Chemical Strategies for the Control of the Golden Mussel (*Limnoperna fortunei*) in Industrial Facilities

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Abstract There are a number of available chemical strategies for the control of macrofouling by bivalves within piping systems such as those that carry raw water into water treatment plants, cooling water to vital areas of power plants and industrial facilities, and fire protection water in any industry. A number of these strategies have been tested for the control of golden mussels and some of the methods tested hold promise for industrial use. Treatment strategies generally fall into proactive and reactive treatments. Proactive treatments will prevent settlement of mussels or prevent the development of adults. Reactive treatments will allow the settlement and growth of adult mussels, periodically removing settled adults. The chemicals used for both strategies are divided into nonoxidizing and oxidizing chemicals. The actual choice of chemicals and the mode of application will depend on several factors such as regulatory approval, economic viability, and preference of the individual user.

Keywords *Limnoperna fortunei* · Golden mussel · Biological invasion · Biofouling control · Impact · Industrial plants · Power plants · Irrigation facilites · Chemical treatment · Biocides · Molluscicides

Introduction

The golden mussel, a macrofouling bivalve, was introduced into Argentina from Asia around 1990 (Pastorino et al. 1993) and within a decade spread to four other South American countries (see Chapter "Colonization and Spread of *Limnoperna fortunei* in South America" in this volume). Before the widespread invasion of the golden mussels in South America, the invasion of the zebra mussel

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(a morphologically similar bivalve) into North American waters demonstrated the vulnerability of industrial raw water systems to macrofouling species and the need for control of infestation.

The golden mussel (Mytilidae) and the zebra and quagga mussels (Dreissenidae) share many characteristics. Both the dreissenids and the golden mussel cause serious problems for industry because they possess byssal threads with which they hold on to the substrate enabling them to settle in cooling water pipes. Their free-living larvae are carried by raw water and can gain access and settle in most industrial raw water systems. These two characteristics allow both the dreissenids and the golden mussel to foul all structures and surfaces exposed to raw water. Mussel settlement and growth inside cooling water piping can decrease flow and cause numerous maintenance problems for most industrial facilities.

Different industrial facilities face different problems depending on their materials of construction, cooling water system configuration, and the way they use raw water. A vulnerability assessment can pinpoint the areas of a facility most likely to suffer from infestation and allow management to focus control efforts on those areas.

In this chapter, we shall review available information on chemical strategies for the control of golden mussels within piping systems such as those which carry raw water into water treatment plants, cooling water to vital areas of power plants, and fire protection water in any industry. The methods and chemicals included in this review are restricted to those which have been tested with *L. fortunei* and which hold promise for industrial use. The actual number of chemicals that could be potentially used is far larger (Sprecher and Getsinger 2000; Mackie and Claudi 2010; Rajagopal et al. 2012).

Control Strategies for Internal Piping Systems

The approach and the materials used for the control of golden mussels are very similar to those used for the control of dreissenid mussels in Europe and North America. However, it is important to note that in some instances there are differences between the response of the dreissenids and the response of the golden mussel to some strategies. The golden mussel exhibits a wider tolerance of some ecological parameters than the dreissenids (Karatayev et al. 2010) (see Chapter "Parallels and Contrasts Between *Limnoperna fortunei* and Species of *Dreissena*" in this volume). Golden mussels often thrive under extremely adverse environmental conditions (e.g., high pollution levels, low oxygen concentrations, very low calcium levels, low pH; Karatayev et al. 2007), an indication that it is a highly tolerant species. For example, under anoxic conditions at 25 ºC, 100 % mortality of *Dreissena* is achieved in only 4 days (Matthews and McMahon 1994). For *L. fortunei* 13 days are required for 100 % mortality under similar conditions (Perepelizin and Boltovskoy 2011).

Due to wide tolerances of environmental conditions by the golden mussels, individual chemical strategies that have been used successfully for the control of dreissenids need to be retested on the golden mussel to verify their effectiveness.

Proactive versus Reactive Treatments

Prior to discussing individual control strategies, the concept of proactive versus reactive treatment must be considered. Depending on the vulnerability of the individual facility or system, the operators must decide if they will treat in a manner which will prevent settlement of mussels (proactive treatment), or if they will allow settlement to occur and periodically remove settled adults (reactive treatment). The more vulnerable the system and the larger the population density of the mussels, the more likely it is that the operators will choose preventative treatment.

The choice of proactive versus reactive treatment will dictate both the choice of treatment strategy and the method of application. For example, the use of small-pore self-cleaning filters to remove veligers and continuous irradiation of the raw water flowing within a pipe with UV lamps to disable the veligers are both considered proactive treatments. Continuous pH adjustment and low-level continuous dosing with oxidizing and nonoxidizing chemicals to prevent settlement (without necessarily causing mortality of ready to settle veligers) are also proactive treatment strategies.

In contrast, periodic application of oxidizing or nonoxidizing chemicals, mechanical removal and application of hot water to remove adults are examples of reactive treatment.

Proactive Application of Chemicals for the Control of L. fortunei

Nonoxidizing Chemicals

pH Adjustment

Salto de Cashias is a hydroelectric plant in Brazil that belongs to the COPEL company. The facility has been coping with golden mussels for a number of years. The population density of the mussels has been reported to be as high as 150,000 ind./m^2.

The initial system installed for control of mussels in 2000 was that of pH adjustment. Sodium hydroxide was added to the service water stream to achieve a pH of approximately 9. This pH appeared to eliminate mussels from the service water system. In addition, biofilm was removed from service water piping. The removal of the biofilm did expose some underdeposit corrosion in the piping system, including some pinhole leaks (Calazans and Fernandes 2012). The control of settlement is thought to have been achieved by adjustment of the pH to outside of the preferred range of the golden mussel rather than by the direct toxicity of sodium hydroxide.

Before considering pH adjustment as a mitigation strategy, it is important to test pH adjustment in the raw water. In water with high alkalinity or high calcium content, increasing the pH can cause precipitation of calcium carbonate. This would be an undesirable side effect and the use of pH adjustment would not be practical under those circumstances.

Montresor et al. (2013) used concentrations of NaOH from 40 to 800 mg/L, which resulted in a pH range of 11.24–13.04. The authors recorded a LC_{50} (the concentration capable of killing 50% of the individuals exposed) in 96 h exposures to 88.51 mg/L (pH ~ 11.5). This is comparable to the response of zebra mussels observed by Claudi et al. (2012). Zebra mussels exposed to very high pH levels (i.e., pH 10, 11, and 12) caused by addition of NaOH experienced 90% mortality after 120 h at pH 12. Results from both studies suggest that very high pH could be used as an end of season treatment for elimination of adult mussels, as well as a preventative strategy using a modest pH adjustment for settlement prevention.

To continuously adjust the pH at Salto de Cashias had a cost of approximately US$ 200/day. In 2009, plant management decided to switch to the use of the MXD-100 product (see below). One of the primary reasons was worker safety when handling large volumes of sodium hydroxide.

MXD-100

MXD-100 is a product of the Brazilian Company Maxclean Ambiental e Química. It is considered an antifouling and antimicrobial agent. The composition of this product appears fairly complex. According to the manufacturer, it contains plant-derived tannins, isothiazolone, EDTA (ethylenediaminetetraacetic acid), active cationic surfactants, and nonionic glycols. Given the complexity of the product, it is hard to speculate on the mode of action. However, when Pereyra et al. (2011) tested the toxicity of three plant-derived tannin preparations to *L. fortunei*, the LC_{50} values ranged from 138.53 to 1273.73 mg/L, depending on the size of the specimens (larvae or 13 and 19 mm adults) and on the chemical compound tested (ECOTECs-UA, ECOTECs-L and ECOTECs-MC) (Table 1). The observed tannin toxicity was far below the toxicity documented for MXD-100 by Montresor et al. (2013), which suggests that the importance of tannins in the formulation of this compound is marginal. Montresor et al. (2013) tested the toxicity of several concentrations of MXD-100 (0.05, 0.5, 1, 10, 100, and 500 mg/L) on adult *L. fortunei* at ambient water temperature of 23 to 27ºC. The authors found LC_{50} concentrations of 45.49 mg/L after 48 h, 13.69 mg/L after 72 h, and 11.1 mg/L after 96 h (Table 1). When Salto de Cashias switched to the use of MXD-100, the product was injected in the same location as was used for sodium hydroxide but it was added three times each day for 11 min at a time. The amount injected is approximately 0.8 ppm of MXD, and the cost is similar to the sodium hydroxide treatment (L. C. Montresor pers. comm.). In Brazil, the treatment with MXD-100 is usually applied over a period of 90 days, three times a day for 10 min each, at a concentration range of 1–7 mg/L (as registered by the Brazilian Environmental Institute–IBAMA–under the number 4722/11-10) (Mata et al. 2013; Montresor et al. 2013). The three times per day injection appears to eliminate freshly settled individuals and is therefore a preventative strategy.

Oxidizing Chemicals

An oxidizing chemical is the element or compound in a chemical reaction that accepts an electron from another species. Because the oxidizing chemical is gaining electrons, it is said to have been reduced. The reactant element or compound in the chemical reaction is oxidized by having its electrons taken away by the oxidizing agent. This oxidation-reduction chemical reaction is often referred to as a redox reaction.

Ozone

The hydroelectric plant Itaipu (Brazil-Paraguay, upper Paraná River) has tested an ozone addition system in the service water of Unit 10. The ozone was injected continuously into the service water system resulting in a concentration of 0.15–0.20 mg/L. Even at this low concentration there appeared to be good control of mussels downstream of the injection. Furthermore, ozone appeared to provide excellent control of biofilm (Rothe 2007).

Chlorine Dioxide

Chlorine dioxide has been used for the control of *L. fortunei* by a municipal water system in Southern Brazil by Nalco (Nalco Inc. 2013). The water utility used Purate™ chlorine dioxide technology as the primary oxidant starting in 2007. The objective was to prevent the attachment of the *L. fortunei* veligers in the incoming water pipeline. Chlorine dioxide was injected at the point of water withdrawal in the river and again at the intake to the treatment plant. A continuous dose of approximately 0.8 ppm of chlorine dioxide at the intake prevented settlement in the pipeline. The addition of chlorine dioxide was also effective for color and odor removal caused by Cyanobacteria. According to the study, by using chlorine dioxide, this municipality saved more than 80% of the expenses previously used to manually remove golden mussels. In 2006, the expenses for golden mussel control were US$ 260,000. In 2008, the expense was less than US$ 50,000.

Chlorine

Chlorine as chlorine gas or either sodium or calcium hypochlorite has been widely used for prevention of macrofouling and water disinfection. Morton (1976) reported on the efficacy of chlorine for control of *L. fortunei* in Hong Kong's raw water supply system. Continuous application of 0.5 mg chlorine/L was found to be sufficient to prevent infestation of the system without adversely affecting sand filters or leaving a taste in the water (Table 1). The prevention of settlement by chlorine has also

Table 1 Summary of results of chemical control methods assayed with *Limnoperna fortunei*. Toxicant [commercial name]: (1) 2,5′ -dichloro-4′ -nitrosalicylanilide (active ingredient: 70%) [Bayluscide WP70]; (2) Chlorine dioxide; (3) Diallyl dimethyl ammonium chloride polymer (active ingredient: 40%) [Veligon TL-M]; (4) Didecyl dimethyl ammonium chloride (active ingredient: 50%) [H130M]; (5) N-alkil dimethylbenzyl ammonium chloride (active ingredient: 50%) [Clam-Trol CT-2]; (6) N-alkil dimethylbenzyl ammonium chloride (active ingredient: 50%) [Spectrus CT1300]; (7) Poly(oxyethylene (dimethyliminio) ethylene (dimethyliminio) ethylene dichloride) [BULAB 6002]; (8) Poly(oxyethylene (dimethyliminio) ethylene (dimethyliminio) ethylene dichloride) [BULAB 6002]; (9) Poly-diallyldimethyl ammonium chloride (microencapsulated); (10) Potassium chloride; (11) Potassium chloride (microencapsulated); (12) Quaternary ammonium+tannin extracts [MXD-100]; (13) *Schinopsis balansae* tannins at 70% [ECOTEC-L]; (14) *Schinopsis balansae* tannins at 74% [ECOTEC-UA]; (15) *Schinopsis balansae* tannins at 86.5% [ECOTEC-MC]; (16) Sodium chloride; (17) Sodium dichloroisocyanurate; (18) Sodium hydroxide; (19) Sodium hypochlorite; (20) Total ammonia; (21) Trichloroisocyanuric acid; (22) Unionized ammonia (NH_3-N); (23) Potassium permanganate; (24) Copper sulfate

Toxicant	LC_{50} or % dead	Exposure [postexposure] times (h)	No. of concentrations tested (range, ppm)	Stage (size in mm)	Temp. (ºC)	Test conditions	Reference
1	1.0 ppm	48 [24–264]	7 (0.25-8)	A (15–25)	15	Static	Cataldo et al. (2003)
1	0.8 ppm	48 [24–264]	7 (0.25-8)	A (15–25)	20	Static	Cataldo et al. (2003)
1	0.3 ppm	48 [24–264]	7 (0.25-8)	A (15–25)	25	Static	Cataldo et al. (2003)
2	427.6 ppm	48 [48]	14 (1-800)	A (15–25)	25	Static	Calazans et al. (2013)
3	3.88 ppm	720 [0]	5 (2–50)	A	20–22	Static	Boltovskoy and Cataldo (2003)
3	815.04 ppm	264 [0]	5 (0.5–10)	L	20–22	Static	Boltovskoy and Cataldo (2003)
4	0.56 ppm	720 [0]	5 (0.5–10)	A	20–22	Static	Boltovskoy and Cataldo (2003)
4	1.03 ppm	264 [0]	5 (0.5–10)	L	20–22	Static	Boltovskoy and Cataldo (2003)
4	2.9 ppm	48 [24–264]	8 (0.5–30)	A (15–25)	15	Static	Cataldo et al. (2003)
4	1.7 ppm	48 [24–264]	8 (0.5–30)	A (15–25)	20	Static	Cataldo et al. (2003)
4	0.8 ppm	48 [24–264]	8 (0.5–30)	A (15–25)	25	Static	Cataldo et al. (2003)
5	2.43 ppm	36 [252]	5 (1–3)	A	20–22	Static	Boltovskoy and Cataldo (2003)

Table 1 (continued)

Toxicant	LC_{50} or % dead	Exposure [postexposure] times (h)	No. of concentrations tested (range, ppm)	Stage (size in mm)	Temp. (°C)	Test conditions	Reference
5	0.98 ppm	36 [480]	5 (1–3)	A	30	Static	Boltovskoy and Cataldo (2003)
5	1.28 ppm	36 [480]	5 (1–3)	A	25	Static	Boltovskoy and Cataldo (2003)
5	2.43 ppm	36 [480]	5 (1–3)	A	20	Static	Boltovskoy and Cataldo (2003)
5	0.88 ppm	48 [480]	5 (1–3)	A	30	Static	Boltovskoy and Cataldo (2003)
5	1.38 ppm	48 [480]	5 (1–3)	A	25	Static	Boltovskoy and Cataldo (2003)
5	2.52 ppm	48 [480]	5 (1–3)	A	20	Static	Boltovskoy and Cataldo (2003)
5	0.90 ppm	720 [0]	5 (0.5–10)	A	20–22	Static	Boltovskoy and Cataldo (2003)
5	0.71 ppm	264 [0]	5 (0.5–10)	L	20–22	Static	Boltovskoy and Cataldo (2003)
5	34.9 ppm	48 [24–264]	5 (1–30)	A (15–25)	15	Static	Cataldo et al. (2003)
5	1.3 ppm	48 [24–264]	5 (1–30)	A (15–25)	20	Static	Cataldo et al. (2003)
5	1.2 ppm	48 [24–264]	5 (1–30)	A (15–25)	25	Static	Cataldo et al. (2003)
6	% dead: 41.75	12 [120]	1 (2.3)	A (5–35)	22.5–23.5	FT	Boltovskoy and Cataldo (2003)
6	% dead: 41.45	24 [120]	1 (2.3)	A (5–35)	22.5–23.5	FT	Boltovskoy and Cataldo (2003)
6	% dead: 62.15	36 [120]	1 (2.3)	A (5–35)	22.5–23.5	FT	Boltovskoy and Cataldo (2003)

Table 1 (continued)

Toxicant	LC_{50} or % dead	Exposure [postexposure] times (h)	No. of concentrations tested (range, ppm)	Stage (size in mm)	Temp. (°C)	Test conditions	Reference
6	% dead: 92.85	48 [120]	1 (2.3)	A (5–35)	22.5–23.5	FT	Boltovskoy and Cataldo (2003)
6	% dead: 92.05	72 [120]	1 (2.3)	A (5–35)	22.5–23.5	FT	Boltovskoy and Cataldo (2003)
6	% dead: 63	24 [168]	1 (2.5)	A (6–43)	24–25	FT	Boltovskoy et al. (2005)
6	% dead: 94	48 [168]	1 (2.5)	A (6–43)	24–25	FT	Boltovskoy et al. (2005)
6	% dead: 99	72 [168]	1 (2.5)	A (6–43)	24–25	FT	Boltovskoy et al. (2005)
7	7.185 ppm	24 [0]	5 (5–75.5)	L	ND	Static	Darrigran et al. (2001)
8	0.88 ppm	720 [0]	4 (0.5–20)	A	20–22	Static	Boltovskoy and Cataldo (2003)
8	1.51 ppm	264 [0]	4 (0.5–10)	L	20–22	Static	Boltovskoy and Cataldo (2003)
8	% dead: 77–100 %	116–168 [0]	3 (8–20)	A	24	Static	Darrigran and Damborenea (2001)
9	1313.3 ppm	6 [48]	1 (90)	A (15–25)	25	FT	Calazans et al. (2013)
9	270.9 ppm	48 [48]	11 (12–1000)	A (15–25)	25	Static	Calazans et al. (2013)
10	1439.0 ppm	48 [48]	8 (10–10000)	A (15–25)	25	Static	Calazans et al. (2013)
11	8303.1 ppm	6 [48]	4 (90–1000)	A (15–25)	25	FT	Calazans et al. (2013)
11	2536.9 ppm	48 [48]	8 (12–6000)	A (15–25)	25	Static	Calazans et al. (2013)
12	45.49 ppm	48 [0]	6 (0.05–500)	A (21–26)	23–27	Static	Montresor et al. (2013)

Table 1 (continued)

Toxicant	LC_{50} or % dead	Exposure [postexposure] times (h)	No. of concentrations tested (range, ppm)	Stage (size in mm)	Temp. (ºC)	Test conditions	Reference
12	13.69 ppm	72 [0]	6 (0.05–500)	A (21–26)	23–27	Static	Montresor et al. (2013)
12	11.10 ppm	96 [0]	6 (0.05–500)	A (21–26)	23–27	Static	Montresor et al. (2013)
12[a]	% dead: 99	8760 [0]	1 (1)	A	18–26	FT	Netto (2011)
13	138.54 ppm	24 [0]	ND	L	21.8–23.7	Static	Pereyra et al. (2011)
14	160.21 ppm	24 [0]	ND	L	21.8–23.7	Static	Pereyra et al. (2011)
15	983.27 ppm	168 [0]	ND	A (13)	21.8–23.7	Static	Pereyra et al. (2011)
15	309.92 ppm	168 [0]	ND	A (13)	21.8–23.7	Static	Pereyra et al. (2011)
15	160.1 ppm	168 [0]	ND	A (13)	21.8–23.7	Static	Pereyra et al. (2011)
15	1273.73 ppm	168 [0]	ND	A (19)	21.8–23.7	Static	Pereyra et al. (2011)
15	442.14 ppm	168 [0]	ND	A (19)	21.8–23.7	Static	Pereyra et al. (2011)
15	283.4 ppm	168 [0]	ND	A (19)	21.8–23.7	Static	Pereyra et al. (2011)
15	138.53 ppm	24 [0]	ND	L	21.8–23.7	Static	Pereyra et al. (2011)
16	% dead: 90	240 [0]	1 (2000)	A	15–22	Static	Angonesi et al. (2008)
16	% dead: 92	240 [0]	1 (4000)	A	15–22	Static	Angonesi et al. (2008)
16	% dead: 100	240 [0]	1 (6000)	A	15–22	Static	Angonesi et al. (2008)
16	% dead: 100	240 [0]	1 (8000)	A	15–22	Static	Angonesi et al. (2008)
16	% dead: 100	240 [0]	1 (12000)	A	15–22	Static	Angonesi et al. (2008)
16	8336.7 ppm	48 [48]	8 (1000–20000)	A (15–25)	25	Static	Calazans et al. (2013)
17	376.0 ppm	48 [48]	9 (1–2000)	A (15–25)	25	Static	Calazans et al. (2013)
17[a]	% dead: 86	8760 [0]	1 (1)	A	18–26	FT	Netto (2011)
18	344.95 ppm	48 [0]	7 (40–800)	A (21–26)	23–27	Static	Montresor et al. (2013)

Table 1 (continued)

Toxicant	LC_{50} or % dead	Exposure [postexposure] times (h)	No. of concentrations tested (range, ppm)	Stage (size in mm)	Temp. (ºC)	Test conditions	Reference
18	113.14 ppm	72 [0]	7 (40–800)	A (21–26)	23–27	Static	Montresor et al. (2013)
18	88.51 ppm	96 [0]	7 (40–800)	A (21–26)	23–27	Static	Montresor et al. (2013)
18[a]	% dead: 99	8760 [0]	ND	A	18–26	FT	Netto (2011)
19	% dead: 2	24 [168]	1 (0.5)	A (6–43)	24–25	FT	Boltovskoy et al. (2005)
19	% dead: 1	48 [168]	1 (0.5)	A (6–43)	24–25	FT	Boltovskoy et al. (2005)
19	% dead: 0.2	72 [168]	1 (0.5)	A (6–43)	24–25	FT	Boltovskoy et al. (2005)
19	663.6 ppm	48 [48]	4 (10–1000)	A (15–25)	25	Static	Calazans et al. (2013)
19	% dead: 100	720–2160 [0]	1 (1)	A	ND	FT	Cepero (2003)
19	300 ppm: 6 d	144 [0]	9 (0.2–400)	A	~20	FT	Morton et al. (1976)
19	400 ppm: 6 d	144 [0]	9 (0.2–400)	A	~20	FT	Morton et al. (1976)
19	200 ppm: 6.5 d	156 [0]	9 (0.2–400)	A	~20	FT	Morton et al. (1976)
19	1 ppm: 15.3 d	367.2 [0]	9 (0.2–400)	A	~20	FT	Morton et al. (1976)
19	0.8 ppm: 23 d	552 [0]	9 (0.2–400)	A	~20	FT	Morton et al. (1976)
20	46.54 ppm	24 [0]	5 (5–80)	A (21–26)	23–27	Static	Montresor et al. (2013)
20	19.84 ppm	48 [0]	5 (5–80)	A (21–26)	23–27	Static	Montresor et al. (2013)
20	14.29 ppm	72 [0]	5 (5–80)	A (21–26)	23–27	Static	Montresor et al. (2013)

Table 1 (continued)

Toxicant	LC_{50} or % dead	Exposure [postexposure] times (h)	No. of concentrations tested (range, ppm)	Stage (size in mm)	Temp. (ºC)	Test conditions	Reference
20	11.53 ppm	96 [0]	5 (5–80)	A (21–26)	23–27	Static	Montresor et al. (2013)
21	368.2 ppm	48 [48]	6 (10–2000)	A (15–25)	25	Static	Calazans et al. (2013)
22	0.58 ppm	24 [0]	5 (5–80)	A (21–26)	23–27	Static	Montresor et al. (2013)
22	0.35 ppm	48 [0]	5 (5–80)	A (21–26)	23–27	Static	Montresor et al. (2013)
22	0.29 ppm	72 [0]	5 (5–80)	A (21–26)	23–27	Static	Montresor et al. (2013)
22	0.25 ppm	96 [0]	5 (5–80)	A (21–26)	23–27	Static	Montresor et al. (2013)
23	% dead: 100	720–2160 [0]	1 (1)	A	ND	FT	Cepero (2003)
24	% dead: 100	720–2160 [0]	1 (1 ppm of Cu2+)	A	ND	FT	Cepero (2003)
24	% dead: 100	2160 [0]	3 (0.25–1 ppm of Cu2+)	A	ND	FT	Cepero (2003)

ND no data, *A* adults, *L* larvae, *FT* flow through

[a] Testing was carried out under plant conditions, comparing fouling developing on clean test vouchers enclosed in a steel pipe through which untreated (control) and treated water was circulated. Animals exposed to pH 9 for 24 h/day during 1 year. Mortality assessed by comparison with simultaneous control

been documented by numerous authors for dreissenids (Mackie and Claudi 2010). The mechanism appears to be the closing of the valves by veligers in the presence of an oxidant, thus preventing attachment to the substrate. Semicontinuous application of chlorine has been used with varying success. In adults, the application has to be such as to prevent them from opening their shells and recovering between chlorination events. Chlorination regime of 15 min on and 45 min off has been used successfully by several facilities in Canada for a number of years. Twice per day treatment of 1 h at 2 ppm has been shown to eliminate new settlement that has occurred in the last 12 h. However, this regime will not eliminate adult mussels (Mackie and Claudi 2010).

Reactive Application of Chemicals for Control of *L. fortunei*

Proprietary Nonoxidizing Chemicals

Nonoxidizing chemicals are generally used as reactive treatment to eliminate established adult population. This is primarily due to the fact that many of the proprietary nonoxidizing molluscicides must be detoxified prior to their release into open water environment. The proprietary chemicals also tend to be costly, making continuous application for settlement prevention not feasible. It is therefore their effect on adults that is of greatest interest. The effect of nonoxidizing chemicals on *L. fortunei* has been reported to be quite different from the effect some of these chemicals have on dreissenids.

Clam-Trol CT-2/Spectrus CT1300

The primary active ingredients of this product [50% n-alkyl (C12-50%, C14-40%, and C16-10%) dimethylbenzyl ammonium chloride] are cationic surfactants of the alkyldimethyl-benzyl ammonium chloride (ADBAC) family. There were three formulations of the product: CT-1, CT-2, and CT-3. The proportions of the various components varied in each formulation. Clam-Trol CT-2 was the product most tested on the golden mussel and it has been renamed relatively recently to Spectrus CT1300.

Boltovskoy and Cataldo (2003) tested the effect of Clam-Trol CT-2 at different concentrations (0.5–10 ppm). At the lowest concentration of 0.5 ppm no mortalities were observed. At 1 ppm LC_{50} was reached in 192 h, at 2 ppm it was 120 h, at 5 ppm it was 96 h, and at 10 ppm it was 72 h (Table 1). Interestingly, total mortality for 2 ppm, 5 ppm, and 10 ppm concentrations was reached at the same time: 192 h (Table 1). By comparison, the North American experience for dreissenids is that 2–5 ppm applied for 6–24 h will result in 100% adult mortality (McMahon 2008).

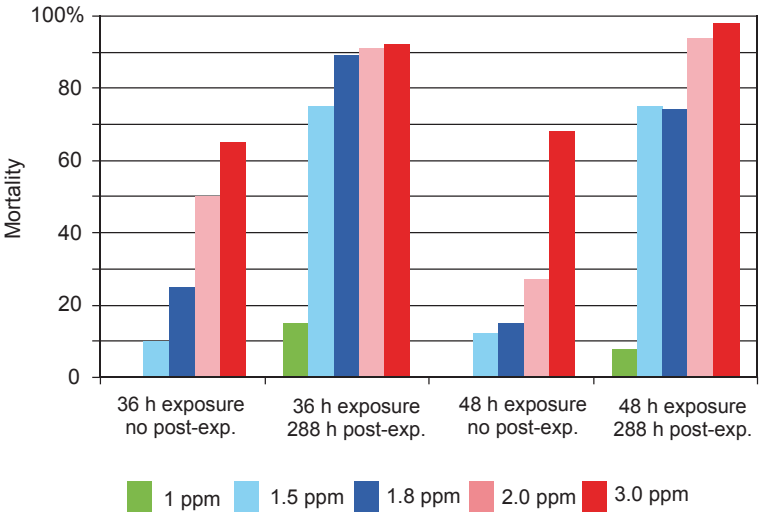

Fig. 1 Clam-Trol CT-2 exposure for 36 h followed by 288 h recovery. (Based on data from Cataldo et al. 2003)

Boltovskoy and Cataldo (2003) tested the effect of short-term exposure on adult *L. fortunei* followed by a period of recovery. They exposed the adult mussels to five concentrations (1, 1.5, 1.8, 2.0, and 3 ppm) for 36 and 48 h. These exposures were followed by a 12-day recovery during which the mussels were observed. After the recovery period, the authors observed significant postexposure mortality at all concentrations above 1 ppm (Fig. 1). This finding of postexposure mortality has significantly reduced the amount of product that needs to be used for reactive treatments in industrial facilities.

These postexposure mortality results are similar to those recorded by Cataldo et al. (2003). The authors report that 48 h exposure to concentration of 2.5 mg/L (ambient water temperature of 20 and 25 °C) resulted in 80–90 % mortalities after 3 days in recovery. It is interesting to note that at higher concentrations of the chemical, the authors noted that mussels in the test closed their shells and ceased filtering.

In a field experiment at the Itaipu Hydroelectric Power Plant in October 2002, the effect of Spectrus CT1300 (produced by GE Betz Inc.), with identical composition as Clam-Trol CT-2, was tested on *L. fortunei* in a continuous flow environment using bioboxes (Boltovskoy and Cataldo 2003). Adult mussels of various size classes were exposed to concentrations between 2.2 and 2.4 ppm of the product. The ambient water temperature was 22.5–23.5 ºC. Mussels were exposed for periods of 12, 24, 36, 48, 60, and 72 h. After the exposure period mussels were moved to the control biobox for a period of 5 days. Mortalities in the controls were less that 5 %. The mortalities in the treated groups increased with exposure time, and ranged from 42 % mortality at 12 h to 90 % at 72 h. The mortality rate began to decrease after 48 h (Table 1). There were no significant differences in mortality between different size classes.

Boltovskoy et al. (2005) tested Spectrus CT1300 once again on adult *L. fortunei* at Embalse Rio Tercero Nuclear Power Plant (Argentina). Chlorine as sodium hypochlorite was also included in the study. The tests were carried out using flow-through bioboxes. The ambient temperature was 24–25 ºC. Spectrus CT1300 was tested with a nominal concentration of 2.5 ppm (1.25 ppm active ingredient). Exposure times were 24, 48, and 72 h and postexposure (recovery) time was between 4 and 7 days. Chlorine was added for 4.5 h per day, and the nominal concentration was 1.5 ppm at the injection point and 0.5 ppm in the biobox. The flow rate to the bioboxes was set at 100 L per minute.

After 72 h, there was zero mortality in the control and only 1.2 % mortality in the biobox treated with chlorine. In the biobox treated with Spectrus CT1300, mortalities increased with time: 63 % of the mussels were killed in 24 h, 94 % in 48 h and 99 % in 72 h (Table 1). No significant differences were found between the mortality of different size classes of adult mussels indicating that the product works similarly across age classes. These results are comparable to those obtained by the authors at Itaipu in 2002 (Boltovskoy and Cataldo 2003).

The lack of difference in response by various size classes is in contrast to findings by Waller et al. (1993) working with dreissenids. These authors found a significant difference between the amount of chemical required to cause an LC_{50} in 48 h in 20–25-mm-long dreissenid mussels (0.738 mg/L of active ingredient) and 5–10-mm-long dreissenids (0.29 mg/L).

McMahon et al. (1994) found that Clam-Trol CT-1 had rapid zebra mussel toxicity at relatively low use rates (1.0–2.0 mg/L for 6–24 h).

Currently, the nuclear power plant Embalse de Río Tercero is using Spectrus at 2.5 ppm 2–3 times a year for 2 days. The 2-day treatment eliminates all settled adult golden mussels.

BULAB 6002

BULAB 6002 (Poly [oxyethylene (dimethyliminio) ethylene (dimethyliminio) ethylene dichloride]) is a liquid cationic polyquaternary ammonium compound used for the control of algae in swimming pools and as a microbicide for the control of microorganisms in commercial and industrial water systems. It also considered an effective molluscicide (Waller et al. 1993). Darrigran and Damborenea (2001) tested the effectiveness of BULAB 6002 on both juvenile and adult golden mussels. Cumulative mortality was assessed in six experiments conducted with different size classes of mussels and different concentrations of the product (8, 12, and 20 mg/L of active substance). At 24 ºC, the results show that 20 mg/L resulted in 100 % mortality of adult golden mussels in 144 h (Table 1). Boltovskoy and Cataldo (2003) tested several concentrations of BULAB 6002 (2, 5, 10, and 20 ppm) on adult golden mussels. At ambient temperature of 20–22 ºC LC_{50} was reached in 408 h at a concentration of 2 ppm of BULAB 6002, and 288 h for concentration of 20 ppm. Total mortality was reached after 672 h for the 2 ppm concentration and 552 h for the 20 ppm concentration. Interestingly, there was only 24 h difference

between reaching total mortality at 10 ppm versus 20 ppm (Table 1). In comparison, during trials with dreissenids, adult mortality at 0.5 ppm was reached in 826 h, at 2 ppm the time required was 313 h, and at 8 ppm total adult mortality was reached in 197 h (McMahon et al. 1993).

H-130 (Didecyl Dimethyl Ammonium Chloride)

This compound is a nonoxidizing liquid containing a solution of polyquaternary alkyl ammonium registered in North America for use as a molluscicide in industrial once-through freshwater cooling water systems. Because of its need for proper deactivation prior to discharge, in North America, it is sold only as part of a complete Calgon mollusk treatment application service, and is to be used only with supervision from a Calgon representative.

Boltovskoy and Cataldo (2003) tested the effect of several concentrations of H130 (0.5, 1, 2, 5, and 10 ppm) on adult golden mussels. At 0.5 ppm there was less than 50% mortality after 30 days. At 1 ppm LC_{50} mortality was reached in 96 h, while concentrations of 2,5 and 10 ppm reached LC_{50} in 48 h and total mortality in 120 h.

Cataldo et al. (2003) tested several concentrations of H130 (2.5, 5, 10, 20, and 30 mg/L) at 15, 20, and 25 °C ambient temperature using a 48 h exposure followed by a recovery period in clean water. At 15 °C, none of the tested concentrations achieved 100% mortality in postexposure recovery experiments. At 20 °C, only doses >10 mg/L achieved 100% mortality. At 25 °C, all concentrations down to 2.5 mg/L were 100% effective in 1 week or less in causing 100% mortality. Once again, during treatments with the higher concentrations of the chemical, authors noted that the mussels in the test chamber closed their shells and ceased filtering.

For comparison, in dreissenids, 1 ppm for 24 h causes 100% mortality in adults (McMahon 2008).

Bayluscide

Bayluscide (dichloro-2′nitro-4′ salicylanilide) was originally developed as a molluscicide to eliminate snails. It is not considered to be persistent in the environment; it breaks down in natural water and sediment systems through hydrolysis, photolysis, and microbial degradation (Dawson 2003).

Cataldo et al. (2003) tested several concentrations of Bayluscide (0.25, 0.5, 1, 2, 4, 6, and 8 mg/L) at 15, 20 and 25 °C using a 48 h exposure followed by a recovery period in clean water. At 15 °C, 48 h exposure at concentrations of 4 mg/L caused greater than 80% mortality. Concentrations between 1 and 2 mg/L killed 60–70%, and concentrations of 0.5 mg/L eliminated <20% of adults. All concentrations yielded postexposure mortalities only for the first 2–3 days during recovery. At 20 °C the final mortalities were generally higher and occurred faster than with

comparable concentrations at 15 °C with similar postexposure mortalities for up to 5 days. At 25 °C, the adult mussel response to concentrations between 0.5 and 8 mg/L was almost identical and >90% of the adult mussels were eliminated after 4 days during recovery. At 0.25 mg/L, mortality rate peaked to 30% (Table 1).

By comparison, a concentration of 0.05 mg/L has been reported to cause 70% mortality in 24 h in dreissenids, and 0.1 mg/L achieved 100% mortality in the same time period (Waller et al. 1993).

Veligon [Poly (Dimethyl Diallyl Ammonium Chloride)]

High-charge cationic coagulant dimethyl diallyl ammonium chloride (DMDAAC) compounds have been used in water treatment plants as flocculants and clarification aids. There are various Veligon formulations of these compounds and they differ in their molecular weight and cationic charge density. Flocculation binds up the veligers in the floc and there is also reported impact on adult dreissenids. In North America, it is approved for use in potable water.

Boltovskoy and Cataldo (2003) tested the effect of several concentrations of Veligon TL-M (2, 5, 10, 20 and 50 ppm) on adult golden mussels. At a concentration of 2 ppm there was no mortality during the 48 h exposure or after 30 days in recovery. For concentrations of 5, 10, 20, and 50 ppm the following mortalities were noted after 48 h: 30, 50, 75, and 80%. However, all the concentrations tested resulted in 100% delayed mortality with 11 and 30 days (Table 1).

By comparison, median reported LC_{50} at 96 h for adult zebra mussels is between 1.5 and 3.0 mg/L (Blanck et al. 1996).

Nonproprietary Nonoxidizing Chemicals

There are numerous other nonoxidizing chemicals that impact both adult and larval golden mussels. The challenge is to select those that are economical and have the least possible negative impact on the environment.

Copper Sulfate

The toxicity of copper to marine life has been recognized for centuries, and hence the use of sheets of copper on bows of sailing ships to prevent hull fouling. Copper-based antifouling paints are widely used on marine and freshwater watercraft. Mollusks are particularly sensitive to the presence of copper in the environment. Elevated levels of copper can result in such diverse effects as decreased growth rate, reproductive impairment, enzyme inhibition, reductions or alterations in protein synthesis, and disruptions of ATP synthesis and Ca^{2+} homeostasis (Clayton et al. 2000). The toxicity of copper in freshwater systems is greatly influenced by the total

Table 2 Effect of copper-based algaecides on dreissenid mussel adults. Average percent mortality after 96 h of exposure, and mortality following maximum recovery time allowed. (After Claudi et al. 2014)

	Low concentration				High concentration			
	Zebra		Quagga		Zebra		Quagga	
Algaecide	96 h	Max	96 h	Max	96 h	Max	96 h	Max
Copper sulfate (July)	0.8%	6.7%	–	–	25.0%	69.9%	–	–
Copper sulfate (Nov)	10.4%	14.2%	65.0%	92.4%	36.3%	52.1%	96.5%	99.4%
Natrix™ (July)	16.2%	62.2%	–	–	63.0%	97.2%	–	–
Natrix™ (Nov)	55.3%	64.4%	93.0%	99.6%	70.7%	84.6%	100%	–
Captain™	26.0%	43.5%	94.1%	–	72.5%	86.7%	94.1%	–
EarthTec®	100%	–	97.1%	–	100%	–	99.3%	–

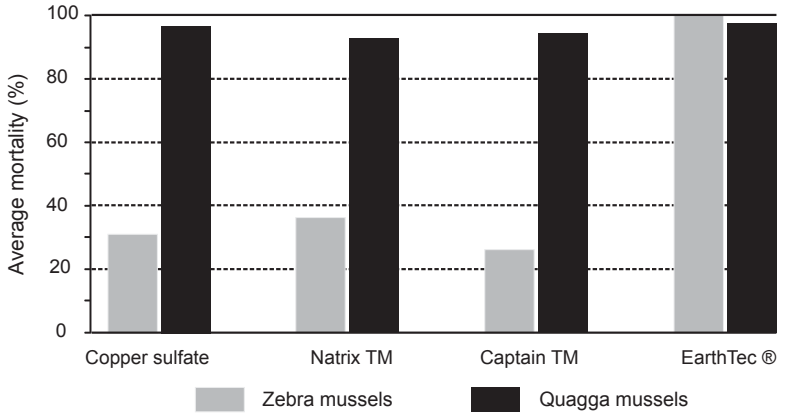

Fig. 2 Effect of copper-based algaecides (0.5 mg/L copper equivalent) on dreissenid mussel adults. (After Claudi et al. 2014)

hardness of water. Claudi et al. (2014) found four copper-based algaecides to be a viable tool for managing adult zebra and quagga mussel infestations, particularly in water bodies which require the use of these chemicals for the control of algae or aquatic plants (Table 2). Figure 2 shows the observed mortality in zebra and quagga mussels after 96 h exposure to 0.5 mg/L copper concentration of the four algaecides tested.

Soares et al. (2009) evaluated copper sulfate toxicity to golden mussels. The aim was to establish LC_{50} and LC_{95} doses which could be used for adult mussel control. Adult *L. fortunei* were exposed to 1.24, 2.33, 3.88, 5.43, 7.76, 10.08, 13.19, and 15.50 mg/L copper concentrations through analogous copper sulfate solutions ($CuSO_4$ $5H_2O$). Test organisms (1.8–2 mm in length) were acclimatized

(20 ± 1 °C, 24 h) in aquaria with air pumps in river water prior to exposure to the copper solution for 48 h. The average, minimum, and maximum LC_{50} and LC_{95} values were 2.16 (1.70 and 2.65), and 4.86 (3.97 and 6.47) mg/L, respectively.

Salinity

Salinity tolerance of the golden mussel was investigated by Angonesi et al. (2008). Ninety percent of the individuals of *L. fortunei* survived for at least 10 days after they were placed in salinity of 2‰. When exposed to salinity of 4 and 6‰, the adult mussels exhibited high tolerance only in the first 96 and 72 h, respectively, with a sharp decline thereafter. Similar salinity tolerance can be found in *L. fortunei* in the Asian and South American estuarine regions, such as the Changjiang River (China) (Huang et al. 1981), Río de la Plata estuary (Argentina) (Darrigran and Damborenea 2006), and in the Patos Lagoon (Brazil) (Capítoli and Bemvenuti 2004). In the studies above, *L. fortunei* survival differs from the findings by Deaton et al. (1989), where 80% of the animals survived in a salinity of 6.8‰ for time periods of 3 weeks or more, and less than a week in a salinity of 13.6‰. Angonesi et al. (2008) found *L. fortunei* specimens could only tolerate a salinity of 2‰ for a period of up to 10 days. For this same time period, salinities starting at 4‰ were fatal for at least 80% of the organisms. The higher tolerance found by Deaton et al. (1989) through salinity and osmotic regulation experiments (hemolymph osmotic and ionic composition and tissue amino acid content) may be due to the species' ecological adaptation in Asia, the species' origin (Deaton et al. 1989).

In agreement with the results of Angonesi et al. (2008), *L. fortunei* was found to be restricted to salinities below 2–3‰ in several South American brackish waterbodies (Capítoli and Bemvenuti 2004; Brugnoli et al. 2005; Darrigran and Mansur 2006; Darrigran et al. 2011). However, in 2004 and 2009 *L. fortunei* was recorded in the Río de la Plata estuary in the vicinity of Montevideo (Uruguay) (Giberto and Sardiña 2009), in an area periodically influenced by salinities in excess of 20‰ (Sylvester et al. 2013). This suggests that while the mussel is not tolerant of continuous salinities above 2 or 3‰, it is able to tolerate relatively high intermittent salinity exposure. Sylvester et al. (2013) evaluated the tolerance of *L. fortunei* to intermittent higher salinity by testing mussel mortality in 30-day experiments using both constant and fluctuating salinities at different temperatures in the laboratory. Test conditions simulated different seasons of the year and locations with increasing influence of marine waters in the Río de la Plata estuary. Significant mortality (31% after 30 days) was observed at a constant salinity of 2‰, increasing to 45 and 57% at 5 and 10‰, respectively. In contrast, considerably greater tolerances were observed when conditions in the experimental chamber fluctuated between saltwater and freshwater. No significant mortality was observed in mussels exposed to a salinity cycle with abrupt salinity changes ranging from 1 to 23‰ (mean 2.68‰) over a month. Tolerance was not affected by different temperatures. As mussels were observed to close their shells in higher salinity regimes (Boltovskoy pers. comm.), the tolerance of *L. fortunei* to short term salinity changes is likely due to the ability of the adults to avoid noxious conditions.

Calazans et al. (2013) observed LC_{50} of sodium chloride to be 8.3 ppt for a 48 h exposure. This suggests that high salinity could be used to eliminate settled adult golden mussels on freshwater ship hulls or ballast water tanks. By taking ships in need of clearing golden mussel infestation to very saline parts of the estuary for more than 2 days could help eliminate any fouling of the hull and of the cooling water systems. Filling ballast water tanks with saline waters could have the same effect thereby limiting further expansion of the geographic range of the mussel through ballast water introduction.

Ammonium Chloride

Montresor et al. (2013) tested the effect of unionized ammonia (TA-N) on adult *L. fortunei* using a solution of ammonium chloride. The concentrations of NH_3-N tested were 0.14, 0.21, 0.31, 0.50, and 0.72 mg/L at temperatures between 23 and 27 °C. The authors note that the current legal limits for concentrations of TA-N in Brazil are 5.6 mg/L TA-N at pH between 7.5 and 8.0. Given the high limits for TA-N in Brazil, ammonium chloride could be considered a viable treatment for elimination of adult mussels in this country.

Biobullets

Microencapsulated poison has been created in England to combat *D. polymorpha* in Britain. Calazans et al. (2013) tested microencapsulated chemicals, along with the traditional dissolved chlorine and potassium chloride (KCl), for the control of *L. fortunei*. The "biobullets" tested were a commercial blend of microencapsulated KCl and quaternary ammonia. The encapsulation coating was made of a mixture of binder starch, oil, and wax. The amount of microencapsulated KCL required to cause 50% mortality was ten times lower than for the dissolved form of the same chemical. The same study demonstrated similar effects for other microencapsulated substances. Since the amount of chemical released into the environment in microencapsulated form is substantially lower, Biobullets may be a more environmentally friendly alternative to deliver chemical treatment to adult mussels. At this point, the technology may still require some improvement. In a study commissioned by the Spanish government, the use of Biobullets was not rated as the best available technique due to the need to improve the stability of the microparticles and standardizing the Biobullet size to enhance particle retention by the target molluscs (Calazans pers. comm.).

Oxidizing Chemicals

Chlorine

Chlorine as chlorine gas, liquid sodium hypochlorite, calcium hypochlorite pellets, or pellets of sodium dichloroisocyanurate are used by a large majority of the plants in South America that use chemical control. Porto Primavera CESP (Brazil) uses sodium dichloroisocyanurate; Bariri Ibitinga AES Tietê (Brazil) use chlorine gas; Rosana, Taquaruçu, Canoas I and Canoas II of Duke Energy (Brazil) use chlorine gas, Itaipu (Brazil-Paraguay) uses chlorine gas and sodium hypochlorite; Tucuruí Eletronorte (Brazil) uses granular chlorine; Central Puerto and Termoeléctrica General Belgrano (Argentina) use sodium hypochlorite. Whatever the source, chlorine is consistently toxic at approximately the same concentration of Total Residual Chlorine (TRC) or Free Available Chlorine (FAC).

Numerous studies exist detailing the toxicity of chlorine to adults of *L. fortunei*. Cataldo et al. (2003) tested sodium hypochlorite concentrations (as free available chlorine) of 1, 5, 10, 25, 50, and 100 mg/L at three different exposure temperatures (15, 20, and 25 °C). The authors report that at 15 °C, there were no mortalities at any concentration of chlorine for 2 weeks. After that, except for the lowest dose of 1 mg/L, all concentrations resulted in 100 % mortality in 2–4 weeks. At 1 mg/L, there was only 30 % mortality at the end of the experiment on day 47. At 20 °C, chlorine concentrations between 5 and 100 mg/L resulted in 100 % mortality after about 1 month of exposure; at a dose of 1 mg/L 50 % mortality was observed at the end of the experiment. At 25 °C, the effects of chlorine were almost identical throughout the entire range of concentrations tested; 100 mg/L chlorine required 11 days, and 1 and 5 mg/L chlorine required 17 days for total mortality. The calculated LC_{50} values at 15 °C were 25 days at 93.2 mg/L, 30 days at 51.7 mg/L, 35 days at 27.2 mg/L, 40 days at 14.0 mg/L, and 45 days at 2.1 mg/L (Table 1). At 20 °C the calculated LD50 values were 20 days at 3.3 mg/L and 25 days at 1.2 mg/L. At 25 °C, the calculated LD50 value was 10 days at 5.5 mg/L (Table 1).

Adult mussels recognize chlorine as a noxious substance and keep their shell closed as long as possible before they are forced to take in water for respiration. The length of time they are able to keep the shells closed depends on the ambient water temperature. The warmer the water, the shorter is the time of shell closure. This explains why no mortality was observed at 15 °C for 2 weeks. Higher doses of chlorine do not translate into significantly lower time to mortality as reflected by the LC_{50} values: at 15 °C 25 days were needed at 93.2 mg/L, and 45 days at 2.1 mg/L (Cataldo et al. 2003). These results generally align with earlier studies on *L. fortunei* (Morton et al. 1976).

CESPI (Companhia Energetica de São Paulo) reports good results when treating cooling water systems for 2 h/day by addition of sodium hypochlorite at a concentration of 1.5 ppm residual in the system. This type of strategy has been shown to eliminate freshly settled juveniles of *Dreissena*. The freshly settled juveniles have relatively fragile shells that can be oxidized, exposing the juvenile to chlorine attack. Adults are not affected as their shells are robust and can be kept closed during short periods of chlorination.

Sodium Dichloroisocyanurate

This compound is mainly used as a disinfectant, biocide, industrial deodorant, and detergent. It is found in some modern water purification tablets and filters. In these applications, it is a source of slow release of chlorine in low concentrations at a relatively constant rate. CESPI has successfully tested the use of sodium dichloroisocyanurate as an alternative source of chlorine for golden mussel control. The advantage is easier storage and no formation of trihalomethanes. On the negative side, the product is effervescent and forms microbubbles that can negatively impact pump performance and must be eliminated prior to the addition of the dissolved product. The strategy is to maintain 1.5 ppm residual of chlorine in the pipe for 2 h during the breeding season and to cut back to 0.5 ppm residual for 2 h in the winter.

Concluding Remarks

The advantage of chemical control is the ability of such a strategy to protect the entire system, from the point of chemical injection to the point of water discharge. It can be applied continuously or semicontinuously to eliminate settlement or it can be applied periodically to eliminate adults. Chemical addition is quick to implement with generally modest capital expenditure. However, in many parts of North America and Europe, chemical controls for macrofouling are more and more difficult to implement due to strict regulatory limits on the presence of chemicals in the discharge water.

Many chemicals will cause mortality in golden mussels; however, worker safety, cost, and protection of the environment, protection of materials of construction and ambient water quality all have to be evaluated when contemplating a chemical protection strategy. As requirements and conditions differ between facilities, no one method or chemical will be suitable at every location. The same is true of proactive versus reactive treatments. Facilities coping with massive infestation levels are more likely to require proactive or frequent periodic treatment to keep sensitive components from failing. During the periodic treatments, it is essential to apply the chemical continuously until mortality of all adults has been reached. Any break in chemical application will allow the adults to recover and the overall length of treatment will increase.

The timing of periodic or end of season treatments needs to take ambient water temperature into consideration. Most, if not all chemicals will cause mortality more quickly and at lower concentration in warm water (20–30 °C) then in temperature below 15 °C (Fig. 3). To minimize treatment time and lower the cost of chemicals required, warm water treatments are recommended.

Finally, from the above review of the chemical methods of control we must conclude that the adults of *L. fortunei* appear to require higher doses or longer treatment periods than adult dreissenids. This observation is likely the result of the higher tolerance *L. fortunei* has for a variety of environmental conditions and polluted water

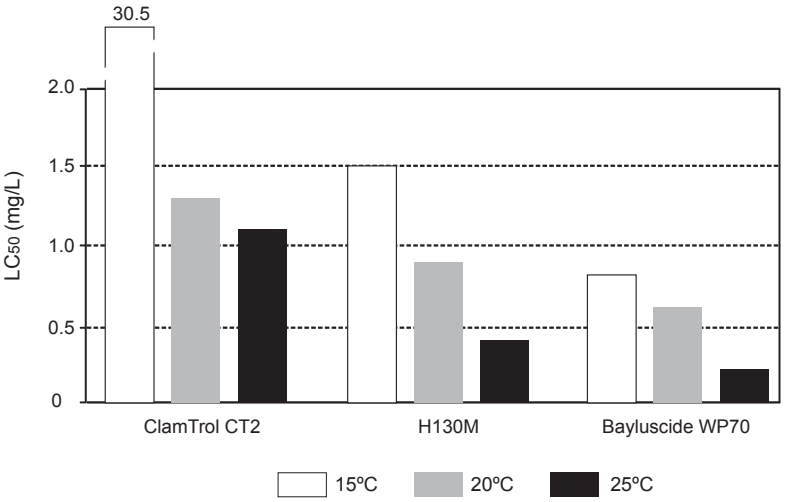

Fig. 3 LC_{50} values for three different molluscicides at 15, 20 and 25 ºC. In all cases exposure times are 2 days; concentrations are based on percent active ingredient. (Based on data from Cataldo et al. 2003)

(Villar et al. 1999; Belaich et al. 2006; Boltovskoy et al. 2006; Young et al. 2014). It is, however, important to note that the impact of oxidizing chemicals on veligers or freshly settled juveniles is identical between the two species. In proactive treatments preventing settlement, the veligers do detect the oxidant as a noxious substance, close their shells and do not attach. In freshly settled juveniles, the impact of the oxidizing chemical is due to an attack on the fragile shell provided the oxidant dose is high enough for a long period of time.

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