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Acute Metabolic Responses of Lotic Epilithic Communities to Total Residual Chlorine

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Numerous workers have examined the effects of chlorine on individual epilithic community components (e.g., CAIRNS & PLAFKIN 1975; BROOKS & LIPTAK 1979), but little attention has been given to the effects of residual chlorine on the whole lotic epilithic community. Epilithic organisms are those microfloral and fauna assemblages living on the surface of rocks; the primary constituents include bacteria, algae, fungi, and protozoans. Epilithic communities are an important functional component of aquatic ecosystems. Thus, deterioration of the community due to the introduction of a stress agent could drastically impair the functional capabilities of the entire system. As the respiration rates of biological systems are reflective of energy flow through the system, and primary productivity is a measure of carbon fixation capabilities, the purpose of this investigation was to determine the acute metabolic responses of lotic epilithic communities to various concentrations of total residual chlorine (TRC).

STUDY AREA AND METHODS

The study was conducted in an undisturbed reach of the Sheep River, Alberta, a fifth-order Rocky Mountain foothills stream. A detailed description of the area and watershed characteristics are presented in MOORE et al. (1980) and OSBORNE et al. (1981).

The acute metabolic responses of the epilithic communities inhabiting natural substrates (less than 31 mm diameter) to total residual chlorine were individually determined using 10 benthic in situ respirometers (OSBORNE & DAVIES 1981) from August 19 through 21, 1980. Previous studies on the same 10 communities indicated that the metabolic rates had been at a constant level for approximately three weeks prior to the beginning of this study (OSBORNE 1981). Thus, it was assumed that any changes in the metabolic rates during this study would be directly attributable to the effects of TRC exposure.

On August 19, background community respiration (CR) and net primary production (NP) rates were simultaneously determined in each of five respirometry units using pH combination electrodes attached to a 5-channel junction box (Fisher Scientific) connected to an Accumet pH meter. Following the attachment of either a dark

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or transparent respirometry top (OSBORNE & DAVIES 1981), the pH in each chamber was determined every 5 min for one hour. Temperature and total alkalinity (A.P.H.A. 1971) of the water within each respirometer were also determined before and after each series of pH readings. Following the one hour period the respirometry tops were removed until the next day of readings. The calculations of CO_2 for each 5-min period were made according to VOLLENWEIDER (1969). Hourly rates of CR and NP were determined using linear regression analysis (SOKAL & ROHLF 1969) with times as the independent variable and CO_2 concentration for each 5-min period the dependent variable.

On August 20, the respirometry tops were again attached and freshly field-prepared hypochlorous acid solutions were injected into each respirometer to attain a total chlorine residual concentration within each chamber of either 0.1, 0.5, 0.7, 1.0, or 2.0 mg/L. One hour later the respirometry tops were removed and the system allowed to fill with river water. Community respiration was again determined in each unit as previously described. On August 21, CR and NP rates were determined and used as the 24-h post-exposure treatments.

The rates of CR and NP for each TRC concentration and day were statistically compared on the basis of the slopes of the respiration or production lines according to SNEDECOR & COCHRAN (1977). P/R (production-respiration) ratios were calculated as GP/CR for each one hour period; where GP (gross primary productivity) = NP + CR.

RESULTS AND DISCUSSION

Significant differences occurred in the calculated CR and NP rates of epilithic communities following a one hour exposure to TRC (Table 1). The CR rates of epilithic communities exposed to TRC concentrations of 0.5 mg/L and greater significantly decreased, while those exposed to 0.1 mg/L TRC significantly increased when compared to the control period (ie. August 19) (Figure 1).

Increases in the respiration rates of aquatic organisms following exposure to sublethal concentration of a toxicant have generally been recognized as a metabolic response to stress (MAKI & JOHNSON 1976; OSBORNE et al. 1980). Thus, the significant increase in CR in the 0.1 mg/L exposed samples may be interpreted as a metabolic stress response to sublethal concentrations of TRC. The chronic ramifications of a prolonged respiratory increase would likely be a decrease in the functional capabilities of the community as more energy is required for simple system maintenance.

The significantly higher CR rates on August 21 in the 0.1 mg/L treatment (Table 1) relative to the August 20 rates indicate that the communities were responding to the initial TRC exposure 24 h later. The significantly lower August 21 NP rates further

Table 1. Statistical comparisons of calculated total respiration rates with respect to TRC concentrations for control with chlorination rates, control with 24-h post-exposure rate, and chlorination with 24-h post-exposure rates. The net primary productivity results were exactly the same. ** = significant at 0.01; *** = significant at 0.001; NS = not significant at 0.05. The plus and minus values indicate significantly higher or lower values relative to the controls.

TRC Conc.	Control with chlorination	Control with 24-h post	Chlorination with 24-h post
0.1	*** (+)	*** (+)	** (+)
0.5	*** (-)	*** (-)	*** (-)
0.7	*** (-)	*** (-)	** (-)
1.0	*** (-)	*** (-)	NS
2.0	*** (-)	*** (-)	NS

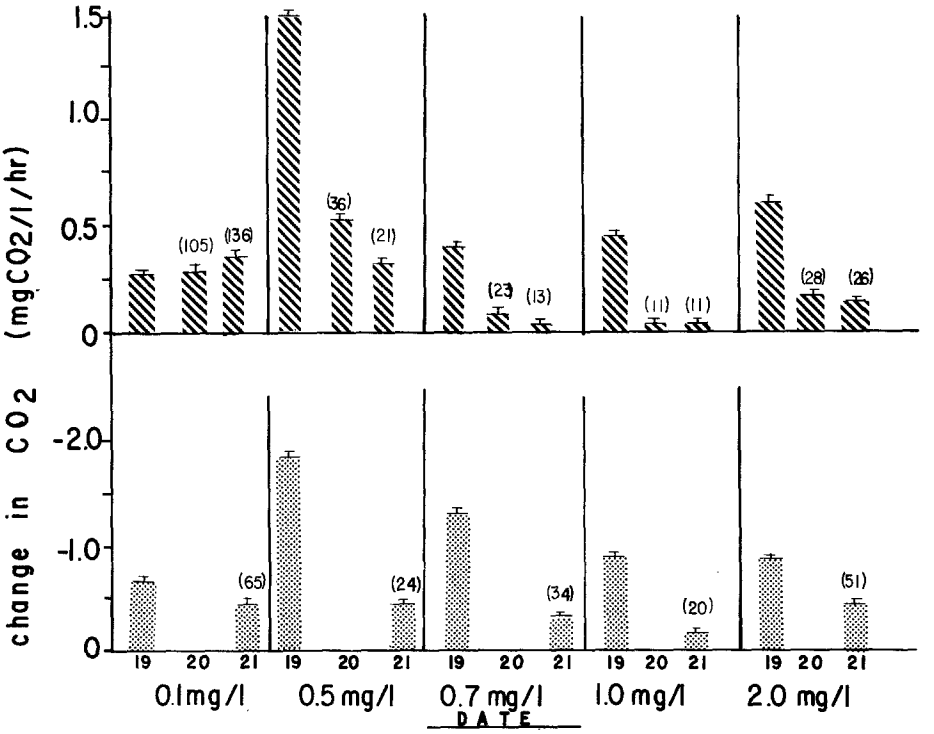

Figure 1. Calculated total community respiration (slash bars) and net primary production (dotted bars) rates ($mgCO_2/L/h$) for each TRC treatment and standard errors for the period August 19-21, 1980. The percent relative to the control period are noted in ().

indicates that photosynthesis was inhibited (Table 1; Figure 1). These data suggest that a substantial proportion of the epilithic organisms survived the initial 1-h exposure, but that the functional capabilities of the communities were significantly reduced. The significant reductions in the P/R values further support these conclusions (Table 2).

Exposure to TRC concentrations greater than or equal to 0.5 mg/L decrease CR rates by 65-90% within the first hour of exposure (Figure 1). Communities exposed to 0.5 and 0.7 mg/L TRC also had significantly lower respiration rates 24 h later. No differences were found between the August 20 and 21 CR rates of the communities treated with 1.0 and 2.0 mg/L TRC (Table 1).

The significantly lower respiration rates of samples following a one hour exposure are indicative of the immediate (acute) toxic effects of TRC to epilithic communities and corresponds with previous bacterial (VENKOBACHAR et al. 1977), protozoan (CAIRNS & PLAFKIN 1975), and marine diatom (HIRAYAMO & HIRANO 1970) toxicity results. These data indicate that a one hour exposure to 0.5-2.0 mg/L TRC significantly reduces the metabolic rates of epilithic communities following a one hour exposure. The significantly lower 24 h post-exposure CR rates in the 0.5 and 0.7 mg/L treatments suggests a continued toxicity and deterioration of the functional capabilities of the epilithic communities (Figure 1; Table 1), while the similarities in the 1.0 and 2.0 mg/L of the damage occurred within the first hour. The differences in the 24 h post-exposure rates relative to the initial TRC exposure rates (ie. August 20) appear directly related to the concentration of TRC employed as the maximum adverse effects at 0.5 and 0.7 mg/L were delayed at 24 h.

Table 2 Calculated P/R ratios for August 19 (control) and 21 and the percent of P/R (August 21) to the control period for each TRC concentration.

TRC Con.	Aug 19	Aug 21	(Aug 19/Aug 21)X100
0.1	2.47	1.23	49.8
0.5	1.25	1.35	108.5
0.7	3.28	6.17	188.5
1.0	1.67	3.07	184.1
2.0	1.43	2.80	196.1

Although TRC significantly reduced the metabolic rates of epilithic communities over a 24-h period, not all of the individuals within the samples succumbed to the toxicant as indicated by the 24 h CR and NP post-exposure rates. This suggests a variability in the metabolic sensitivity of organisms within the

communities which coincides with earlier chlorine toxicity studies (VIDEAU et al. 1979; HIRAYAMO & HIRANO 1970). A significant reduction in NP ranging from 35-75% occurred at all TRC concentrations within 24 h of exposure. These results concur with previous lentic and marine phytoplankton studies which indicated that TRC inhibits photosynthesis (BROOKS & LIPTAK 1972; CARPENTER et al. 1972). Comparisons of the calculated control and 24 h post-exposure P/R values. BROOKS & LIPTAK (1979) have examined the effects of sublethal TRC concentrations (ie. less than 0.1 mg/L) on lentic phytoplankton and reported decreases in chlorophyll a concentrations and carbon uptake rates which returned to control levels within 24 h of exposure. The 108-196% increase in the P/R values (Table 2) in the 0.5-2.0 mg/L TRC treatment suggests that a substantial proportion of the surviving autotrophs were functionally active during the 24-h post-exposure period, although NP was significantly lower as previously discussed. These data further suggest that a greater proportion of heterotrophs were made metabolically inactive by TRC exposure then were autotrophs. It is likely then that the 24 h post-exposure CR values in the 0.5-2.0 mg/L treatment were primarily a function of autotrophic respiration. Thus, the results of this study indicate that TRC concentrations greater than 0.5 mg/L inhibit a greater proportion of heterotrophs relative to autotrophs, while the lower TRC concentration (ie. 0.1 mg/L) permitted the survival of a majority of heterotrophs, but inhibited the functional capabilities of the autotrophs to approximately the same degree as did higher TRC concentrations.

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