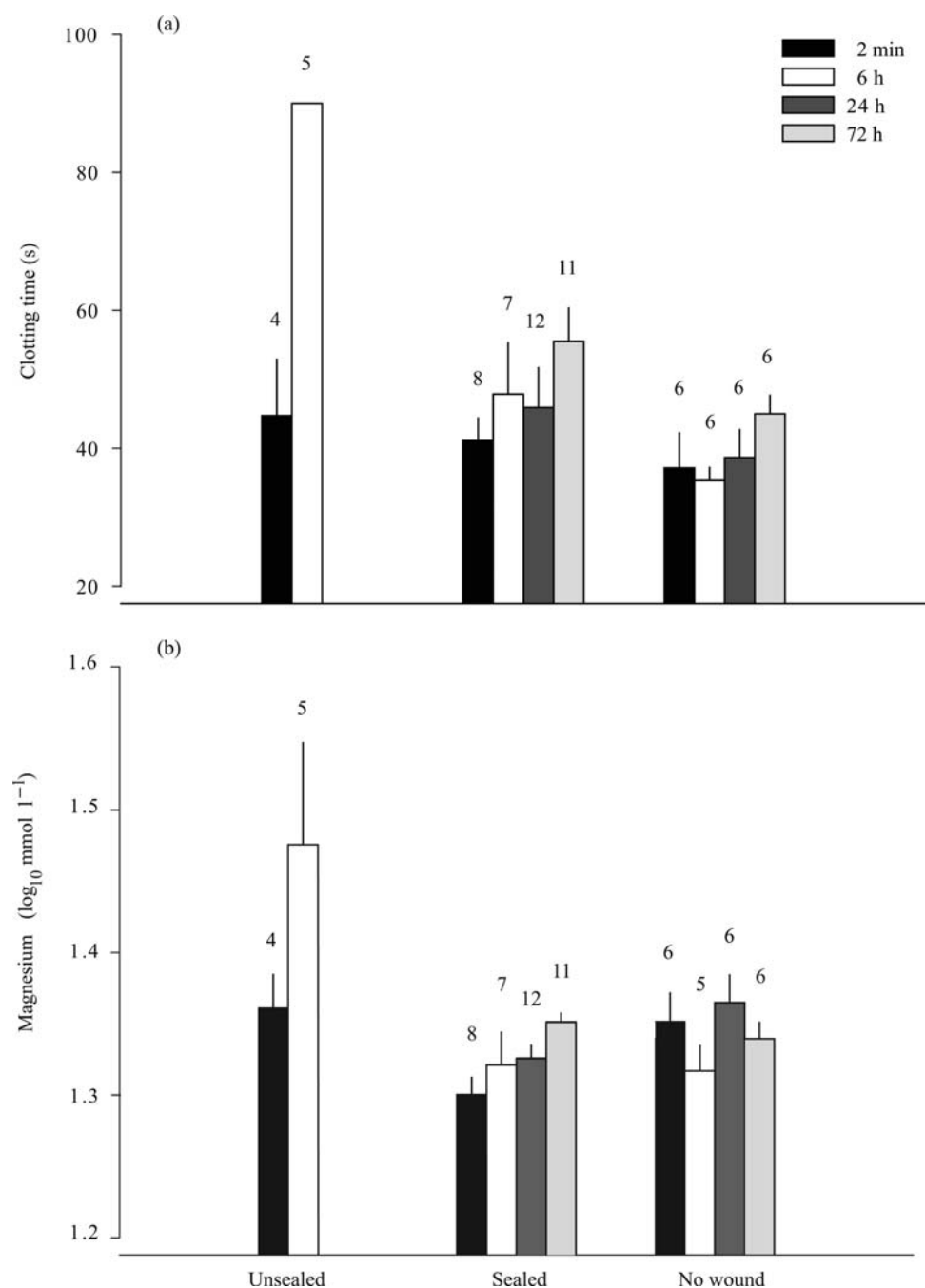


Figure 3. Means (+s.e.) of (a) blood-clotting times and (b) magnesium ion concentrations of blue swimmer crabs with unsealed, sealed, and no wounds, 2 min, and 6, 24, and 72 h after selection and/or discarding. The numbers of crabs at each sampling time are given above each histogram.

Table 1. Summaries of *F*-ratios from unbalanced analyses of variance to determine the effects on the physiology of blue swimmer crabs attributable to wound type (unsealed, sealed, or no wound) and sampling time (2 min, and 6, 24, and 72 h) in Experiment 2.

Parameter	d.f.	Haemocyte count (10 ⁶ cells ml ⁻¹)	Clotting time (s)	Refractive index	Calcium (mmol l ⁻¹)	Magnesium (log ₁₀ mmol l ⁻¹)	Potassium (mmol l ⁻¹)	Sodium (mmol l ⁻¹)
Wound type	2	4.0*	16.4**	7.0**	5.5**	11.0**	2.6	3.2*
Sampling time	3	1.8	4.9**	0.4	1.7	1.2	11.4**	1.1
Interaction	4	0.7	4.1**	0.8	1.9	2.9*	1.7	0.3

The residual degrees of freedom were 61 for haemocyte count, clotting time, and refractive index, and 58 for the remaining variables.

p* < 0.05; *p* < 0.01.

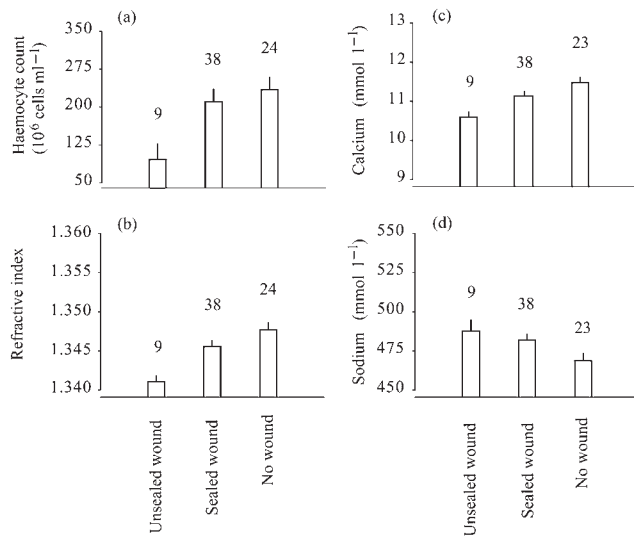


Figure 4. Means (+s.e.) of physiological parameters: (a) total haemocyte count; (b) refractive index; (c) calcium; and (d) sodium ion concentrations of blue swimmer crabs with unsealed, sealed, or no wound in Experiment 2. The numbers of blue swimmer crabs for each wound type are given above each histogram.

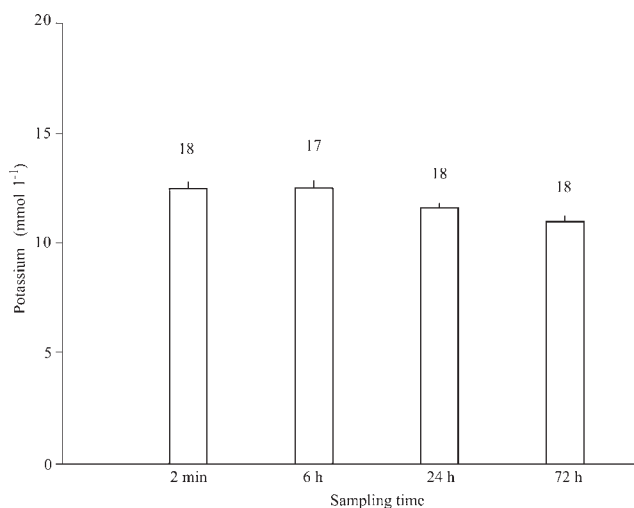


Figure 5. Means (+s.e.) of potassium ion concentrations of blue swimmer crabs sampled for blood 2 min, and 6, 24, and 72 h after selection and/or discarding. The numbers of blue swimmer crabs at each sampling time are given above each histogram.

cells are involved with the blood-clotting cascade and that clotting time is an easily measured indicator of physiological stress in blue swimmer crabs.

Interestingly, clotting times increased for blue swimmer crabs with sealed or no wounds up to 72 h after the start of Experiment 2. Increasing clotting times following up to 4 d after capture were also observed by Fotedar *et al.* (2006), who examined the effects of holding duration on this parameter for western rock lobster. Factors that may have contributed towards this delayed physiological response in blue swimmer crabs could include the exposure to artificial light and/or a lack of shelter in the experimental tanks.

Previous studies have shown that a loss of blood in crustaceans can alter concentrations of ions and, therefore, that ions may be used as indicators of physiological stress (Paterson *et al.*, 1997, 2005). However, the exact mechanism of ionic change is difficult to explain. Although the elevation of blood magnesium levels in open-wounded crabs 6 min after injury is consistent with the entry of magnesium-rich seawater (Figure 3b), the changes observed in other ions may have a physiological cause. As a weak hyperosmotic regulator (Romano and Zeng, 2006), blue swimmer crabs would have a similar, or even slightly higher, blood sodium level than the medium, so the further rise in blood sodium in wounded crabs is unlikely to be seawater contamination. Moreover, some calcium may “disappear” from these anaemic crabs because the circulating protein normally carries a significant fraction of calcium ions (Greenaway, 1985). The temporal decrease of potassium ions, which are normally tightly regulated in crustacean blood (Paterson *et al.*, 1997), indicates a delayed physiological response. Irrespective of the treatment, this may be due to the same environmental holding conditions, which could be responsible for the contrary relationship with clotting times.

Notwithstanding the above, any reduction in wounding and associated blood loss would benefit the survival of discarded blue swimmer crabs and hence contribute towards their commercial sustainability. Further, this would address obvious welfare issues associated with discarding the species. Such an outcome could be achieved if undersized crabs are carefully removed from meshes. Care should be given in particular to entanglements around the fifth pereopods, because damage to these has the potential to be more harmful than damage to the chelipeds (Figure 1). To reduce potential injury to fishers (from the chelipeds), entangled crabs might be pacified by placing them into ice slurries for 30 s (Bellchambers *et al.*, 2005), although the impacts of such a treatment on survival and any associated physiological impacts would need to be investigated. If the removal of an appendage is inevitable, autotomy could be induced by using pliers and exerting pressure to or cutting off the appendage distal to the fracture plane (Bergmann and Moore, 2001; Patterson *et al.*, 2007). Induced autotomy has been demonstrated to reduce significantly the discard mortalities in the edible crab (*Cancer pagurus*; Patterson *et al.*, 2007) and the squat lobster (*Munida rugosa*; Bergmann and Moore, 2001). Further research is required to establish appropriate techniques for blue swimmer crabs, because very little is known about their responses to stimuli that evoke autotomy, the associated wounds, and subsequent regeneration and recovery.

Although the survival of discarded blue swimmer crabs may be improved by induced, rather than forced, autotomy, induced appendage loss could still have some functional and ecological cost by impairing an individual's ability to evade predation (Potter *et al.*, 1991), forage, grow, and reproduce (Juanes and Smith, 1995). By not accounting for these effects and other cumulative *in situ* impacts of gear-, capture- and post-capture parameters (e.g. mesh configurations, gear deployment duration, soak time, retrieval techniques, inter- and intraspecific competition, and predation), the mortality estimates derived here should be considered as conservative.

Given the above, the results from this study should not be extrapolated to estimate damage and discard mortality rates of blue swimmer crabs caught in gillnets throughout NSW. However, by simulating the capture of blue swimmer crabs, then

exposing them to air and removing appendages during disentanglement (which are common procedures employed by most commercial and recreational fishers), the results are nevertheless indicative of the effects on short-term mortality and sublethal stress.

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