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The Toxicity and Bioaccumulation of Selenate, Selenite and Seleno-L-Methionine in the Cyanobacterium *Anabaena flos-aquae*

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Abstract. A laboratory investigation was conducted to study the toxicity and bioaccumulation of seleno-L-methionine, selenate and selenite in the cyanobacterium *Anabaena flos-aquae*. The first sub-lethal effects of seleno-L-methionine, selenite and selenate occurred at 0.1, 3.0, and 3.0 mg/L, respectively with a decrease in chlorophyll *a* concentration ($P < 0.0001$). Selenium bioconcentration factors (BCF) were in the order of seleno-L-methionine, selenite, and selenate. Significant decreases in intracellular selenium concentration were observed at both the no effect (NOEL) and low effect levels (LOEL) at each oxidation state tested in the given experiment ($p < 0.0001$). Mechanisms for the assimilation, toxicity and regulation of selenium are presented.

Selenium, a natural trace element in aquatic ecosystems, is an essential micronutrient which becomes toxic at slightly higher levels than nutritional. Like other Group VI elements, such as sulfur, dissolved waterborne selenium occurs in three oxidation states: Se^{+6} which is present as selenate, an analogue of sulfate; Se^{+4} which occurs as selenite; and Se^{-2} which can occur as organic or inorganic selenides. The organic selenides can be incorporated into sulfur's biochemical pathways, forming selenomethionine and selenocysteine, analogues to the essential amino acids methionine and cysteine.

Selenate and selenite are the dominant waterborne species, but a substantial portion of organic selenium has been recorded in aquatic systems (Cutter and Bruland 1984; Robberecht and Van Grieken 1982; Takayanagi and Wong 1985). Several other investigators have reported on the toxicity or bioaccumulation of selenite, selenate and selenomethionine (Shrift 1954a, 1954b; Kumar and Prakash 1971; Moede *et al.* 1980; Wheeler *et al.* 1982; Nimi and LaHam 1976) to various aquatic organisms. However, to our knowledge, there has yet to be a study published comparing the bioaccumulation and toxicity of all three selenium species employing aquatic microorganisms.

With this in mind, we conducted an investigation into the bioconcentration and toxicity of selenate, selenite and seleno-L-methionine in the cyanobacterium *Anabaena flos-aquae*, a filamentous nitrogen-fixing species common to eutrophic systems throughout the United States.

Materials and Methods

An axenic culture of *Anabaena flos-aquae*, (Lyng) Bréb., was obtained from the University of Texas culture collection (UTEX # 1444) (Starr 1987).

The composition of the media, preparation and environmental conditions of the algal cultures are previously described (Kiffney 1989).

Toxicity and bioconcentration of selenite, selenate and seleno-L-methionine were determined in a ten-day experimental period. This test period was selected because it allowed us to monitor the effect of selenium during the three algal growth phases (lag, exponential and stationary). Range-finding experiments were conducted to determine selenium levels which would provide a range of toxic effects (from a NOEL to lethality). For selenite and selenate, the concentrations employed were 1.0, 3.0, 5.0, and 10.0 mg/L while for seleno-L-methionine the treatments consisted of 0.05, 0.1, and 0.3 mg/L. Experimental procedure provided for three replicates of each treatment per sampling period. Each ten-day experiment was conducted once.

All treatments were inoculated with 1×10^5 cells/ml obtained from a stock culture in the log growth phase into a 3 L clear, glass vessel containing 2 L water and modified Woods Hole media. Media water used for algal cultures was double deionized (Barnstead deionizing columns) and sterilized (Ultraviolet Technology ultraviolet column). The cultures were then inoculated with selenium.

Reagent grade selenium (Na_2SeO_4, Na_2SeO_3 and seleno-L-methionine) were supplied by the Sigma Chemical Co. Selenium stock solutions were prepared by dissolving selenium in sterilized, double deionized water.

Samples were collected at 0 hr to determine baseline levels for chlorophyll *a*, dry weight/ml and selenium concentration both in the organism and in the growth medium. Similar samples were obtained at 48, 96, 144, and 240 hr for the selenate and selenite assays. Samples were obtained at 48, 144, and 240 hr for the selenomethionine evaluations, but not at 96 hr because of the expense of seleno-L-methionine. Each sampling period was designed to correspond to a particular growth phase. A 96-hr experiment was performed on each form of selenium to determine the repeatability of the algal growth and bioaccumulation data.

Chlorophyll *a* was measured to monitor alterations in growth. The chlorophyll *a* concentration was determined by the acidification method of Strickland and Parsons (1972) and a model III Turner fluorometer.

The bioaccumulation of selenium by *A. flos-aquae* was determined by filtering a known volume of algal culture through a 0.45 μm Millipore filter. A subsample of the filtrate was collected from each treatment to determine the selenium concentration remaining in the culture media. The algal tissue sample was then rinsed with media water. In preliminary experiments, it was determined that small amounts of selenite absorbed onto the Millipore filter after rinsing. This value was substracted before calculation of the final bioaccumulation values.

Analytical methods for the preparation and analysis of selenium samples are previously described (Cutter 1978; Kiffney 1989).

All data were log transformed to achieve homogeneity of variances using Bartlett's Chi-Squared Test ($p < 0.05$). ANOVA was used to determine effects of among treatments on chlorophyll *a* synthesis and bioconcentrated selenium. Tukey's HSD test was utilized to make multiple comparisons among means at the 95% significance level. Data was back-transformed to report means and standard deviations for parameters measured.

Results

Selenate: Ambient selenium levels for the ten-day period were (Mean ± SD) 0.995 ± 0.08, 2.99 ± 0.23, 4.93 ± 0.48, and 9.72 ± 0.24 mg/L (n = 3).

There were no significant differences in chlorophyll *a* between the control (no added selenate) and the 1.0 mg/L selenate treatment (Table 1). The 5.0 and 10.0 mg/L treatments significantly impaired chlorophyll *a* formation compared to the control, the 1.0, and the 3.0 mg/L treatments beginning on day 2. There were no significant differences in the 5.0 and 10.0 mg/L treatments compared to the initial chlorophyll value, indicating a complete inhibition of growth. Beginning on day 4, there were significant differences in chlorophyll *a* synthesis at the 3.0 mg/L selenate treatment compared to the control and 1.0 mg/L treatments.

The 1.0, 3.0, 5.0, and 10.0 mg/l selenate treated cultures bioaccumulated significantly more selenium and showed a higher BCF (bioconcentration factor ([Se] in the tissue/[Se] in the media)) compared to the control treatment (Table 2 and Figure 1a). The 3.0, 5.0, and 10.0 mg/L selenate treatments bioconcentrated more selenium than the 1.0 mg/L treatment. The 1.0 mg/L treatment reached a maximum selenium concentration on day 4, which was followed by significant decreases in accumulated selenium on day 6 and day 10. The 1.0, 3.0, and 5.0 mg/L treatments exhibited higher BCF than the 10.0 mg/L treatment. The highest BCF (251.6) was observed at the 1.0 mg/L treatment.

Some uptake of selenate and selenite by *A. flos-aquae* was observed in the control treatments and was attributed to a combination of glassware contamination and trace levels of selenium in the experimental water and in the media.

Selenite: The concentrations of selenium in the media for the selenite treatments over the ten-day period were: 1.03 ± 0.08, 2.92 ± 0.12, 4.99 ± 0.26, and 9.87 ± 0.505 mg/L (n = 3).

The control and the 1.0 mg/L treatments were not significantly different in their synthesis of chlorophyll *a* (Table 3). The first significant decreases in chlorophyll *a* occurred on day 2 at the 5.0 and the 10.0 mg/L treatments. The 3.0 mg/L selenite treatment exhibited a significant decrease in chlorophyll *a* beginning on day 4.

The 1.0, 3.0, 5.0, and 10.0 mg/L treatments bioconcentrated significantly more selenium and showed a higher BCF compared to the control (Table 4 and Figure 1b). The 1.0 mg/L treatment attained a maximum selenium level on day 2, which was followed by significant decreases in bioaccumulated selenium. The 3.0 mg/L treatment also reached a maximum bioaccumulated selenium level on day 2, but significant reductions in selenium did not occur until day 6. The 5.0 and 10.0 mg/L treatments attained a maximum bioaccumulated selenium level on day 2 and maintained this level throughout the experiment. The 10.0 mg/L selenite treatment bioconcentrated significantly more selenium than the 5.0 mg/L treatment on days 2, 4, and 6.

Seleno-L-methionine: The concentration of seleno-L-methionine in the culture media decreased over the ten-day experimental period and an odor characteristic of methylated selenium was detected in experimental cultures (our observations). The recovery of selenomethionine (*e.g.*, total selenium) in the growth medium at 48 hr was 0.051 ± 0.026, 0.105 ± 0.07, and 0.280 ± 0.0108 mg/L (n = 3) for the 0.05,

Table 1. The effect of selenate on chlorophyll *a* (μg/L) over a ten-day period. Values are mean ± S.D (n = 3)

Selenium treatment	Day 0	Day 2	Day 4	Day 6	Day 10
Control	0.3 ± 0.01 G[a]; H	0.85 ± 0.4 E; F	8.1 ± 53.5 B	269.9 ± 53.5 A	613.0 ± 158.4 A
1.0 mg/L	0.3 ± 0.01 G; H	0.7 ± 0.1 F	3.78 ± 1.9 B; C	283.8 ± 49.3 A	604.0 ± 250.7 A
3.0 mg/L	0.3 ± 0.01 G; H	0.3 ± 0.3 F; G; H	0.8 ± 0.2 E	1.3 ± 0.2 D; E	2.5 ± 1.9 C
5.0 mg/L	0.3 ± 0.01 H	0.2 ± 0.03 H	0.3 ± 0.04 H	0.1 ± 0.2 H	0.2 ± 0.0 H
10.0 mg/L	0.3 ± 0.01 H	0.2 ± 0.03 H	0.1 ± 0.0 H	0.2 ± 0.0 H	0.2 ± 0.0 H

[a] Values with the same upper case letters are not significantly different at the 95% confidence interval

Table 2. Algal tissue selenium levels (μg/g) exposed to selenate over a ten-day period. Values are mean ± S.D (n = 3)

Selenium treatment	Day 0	Day 2	Day 4	Day 6	Day 10
Control	6.7 ± 0.9 H[a]	5.5 ± 0.7 H	1.3 ± 0.9 H	1.4 ± 0.9 H	3.5 ± 1.7 H
1.0 mg/L	6.7 ± 0.9 H	164.6 ± 43.5 F	251.6 ± 45.9 E; F	42.6 ± 4.2 G	30.44 ± 7.7 G
3.0 mg/L	6.7 ± 0.9 H	411.0 ± 189.5 D; E	393.7 ± 13.7 D; E	728.8 ± 33.7 B; C; D	743.5 ± 128.2 B; C; D
5.0 mg/L	6.7 ± 0.9 H	751.0 ± 127.1 A; B; C; D	608.7 ± 173.8 C; D	924.9 ± 77.2 A; B; C	1217.7 ± 245.0 A; B
10.0 mg/L	6.7 ± 0.9 H	1337.5 ± 644.7 A; B; C	715.0 ± 73.8 B; C; D	1515.5 ± 114.3 A	1312.7 ± 245.0 A; B

[a] Values with the same upper case letters are not significantly different at the 95% confidence interval

0.1 and 0.3 mg/L treatments. By 240 hr, these values had declined to 0.01 ± 0.06, 0.049 ± 0.016, and 0.240 ± 0.016 mg/L, respectively.

A comparison between the control and the 0.05 mg/L treatments did not reveal any significant differences in chlorophyll *a* concentration at any time during the experiment (Table 5). The first significant decrease in chlorophyll *a* concentration occurred on day 2 at the 0.1 and 0.3 mg/L treatment levels. The 0.3 mg/L treatments demonstrated a significant decrease in chlorophyll *a* synthesis compared to the 0.1 mg/L treatment on days 6 and 10.

Selenium levels for the control treatments were below the detection limits of our analytical methods (Detection limit ~1 ng/L). The 0.05, 1.0, and the 0.3 mg/L treatments accumulated significant amounts of selenium at each sampling interval (Table 6 and Figure 1c). The 0.05 mg/L treatment reached a maximum selenium level on day 2, which was followed by significant reductions in selenium on day 6 and day 10. The highest CBF value of 19566 was recorded at the 0.1 mg/L treatment.

Discussion

This study demonstrated *A. flos-aquae* is able to bioconcentrate selenium at ambient concentrations much higher than the national average (1–14 ng/L; Birkner 1978) without inhibiting growth. Our results indicate seleno-L-methionine is more toxic than selenate and selenite, while the inorganic forms are equally toxic. Seleno-L-methionine is assimilated to a greater degree than selenate or selenite, while selenite is bioconcentrated more than selenate. Decreases in bioaccumulated selenium during the experimental period, when exposed to sub-lethal concentrations, suggests *A. flos-aquae* is able to regulate selenium. The regulation of accumulated selenium by *A. flos-aquae* could be a possible mechanism of detoxification.

In comparing the selenium toxicity and bioaccumulation data on microorganisms, it is essential to understand that there are important differences in algal culture techniques and these differences make direct comparisons difficult. Still the levels of selenium that did not affect the growth of *A. flos-aquae* determined in this experiment appear to fall within the range of selenium levels from previous studies. The growth of *A. flos-aquae* in this experiment was not affected by the addition of 0.05 mg/L seleno-L-methionine and 1.0 mg/L for both selenate and selenite. Foe and Knight (1986), utilizing culture methods similar to ours, found chlorophyll *a*/cell was unaffected by the addition of selenite concentrations of 0.05 mg/L in the green alga *Selenastrum capricornutum*. The addition of 1.25 mg/L selenate did not affect the growth rate or the final population density in the green alga *Chorella vullgaris* (Shrift 1954a). The addition of 0.31 to 1.25 mg/L selenomethionine increased the lag time, but did not affect the final population densities when compared to the controls in the green alga *C. vulgaris* (Shrift 1954b).

That the growth of *A. flos-aquae* is not inhibited at these levels is not surprising considering the nutritional value of selenium in other organisms. Naturally occurring selenoproteins have been identified in microorganisms (Stadtman 1980) and demonstrate that selenium is essential for certain enzymes (Stadtman 1979). Other investigators have observed stimulatory effects due to the addition of selenium to culture media. The growth of the cyanobacterium *Microcoleus vaginatus* was stimulated by the addition of 1.0 mg/L selenate (Vocke *et al.* 1980). Growth was improved by addition of 0.005 mg/L selenite and 0.05 mg/l selenocystine and selenomethionine in the dinoflagellate *Perridinium cinctum*. Improved growth was also shown in the diatom *Stephanodiscus hantzschii* var *pusillus* by the addition of 0.11 mg/L selenite and 0.55 mg/L selenocystine and selenomethionine (Lindström 1983). The addition of selenium is either essential for, or markedly improves the growth of six classes of algae (Price *et al.* 1987).

However, at elevated levels, selenium does become toxic. Our results indicate seleno-L-methionine is approximately 30 times more toxic to *A. flos-aquae* than selenate and selenite. It has been reported that selenomethionine is approximately 10 times more toxic than inorganic selenium to algae and fish (Kumar and Prakash 1971; Nimi and LaHam 1976).

A. cylindrica (a cyanobacterium) exhibited a greater tolerance to selenate and selenite levels of 40 and 80 mg/L than *Scenedesmus dimorphus* (a green alga) (Moede *et al.* 1980). The growth of the cyanobacteria, *Phormidium luridum* var *olivacea* and *M. vaginatus*, were inhibited by the addition of

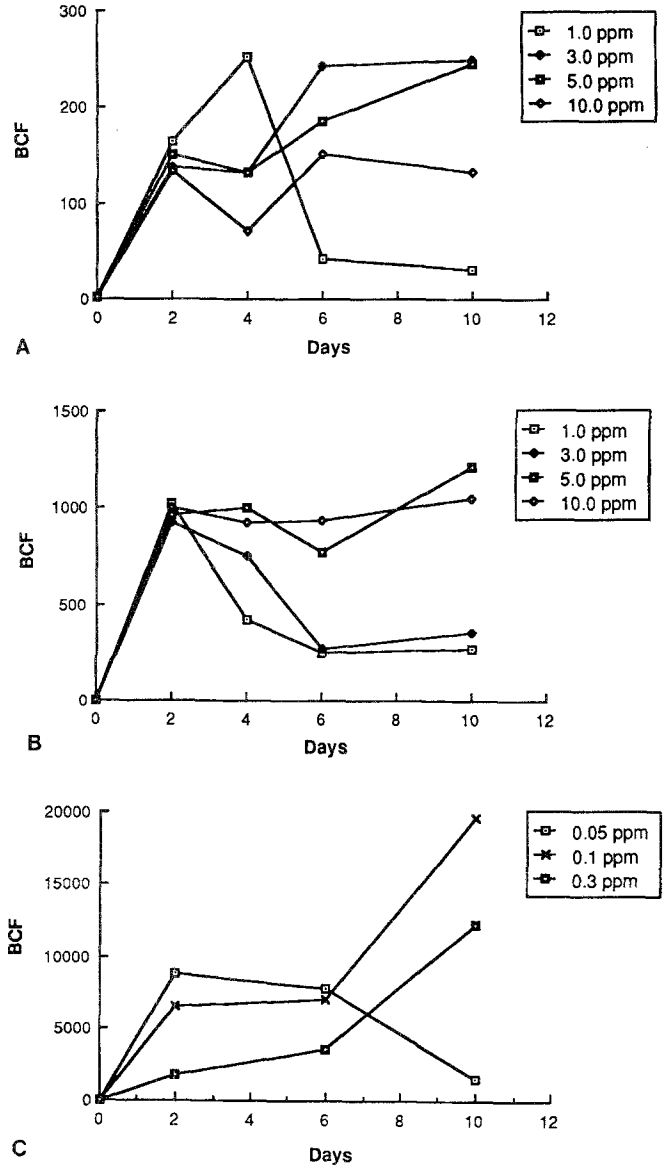

Fig. 1 (a), (b), and (c): The bioconcentration factor ([Se] in the tissue/[Se] in the media) of selenate, selenite and seleno-l-methionine in *Anabaena flos-aquae,* respectively

0.8 mg/L selenite and 10 mg/L selenate, respectively (Sielicki and Burnham 1973; Vocke *et al.* 1980). The first significant inhibition of growth by selenate in the green algae *Ankistrodesmus falcatus, Scenedsmus obliquus* and *S. capricornutum* occurred at 0.01, 0.1 and 0.3 mg/L, respectively (Vocke *et al.* 1980). Foe and Knight (1986) determined that the first sublethal effect of selenite on the green alga *S. capricornutum* occurred at 0.075 mg/L. Shrift (1954a, 1954b) observed impaired growth in the green alga, *C. vulgaris,* at 10.0 mg/L selenate and 0.31 mg/L selenomethionine, respectively.

The toxicity of selenium compounds in plants has been attributed to the disruption of biochemical events within the cell by selenocysteine and selenomethionine (Brown and Shrift 1982b). The formation of dysfunctional enzymes is probably the most strongly implicated cause of selenium toxicity. A significant loss in enzyme function would result from the synthesis of polypeptides in which some or all of the sulfur amino acids had been replaced by their selenium analogues. The physical differences between the sulfur and selenium molecules are thought to be fundamentally responsible for the toxic effects of organic selenium (Brown and Shrift 1982b).

Selenium bioaccumulation by *A. flos-aquae* was much greater for seleno-L-methionine than for selenite and selenate, and greater for selenite than for selenate. The highest BCF was observed for seleno-L-methionine with values ranging from 1520 to 12,193. The bioconcentration factors for selenite ranged from 267 to 1004, while for selenate the BCF ranged from 30 to 115. Similar results were observed in the dinoflagellate *P. cinctum,* which assimilated selenite and organic selenium more readily than selenate (Lindström 1983). The preferential uptake of selenite and selenomethionine was explained by the additional energy consuming metabolic steps necessary for the alga to reduce and incorporate selenate into sulfur amino acids.

The apparent antagonistic relationship between selenate and sulfate provides another possible mechanism for the lack of selenate assimilation. Shrift (1954a) showed that there was competitive antagonism in the uptake of selenate/sulfate in the green alga *C. vulgaris.* There was a decrease in the uptake of selenium as selenate by *C. vulgaris* when the external concentration of sulfate was increased. It is possible the sulfate concentrations used in our experiments were at levels sufficient enough to inhibit the bioconcentration of selenate compared to selenite and seleno-L-methionine, which have yet to show a similar antagonistic relationship with sulfate.

Our results show a inverse correlation between the bioconcentration of selenium and the growth of the *A. flos-aquae,* a phenomenon demonstrated in the green algae *C. vulgaris* (Shrift 1954a) and *S. capricornutum* (Foe and Knight 1986). *A. flos-aquae* appears to utilize and regulate selenium at sublethal levels, but at toxic levels regulation breaks down and selenium rapidly accumulates in the cell disrupting normal metabolic activity. NOEL treatments of selenate, selenite and seleno-L-methionine and the 3.0 mg/L selenite treatment produced significant decreases in intracellular selenium as the experiment progressed. The decrease in intracellular selenium appears to contribute to the amelioration of the toxicity of selenium as there were no significant growth effects at the NOEL treatments while the 3.0 mg/L selenite treatment exhibited significant growth. Foe and Knight (1986) reported that *S. capricornutum* regulated its intracellular selenium concentration when the external media contained less than 100 ppb (μg/kg) selenium and this regulation process appears to be a detoxification mechanism for this alga.

The mechanism by which algae regulate selenium to ameliorate toxicity is not known. Selenium accumulator plants, which are able to tolerate high levels of intracellular selenium, are though to form selenorganic compounds that do not disrupt normal metabolic functions (Brown and Shrift 1982a). The protein fraction from *Astragalus,* a selenium tolerant plant, contained less selenium than protein fractions from three nontolerant plant species (Brown and Shrift 1981). It was hypothesized *Astragalus* is able to exclude se-

Table 3. The effect of selenite on chlorophyll *a* (μg/L) over a ten-day period. Values are mean ± S.D (n = 3)

Selenium treatment	Day 0	Day 2	Day 4	Day 6	Day 10
Control	1.75 ± 0.05 G[a]; H; I	8.95 ± 0.7 E	47.1 ± 3.6 C	291.6 ± 68.3 B	606.9 ± 28.4 A
1.0 mg/L	1.75 ± 0.05 G; H; I	6.82 ± 1.6 E	45.6 ± 15.2 C	191.8 ± 73.2 B	538.6 ± 184.9 A
3.0 mg/L	1.75 ± 0.05 G; H; I	6.8 ± 0.7 E	19.8 ± 2.4 D	52.7 ± 36.3 C; D	195.3 ± 153.2 B
5.0 mg/L	1.75 ± 0.05 G; H; I	3.4 ± 0.76 F; G	5.4 ± 0.9 E; F	2.9 ± 0.9 F; G; H	3.3 ± 2.1 F; G; H
10.0 mg/L	1.75 ± 0.05 G; H; I	2.13 ± 0.03 G; H; I	2.44 ± 0.9 G; H; I	1.73 ± 0.3 H; I	1.4 ± 0.2 I

[a] Values with the same upper case letters are not significantly different at the 95% confidence interval

Table 4. Algal tissue selenium levels (μg/g) exposed to selenite over a ten-day period. Values are mean ± S.D (n = 3)

Selenium treatment	Day 0	Day 2	Day 4	Day 6	Day 10
Control	6.3 ± 0.0 H[a]	6.2 ± 0.2 H	5.3 ± 1.4 I	2.0 ± 1.0 J	0.8 ± 0.4 J
1.0 mg/L	6.33 ± 0.0 H	1024.2 ± 31.4 E	418.9 ± 123.5 F	267.7 ± 30.6 G	270.9 ± 95.0 G
3.0 mg/L	6.33 ± 0.0 H	2784.9 ± 367.2 C; D	2257.7 ± 148.5 D	792.9 ± 13.4 E	1071.0 ± 239.7 E
5.0 mg/L	6.33 ± 0.0 H	4815.6 ± 448.6 B; C	5035.0 ± 285 B	3879 ± 292.8 B; C	6072 ± 377.9 A; B
10.0 mg/L	6.33 ± 0.0 H	10,041.8 ± 775.7 A	9262.2 ± 503.0 A	9332.7 ± 828.7 A	9365.2 ± 1590.4 A

[a] Values with the same upper case letters are not significantly different at the 95% confidence interval

Table 5. The effect of seleno-I-methionine on chlorophyll *a* (μg/L) over a ten-day period. Values are mean ± S.D (n = 3)

Selenium treatment	Day 0	Day 2	Day 6	Day 10
Control	0.83 ± 0.02 E[a]; F	3.0 ± 0.2 D	339.2 ± 6.7 B	491.8 ± 67.0 A
0.05 mg/L	0.83 ± 0.02 E; F	2.6 ± 0.4 D	326.9 ± 12.0 B	563.0 ± 28.13 A
0.1 mg/L	0.83 ± 0.02 E; F	1.0 ± 0.02 E	10.9 ± 1.2 C	8.3 ± 0.5 C
0.3 mg/L	0.83 ± 0.02 E; F	0.8 ± 0.07 E; F	0.6 ± 0.06 G	0.6 ± 0.08 F; G

[a] Values with the same upper case letters are not significantly different at the 95% confidence interval

lenium from polypeptides, which restricts the synthesis of dysfunctional selenium substituted proteins.

The volatilization of methylated selenium compounds has also been suggested as another mechanism of regulation for the detoxification of selenium by other life forms (National Academy of Science 1976). We detected a distinct odor from our seleno-L-methionine treatments and a subsequent loss of selenium from the medium. It is possible this loss of selenium is due to the bioconcentration and subsequent methylation of selenium by *A. flos-aquae*. Lindström (1983) reported a similar unpleasant odor from algal cultures grown in organic selenium, which was attributed to either volatilization of methylated Se, or to sulfur compounds. Niimi and LaHam (1976) reported a 15–50% loss of selenium from a solution of selenomethionine, which was accompanied by a distinct odor and a opaque film on the surface of the aquaria in their experiments; they attributed the loss of selenium to microbial methylation.

In our study, *A. flos-aquae* was able to maintain metabolic functions at sublethal levels of inorganic and organic se-

Table 6. Algal tissue selenium levels (μg/g) exposed to seleno-I-methionine over a ten-day period. Values are mean ± S.D (n = 3)

Selenium treatment	Day 0	Day 2	Day 6	Day 10
Control	BD[a]	BD	BD	BD
	H[b]	H	H	H
0.05 mg/L	BD	442.8 ± 39.5	390.8 ± 83.1	76.8 ± 12.0
	H	E; F	F	G
0.1 mg/L	BD	645.0 ± 124.9	766.9 ± 67.7	1956.6 ± 669.5
	H	D; E	C; D	B
0.3 mg/L	BD	545.0 ± 20.0	1084.26 ± 202.1	3658.8 ± 681
	H	D; E; F	C	A

[a] Below detection limits (1.00 ng/L)

[b] Values with the same upper case letters are not significantly different at the 95% confidence interval

lenium despite bioconcentrating high levels of selenium from the medium. We attribute selenium toxicity to the level of intracellular selenium, because an inverse relationship was observed between the synthesis of chlorophyll *a* and the level of intracellular selenium. We propose, when exposed to NOEL concentrations of selenium, *A. flos-aquae* replaces some of the sulfur atoms with selenium atoms employing the sulfur metabolic pathways in the synthesis of proteins without affecting the metabolic functions necessary for growth. However, above the NOEL, selenium saturates the sulfur metabolic pathways, forming deleterious amounts of selenorganic compounds.

It is believed the poisoning of the upper trophic levels in selenium contaminated systems is due to the bioconcentration of selenium by primary producers and the subsequent biomagnification of the toxic organic forms of selenium up the aquatic food chain. Therefore, this cyanobacterium is a potentially dangerous food source in the aquatic food chain in selenium contaminated systems.

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