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Acute Effects of Potassium on Filtration Rates of Adult Zebra Mussels, *Dreissena polymorpha*

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ABSTRACT. *Valve response and filtration rates of adult zebra mussels,* Dreissena polymorpha, *exposed to elevated levels of K^+ were assessed to determine the efficacy of using K^+ in conjunction with a biocide to control mussel infestations. Mussels, 10 to 15 mm total shell length, were exposed to K^+ at concentrations ranging from ambient to 2.00 mmol/L for 3, 6, 12, and 24 h at temperatures of 12 and 22°C. After exposure, each mussel was tested for valve response by tactile stimulation and filtration rate was estimated by clearance of latex microspheres from test solutions during a 30 minute period. Responsiveness was not affected in mussels exposed to ≤ 1.00 mmol/L K^+ for up to 24 h. Valve closure, however, was inhibited in ≥ 92% of the mussels after exposure to 2.00 mmol/L K^+ for ≥ 12 h. Thus, the use of K^+ to inhibit valve closure in mussels during the application of some biocides may increase contact time between the biocide and the soft tissues of mussels. Filtration rates of mussels, however, decreased as the K^+ concentration increased from 0.50 to 2.00 mmol/L. At the K^+ concentration that inhibited valve closure (2.00 mmol/L), filtration rates of mussels were > 90% lower than mussels at ambient K^+ levels suggesting less biocide may be drawn in the mantle cavity to act on susceptible tissues. Therefore, use of K^+ to prevent valve closure during the application of a biocide may not enhance the efficacy of the biocide due to the inhibitory effects of K^+ on filtration. Furthermore, facilities treated with K^+ at levels necessary to prevent valve closure may threaten native bivalve populations.*

INDEX WORDS: *filtration rate, control, potassium,* Dreissena polymorpha, *zebra mussel.*

INTRODUCTION

The zebra mussel, *Dreissena polymorpha* (Pallas) is one of the most recent exotic species to become successfully established in fresh waters of North America (Mills *et al.* 1993). Zebra mussels, native to the Caspian Sea region, were introduced into the Great Lakes in 1986, most likely as veliger larvae from the discharge of ballast water of transoceanic ships (Hebert *et al.* 1990). Zebra mussels have since become a serious biofouler in municipal and industrial facilities that draw water from infested lakes and rivers (Nalepa and Schloesser 1993). In some facilities, average mussel densities can range up to 1,000,000 individuals/m^2, resulting in clogged screens and filters, obstructed valves, and limited flow rates (Claudi and Mackie 1994).

Various chemical control measures have been implemented in facilities faced with biofouling prob-

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lems. Chlorination is an effective and popular oxidizing agent for control of zebra mussels in Europe and the United States, but results in the production of carcinogenic trihalomethane compounds (Claudi and Evans 1993). In contrast, the use of nonoxidizing chemicals that contain polyquaternary ammonia as the active ingredient do not produce carcinogenic by-products and are toxic to mussels at relatively low concentrations (McMahon *et al.* 1993). Many nonoxidizing agents, however, are more toxic to nontarget organisms than to zebra mussels (Waller *et al.* 1993). Waller *et al.* (1993) found that Bayluscide, Clamtrol CT-1, and potassium chloride were the only three compounds out of 18 chemicals tested that were more toxic to zebra mussels than to fish.

Discovery that several potassium salts are lethal to zebra mussels at concentrations that are not toxic to nontarget organisms has prompted some investigators to suggest K^+ as a possible control agent for zebra mussels (Fisher *et al.* 1991, Waller *et al.* 1993). Potassium chloride (KCl) had the greatest selectivity for zebra mussels over other candidate control agents and ranked intermediate in toxicity to zebra mussels. Other investigators have also considered KCl as a possible control agent for the biofouling Asiatic clam, *Corbicula fluminea* (Imlay 1973, Anderson *et al.* 1976, Daum *et al.* 1977). The use of KCl as a control agent for zebra mussels, however, may not be economically or environmentally feasible since K^+ is toxic to mussels at only high concentrations (24 h LC_{50} = 200 mg/L at 22°C, Wildridge *et al.* 1998).

An alternative method of utilizing K^+ to control zebra mussels involves the use of K^+ in conjunction with a biocide. The valves of the mussels must be parted in order for the surrounding medium containing the biocide to contact the mussel's soft tissues. Several studies have shown that increased periods of valve closure occur in zebra mussels and other bivalves in response to elevated levels of chlorine, organic compounds, and heavy metals (Slooff *et al.* 1983, Doherty *et al.* 1987, Kramer *et al.* 1989). Since valve closure prevents a controlling agent from reaching the susceptible tissues of bivalves, the effectiveness of the chemical treatment program is reduced. For example, valve closure contributed to the low toxicity of some heavy metals to Asiatic clams (Rodgers *et al.* 1980).

In contrast, exposure to elevated levels of K^+ has been shown to prevent valve closure in the Asiatic clam (Anderson *et al.* 1976). If K^+ similarly inhibits valve closure in zebra mussels, then exposure of mussels to K^+ prior to application of a biocide may enhance chemical control programs (Neuhauser *et al.* 1994). Preliminary studies indicate that the addition of K^+ (100 mg/L as KCl) to various chemical agents significantly reduces the time to obtain 95% mortality in zebra mussels (Lewis 1996). Therefore, prevention of valve closure in mussels may reduce the biocide concentration and exposure time necessary to achieve a desired mortality.

In addition, filtration rates of mussels exposed to K^+ may affect the rate of biocide reaching susceptible tissues since filtration draws the surrounding medium containing the biocide into the mantle cavity. If the K^+ concentration required to prevent valve closure also lowers filtration rates of mussels, then treatment of mussels with K^+ prior to exposure of a biocide may not enhance chemical control programs. Filtration rates of freshwater bivalves in response to elevated levels of K^+ have not been measured, but ciliary activity in excised gill tissue from *D. polymorpha* ceased in mussels exposed to > 4.3 mmol/L K^+ (Fisher *et al.* 1991, O'Donnell *et al.* 1996) suggesting an inhibitory effect of K^+ on mussel filtration.

Another concern with the use of K^+ in control methods is the impact the effluent from treated facilities will have on aquatic organisms that reside in receiving waters. Chronic exposure to extremely low levels of K^+ is lethal to North American freshwater bivalves (Imlay 1973). Imlay (1973) reported that a K^+ concentration of 0.27 mmol/L is lethal to 90% of the individuals of three unionid bivalve species in 52 days and only 0.18 mmol/L is lethal to two species within 8 months. Several investigators also report that valve activity patterns of the freshwater clam *Anodonta cygnea* are altered at K^+ concentrations of 1 mmol/L (Koshtovants and Salanki 1958; Lukacsovics and Salinki 1968). Thus, the use of K^+ in methods to control mussel infestations may threaten native unionids in some areas of the Great Lakes and surrounding waterways that have not already been extirpated by zebra mussels (Schloesser *et al.* 1996).

The objective of this study was to assess valve response in zebra mussels exposed to elevated levels of K^+ to determine if K^+ inhibits valve closure. Since K^+ may also affect filtration rates of mussels, filtration was measured in mussels exposed to elevated concentrations of K^+. Thirdly, this study examines if the use of K^+ in mussel control programs at concentrations necessary to maintain valve par-

ture is potentially harmful to native unionid bivalves.

METHODS

Rocks colonized by *D. polymorpha* were collected by hand May through August at a water depth of 2 to 3 m from Little Sodus Bay, Lake Ontario, New York. Mussels were removed from the rocks by severing byssal threads with a scalpel and transported to the laboratory in a cooler containing aerated lake water within 24 h. Mussels (ca. 1,000) were held for a maximum of 2 weeks in a 12-L aquarium containing filtered (10 μm) lake water that was aerated and recirculated through an internal filter of activated carbon chips (Ammo-Chips®, Aquarium Pharmaceuticals, Inc., Chalfont, PA) to remove ammonia. Lake water in the aquarium was exchanged at least weekly (Nichols 1993) and mussels were not fed (Kilgour and Baker 1994). The aquarium was placed in a circulating water bath that maintained water temperature within ±1°C of the test temperature and received a light:dark photoperiod of 16:8 h.

Mussels selected for testing were 10 to 15 mm in total shell length (14.2 ± 0.31 mm, $\bar{X}$ ± S.E.) and were not significantly different among the tests or K^+ concentrations. Mussels exhibited normal activity of having valves parted, siphons extended, and byssal thread attachment. Forty-eight hours prior to testing, each mussel was secured to a polystyrene weighing dish to allow transfer of mussels with minimal handling. Mussels were secured by adhering the lateral surface of one valve to the raised edge of the dish with silicone. Ventral surfaces of mussels rested on the dish bottom to allow byssal thread attachment. Mussels that did not remain secured to the polystyrene were discarded and replaced.

Filtered (10 μm) lake water was used as control and dilution water. Chemical characteristics of the lake water ($\bar{X}$ ± S.D.) were: K^+ concentration of 0.05 ± 0.009 mmol/L, pH of 8.0 ± 0.15, total hardness of 123 ± 4.2 mg $CaCO_3$/L, and total alkalinity of 1.3 ± 0.13 meq/L (70 ± 5.1 mg $CaCO_3$/L). Actual K^+ concentrations of the test solutions were measured with a K^+ electrode (Orion Research, Inc., Boston, MA) and were within 5% of the target concentration.

Tests were conducted during the early and late summer months at water temperatures of 12 and 22°C, respectively. Each beaker (100 mL) contained one immobilized mussel and 50 mL of either lake water (control) or test solution. Twenty mussels each were simultaneously exposed to K^+ concentrations of 0.05 (ambient), 0.12, 0.25, 0.50, 1.00, and 2.00 mmol/L. After 3, 6, 12, and 24 h exposure, five mussels at each concentration were assessed for valve response and filtration rate was measured in each mussel. Dissolved oxygen, pH, and free ammonia levels from each test concentration were measured initially and at the end of each exposure period, but no significantly changes ($p < 0.05$) in these values were observed.

Valve response was assessed by tactile stimulation with a blunt probe. Mussels were designated responsive if valve movement was observed during stimulation or nonresponsive if valve movement was not observed. Mussels with their valves closed could not be tested for responsiveness and were counted separately.

Filtration rate of each mussel was estimated from clearance of latex microspheres during a 30 minute period. After assessing valve response, each mussel was transferred to a new beaker containing 50 mL of the same K^+ concentration and a known concentration of microspheres (2.01 ± 0.05 μm in diameter; Duke Scientific, Palo Alto, CA). Microsphere concentration in each beaker was estimated by measuring the absorbance of the test solution prior to the addition of a mussel and 30 minutes later. A 30-minute period was selected to minimize any effects probing and transferring the mussels had on filtration. Absorbance was measured with a Milton Roy 21D Spectrophotometer at a wave length of 950 nm and converted to microsphere concentration (mg/L) based on a standard curve. Filtration rate was calculated according to Coughlan's (1969) formula.

A separate test was conducted to measure the settling rate of microspheres since the test solutions were not stirred and the microspheres are slightly more dense than water (1.05 g/L). Settling rate was estimated in 30 beakers without mussels and calculated from the change in microsphere concentration during a 30 minute period. The mean settling rate was deducted from each estimate of filtration rate.

After measuring filtration rate, mussels were placed in fresh lake water for 24 h to assess mortality. The test was repeated five times at each temperature using a different set of mussels for each test. Mean filtration rate and mean percent of nonresponsive mussels were calculated at each K^+ treatment for each test. Means for all tests were averaged and data are presented as the cumulative mean ± standard error of the five tests for each treatment. Means were statistically compared by

one and two-way ANOVA followed by Fisher's Protected Least Significant Difference post hoc Multiple t-test (Sokal and Rohlf 1981). Filtration data were log transformed to achieve homogeneous variances for statistical analysis (Bartlett's test, Sokal and Rohlf 1981). Analyses were performed using StatView (Abacus Concepts, Inc., Berkeley, CA, Version 4.01) and differences were considered significant at $p \leq 0.05$.

RESULTS

Valve Response

Nonresponsiveness was induced in mussels that were exposed to 2 mmol/L K^+ for ≥ 3 h (Table 1). At least 92% of the mussels were nonresponsive after 12 h exposure to 2.00 mmol/L K^+. Valves of affected mussels were parted, but were not widely gaping, and the mussels often exhibited siphon extension. The valves showed no movement in response to tactile stimulation of the valves, extended siphons, or other exposed mantle tissue. Valve response was not affected in mussels exposed to K^+ concentrations ≤1.00 mmol/L for up to 24 h at either temperature.

The percent of nonresponsive mussels produced at 12°C (early summer) was not significantly different from the percent of nonresponsive mussels produced later in the summer at 22°C at any exposure period (Table 1). Exposure to 2.00 mmol/L K^+ for ≥3 h was necessary to inhibit valve closure at both temperatures. Thus, the affect of K^+ on valve response was similar in early and late summer mussels.

Although a higher water temperature had no affect on the percent of nonresponsive mussels, the duration of the exposure period was positively related to the percent of nonresponsive mussels. The percent of nonresponsive mussels exposed to 2.00 mmol/L K^+ significantly increased from 12 to 96% as the duration of exposure increased from 3 to 12 h. There was, however, no significant increase in the number of nonresponsive mussels as the exposure time increased from 12 to 24 h.

All nonresponsive mussels recovered within 10 minutes when placed in lake water at ambient K^+ levels, indicating the K^+ treatments were not lethal. Furthermore, no mortalities occurred in the controls or any test solution following the 24 h recovery period. Valve closure was observed in some mussels exposed to ambient and elevated K^+ levels preventing the assessment of valve response at the end of an exposure period, but valve closure occurred in less than one percent of mussels at each treatment.

Filtration Rate

Filtration rates of mussels decreased as the K^+ concentration increased from 0.25 to 2 mmol/L after only 3 h of exposure (Fig. 1). Filtration was significantly reduced at K^+ concentrations that were lower than the concentration necessary to induce nonresponsiveness. At the K^+ concentration that induced nonresponsiveness (2 mmol/L), filtration rates were 90% lower than mussels at ambient K^+ levels. Filtration rates were significantly reduced at

TABLE 1. Percent of* Dreissena polymorpha *that were nonresponsive to tactile stimulation after 3, 6, 12, and 24 h exposure to 2 mmol/L K^+ at 12°C and 22°C (mean ± s.e.m.)[1,2,3]

Temperature (°C)	Exposure Period (h)			
	3	6	12	24
12	40 ± 9[a]	64 ± 7[a]	92 ± 8[b]	96 ± 4[b]
22	12 ±12[a]	68 ± 10[b]	96 ± 4[c]	99 ± 1[c]

[1]Means within a row with different letters are significantly different ($p \leq 0.05$).
[2]Means within a column are not significantly different ($p \leq 0.05$).
[3]n = 20 for all treatments.

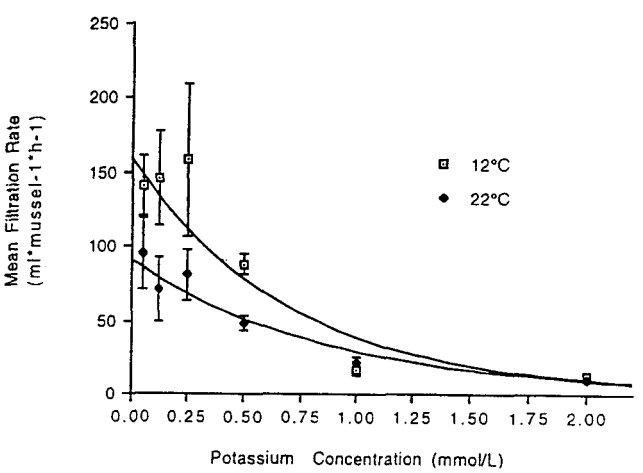

FIG. 1. Filtration rate (mean ± s.e.m.) of* Dreissena polymorpha *after 24 h exposure to increasing K^+ concentrations at 12°C and 22°C. (If no error bar, then the s.e.m. is smaller than the symbol; 12°C: $y = 159*10^{-0.02x}$, $r^2 = 0.85$; 22°C: $y = 91*10^{-0.01x}$, $r^2 = 0.99$).

TABLE 2. Filtration rate (mL/mussel/h) of* Dreissena polymorpha *after 3, 6, 12, and 24 h exposure to increasing K^+ concentrations at 12°C (mean ± s.e.m.)[1, 2, 3]

K^+ conc. (mmol/L)	Exposure Period (h) 3	6	12	24
0.05	239 ± 45^a	206 ± 41^a	157 ± 16^a	141 ± 20^a
0.12	169 ± 15^a	213 ± 38^a	190 ± 23^a	146 ± 32^a
0.25	$184 \pm 30^{a,b}$	$145 \pm 40^{a,b}$	$148 \pm 18^{a,b}$	$158 \pm 51^{a,b}$
0.50	83 ± 14^b	67 ± 7^b	79 ± 17^b	88 ± 7^b
1.00	12 ± 2^c	17 ± 4^c	15 ± 3^c	16 ± 3^c
2.00	9 ± 1^c	11 ± 1^c	16 ± 3^c	13 ± 1^c

[1]Means within a column with different letters are significantly different ($p \leq 0.05$).
[2]Means within a row are not significantly different ($p \leq 0.05$).
[3]n = 20 for all treatments.

TABLE 3. Filtration rate (mL/mussel/h) of* Dreissena polymorpha *after 3, 6, 12, and 24 h exposure to increasing K^+ concentrations at 22°C (mean ± s.e.m.)[1, 2, 3]

K^+ conc. (mmol/L)	Exposure Period (h) 3	6	12	24
0.05	86 ± 11^a	56 ± 9^a	85 ± 15^a	95 ± 24^a
0.12	100 ± 15^a	79 ± 18^a	70 ± 16^a	71 ± 22^a
0.25	70 ± 16^a	70 ± 10^a	58 ± 21^a	81 ± 17^a
0.50	$68 \pm 20^{a,b}$	$50 \pm 11^{a,b}$	$48 \pm 11^{a,b}$	48 ± 5^a
1.0	$21 \pm 5^{b,c}$	$17 \pm 4^{b,c}$	$18 \pm 3^{b,c}$	22 ± 3^c
2.0	8 ± 2^c	11 ± 4^c	12 ± 2^c	10 ± 1^c

[1]Means within a column with different letters are significantly different ($p \leq 0.05$).
[2]Means within a row are not significantly different ($p \leq 0.05$).
[3]n = 20 for all treatments.

K^+ concentrations as low as 0.5 mmol/L at 12°C (Table 2) and 1.0 mmol/L at 22°C (Table 3).

The inhibitory effect of K^+ on filtration occurred within 3 hours exposure to elevated K^+ concentrations. Increasing the duration of exposure from 3 to 24 h did not cause a further reduction in filtration rates at 12°C (Table 2) or 22°C (Table 3). Similarly, increasing the K^+ concentration from 1.00 to 2.00 mmol/L did not result in lower filtration rates of mussels (Tables 2 and 3). Therefore, filtration rates of nonresponsive mussels at 2.00 mmol/L K^+ were similar to that of responsive mussels at 1.00 mmol/L K^+.

Filtration rates were significantly reduced to 8 to 15 mL/mussel/h in mussels exposed to the K^+ concentration that induced nonresponsiveness (2.00 mmol/L K^+). Although ambient filtration rates of mussels at 12°C were significantly higher than rates of mussels at 22°C, the effect of K^+ on filtration was similar for early summer mussels at 12°C compared to late summer mussels at 22°C.

DISCUSSION

This study demonstrates that valve closure can be prevented in zebra mussels following > 3 h exposure to 2.00 mmol/L K^+. Affected mussels had their valves parted and were not capable of valve movement in response to tactile stimulation. Furthermore, mussels exposed to K^+ at levels necessary to inhibit valve closure had significantly reduced filtration rates. Filtration rates of mussels were found to be more sensitive to elevated environmental K^+ levels, since rates were lowered at K^+ concentrations as low as 0.50 mmol/L whereas valve closure was not prevented in mussels until the K^+ concentration exceeded 1.00 mmol/L.

The mechanism by which K^+ inhibits contraction of the posterior adductor muscle preventing valve closure in bivalves is not known. Dietz and Byrne (1990) hypothesized that excess K^+ in the blood may depolarize nervous tissue resulting in the inability of *C. fluminea* to close their valves when stimulated. Salanki (1961) discovered that when nerves from the siphons to the visceral ganglia were severed the effect of K^+ on the rhythmic activity of the valves of *A. cygnea* ceased, indicating that K^+ ions act on receptors on the body surfaces which are in direct contact with the surrounding medium. Evidence from this study suggests that K^+ may also act on external receptors of *D. polymorpha* preventing valve closure since nonresponsive mussels recover rapidly (< 10 minutes) when transferred to freshwater.

Filtration rates of zebra mussels were found to decrease with increasing K^+ concentration of the surrounding medium. Similarly, elevated levels of copper, cadmium, zinc (Kraak *et al.* 1994), lead (Bleeker *et al.* 1992), and nickel (Stuijfzand *et al.* 1995) lower filtration rates of *D. polymorpha*. Lower filtration rates of bivalves exposed to heavy metals and other toxicants has been attributed to increased periods of valve closure (Salanki and V.-Balogh 1989). Lower filtration rates of zebra mussels exposed to K^+, however, occurred in mussels not capable of valve closure, suggesting filtra-

tion is reduced due to the affects of K^+ on gill ciliary activity. Evidence to support this is given by studies conducted by Fisher *et al.* (1991) and O'Donnell *et al.* (1996) that demonstrate elevated levels of K^+ inhibit gill ciliary activity in *D. polymorpha*.

The mechanism by which K^+ affects ciliary activity is not known. Using noninvasive phosphorus-31 nuclear magnetic resonance spectroscopy, O'Donnell *et al.* (1996) showed that exposure of mussels to 2.1 mmol/L KCl causes some loss in ATP pools suggesting loss of energy necessary to sustain ciliary beating. Dietz and Byrne (1990) found that elevated extracellular K^+ is rapidly taken up by zebra mussels, most likely across the gill epithelium, and may lead to an inability to regulate cell volume. Widespread vacuolation and disruption of the gill membrane were reported by Fisher *et al.* (1991) in gill tissue of zebra mussels exposed to elevated K^+ levels. Furthermore, Dietz *et al.* (1994) hypothesized that elevated K^+ levels may cause electrochemical imbalances in excitable tissues such as the gill epithelium, therefore affecting gill ciliary activity and ultimately filtration.

This study demonstrates that K^+ can be used to prevent valve closure in zebra mussels. Thus, the use of K^+ in conjunction with a biocide may increase mussel mortality since K^+ prevents valve closure allowing for continuous contact of the biocide to soft tissues of mussels. The efficacy of one commercial nonoxidizing biocide, however, was not enhanced in mussels exposed to K^+ at levels that inhibited valve closure during exposure to the biocide (Wildridge *et al.* 1998). In fact, in several instances mortality was reduced in mussels exposed to K^+.

This study shows that filtration rates of mussels are also significantly lower in mussels exposed to K^+ at levels that prevent valve closure. This finding may explain why Wildridge *et al.* (1998) observed reduced mortality in K^+-treated mussels. Bivalves are suspension feeders ingesting mostly particulate matter and not dissolved substances such as many biocides (Morton 1971). Therefore, as long as a biocide dissolved in the medium contacts the soft tissues of bivalves, mortality can occur. The currents created by ciliary beating, however, appear to be more important then simply maintaining valve parture in determining the amount of a dissolved biocide actually reaching and acting on susceptible tissues.

There is also concern for using K^+ to control zebra mussels in facilities that discharge their effluent directly into areas of the Great Lakes that harbor native unionid bivalves. Many populations of unionid bivalves in the Great Lakes basin have already been extirpated or nearly extirpated since the arrival of zebra mussels, but there are some areas where unionids and zebra mussels coexist (Schloesser *et al.* 1996). This study shows that a relatively high level of K^+ is necessary to prevent valve closure (2.00 mmol/L K^+) in zebra mussels. Although the K^+ discharged from treated facilities would be diluted since ambient K^+ levels in the Great Lakes is low (0.04 to 0.08 mmol/L; Great Lakes Basin Commission 1975), long-term use of K^+ may elevate levels to levels that are harmful to native bivalves. Imlay (1973) predicted the maximum safe level of K^+ for the continued existence of most freshwater bivalves is 0.10 to 0.25 mmol/L, which is only slightly higher than ambient levels.

This study finds that acute exposure to low levels of K^+ (0.50 mmol/L) causes lower filtration rates of zebra mussels. If filtration rates are also lowered in unionid bivalves following chronic exposure to waters with elevated K^+ levels, then long-term survival of native bivalves may be reduced by preventing bivalves from obtaining nutrients adequate for survival. This may explain Imlay's (1973) finding that waters with a K^+ concentration > 0.25 mmol/L do not support native unionid bivalves. Therefore, approval for use of K^+ in methods to control mussels in the Great Lakes region is questionable. Potassium, however, may be useful in closed systems or facilities with high dilution capabilities, or facilities that utilize precipitation methods or other measures to avoid elevating K^+ levels in areas that support native unionid bivalves.

In conclusion, use of K^+ to control macrofouling populations of *D. polymorpha* by preventing valve closure in mussels may not be practical due to the inhibitory effects of K^+ on filtration and the potential impacts on native bivalves. At K^+ concentrations required to prevent valve closure, filtration rates are significantly lower and appear to minimize the affects of at least one biocide. In addition, K^+ concentrations as low as 0.50 mmol/L affect filtration rates in zebra mussels and may similarly inhibit filtration rates of native mussels. Thus, the use of K^+ may limit the long-term survival of native bivalves in some areas of the Great Lakes.

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