Nordic J. Freshw. Res. (1995) 70: 21-30

Rotenone Tolerance in the Freshwater Pearl Mussel *Margaritifera margaritifera*

DAG DOLMEN[1], JO VEGAR ARNEKLEIV[1] and TROND HAUKEBØ[2]

[1] University of Trondheim, The Museum, N-7004 Trondheim, Norway.

[2] County Governor of Møre og Romsdal, N-6400 Molde, Norway.

Abstract

In connection with rotenone treatments of Norwegian rivers against the salmon parasite *Gyrodactylus salaris*, knowledge of the toxic effect of rotenone on the vulnerable freshwater pearl mussel *Margaritifera margaritifera* was needed. In a field experiment the mussels survived treatments with 5 ppm rotenone solution for 12 h. In a laboratory experiment the mussels survived 30 ppm for 12 h. At 40 ppm the mussels survived the treatment, but died less than a week later. The lethal concentration of rotenone for the freshwater pearl mussel, over a 12 h exposure period in the laboratory, is thus estimated at 30-40 ppm. Compared to fish, the freshwater pearl mussel is highly resistant to rotenone. Rotenone treatments, such as those carried out in Norwegian rivers to get rid of the salmon parasite (<5 ppm rotenone solution for <8 h), would not represent a threat to a population of the freshwater pearl mussel.

Keywords: Pearl mussel, *Margaritifera margaritifera,* rotenone, tolerance.

Introduction

The freshwater pearl mussel *Margaritifera margaritifera* (L.) is distributed throughout northern Europe, Eurasia and eastern North America (Wells et al. 1983, Collins and Wells 1986). The species has a many centuries' long tradition in Europe as a source of excellent pearls. For this reason many local populations were on the point of extinction during the latter half of the 18th century, e.g. in southern Norwegian rivers (Kleiven et al. 1989). The mussel shell makes a valuable record of long-term water qualities of the watercourse (Carell et al. 1987). Due to pollution, especially of lotic habitats, but also to over-collecting, it is now considered to be a vulnerable or endangered species in most European countries, and is therefore included in the Berne Convention app. III (Council of Europe 1992, cf. Collins and Wells 1986, United Nations 1991). In Sweden the number of reproductive mussel populations has declined drastically (Grundelius 1987, Bergquist 1993). Although the freshwater pearl mussel has been eradicated over wide areas of southern Norway today, probably because of acid precipitation (Dolmen and Kleiven 1993), its status seems still satisfactory in large parts of this country. Its distribution in Norway has been dealt with by Økland (1976) (cf. Kleiven et al. 1988).

A number of Norwegian rivers have been treated with rotenone (usually around 2 ppm) to exterminate the monogenean salmon parasite *Gyrodactylus salaris* Malmberg (e.g. Johnsen and Jensen 1986, Dolmen 1987, Johnsen et al. 1989, Direktoratet for naturforvaltning 1992). The use and effect of rotenone in fishery management in North America and Scandinavia have been dealt with by e.g. Soleman (1950), Quenild (1977), Tobiasson (1979), Fox (1985), Sousa et al. (ca. 1985-90), Næss et al. 1991, see also Haley (1978) and Ugedal (1986) for literature reviews.

This paper investigates the toxic effect of rotenone on the pearl mussel, which often occurs in Norwegian salmon rivers. Earlier experiments (e.g. Marking and Bills 1976) indicate a very high tolerance to rotenone in molluscs.

Material and methods

Sites of experimentation

Mussels for field experiments were collected from the upper reaches of the River Hustadelva, Fræna. Those used in the laboratory experiment came (for practical reasons) from the River Aureelva, Sykkylven. Both are medium-sized (ca. 2-5 $m^3 s^{-1}$) and unpolluted rivers with populations of the freshwater pearl mussel, locally up to approximately 100 $m^{2\,-1}$. The field experiment was carried out in the River Haukebøelva, Molde, which much higher up in the watercourse (Arstadelva) has a small population of the pearl mussel. The mussels from this experiment were afterwards released some few hundred meters above the study area. The water for the laboratory experiment was tap water from the Lake Jonsvatnet, near Trondheim, a watercourse in which also the mussel is known to live. The surviving mussels from the laboratory experiment were released into the Creek Trollbekken near Trondheim with no known mussel population. The geographical positions and some water quality parameters of the different localities are shown in Table 1.

The field experiment

The experiment in the River Haukebøelva took place in a small side channel on 5 September 1989. The water-flow could be manipulated and was kept constant during the experiment. The water temperature was about 11 °C, which is normal for Norwegian rivers in autumn when most rotenone treatments are carried out. Mussels collected from the River Hustadelva were placed into the River Haukebøelva in two baskets for acclimatization. The mussels (total 111) were

Table 1. Water quality and other biotope characteristics of sites where freshwater pearl mussels were collected, tested or released.

Locality UTM 32V	Date	pH	Cond. K_{25} µS cm^{-1}	Tot. hard. ° dH	Ca^{2+} mg L^{-1}	Alk. µeq L^{-1}	Cl^- mg L^{-1}	Water colour mg Pt L^{-1}	Temp. °C	Biotope: current, substrate etc.
Hustadelva	1986-08-05	7.1	25	1.22	-	0.31	-	-	-	Slowly-flowing;
MQ 080788	1989-09-06	6.9	97	1.3	6.8	-	13.5	20	12.3	sandy bottom; *Potamogeton perfoliatus*, mosses
Haukebøelva	1986-08-05	6.6	31	0.26	-	0.03	-	-	-	Slowly- and
MQ 003577	1989-09-05	6.4	35	0.2	1.4	-	6.5	50	11.4	swiftly-flowing;
(release: MQ 011586)										mud- and sandy bottom
Aureelva LQ 775212	1985-11-11	6.8	50	0.48	-	0.10	-	-	-	Slowly-flowing; sand and stones
Trollbekken	1990-12-04	-	60	0.85	4.3	-	6.75	40	-	Slowly-flowing,
NR 649361	1992-09-25	6.9	71	-	-	-	-	10	-	sand and gravel
Tap water (Jonsvatnet) NR 7829	1994-10-24	7.0	50	-	-	-	-	20	-	Source of exper. water

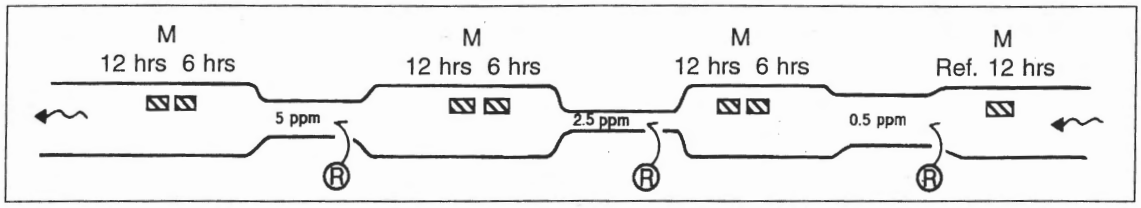

Fig. 1 The position of the baskets of mussels (M), the tanks of rotenone (R) and the different rotenone concentrations along the channel. The river current is from right to left. The distance between the upper and lower baskets is about 100 m.

grouped according to size: 83 large (12-13 cm), 24 medium-sized (9-11 cm) and 4 less than medium (6.5-7 cm). They were then put into seven baskets (cages, 30 x 50 x 25 cm), each containing 15-16 mussels of roughly equal size distribution. The mussels had no possibility to dig down into the substrate. Except for the control group, placed furthest up the river, two baskets were placed at each of three different downstream sites, at a depth of approximately 30-40 cm (Fig. 1). The water velocity varied between approximately 0.2 and 0.5 cm s^{-1} at the different sites. The rotenone solution of known concentration was emptied slowly into the water from tanks fitted with narrow plastic tubes. The river current mixed it well into the water-flow, as shown by help of rhodamine B (see later). The rotenone concentration in the channel was calculated on basis of the cross section of the channel, the water velocity at the site, as measured by help of rhodamine B, and the rate of rotenone release. The mussels were exposed to 0.5, 2.5 and 5.0 ppm of rotenone solution for 6 h (Table 2). After 6 h, one of each pair of baskets was removed from the channel and transferred to the site of the control group, while the second baskets were exposed to rotenone for a further 6 h. Rotenone treatments of Norwegian rivers last usually for 5-8 h.

Table 2. Response of *Margaritifera margaritifera* exposed to different concentrations of rotenone solution in the field experiment. No data = missing observation. Shaded area = mussels after having been transferred to fresh water.

h from start of exposure	Days after exposure	Number of open - narrow slit/closed mussels and responding mussels (in brackets)						
			Total exposure period 6 h			Total exposure period 12 h		
		Contr	0.5 ppm	2.5 ppm	5 ppm	0.5 ppm	2.5 ppm*	5 ppm
0.25		11- 5 (-)	7- 9 (-)	6-10 (-)	4-12 (-)	6-10 (-)	8- 7 (-)	4-12 (-)
4		-	8- 8 (15)	10- 6 (15)	4-12 (-)	10- 6 (15)	10- 5 (-)	7- 9 (-)
6		-	9- 7 (-)	6-10 (-)	3-13 (-)	-	-	-
8.5		-	-	-	-	6-10 (-)	6- 9 (-)	3-13 (-)
12		10- 6 (10)	14- 2 (14)	8- 8 (8)	4-12 (4)	10- 6 (10)	15- 0 (15)	12- 4 (12)
	1	13- 3 (16)	10- 6 (15)	5-11 (15)	7- 9 (15)	3-13 (16)	11- 4 (10)	4-12 (16)
	3	8- 8 (15)	8- 8 (14)	1-15 (16)	0-16 (16)	1-15 (16)	0-15 (15)	0-16 (16)
	7	6-10 (16)	0-16 (16)	0-16 (16)	3-13 (16)	3-13 (16)	8- 7 (15)	3-13 (16)
	11	6-10 (16)	5-11 (16)	8- 8 (16)	5-11 (16)	4-12 (16)	4-11 (15)	10- 6 (16)
	25	6-10 (16)	4-12 (16)	4-12 (16)	3-13 (16)	7- 9 (16)	8- 7 (15)	6-10 (16)
	55	4-12 (16)	0-16 (16)	3-13 (16)	2-14 (16)	3-13 (16)	4-11 (15)	3-13 (16)

* 15 mussels used in this group, 16 in all other groups

After the rotenone exposure, the mussels were kept in the cages and examined after 1, 3, 7, 11, 25 and 55 days. They were then released into the river and re-examined after 1 and 3 years in the river by divers.

During the experiment the mussels were observed at regular intervals, and the number of wide-open shells (the whole shell open, at mid-body >2-3 mm), supposed to indicate healthy condition, and narrow-slit/closed shells (gap at mid-body <2-3 mm), were counted. They were then prodded to control closure response. If open shells did not respond to touch by closing, they were considered to be much weakened (Burress 1982). Any differences observed in the number of open (or responding) mussels between the groups were tested in a chi-square test with two variables and without expected values.

The laboratory experiment

Eight 15 L aquaria, with stagnant tap water from the Lake Jonsvatnet were kept at 10 °C, with continuous light and air-bubbling. After an acclimatization period of 4.5 days to the experimental conditions, on 16 October 1990, 9 medium-sized to large mussels were placed into each aquarium and allowed to remain undisturbed for 1 h before the experiment started. The rotenone solution was then mixed into the water, and the mussels were exposed to 5, 10, 15, 20, 30, 40, and 50 ppm solution for 12 h. One aquarium was kept as a control. Every hour the mussels were examined for shell opening, more detailed in this experiment, since the mussels were easier to observe (wide-open: >1-3 mm, narrow-slit: 0.1-2 mm, closed: 0) and response to touch (strong: immediate response, not so strong: clearly delayed response, weak: almost no response at all) (Table 3). After 12 h of exposure the aquaria were replenished with fresh water, and shell opening and touch response were observed after 2 and 7 days. At the end of the experiment the mussels were marked and then released into the Creek Trollbekken and re-examined after 2 and 3 years.

The rotenone solution used for these experiments was "Gullvik's rotenone" manufactured in Sweden, and which is almost identical to the American Pro-Noxfish, a rotenone solution containing 2.5% rotenone and 2.5% of a synergist (sulfoxide); the overall effect is that of a 5% solution of rotenone. Gullvik's rotenone has been the most widely used rotenone product in fishery management in Scandinavia during the past few years, and is also the one used so far to exterminate *G. salaris* in infested Norwegian rivers.

The water temperature and light intensity used in these experiments were not much different from those found in Norwegian rivers by daytime in autumn, when most rotenone treatments take place. The water quality also lay within the range preferred by the freshwater pearl mussel (Table 1, cf. Grundelius 1987).

Results

The field experiment

During the rotenone treatment of the channel, sticklebacks *Gasterosteus aculeatus* were first affected, then after 20 min trouts *Salmo trutta* were also seen dying; and at last, after approximately 2 h, two eels *Anguilla anguilla* came creeping up from the water and going on land.

Before the rotenone treatment started, approximately the same number of open and narrow slit/closed shells was recorded in all the baskets. After only 15 min (Table 2), about one third of the mussels in the 5 ppm groups were narrow-slit/closed ($P<0.01$, pooled data) compared to the control group. After 4 h and 6 h a higher number of mussels had more or less closed shells in the 5 ppm groups than in the 2.5 ppm group ($P<0.02$, two baskets) and the 0.5 ppm group ($P<0.05$, one basket).

After 12 h, a greater number in the 5 ppm group of mussels which had been transferred to fresh water 6 h earlier, were narrow-slit/closed than in the control group ($P<0.05$). Besides, a greater number of mussels were narrow-slit/closed, both in the 2.5 ppm group and the 5 ppm group, than in the 0.5 ppm group ($P<0.05$ and $P<0.001$, respectively).

All mussels treated with rotenone, both for 6 h and 12 h, did not keep, or only occasionally

kept, thei
sure, whil
group did
$P<0.01$). I
mussels in
especially
ter 12 h n
diagnosed

During
of the roter
closed she
touch (Tab
with the n
row-slit/cl

Table 3. Re
concentrati
Shaded are

Rotenone concentrati						
Time (h)						
Shell gap						
Wide-open						
Narrow slit						
Closed/alm						
Response t						
Strong						
Not so stro						
Weak						
No respons						

Rotenone concentrati						
Time (h)						
Shell gap						
Wide-open						
Narrow sli						
Closed/alr						
Response						
Strong						
Not so stro						
Weak						
No respon						

kept, their foot outside the shell during exposure, while most of the mussels in the control group did (all groups, except for 6 h at 0.5 ppm, $P<0.01$). In addition, the response to touch by mussels influenced by rotenone was much slower, especially for the groups exposed to 5 ppm. After 12 h no mussels in the experiment could be diagnosed as dead, however.

During 1-2 days in clean, running water, most of the rotenone-exposed mussels had narrow-slit/closed shells, but almost all still responded to touch (Table 2). Also during the next 2.5 months, with the mussels still in the cages, more narrow-slit/closed mussels than open mussels were usually observed, but this was also the case for the control group (Table 2). Practically all mussels responded to touch.

When the released mussels after 1 and 3 years were examined in the river by divers, 75 and 91, respectively, of the total of 110 mussels, were located. All appeared to be healthy, and no dead ones were discovered.

The laboratory experiment

During 12 h of exposure there were no significant differences in shell gap and response to touch between the 5, 10 and 15 ppm groups and the control group (Table 3), although there was

Table 3. Response of *Margaritifera margaritifera* exposed to rotenone in a laboratory experiment. For each concentration $N = 9$. After 12 h of rotenone treatment the mussels were transferred to clean water. * = dead. Shaded area = control group or mussels after having been transferred to fresh water.

Rotenone concentration	0 ppm control group						5 ppm group 5 ppm				0 ppm		10 ppm group 10 ppm				0 ppm		15 ppm group 15 ppm				0 ppm	
Time (h)	3	6	9	12	58	168	3	6	9	12	58	168	3	6	9	12	58	168	3	6	9	12	58	168
Shell gap																								
Wide-open	9	9	9	8	8	9	9	9	9	9	9	7	8	7	7	7	6	9	5	5	7	7	8	9
Narrow slit				1	1							2	1	1	1		3		3	4	2	2	1	
Closed/alm. closed														1	1	2			1					
Response to touch																								
Strong	9	9	9	9	9	9	7	9	8	8		8	6				9	9	7					
Not so strong							2		1	1	9	1	2	7	8	8			1	7	9	9	8	9
Weak													1	1					1	2			1	
No response														1	1	1								

Rotenone concentration	20 ppm group 20 ppm				0 ppm		30 ppm group 30 ppm				0 ppm		40 ppm group 40 ppm				0 ppm		50 ppm group 50 ppm				0 ppm	
Time (h)	3	6	9	12	58	168	3	6	9	12	58	168	3	6	9	12	58	168	3	6	9	12	58	168
Shell gap																								
Wide-open	6	4	6	4	8	9	3		1	1	9	6	2		1		9	9						
Narrow slit	3	2	1	2			3	3	4	1		3	6	6	8	4								
Closed/alm. closed		3	2	3	1		3	6	4	7			1	3		5			9	9	9	9	9	9
Response to touch																								
Strong	4	1				9						5	1											
Not so strong	3	4			8		2					2	4											
Weak	1	2	6	5			4	2	4	2	9	2	1	2	3	4	6							
No response	1	2	3	4	1		3	7	5	7			3	7	6	5	3*	9*	9	9	9	9	9*	9*

a trend towards an increase in the numbers of less open and slowly-responding mussels with increasing concentration of rotenone. At the concentrations 30, 40 and 50 ppm, at 12 h, however, significantly more mussels were closed than in the control group ($P<0.001$, $P<0.01$, $P<0.001$, respectively). The response to touch decreased in a similar pattern: for both strong and weak responses taken together compared to no response at all; $P<0.05$, $P<0.001$, $P<0.01$ and $P<0.001$ for 20, 30, 40 and 50 ppm, respectively.

After the 12 h exposure period, the mussels exposed to 40 and 50 ppm were dead within one week. The exact time at which the first mussels in the 40 ppm group died is uncertain, but some mussels responded to touch during the whole course of the exposure period, and even two days thereafter. In the 50 ppm group all the mussels remained closed and never responded to touch after 2 h; they probably died at an early stage of the rotenone exposure.

The mussels exposed to ≤30 ppm all recovered completely within one week in clean water.

After 2 and 3 years, 53 of the 54 mussels which survived the 12 h exposure and were placed in the Creek Trollbekken, were located, still alive and appeared healthy (one mussel was not found).

Discussion

The rationale for the present experiment was the recent attempts to get rid of the salmon parasite *G. salaris*. Rotenone treatment of rivers, in order to kill the parasite with the host, could probably also have a devastating effect on the vulnerable freshwater pearl mussel. Mo (1986) showed that at a temperature of 11 °C, salmon (*Salmo salar*) parr died after ca. 15 min following treatment with 1 ppm rotenone under laboratory conditions. The last of the parasites only died after 45-60 min. Without a host, however, *G. salaris* will die eventually within a few days, anyway (Mo 1986). So far, rotenone concentrations of 1.5-2 ppm have been used in Norwegian rivers, and the procedure may in practice last for up to 8 h, and normally 2-5 h. Locally a rotenone concentration of around 5 ppm should also be expected in the river water.

The toxicity and dissipation time of rotenone depends on factors such as temperature, light intensity, alkalinity, oxygen and organic contents of the water (Post 1958, Örn 1962, Schnick 1974, Tobiasson 1979, Dawson et al. 1991). Since rotenone was continuously added to the water in the field experiment, dissipation would be no problem. In the laboratory experiment the rotenone concentration was set at the start of the experiment, and no replenishment of rotenone was made during the 12 h long experiment. With the experimental conditions used, however, i.e. a temperature of 10 °C, a low light intensity and a low organic content of the water, rotenone dissipation will have progressed only slowly (cf. Næss et al. 1991).

The results of both the field and laboratory experiments, show that, compared to fish the freshwater pearl mussel is highly resistant to rotenone. At 11 °C, salmonids are usually killed after only 0.5-1 h at rotenone concentrations of less than 0.5 ppm (even 0.2 ppm) (e.g. Mo 1986). Double concentration (1 ppm) is usually used with cyprinid fish (Burdick et al. 1955, Snekvik 1967, Meadows 1973, Marking and Bills 1976, Søilen 1984, Mo 1986). The lethal concentration (LC) of rotenone for adult freshwater pearl mussels exposed experimentally over a period of 12 h at 10 °C is here shown to lie between 30 and 40 ppm.

Chandler and Marking (1982) also showed that all species of freshwater invertebrates that they investigated (in laboratory), except for the water-fleas, were more tolerant to rotenone than were salmonids. Most tolerant were the molluscs (3 gastropod and 3 bivalve species were used), which tolerated from 100 to 850 times the lethal dose for salmon (cf. Marking and Bills 1976), for 96 h and 24 h, respectively. The bivalve *Corbicula manilensis* died only at a concentration of 7.5 ppm during an experimental period of 96 h, while the gastropod *Helisoma* sp. died at a rotenone concentration of 30 ppm for a 24 h period. Both these species show a very high tolerance, fully comparable to what we found for

the freshwate
the range 30
luscs to rote
other studie
Holcombe et
90).

In their na
invertebrates
in laboratory
1976, cf. En
and Marking
case for mobi
are more or le
by digging o
tom substrate
effect on rote
1960, Örn 19
fore presume
in nature dug
higher conce
found by us.

At the lowe
the laboratory
the highest c
rivers, the mu
presence of ro
of mussels wh
those that prot
gradually dec
concentration
in the field ex
of rotenone.

Lennart He
similar behavi
shells) in spec
sel when they
water. Practic
among those
rivers, are du
by about two
iour prevents
e.g. during a

Adult muss
by rotenone t
in Norwegian r
mussels small
sume they are

the freshwater pearl mussel, with a 12 h LC in the range 30-40 ppm. High resistance of molluscs to rotenone has also been reported from other studies (Chandler and Marking 1982, Holcombe et al. 1987, cf. Sousa et al. ca. 1985-90).

In their natural habitat, both fish and aquatic invertebrates are less affected by rotenone than in laboratory experiments (Marking and Bills 1976, cf. Engstrom-Heg et al. 1978, Chandler and Marking 1982). This may especially be the case for mobile benthic invertebrates, since they are more or less being able to escape the toxicant by digging or hiding in the substrate. The bottom substrate, in itself, also has an inactivating effect on rotenone, at least in lakes (Lindgren 1960, Örn 1962, Andreasson 1963). We therefore presume that the freshwater pearl mussel, in nature dug down in the river bed, tolerates higher concentrations of rotenone than was found by us.

At the lowest rotenone concentration used in the laboratory experiment, 5 ppm, which exceeds the highest concentrations used in Norwegian rivers, the mussels seemed hardly to notice the presence of rotenone at all. However, the number of mussels which remained wide-open, and also those that protruded their foot out of their shells, gradually decreased with increasing rotenone concentration and time. A similar trend was seen in the field experiment at lower concentrations of rotenone.

Lennart Henrikson (pers. comm.) has seen a similar behaviour (keeping their foot inside their shells) in specimens of the freshwater pearl mussel when they were placed into strongly acidic water. Practically all mussels, however, at least among those we have observed in Norwegian rivers, are dug down into the bottom substrate by about two third of their length. This behaviour prevents them from drifting downstream, e.g. during a rotenone treatment.

Adult mussels are thus not seriously affected by rotenone treatments of the kind carried out in Norwegian rivers. However, we have not tested mussels smaller than 5-6 cm, although we presume they are safe at the concentrations used. Mussels in the youngest stage, the parasitic glochidia larva, will die with its host (salmon or trout). Since the river will have a fairly dense population of fish already the year after the rotenone treatment, only one year-class of mussels will be lost, however.

Within the body, rotenone works primarily by inhibiting the electron transport system of the mitochondria (Lindahl and Öberg 1961, Horgan et al. 1968), leading to a slow-down in oxygen transport through the gills and to reduced cell respiration. Lethal rotenone poisoning probably occurs at the time when the uptake exceeds the animal's capacity to break it down metabolically (Gingerich and Rach 1985, cf. Fukami et al. 1969).

Some mussels which did not respond to touch and appeared to be dead even at a relatively early stage of the experiment, recovered after transfer into clean water (Tables 2 and 3). Recovery from high sub-lethal doses of rotenone, and apparent lethargy, is also known from the literature, e.g. by zooplankton (Almquist 1959), by fish (Gilderhus 1972), and by oysters *Ostrea edulis* (Samuelsen et al. 1988). Oysters tolerated about 1 ppm of rotenone solution for 7-8 days. At that time the rotenone concentration within the animals had risen to 7-8 ppm and many of them died. Others survived, however, and as the rotenone concentration outside the animals gradually began to decrease, so did the concentration within the live mussels, through excretion, and they eventually recovered fully (Samuelsen et al. 1988).

The time needed for the freshwater pearl mussel to recover from high sub-lethal concentrations of rotenone (up to 30 ppm rotenone solution) was less than a week, which is comparable to the four days needed by the blue mussel *Mytilus edulis* fully to recover from sub-lethal concentrations of formaldehyde in the experiments of Nordtug et al. (1991).

Although much weakened, no mussels exposed to 40 ppm were classified as dead at the time when the experiment was finished. After being transferred to clean water, however, final mortality occurred after 2 to 6.5 days. A similar

delay, from exposure of fish to a deadly dose of rotenone to final death, has likewise been recorded by Gilderhus (1972).

Many oxygen-demanding invertebrates have been shown to be especially sensitive to rotenone (Morrison 1977, Engstrom-Heg et al. 1978, Arnekleiv 1992). Gill-breathing animals are also highly vulnerable to rotenone poisoning, since the substance is very effectively taken up through the gills (Öberg 1965). Therefore, it seems strange that a gill-breathing animal that lives exclusively in very clean, running and well-oxygenated water, should have such a high tolerance to rotenone.

A reasonable explanation for the high degree of tolerance to rotenone by the freshwater pearl mussel may well lie in a possible capacity for anaerobic respiration. Such facultative respiration is found in the marine blue mussel (Roberts 1976, George et al. 1977, Nordtug et al. 1991), probably an adaptation to surviving in the littoral zone when the shells close during low tide.

The freshwater pearl mussel may in fact very well be adapted to anaerobic conditions, since some of the creeks in which they live dry out almost completely at times during extremely warm summers with low precipitation. In order to test this hypothesis, however, further investigations are needed.

Conclusion

The conclusion is that rotenone treatments such as those carried out in order to get rid of the salmon parasite *G. salaris,* would not seem to represent a threat to adult freshwater pearl mussels. Such treatments usually last for no longer than 5-8 h and with rotenone concentrations of no more than 5 ppm (locally). The tolerance of adult mussels to rotenone appears to be very high. At 10 °C they survived and recovered concentrations of up to 30 ppm (but not 40 ppm) for 12 h under laboratory conditions, and can probably withstand even higher concentrations in nature.

Acknowledgements

The project was financed by The Directorate for Nature Management. Ove Eide kindly assisted in the rotenone experiments in the River Haukebøelva, Philip Tallantire corrected the English language, and Tor Strømgren commented on an original version of the manuscript. Thanks go also to Lennart Henrikson and referees of Nordic Journal of Freshwater Research for constructive help and criticism.

References

Almquist, E. 1959. Inverkan av rotenonbehandling på fiskens födoorganismer. - Svensk Fisk. Tidskr. 68: 61-62. (In Swedish.)

Andreasson, S. 1963. Rotenonets inverkan på evertebrater i relation til vissa miljöfaktorer. - Svensk Fisk. Tidskr. 72: 90-95. (In Swedish.)

Arnekleiv, J.V. 1992. Giftvirkning av rotenon på bunndyr og reetablering av bunndyr i rotenonbehandlete vassdrag. p. 50-67. - In: Fagseminar om *Gyrodactylus salaris* og sykdoms-/rømmings-problematikken, 15-17 april 1991, Stav Gjestegård, Malvik. Presenterte foredrag. Direktoratet for naturforvaltning, Trondheim. (In Norwegian.)

Bergquist, K. 1993. Gammelmusslan. - Sveriges Natur 1993(1): 3-11. (In Swedish.)

Burdick, G.E., H.J. Dean and E.J. Harris. 1955. Toxicity of emulsifiable rotenone to various species of fish. - N. Y. Fish Game J. 2: 36-37.

Burress, R.M. 1982. Effects of synergized rotenone on nontarget organisms in ponds. - U.S. Fish Wildl. Ser. Invest. Fish Control 91: 1-7.

Carell, B., S. Forberg, E. Grundelius, L. Henrikson, A. Johnels, U. Lindh, H. Mutvei, M. Olsson, K. Svärdström and T. Westermark. 1987. Can mussels shells reveal environmental history? - Ambio 16: 2-10.

Chandler, J.H. Jr. and L.L. Marking. 1982. Toxicity of rotenone to selected aquatic invertebrates and frog larvae. - Prog. Fish Cult. 44(2): 78-80.

Collins, N.M. and S.M. Wells. 1986. Insects and other invertebrates as candidates for the Bern Convention. - European committee for the conservation of nature and natural resources, Strasbourg.

Council of Europe. 1992. Convention on the conservation of European wildlife and natural habitats. Appendices to the convention. - European committee for the conservation of nature and natural resources, Strasbourg.

Dawson, V.K., W.H. Gingerich, R.A. Davis and P.A. Gilderhus. 1991. Rotenone persistence in freshwater ponds: Effect of temperature and sediment adsorption. - N. Am. J. Fish. Mgmt. 11: 226-231.

Direktoratet f
Gyrodactylu
tikken, 15.-
senterte fo
Trondheim.

Dolmen, D. 19
way; infestati
and G. Maln
in natural wa
holm 2-4 De
University o

Dolmen, D. and
30. - In: Ron
1992. FOU-å

Engstrom-Heg, I
tolerances of
31-41.

Fox, R.C. 1985.
fornia Depart

Fukami, J.-I., T.
Oxidative me
sects and its r
17: 1217-122

George, S.G., B.
characteristic
bivalve mollu

Gilderhus, P.A. 1
and rotenone
Res. Board C

Gingerich, W.H.
and elimina
macrochirus)

Grundelius, E. 19
in the provin
Drottningholn

Haley, T.J. 1978
Environ. Path

Holcombe, G.W.,
1987. Simulta
of 13 chemica
invertebrate f
697-710.

Horgan, D.J., T.P
respiratory c
dinucleotide d
piericidin A an
243: 834-843.

Johnsen, B.O. an
salmon, *Salmo*
rivers. - J. Fisl

Johnsen, B.O., A.
tion of *Gyroda*
salar by roten
way. - Fauna n

Direktoratet for naturforvaltning. 1992. Fagseminar om *Gyrodactylus salaris* og sykdoms-/rømmings-problematikken, 15.-17. april 1991, Stav Gjestegård, Malvik. Presenterte foredrag. - Direktoratet for naturforvaltning, Trondheim. (In Norwegian.)

Dolmen, D. 1987. *Gyrodactylus salaris* (Monogenea) in Norway; infestations and management. p. 63-69. - In: A. Stenmark and G. Malmberg (eds.) Proc. Symp. Parasites and diseases in natural waters and aquaculture in Nordic countries. Stockholm 2-4 Dec. 1986. Zoo-tax, Naturhistoriska Riksmuseet, University of Stockholm.

Dolmen, D. and E. Kleiven. 1993. Elvemuslingprosjektet. p. 29-30. - In: Romundstad, A.J. (ed.) Kalking i vann og vassdrag 1992. FOU-årsrapporter. DN-notat 1993-1. (In Norwegian.)

Engstrom-Heg, R., R.T. Colesante and E. Silco. 1978. Rotenone tolerances of stream-bottom insects. - N. Y. Fish Game J. 25: 31-41.

Fox, R.C. 1985. Rotenone use for fisheries management. - California Department of Fish and Game.

Fukami, J.-I., T. Shishido, K. Fukunaga and J.E. Casida. 1969. Oxidative metabolism of rotenone in mammals, fish, and insects and its relation to selective toxicity. - J. Agr. Food Chem. 17: 1217-1226.

George, S.G., B.J.S. Pirie and T.L. Coombs. 1977. Metabolic characteristics of endocytosis of ferritin by gills of a marine bivalve mollusc. - Biochem. Soc. Trans. 5: 136-137.

Gilderhus, P.A. 1972. Exposure times necessary for antimycin and rotenone to eliminate certain freshwater fish. - J. Fish. Res. Board Can. 29: 199-202.

Gingerich, W.H. and J.J. Rach. 1985. Uptake, biotransformation, and elimination of rotenone by bluegills (*Leponis macrochirus*). - Aquat. Toxicol. 6: 179-196.

Grundelius, E. 1987. The decline of the freshwater pearl mussel in the province of Dalarna. - Inform. Inst. Freshw. Res., Drottningholm (4). 72 p. (In Swedish with English summary.)

Haley, T.J. 1978. A review of the literature of rotenone. - J. Environ. Pathol. Toxicol. 1: 315-337.

Holcombe, G.W., G.L. Phipps, A.H. Sulaiman and A.D. Hoffman. 1987. Simultaneous multiple species testing: Acute toxicity of 13 chemicals to 12 diverse freshwater amphibian, fish, and invertebrate families. - Arch. Environ. Contam. Toxicol. 16: 697-710.

Horgan, D.J., T.P. Singer and J.E. Casida. 1968. Studies on the respiratory chain-linked reduced nicotinamide adenine dinucleotide dehydrogenase. XIII. Binding sites of rotenone, piericidin A and amyl in the respiratory chain. - J. Biol. Chem. 243: 834-843.

Johnsen, B.O. and A. Jensen. 1986. Infestations of Atlantic salmon, *Salmo salar*, by *Gyrodactylus salaris* in Norwegian rivers. - J. Fish Biol. 29: 233-241.

Johnsen, B.O., A.J. Jensen and B. Sivertsen. 1989. Extermination of *Gyrodactylus salaris*-infected Atlantic salmon *Salmo salar* by rotenone treatment in the river Vikja, Western Norway. - Fauna norv. Ser. A 10: 39-43.

Kleiven, E., J. Økland and D. Dolmen. 1988. Elveperlemuslingen. - Nor. Nat. 24 (6): 16-18. (In Norwegian.)

Kleiven, E., J. Økland and D. Dolmen. 1989. Elveperlemuslingen - muslingen med kongeleg pondus. - Vår barndoms have. Årbok 1989 Vest-Agder Fylkesmuseum: 42-71. (In Norwegian.)

Lindahl, P.E. and K.E. Öberg. 1961. The effect of rotenone on respiration and its point of attack. - Exp. Cell Res. 23: 228-237.

Lindgren, P.E. 1960. About the effect of rotenone upon benthonic animals in lakes. - Rep. Inst. Freshw. Res., Drottningholm 41: 172-184.

Marking, L.L. and T.D. Bills. 1976. Toxicity of rotenone to fish in standardized laboratory tests. - U.S. Fish Wildl. Serv., Invest. Fish Control 72: 1-11.

Meadows, B.S. 1973. Toxicity of rotenone to some species of coarse fish and invertebrates. - J. Fish Biol. 5: 155-163.

Mo, T.A. 1986. Virkning av rotenon på laks og lakseparasitten *Gyrodactylus salaris*. Laboratorieforsøk. Gyrodactylus-undersøkelsene Zoologisk Museum, Universitetet i Oslo. Rapp. 1986-1: 1-16. (In Norwegian.)

Morrison, B.R.S. 1977. The effects of rotenone on the invertebrate fauna of three hill streams in Scotland. - Fish. Mgmt 8: 128-139.

Næss, T., K.E. Naas and O.B. Samuelsen. 1991. Toxicity of rotenone to some potential predators on marine fish larvae - An experimental study. - Aquacult. Eng. 10: 149-159.

Nordtug, T., J.F. Børseth, A. Olsen and K.E. Zachariassen. 1991. Oxygen consumption during exposure to and recovery from high sublethal concentrations of formaldehyde, benzene and phenol. Effects of organic chemicals on the physiology of the blue mussel (*Mytilus edulis*). Perspectives for toxicity testing and environmental monitoring. p. 93-96. - In: K.E. Zachariassen (ed.) Report from the BECTOS-program 1988-91. University of Trondheim.

Öberg, K.E. 1965. On the principal way of attack of rotenone in fish. - Ark. Zool. 18: 217-220.

Økland, J. 1976. Utbredelsen av noen ferskvannsmuslinger i Norge, og litt om European Invertebrate Survey. - Fauna 29: 29-40. (In Norwegian.)

Örn, B. 1962. Kvantitativ analys av tekniska rotenonpreparat. - Södra Sveriges Fiskeriforskning. Årsskrift 1961-1962: 26-38. (In Swedish.)

Post, G. 1958. Time versus water temperature in rotenone dissipation. - Proceedings of the Western Association of State Game and Fish Commissioners 38: 279-284.

Quenild, T. 1977. Rotenon. - Jakt-fiske-friluftsliv 1977 (4): 26-31, 47. (In Norwegian.)

Roberts, D. 1976. Mussels and pollution. p. 67-80. - In: B.L. Bayne (ed.) Marine mussels: Their ecology and physiology. Cambridge University Press, Cambridge.

Samuelsen, O.B., E. Solheim, H. Otterå and J.P. Pedersen. 1988. The toxicity, absorption and excretion of rotenone in oysters (*Ostrea edulis*), and its degradation in seawater at temperatures near 0°C. - Aquaculture 70: 355-363.

Schnick, R.A. 1974. A review of the literature on the use of rotenone in fisheries. - U.S. Fish Wildl. Serv., Rep. LR-74-15.

Snekvik, E. 1967. Notat om rotenonbehandling av fiskevann. Opptrykk 1984. - Direktoratet for naturforvaltning, Trondheim. (In Norwegian.)

Søilen, P. 1984. Kjemisk utrydding av fisk. p. 360-362. - In: K.W. Jensen (ed.) Sportsfiskerens leksikon. Kunnskapsforlaget, Oslo. (In Norwegian.)

Soleman, V.E.F. 1950. History and use of fish poisons in the United States. - Can. Fish Cult. 8: 3-16.

Sousa, R.J., F.P. Meyer and R.A. Schnick. ca. 1985-90. Better fishing through management. How rotenone is used to help manage our fishery resources more effectively. - Sport Fish Restoration (USA).

Tobiasson, G. 1979. The use of rotenone in Sweden. - Inform. Inst. Freshw. Res., Drottningholm (10). 33 p. (In Swedish with English summary.)

Ugedal, O. 1986. Litteraturstudie av rotenons virkning i ferskvannsøkosystemer. - DN-Reguleringsundersøkelsene 1986-14: 1-52. (In Norwegian.)

United Nations. 1991. European red list of globally threatened animals and plants. - Economic Commission for Europe, Geneva.

Wells, S.M., R.M. Pyle and N.M. Collins. 1983. The IUCN invertebrate red data book. - IUCN, Cambridge.