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Management and Ecological Note

Toxicity of rotenone to topmouth gudgeon *Pseudorasbora parva* for eradication of this non-native species from a tarn in Cumbria, England

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Rotenone is used in various parts of the world in fish population surveys and fisheries management (Ling 2003; McClay 2005), including the eradication of undesirable fishes (e.g. Amey 1981). In response to continued concern surrounding the risks and impacts of non-native fishes (Copp, Garthwaite & Gozlan 2005), use of this plant-derived piscicide in England and Wales has been considered as one possible measure, in support of existing government policy, to control the spread of non-native freshwater fishes (Defra 2003).

Probably the most invasive of the non-native species introduced to the UK since 1950 is the Asiatic cyprinid, topmouth gudgeon *Pseudorasbora parva* (Temminck & Schlegel), which poses a high overall risk (Copp *et al.* 2005) and a particularly serious fish disease risk (Gozlan, St-Hilaire, Feist, Martin & Kents 2005; Pinder, Gozlan & Britton 2005). The introduction of topmouth gudgeon to a tarn near Kendal, Cumbria, Northwest England (54°21′ N, 2°47′ W), probably as a contaminant of fish stocked into the tarn in 2000 and 2002, raised concern because this small natural lake (2 ha) is connected intermittently to a water course that passes through a Special Area of Conservation/Site of Special Scientific Interest (SAC/SSSI). For a variety of reasons (i.e. conservation importance of waters in the English Lake District, existing policy and legislation to control the spread of non-native fishes, angling club interest to have this undesirable species removed), it was deemed necessary to attempt eradication of topmouth gudgeon from this tarn using rotenone (Britton & Brazier 2006). However, the toxicity of rotenone to topmouth gudgeon is not known, and in light of the variable sensitivity of different fish species to rotenone (Gilderhus 1972), it would be undesirable to attempt any such eradication without prior verification that the control agent is effective and efficient. The aim of the present study was to assess the acute toxicity of rotenone to topmouth gudgeon so as to determine the effective rotenone concentration required for complete eradication of the species.

Topmouth gudgeon and water were collected from the tarn in early March 2005 and transferred to CEFAS' Burnham laboratory where they were maintained for a minimum of 14 days before tests began. The rotenone formulation PW Rotenon^{TM} (2.5% rotenone and 2.5% piperonylbutoxide) was used with tarn water (kept refrigerated and in the dark until required) because rotenone efficacy on a given species varies with environmental conditions such as temperature and pH (Ling 2003).

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Over a 5-week period in March to April 2005, two series of tests, range-finding (test concentrations: 0.025–0.500 mg L^{-1} rotenone; one replicate per concentration; three fish per replicate) and definitive (three replicates per concentration, 10 fish per replicate), were conducted in a constant temperature room of 12 ± 2 °C (i.e. a temperature range that matched that expected in the Tarn during the planned eradication procedure). Fish added to each test vessel were randomly selected from the stock population, which consisted of both juveniles and breeding adults (weights ranged from 0.8 to 3.5 g) to ensure that potential size or ontogenetic differences in sensitivity to rotenone were taken into account, and the effective concentration determined from the tests would be applicable to all life stages.

The starting point for the definitive tests was the minimum lethal concentration identified in the range-finding tests. The sequential design employed (Fig. 1) meant that a maximum of 30 fish would be tested at any single concentration to verify that the dose is effective for a representative sample of the population, i.e. accurate and applicable on a larger scale and over a wider distribution of sensitivities to be found in a population. The standard exposure time was 2 h, but longer exposures (up to 7 h) were also conducted in the definitive testing phase to determine, as closely as practical, the minimum dosages and exposure times necessary to achieve 100% mortality. Following the designated period of exposure, test fish were transferred to clean tarn water for 24 h to determine whether or not a moribund state ultimately resulted in death. The numbers of fish dead or recovered after the recovery period were noted. Death was defined as absence of any visible movement before or after stimulation with forceps (Organization for Economic Cooperation & Development 1992; Parrish 1995).

The physical characteristics of the rotenone-treated and clean tarn water varied little within and between tests: mean temperature = 10.5 °C (±0.5 °C), mean pH = 6.98 (±0.04), mean dissolved oxygen = 8.0 mg L^{-1} (±0.7), water hardness = 55 mg L^{-1} as $CaCO_3$ (no change throughout the tests). No mortalities occurred in the control vessels during any of the trials. In the first range-finding test, at 0.025 mg L^{-1} rotenone, no fish died during the exposure or recovery periods; at 0.125 mg L^{-1} rotenone, all fish survived the 120-min exposure, but all died during recovery; at 0.250 mg L^{-1} rotenone, one death occurred between 60- and 120-min exposure, and all others died during recovery; at 0.500 mg L^{-1} rotenone, one death occurred during the first 60-min exposure, and all remaining fish had died by the end of 120-min exposure. In the second range-finding test, at both 0.050 and 0.075 mg L^{-1} rotenone, no fish died during the exposure period, but one fish died during the recovery period at each concentration; at 0.100 mg L^{-1}, one death occurred during 60-min exposure, with all remaining fish dead after 120-min exposure. Fish that survived the rotenone exposure were not moribund but actively swimming.

In the definitive tests (Table 1), full recovery (active swimming) was observed in 30% of fish exposed to 0.125 mg L^{-1} rotenone for 2 h. No fish survived the recovery period after exposure for 2 h to 0.150 mg L^{-1} rotenone. This same result was observed at 0.125 mg L^{-1} rotenone when fish were exposed over longer periods (4 and 7 h) to determine the exposure

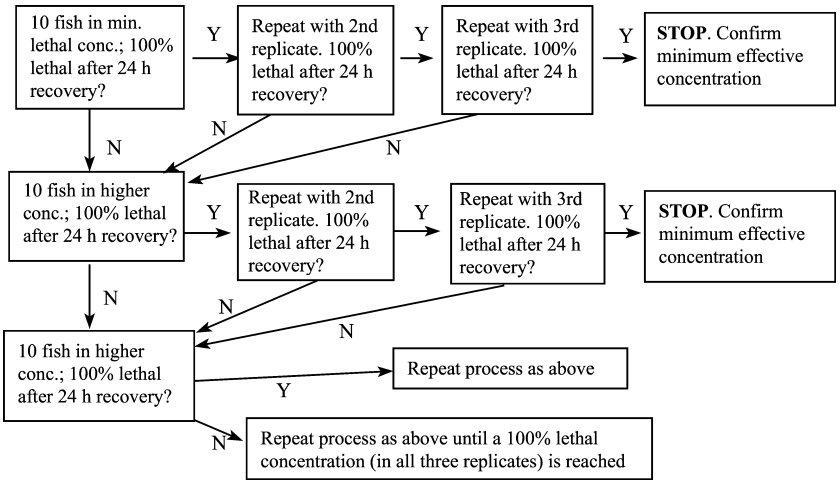

Figure 1. Experimental design used for definitive exposures of topmouth gudgeon to rotenone. The 100% lethal criteria is applied after the 24-h period in clean water.

Table 1. Definitive tests: mortality of 10 topmouth gudgeon per trial (weights ranged from 0.8 to 3.5 g) during replicate (subscript number) exposures to concentrations of rotenone ('PW Rotenon' formulation concentrations in mg L^{-1} given in parentheses) for 2-, 4- and 7-h exposure periods and 24-h 'recovery' period in clean water, given as numbers of dead fish at the end of the: exposure period/ recovery period

Concentration		Replicate					
		0.125 mg L^{-1}(5.0)			0.150 mg L^{-1}(6.0)		
Exposure (h)	Controls	1	2	3	1	2	3
2	0/0	0*/7	–	–	1*/10	10/10	10/10
4	0/0	10/10	10/10	10/10	–	–	–
7	0/0	10/10	10/10	10/10	–	–	–

*Remaining fish were alive but moribund.

time needed for this concentration to incite death. Therefore, for eradication procedures, 0.15 mg L^{-1} is the lowest concentration of rotenone that will result in 100% mortality of a representative sample (30 fish) of topmouth gudgeon over a 2-h exposure time. This concentration is intermediate in the range of recommended rotenone concentrations (0.10–0.20 mg L^{-1}) for removing common carp, *Cyprinus carpio* L., from ponds (Finlayson, Schnick, Cailteux, DeMong, Horton, McClay, Thompson & Tichacek 2000).

Topmouth gudgeon tolerance to rotenone (Table 1) may be greater than that of other cyprinid species, but this tolerance is dependent on exposure time, and 0.125 mg L^{-1} rotenone is sufficient for exposure periods ≥4 h. The toxicity threshold for a 4-h exposure period may be lower than 0.125 mg L^{-1}, however this requires further study. Therefore, for field applications of 2-h exposure, a concentration of 0.15 mg L^{-1} rotenone is required for 100% mortality of a representative sample of topmouth gudgeon. For exposure periods that exceed 4 h, 0.125 mg L^{-1} rotenone would appear to be sufficient to eradicate topmouth gudgeon if: (1) the piscicide is well mixed into the treatment area and (2) no reduction in effective concentration is experienced (e.g. due to organic matter content in the water). In England and Wales, many of the receiving waters suitable to topmouth gudgeon are likely to have elevated organic matter levels, so a higher concentration (e.g. 0.15–0.20 mg L^{-1}) may be necessary (Finlayson *et al.* 2000). Indeed, when rotenone was applied to the Cumbrian tarn at 0.30 mg L^{-1} rotenone, a final concentration of 0.25 mg L^{-1} was achieved. Three post-eradication fisheries surveys of the Cumbrian tarn have taken place, which has been restocked with fish removed from the tarn and quarantined to ensure no contamination by topmouth gudgeon. Data indicate total absence of the non-native species and good recruitment of native species (Britton & Brazier 2006). This suggests that the eradication has been successful but cannot be considered unequivocal until the survey period is completed in 2 years time.

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