# RIVER WATER AND SEDIMENT REDUCE THE TOXICITY OF DELTAMETHRIN TO *PARATYA AUSTRALIENSIS*

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## ABSTRACT

Deltamethrin is a pyrethroid insecticide used extensively to control cotton pests in Australia and worldwide. Deltamethrin readily binds to organic and particulate matter in the environment, thereby reducing its bioavailability and toxicity, yet most toxicity data come from studies using clean, organic matter-free water that were conducted under conditions that differ greatly from those in the turbid rivers of the cotton-growing regions of Australia.

The aim of this study was to assess the toxicity of deltamethrin to the native glass shrimp, *Paratya australiensis*, and to consider the role of suspended and bottom sediment in the amelioration of deltamethrin toxicity. We conducted a series of acute single-species toxicity tests in the laboratory and in the field in the Namoi-Gwydir cotton region of northwest New South Wales, Australia.

The toxicity of deltamethrin was significantly ($p \leq 0.05$) reduced in river water compared with that in laboratory water in laboratory but not field-based tests. The toxicity of deltamethrin in river water was further reduced with the addition of bottom sediment. Despite reductions in toxicity in natural waters, deltamethrin remained highly toxic (i.e. 60-h EC50 values <200 ng/L) to *P. australiensis*, and thus further investigation of the hazard of deltamethrin is warranted.

**Key words:** *Paratya australiensis*; deltamethrin; laboratory-field comparison; river water; sediment.

## INTRODUCTION

In Australia, the pyrethroid insecticide deltamethrin is an integral component of pesticide resistance management strategies for cotton pests (Farrell and Johnson 2005). The dependence on deltamethrin for cotton production, and the proximity of cotton farms to rivers to facilitate irrigation, poses a substantial risk of contamination for aquatic ecosystems. The off-site migration of deltamethrin and contamination of river water has been linked to effects on riverine biota (Everts et al. 1983; Brooks 1998). Deltamethrin is the most toxic of the cotton pyrethroids across all groups of aquatic organisms (Solomon et al. 2001), and is particularly toxic to aquatic crustaceans (Caquet et al. 1992; Solomon et al. 2001; Beketov 2004).

Deltamethrin is lipophilic (log P = 4.6) (Tomlin 1994). It has low aqueous solubility and preferentially moves out of water to bind with dissolved organic matter and sediments in natural systems (Tomlin 1994). As a result, toxicity is often reduced in natural waters compared with clean, filtered waters (Karim et al. 1985; Day 1991; Muir et al. 1994; Tomlin 1994). The influence of binding processes on toxicity is dependent upon site-specific factors governing the quality and quantity of particulate material in the system (Murphy et al. 1990; Yang et al. 2006a). Consequently, the reduction in deltamethrin toxicity due to sediment binding is difficult to predict and therefore, toxicity tests that do not take account of dissolved organic matter, suspended and bottom sediments, and natural environmental conditions may provide inaccurate estimates of its toxicity in the field. Despite this, most of the toxicity data available for deltamethrin are based on northern hemisphere laboratory studies using clean, organic matter-free water; conditions that differ greatly from those in the rivers of the cotton-growing regions of Australia, which are characterised by high levels of total dissolved solids and suspended sediments (Williams and Wan 1972; Olive and Walker 1982).

In this study we conducted a series of acute toxicity tests in the laboratory and field using the glass shrimp, *Paratya australiensis* Kemp 1917 (Decapoda: Atyidae). Field tests were conducted in the Namoi River in the Namoi-Gwydir cotton region of north-west New South Wales, Australia, with the aim of generating site-specific toxicity data for this region. The specific aims of this study were to determine:

- the toxicity of deltamethrin to *P. australiensis* in river water compared with that of laboratory water;
- the effect of river sediment on the toxicity of deltamethrin to *P. australiensis*; and

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- the effect of *in situ* testing (under natural environmental conditions) on the toxicity of deltamethrin to *P. australiensis*.

## MATERIALS AND METHODS

### Test solutions

Technical grade (≥98% a.i.) deltamethrin ((*S*)-α-cyano-3-phenoxybenzyl (1*R*,3*R*)-3-(2,2-dibromovinyl)-2,2-dimethyl-cyclopropanecarboxylate; CAS no. 52918-63-5) was provided by Rhone-Poulenc. Stock solutions of 40 mg/L and 400 mg/L were prepared in nanograde methanol and stored in the dark at 4°C. Due to difficulties in analysing concentrations of deltamethrin in turbid river water, all concentrations are reported as nominal values.

### Test organism

The glass shrimp, *Paratya australiensis,* is the most common atyid in south-eastern Australia (Walsh and Mitchell 1995). *Paratya australiensis* are small, transparent and quick-moving animals, which live in a wide range of ecological conditions (Richardson et al. 2004) but seemingly prefer still littoral habitats (Richardson and Cook 2006). This species is an omnivorous scavenger-browser that feeds on detrital and particulate material (Richardson et al. 2004). Field-collected *P. australiensis* are widely used in Australian toxicity tests (e.g., Daly et al. 1992; Phyu et al. 2005; Hose and Wilson 2005).

### Field toxicity tests

The field tests were conducted in the Namoi River, upstream of Gunnedah (30° 58' 22" S, 150° 20' 55" E) in northwest New South Wales, Australia. The Namoi River valley is intensively farmed with irrigated and dryland cotton, especially in the mid to lower reaches. Our study site was upstream of irrigated cotton growing areas in the catchment but had similar altitude, climatic, geological and hydrological characteristics to the cotton-growing reaches of the Namoi River (Thoms 1999). Previous analyses of water samples from the site indicated the water was free from pesticides used in cotton growing including organochlorine and organophosphate pesticides (Muschal 1997). River water typically contained 7 mg/L DOC, which accounted for 87% of the TOC in the water (Westhorpe et al. 2008). Bottom sediment was collected from the main channel of the Namoi River at the study site. Sediment collected for this study and used in toxicity tests typically had <1% TOC (Leonard et al. 2001).

To achieve the aims of this study it was necessary to conduct toxicity tests for this pesticide using laboratory water and river water with and without sediment. Due to logistical constraints it was not possible to conduct all three experiments concurrently. Therefore, two sets of experiments were conducted. The first set compared the toxicity of deltamethrin in Namoi River water (RW) with that in filtered laboratory water (LW). The second set compared the toxicity of deltamethrin in Namoi River water containing a 1 cm deep layer (200 g ± 10 g wet weight) of fine settled Namoi River sediment (RW+Sed) with that of laboratory water (LW II).

Laboratory water (LW) was Sydney tap water that had been passed through a mixed-bed filter, activated carbon filter, 5 µm pore size filter, a second activated carbon filter and finally a UV steriliser. This water was transported from the Sydney laboratory to the field site. The first (LW I) and second (LW II) tests were used as reference tests for the RW and RW+Sed tests, respectively. If the outcomes of the LW I and LW II tests were not significantly different ($p>0.05$), temporal variations in test conditions and/or the sensitivity of the test organisms would be judged to have not significantly affected deltamethrin toxicity, and direct comparisons could then be made between the RW and RW+Sed tests.

*Paratya australiensis* were collected from the Namoi River at the study site and animals 1.5 – 2.0 cm long were used for the toxicity tests. The animals were acclimated for at least 48 h prior to testing and during this period were fed Seramin® fish flakes, but were deprived of food for ten hours prior to and during the toxicity tests.

Tests were conducted in 1-L beakers that contained 800 mL of test solution. Each experiment consisted of seven treatments - five nominal deltamethrin concentrations (i.e., 10, 26, 71, 190, and 500 ng/L deltamethrin), a control and a solvent control treatment, each with three replicates. Treatment concentrations for field and laboratory tests were chosen to cover the range of deltamethrin concentrations commonly reported in the environment (Pawlisz et al. 1998; Moraes et al. 2003; Feo et al. 2010). Five animals were randomly allocated to each replicate.

Deltamethrin was injected below the water surface using a solvent-rinsed borosilicate glass bore microsyringe and the water stirred gently to aid initial dispersal. Each treatment and the solvent control received the same volume (40 µL) of solvent. Test vessels were sealed with plastic film to reduce volatilisation and evaporation of the test chemical. The beakers for both tests in each test set were arranged randomly in plastic crates. The crates were tied together and tethered to the shoreline so that the beakers were approximately 75% submerged in the river. Beakers were covered with 90% shadecloth because *P. australiensis* seek shade during daylight hours. The shadecloth also moderated the temperature of the test water.

The beakers were not aerated during the tests to avoid disturbing bottom sediments in the RW+Sed test. Instead, test solutions in the beakers were renewed every 12 h to maintain dissolved oxygen concentrations at riverine levels throughout the test. The water for renewal solutions was drawn from a swiftly flowing region of the river. At each renewal, physico-chemical variables and turbidity were monitored (see Appendix 1), and immobilised animals were recorded and removed. Immobilisation was defined as the failure of appendages such as swimmerets, gills, and antennae to move within five seconds of being gently prodded. As the immobilised state was usually accompanied by muscular opacity (indicative of denatured protein), death, rather than chemical knockdown, could be assumed for most immobilised organisms. Preliminary research indicated there was no detectable change in toxicity with exposure beyond 60 h (up to 96 h) in river water so all tests were limited to 60 h duration.

Field tests were conducted in autumn during a period of moderate river flow. The natural photoperiod was 12:12 h light:dark. The conductivity of the RW during the first set of experiments was 550 – 650 µS/cm but ranged from 750 – 950 µS/cm during the second set of experiments. An additional laboratory test was conducted to test the effect of this conductivity change on deltamethrin toxicity (see below).

### Laboratory toxicity tests

Separate laboratory tests were conducted using both LW and RW. River water was collected from the field study site and transported to the laboratory in Sydney. Colloidal aggregates form in river water samples soon after collection so one-litre aliquots of river water were sonicated (Branson Sonifier 450, at maximum power for five minutes) to break up colloidal aggregates prior to using the water for toxicity testing (Leigh and Hyne 1999). Sonicated aliquots of water were combined and allowed to cool for an hour before use. Water treated in this way is, hereafter, referred to as resuspended river water ($RW_{Resus}$).

Three laboratory tests were conducted using both LW (two tests) and $RW_{Resus}$ (one test) to allow comparison with the field tests. The first LW test was conducted with water at 200 µS/cm (hereafter referred to as $LW_{200}$). A second test was run using LW with its conductivity adjusted to 750 ± 50 µS/cm by adding clean seawater treated with 1 µm filtration and ultraviolet irradiation. This conductivity-adjusted water is, hereafter, referred to as $LW_{750}$. The $LW_{750}$ test was conducted in order to have the same conductivity as the RW during the second set of field tests.

Animals collected from the Namoi River appeared stressed after the 6-h road trip to the laboratory and so were not used in laboratory tests. Instead, *P. australiensis* used for laboratory testing were collected from the upper Colo River (33° 26' S, 150° 51' E) located approximately 100 kilometres north-west of Sydney, Australia. The site of collection is immediately downstream of the Wollemi National Park. This river is a "Protected river" indicating it is one of the cleanest and least polluted rivers in NSW, Australia (NSW Dept. Environment and Planning 1983; Birch et al. 1998). The animals were acclimated to laboratory conditions for one week prior to use in toxicity tests. *Paratya australiensis* were fed Seramin® tropical flake food during the acclimation period, but were not fed 24 hours prior to or during testing. Animals used for laboratory tests had the same size range (1.5 - 2 cm long) as those used in the field tests.

Laboratory tests were conducted at 23 ± 1°C with a 16:8 h light:dark regime. Test solutions were renewed every 48 hours and water quality of new and old solutions was monitored at each change (see Appendix 1). As in the field, tests were conducted in one-litre beakers that contained 800 mL of test solution and were covered with transparent plastic film. All laboratory tests involved a control, a solvent control, and five treatment concentrations. The nominal deltamethrin concentrations used for $LW_{750}$ and $RW_{Resus}$ tests were 10, 26, 69, 190 and 500 ng/L with each concentration having three replicates and five randomly allocated animals per replicate. The nominal deltamethrin concentrations used for the $LW_{200}$ test were 10, 20, 60, 160 and 400 ng/L. Toxicant was added as per field tests, with each treatment and the solvent control receiving the same total volume (40 µL) of solvent.

### Statistical analysis

Concentration response curves were estimated by fitting a two-parameter non-linear regression function with a binomial error structure using the DRC package (Ritz and Streibig 2005) in R version 2.10.1 (R Development Core Team 2009). Weibull, log-logistic and log-normal models were fitted to each dataset, with the best-fitting model selected based on Akaike's Information Criterion. The model parameters were estimated using maximum likelihood, with starter values determined by the program's self-starter function. Concentrations affecting 10 and 50% of the shrimps (EC10 and EC50 values) were estimated from the fitted curve. EC10 and EC50 values were compared among tests using ratio tests (Wheeler et al. 2006). The significance level for the ratio test was 0.05.

## RESULTS

### Field toxicity tests

Deltamethrin was highly toxic to *P. australiensis.* Mortality in the control and solvent control treatments was less than 7% in all tests (Appendix 1). There was no significant difference in the EC50 and EC10 values of the RW test compared to the LW I test, indicating that river water alone did not reduce the toxicity of deltamethrin under field conditions (Table 1). However, there was an approximately four-fold, reduction in the toxicity (increased EC10 and EC50 values) of deltamethrin in the RW+Sed test compared with that of the LW II test (Table 1).

The EC10 and EC50 values of the LW I and LW II tests were not significantly different ($p>0.05$). Therefore, the significant differences in the EC50 values between the RW and RW+Sed tests (Table 1) could be ascribed to the presence of sediment, rather than temporal differences between the two test series.

### Laboratory toxicity tests

The toxicity of deltamethrin in the $LW_{200}$ test was only recorded at 48 and 72 h, not at 60 h. In the $LW_{200}$ test, only the 60 ng/L treatment exhibited any increase in mortality (from 3 to 5 dead shrimps) between 48 and 72 h. We, therefore, calculated the geometric mean of mortality in each treatment between 48 and 72 h as an estimate of mortality at 60 h and used these data to estimate a 60-h EC50 value. The estimated 60-h EC50 value is presented in Table 1 and was used in all subsequent comparisons of EC50 values.

There was no mortality in the control and solvent control treatments in all tests (Appendix 1). There was no significant ($p>0.05$) difference in the 60-h EC10 or EC50 values from the $LW_{200}$ and $LW_{750}$ tests (Table 1) suggesting that the change in conductivity did not affect toxicity. There was also no significant difference in the 48- and 72-h EC50 values from the $LW_{200}$ test and the 60-h EC50 value from the $LW_{750}$ test ($p>0.05$). Given this, the estimated 60-h EC50 value from the $LW_{200}$ test is considered suitable for further comparisons in this study.

**Table 1.** Summary of 60-h median effect (EC50) values for the toxicity of deltamethrin to *P. australiensis* in different exposure media. Superscript letters within a column denote significant differences ($p<0.05$) between EC values. All values in (ng/L).

| | EC10 | 95% CI[#] | EC50 | 95% CI |
|---|---|---|---|---|
| **Field tests** | | | | |
| Laboratory Water (LW I) | 27[a] | 22 - 33 | 38[a] | 29 - 48 |
| River Water (RW) | 16[a] | 7 - 24 | 46[a] | 30 - 62 |
| Laboratory Water (LW II) | 29[a] | 16 - 42 | 42[a] | 23 - 61 |
| River Water + Sediment (RW+Sed) | 157[a] | 0 - 457 | 181[c] | 154 - 220 |
| **Laboratory tests** | | | | |
| Laboratory Water - 200 µS/cm ($LW_{200}$)* | 13[a] | 5 - 22 | 42[a] | 24 - 60 |
| Laboratory Water - 750 µS/cm ($LW_{750}$) | 28[a] | 17 - 40 | 46[a] | 34 - 59 |
| Resuspended River Water ($RW_{Resus}$) | 59[a] | 0 - 132 | 70[b] | 63 - 76 |

*60-h EC values based on geometric mean mortality of 48- and 72-h mortality data. # Negative lower 95% CI values reported as zero.

The toxicity of deltamethrin was significantly reduced ($p<0.05$) in the $RW_{Resus}$ test compared with that of the LW tests which were conducted under laboratory conditions. This was evident as a significant difference in the EC50 values, but not the EC10 values (Table 1).

### Laboratory vs field tests

There was no significant difference in the EC50 values of the LW I and $LW_{200}$ tests (Table 1, $p>0.05$), suggesting that the toxicity of deltamethrin in laboratory water was similar under laboratory and field conditions. However, there was a significant difference in the EC50 values of the RW and $RW_{Resus}$ tests (Table 1, $p<0.05$), with deltamethrin being more toxic in laboratory than field-based tests.

## DISCUSSION

### Conductivity effects

In the laboratory, changes in conductivity (i.e., $LW_{200}$ vs $LW_{750}$) did not alter the toxicity of deltamethrin to *P. australiensis*. Similarly, Thomas et al. (2008) also found no difference in deltamethrin toxicity to cladocerans, shrimp and fish over a similar conductivity range, and Dyer et al. (1989) reported no significant difference in the toxicity of fenvalerate to bluegill sunfish (*Lepomis macrochirus*) as conductivity increased from 431 to 735 µS/cm. The change in conductivity we observed spans the range of conductivities expected at the field site. Wood (1997) reported the median and maximum conductivities at a nearby site on the Namoi River as 359 and 816 µS/cm, respectively, during the year of the study. Similar ranges have been recorded since (Gordon 2000; 2001).

### Laboratory vs field comparison

There was no significant difference in the toxicity of deltamethrin in laboratory water in the laboratory or field (LW I vs $LW_{200}$). This suggests that the *P. australiensis* populations from the Namoi and Colo Rivers did not differ in their sensitivity to deltamethrin (cf. Olima et al. 1997), which is consistent with populations from these locations being of a similar genotype (Cook et al. 2006). However, response of *P. australiensis* in river water differed between the laboratory and field, a finding at odds with that of Hose et al. (2003) who showed laboratory or field test conditions did not affect the responses (LC50 values) of the mayfly *Atalophlebia* spp. when exposed to endosulfan in Namoi River water. We suspect that the difference in LC50 values between $RW_{Resus}$ and RW tests is due to the increased time between renewal that was used in the laboratory (48 h) compared with that of field tests (12 h). The less frequent renewal period would increase the amount of deltamethrin that would bind to the organic matter in the river water and the glass of the test containers and thus decrease the bioavailable concentration and hence toxicity of deltamethrin to *P. australiensis*.

### Effect of suspended and bottom sediment

Deltamethrin toxicity was significantly lower in laboratory tests using river water (which contained suspended sediments) compared with that of clean laboratory water. Similarly, Thomas et al. (2008) showed a significant reduction in deltamethrin toxicity to *Ceriodaphnia dubia* in river water compared to clean laboratory water, although no such conclusion was drawn for the rainbowfish, *Melanotaenia duboulayi.*

The inclusion of bottom sediment in field tests with river water significantly ($p<0.05$) reduced the toxicity of deltamethrin (measured as EC50). This reduced toxicity is likely to have occurred through decreased bioavailability (Yang et al. 2006a, b). Indeed, the adsorption of hydrophobic organic contaminants, including pyrethroids, to particulate organic matter can significantly reduce their bioavailability without saturating the sorbent (Garbarini and Lion 1986; House and Ou 1992). Day (1991) showed that 60-80% of the deltamethrin added to test solutions became bound to organic carbon. Reductions in deltamethrin toxicity of between 2.5 and 13-fold have been reported as a result of sorptive processes (Day 1991; Yang et al. 2006b).

At the same time, the reduced bioavailability of deltamethrin may occur through increases in its degradation. The presence of suspended and bottom sediments may increase the degradation rate of deltamethrin via humus-mediated photosensitisation, as has been reported for other pyrethroid insecticides (Jensen-Korte et al. 1987). Alternatively, the reduced toxicity of deltamethrin in the presence of sediments may be due to the degradation of the compound by bacteria (e.g., Haider 1983; L'Hotellier and Vincent 1986; Das and Mukherjee 1999). Because of the close relationship between microorganism population size and the amount of dissolved or particulate organic material in water (Rao et al. 1991), the presence of suspended and bottom sediments may reduce toxicity indirectly through the degradation of deltamethrin by the associated microbial community.

Elsewhere, the bioaccumulation and bioconcentration rates of deltamethrin, and other pyrethroids in the chironomid, *Chironomus tentans*, were significantly reduced in the presence of sediment and particulate matter (Muir et al. 1985). The toxicity of formulated deltamethrin was also reduced in the turbid water of field tests compared with laboratory water tests for several species of fish and macroinvertebrates (Karim et al. 1985; Day 1991). Likewise, we have also shown a significant reduction in the toxicity of deltamethrin in natural waters in the laboratory.

By using nominal concentrations, we have no certainty of the actual exposure concentrations; however, it is most likely that the actual exposure concentrations are below the nominal values given the likely adsorption of deltamethrin onto glassware (Sharom and Solomon 1981; Day 1991). Day and Kaushik (1987) suggested that the loss of pyrethroids by adsorption to glass beakers in bioassays is around one-third after 48 h. Indeed, our preliminary data suggest similar losses (37%) after 48 h, but smaller losses of 6% after 8 h and 10% after 24 h (Thomas 2001). This might suggest that losses by adsorption should be greater in the laboratory tests compared to the field tests and may explain the reduction in toxicity in river water seen only in laboratory tests. Assuming an exponential rate of loss of deltamethrin, the geometric mean concentrations (and hence toxicity estimates) could be around 16% and 5% lower than nominal concentrations in the laboratory and field tests, respectively. The consequence of overestimating our test concentrations by using nominal concentrations is that we are likely to have underestimated the toxicity of deltamethrin.

Existing data suggest a broad range of sensitivities to deltamethrin among crustaceans. McKenney and Hamaker (1984) reported that >50% of estuarine grass shrimp larvae (*Palaemonetes pugio*) died within 96 h of continuous exposure to (nominal) 3.2 ng/L fenvalerate. This value is an order of magnitude lower than that obtained for deltamethrin to *P. australiensis*, and may be due to differences in species, life stage, salinity or test chemical. The findings of L'Hotellier and Vincent (1986) were at the other extreme, reporting a 96-h LC50 of 350 ng/L for formulated deltamethrin to the marine prawn *Penaeus duorarum*. Barata et al. (2006) reported a measured 48-h LC50 for deltamethrin exposure to *Daphnia magna* in clean water of 157 ng/L which contrasts with the 48-h EC50 of 20 ng/L for *C. dubia* exposed in clean water (Thomas et al. 2008).

Clark et al. (1989) measured the mortality of mysids and grass shrimps over ten days, and reported that mortality only occurred at sediment pyrethroid concentrations that were high enough to contaminate overlying water via sediment/water partitioning. They also reported that direct contact with or ingestion of contaminated sediment did not appear to enhance the toxicity of fenvalerate or cypermethrin to mysids, grass shrimps or pink shrimps (Clark et al. 1989 but see Schulz and Liess 2001). This is consistent with our findings that *P. australiensis* was less sensitive to deltamethrin in the presence of bottom sediment, despite its non-selective, omnivorous, scavenging and filter-feeding habit (Richardson et al. 2004), and high likelihood of ingesting contaminated sediment.

Cairns et al. (1984) and Adams et al. (1985) similarly claimed that the dissolved fraction of most hydrophobic xenobiotics appears to be more available to benthic macroinvertebrates than sorbed fractions. For relatively short-lived compounds such as deltamethrin (half-life in soil <23 days; Tomlin 1994), exposure from ingested sediments may not be a critical exposure pathway. Investigation of this aspect of the ecotoxicology of deltamethrin is required, including an assessment of chronic toxicity of deltamethrin in RW to sensitive organisms.

Future studies of deltamethrin should consider the influence of suspended and bottom sediments in reducing toxicity. From our results, it is not surprising that pyrethroids were less toxic in field-based mesocosm tests than predicted by laboratory studies (Giddings et al. 2001). Because mesocosm studies often include natural water and sediment, they should be particularly useful for setting safe environmental levels for pyrethroids. Risk assessments that fail to consider sediment interactions will overestimate deltamethrin toxicity; our field results suggest by a factor of approximately four. While basing environmental protection targets on toxicity data from clean-water tests is conservative, the costs to industry to meet tighter targets may be high.

## CONCLUSIONS

The toxicity of deltamethrin was significantly reduced in river water compared to clean laboratory water which did not contain suspended sediments. This trend was apparent in laboratory but not field studies. Field-based studies, however, showed a further reduction in the toxicity of deltamethrin to *P. australiensis* in river water with the addition of bottom sediment, probably due to adsorption to suspended and dissolved organic matter, and degradation processes. Despite reductions in toxicity with natural waters and sediments, deltamethrin remained toxic (i.e., 60-h EC50 values <200 ng/L) to *P. australiensis* at concentrations similar to those recorded in the field. Alarmingly, our use of nominal concentrations is likely to underestimate toxicity, suggesting that deltamethrin poses a significant ecological risk at or below current field-relevant concentrations.

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**Appendix 1.** Water quality and mortality in controls of laboratory and field-based toxicity tests using *P. australiensis*.

| | pH | Temperature (°C) | Conductivity (µS/cm) | Turbidity (NTU) | % Control Mortality (Solvent) |
|---|---|---|---|---|---|
| **Field tests** | | | | | |
| Laboratory Water (LW I) | 7.48 - 7.90 | 13.4 - 18.9 | 210 - 240 | 0 | 0 (7) |
| River Water (RW) | 7.97 - 8.18 | 17.0 - 18.6 | 550 - 650 | 11 - 34 | 0 (0) |
| Laboratory Water (LW II) | 7.64 - 7.98 | 12.8 - 18.0 | 210 - 260 | 0 | 0 (0) |
| River Water + Sediment (RW+Sed) | 7.92 - 8.17 | 13.2 - 16.7 | 740 - 950 | 11 - 34 | 0 (0) |
| **Laboratory tests** | | | | | |
| Laboratory Water - 200 µS/cm ($LW_{200}$) | 7.14 - 8.28 | 21.6 -22.9 | 178 - 184 | 0 | 0 (0) |
| Laboratory Water - 750 µS/cm ($LW_{750}$) | 7.94 - 7.99 | 23.1 - 24.6 | 710 - 810 | 0 | 0 (0) |
| Resuspended River Water ($RW_{Resus}$) | 7.78 - 8.25 | 23.2 - 24.2 | 694 - 752 | 11 - 20 | 0 (0) |