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EFFECTS OF ENDOTHALL TREATMENT ON PHOSPHORUS CONCENTRATION AND COMMUNITY METABOLISM OF AQUATIC COMMUNITIES

by

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Herbicide treatment and macrophyte senescence may result in the recycling of phosphorus from aquatic weeds to nontarget species because the nutrient is rapidly leached from plant tissue. A field study and two microcosm experiments were conducted to examine changes in phosphorus, oxygen, and chlorophyll a concentrations after the application of the herbicide endothall. In each microcosm experiment, *Potamogeton crispus* communities

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ABSTRACT (Continued).

were housed in 57-ℓ aquaria containing known standing crops of the test plant and sediment with a specific phosphorus-adsorbing capacity.

In the first microcosm experiment (experiment I), the application of 2.0 ppm endothall caused plant death and a rapid, short-term increase in soluble reactive phosphorus, presumably from excessive leaching during senescence. Although the sediment-adsorbing capacity for KH_2PO_4 was high and the microzone appeared to be oxidized throughout the experiment, leached soluble reactive phosphorus was still directly available for algal uptake. This resulted in an epipelic chlorophyll a increase after herbicide application.

A second microcosm experiment, which employed six control and six experimentally treated systems, differed from microcosm experiment I in that the sediment used had a weak phosphorus-adsorbing capacity. In addition, the metabolism of the total microcosm and three autotrophic components (macrophyte-epiphyte, planktonic, and benthic) were monitored to assess the effects of an herbicide perturbation on nontarget assemblages. Unlike experiment I, no soluble reactive phosphorus pulse was observed during plant senescence. However, benthic gross productivity and chlorophyll a again increased, suggesting a rapid transfer of leached phosphorus and other nutrients to the sediment algae.

The apparent differences in the soluble reactive phosphorus responses between the two microcosm experiments may be attributed to differences in the standing crops of macrophyte phosphorus since the concentration was three times higher in microcosm experiment I. Endothall also caused a significant deflection in microcosm productivity rates but an increase in microcosm respiration relative to control rates. The opposite deflection pattern was attributed to destruction of photosynthetic activities by the macrophytes and an abundance of leached labile organic material which potentially stimulated heterotrophic consumption. Microcosm metabolism recovered from herbicide treatment because photosynthetic dominance shifted from the macrophyte-epiphyte component to the benthic algae. Furthermore, the shift in photosynthetic dominance appeared to be stimulated by the uptake of leached macrophyte phosphorus by the sediment algae which were resistant to the herbicide perturbation. These results indicated that phosphorus was rapidly leached from herbicide killed plants and algae could potentially assimilate this phosphorus for growth.

PREFACE

The study presented in this report was sponsored by the Department of the Army, Office of the Chief of Engineers (OCE), Directorate of Civil Works (DAEN-CW), through the U. S. Army Corps of Engineers (CE) Aquatic Plant Control Research Program (APCRP). Funds for the study were provided by DAEN-CW under Department of Army Appropriation No. 96X3122, Construction General. The APCRP is managed by the U. S. Army Engineer Waterways Experiment Station (WES), Vicksburg, Miss. Technical Monitor for OCE was Mr. Darrell Lewis.

The research was performed and this report was prepared by Mr. William F. James while he was employed by the Environmental Laboratory (EL), WES, under an Intergovernmental Personnel Act agreement with Kent State University. Dr. G. Dennis Cooke was Mr. James' faculty advisor. Portions of this work were performed under the direct supervision of Dr. Robert H. Kennedy, EL, and under the general supervision of Mr. Donald L. Robey, Chief, Ecosystem Research and Simulation Division, EL. Mr. J. Lewis Decell was Manager, APCRP; Dr. John Harrison was Chief, EL.

Commander and Director of the WES during the preparation and publication of this report was COL Tilford C. Creel, CE. Technical Director was Mr. F. R. Brown.

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CONTENTS

EFFECTS OF ENDOTHALL TREATMENT ON PHOSPHORUS CONCENTRATION AND COMMUNITY MATABOLISM OF AQUATIC COMMUNITIES

I. INTRODUCTION

A. Background.

The importance of the littoral flora as a site for phosphorus flux in lakes has received attention, particularly in the management of aquatic weed problems (Brooker and Edwards, 1975; Carpenter and Adams, 1977). Aquatic weeds, often growing rapidly and attaining high densities, interfere with irrigation systems, reservoirs, and the aesthetic and recreational qualities of water (Holm et al., 1969). Macrophytes also contain a considerable amount of tissue phosphorus which can become a major source of orthophosphate to the water through plant decomposition (Carpenter and Adams, 1978). Orthophosphate can thereafter be assimilated by other autotrophs which may then reach nuisance levels as well. Plans for macrophyte control must consider the impact of a chosen method on the fate of phosphorus in aquatic ecosystems. Although herbicide treatment is presently a feasible method for aquatic plant control, plant decomposition and nutrient availability may alter the physical, chemical, and biological state of a lake.

Phosphorus composes roughly 0.1 to 1 percent of the macrophyte standing crop, the percentage fluctuating seasonally. Although evidence is sparse for submerged macrophytes, the trend appears to be a high phosphorus content per plant standing crop early in the growing season which decreases as a population matures (Carpenter and Adams, 1977). The percentage may increase later in the life cycle with the development of flowers and fruit (Caines, 1965). Herbicide is usually applied when macrophytes reach a nuisance density and the tissue phorphorus pool is large. It follows that phosphorus can be lost from the tissue in excessive amounts.

Laboratory and field work provide insight into the pattern of organic matter decomposition and phosphorus loss in a variety of aquatic macrophyte types (submerged, floating-leaved, emergent). Submerged macrophyte decomposition generally follows an exponential decay pattern (Jewell, 1971; Novak et al., 1975; Howard-Williams and Davies, 1979), but a biphasic weight loss,

or a distinctly rapid loss followed by slow decomposition, has been reported (Mason and Bryant, 1975). In both cases, phosphorus loss is greatest during the early stages of decomposition. Leaching (water-soluble compound removal) and autolytic processes (enzymatic degradation) are dominant during early senescence, followed by microbial breakdown (Harrison and Mann, 1975). For instance, greater than 60 percent of the phosphorus in decomposing *Potamogeton pectinatus* was lost within fifteen days (Howard-Williams and Davies, 1979) and similarly rapid phosphorus loss was reported for *Myriophyllum spicatum* (Carpenter and Adams, 1978). Apparently, much of the phosphorus is in the soluble or readily solubilized form, particularly for submerged species which have a high percentage of the standing crop in the noncell-wall fraction (Polinisi and Boyd, 1972).

The phosphorus lost from aquatic macrophytes is almost entirely in the fine particulate and orthophosphate forms (Jewell, 1971; Nichols and Keeney, 1973; Carpenter and Adams, 1978). Organic phosphorus is not a major breakdown product of plant decay, but rather appears in the water as a result of microbial transformations (Carpenter and Adams, 1978).

The fate of macrophyte phosphorus after herbicide treatment depends on abiotically and biotically mediated processes of an aquatic ecosystem, and subsequent changes caused by plant decomposition. Nichols and Keeney (1973) realized the importance of the sediment and the effect of oxygenation on the fate of tissue phosphorus in the laboratory. Endothall-treated plants allowed to decompose in systems containing only filtered lake water displayed no difference in phosphorus loss under oxygenated and nonoxygenated conditions. A reported 31.6 to 54.4 percent of the tissue phosphorus was lost within the first 28 days under both conditions (Nichols and Keeney, 1973). Total phosphorus concentrations in the water increased markedly within 14 days, remaining constant thereafter. In systems containing oxidized sediment, total phosphorus showed substantially less to no increase in the water following herbicide treatment. The adsorbing capacity of the sediment for phosphorus (Mortimer, 1971) was hypothesized to be the factor responsible for the results Nichols and Keeney (1973) obtained.

Changes in oxygen and phosphorus during plant decomposition in small lakes and aquaria have been followed to determine whether or not the redox state of an aquatic ecosystem influences the movement of recycled phosphorus. Soluble reactive phosphorus (SRP) increases were reported only during oxygen

depletion (Walker, 1963; Simsiman et al., 1972). Alternatively, Fish (1966), Simpson and Pimental (1972), and Walsh et al. (1971) found no SRP increases after herbicide treatment, and the water remained oxygenated.

The commonly reported scarcity of SRP after herbicide treatment may also be the result of its rapid turnover time in the water (Lean, 1973) and uptake by other organisms. Several workers have observed substantial increases in the chlorophyll a concentration after herbicide treatment (Fish, 1966; Newbold, 1975; Brooker, 1976). In all cases the water remained oxygenated with no increases in the SRP levels. None of the studies considered that the rapid leaching process of plant decomposition could cause an SRP pulse of short duration in the water at the onset of senescence. In the studies reported herein, samples were taken at weekly intervals after treatment.

Community productivity may be sustained or increased after herbicide application by the occurrence of algal blooms, although there is a paucity of data to support this contention. Some herbicides are species-specific in that they destroy only intolerent organisms of a community, allowing resistant or opportunistic species to invade and multiply. Walsh et al. (1971) observed that chlorophyll a, phytoplankton abundance, and phytoplankton productivity increased to a maximum during the most intense period of weed decay in a herbicide-treated pond. At that time, phytoplankton contributed 94 percent of the total community productivity in the experimental pond, while, in the control pond, phytoplankton accounted for only 17 percent. Phytoplankton productivity decreased as macrophytes reinvaded, eventually reaching a level similar to that of the control. Walsh et al. suggested that nutrient availability sustained algal productivity in the herbicide-treated pond.

It has been hypothesized (Rhyther, 1970; Menzel et al., 1970) that a perturbation imposed specifically on an autotrophic component of a community will cause a change in (1) the species dominating primary productivity, and (2) the flow of energy through a community. Furthermore, when an autotrophic component is perturbed, both community productivity and respiration should decrease initially because that component will no longer be contributing to either metabolic pathway (Beyers, 1963). To illustrate, Copeland (1965) observed a decrease in both community productivity and respiration in a turtlegrass-dominated microcosm after a light reduction perturbation. The community rates increased to the pretreatment levels as the community went through succession at the lower light levels. The dominant primary producer

after light reduction was the much faster growing blue-green algae rather than the slow-growing turtlegrass. The transition may have resulted from a competitive adaptation by the algae to lower light levels, or nutrient loss from the decomposing grass may have stimulated algae growth.

Herbicide treatment perturbs the community metabolism of an aquatic ecosystem and causes the release of nutrients from macrophytes. While phosphorus can both adsorb to the sediment and be taken up by other autotrophs, the influence of each on the fate of phosphorus has not been determined in herbicide-perturbed systems. Phytoplankton and macroalgae may be functionally important in conserving phosphorus which would otherwise be lost to the sediment or lake outflow. Analogously, the pin cherry (*Prunus pensylvanicas*) invaded and accumulated biomass and nutrients rapidly, reducing nutrient loss after clearcutting and herbicide treatment at the Hubbard Brook experimental station (Marks and Johnson, 1972). Furthermore, phosphorus is usually the element that limits algal growth, and its availability after herbicide treatment may cause aesthetically unpleasing algal blooms. Therefore, the effects of herbicide treatment on phosphorus release from macrophytes and community metabolism merit further study.

B. Purpose and Scope.

In the present study, microcosms were used to observe phosphorus leaching and changes in community metabolic rates after endothall treatment. Microcosms were constructed in 57-ℓ aquaria with lake water, sediment, and an initial known standing crop of the test plant *Potamogeton crispus*, a nuisance aquatic weed occurring in the midwestern United States. Two experiments were conducted, one with microcosms containing sediment which was highly adsorptive to SRP, and the second with sediment which had a weaker adsorbing capacity. In addition, a field study was conducted at a local lake in northeast Ohio. The specific research questions were:

(1) Is there an SRP pulse in the water after endothall treatment?

(2) Does community productivity shift from macrophyte to algal dominance after an herbicide perturbation?

II. MATERIALS AND METHODS

Endothall (7-oxabicyclo [2, 2, 1] heptane-2,3-dicarboxylic acid) is effective for the control of terrestrial and aquatic weeds. The compound is selectively toxic and is used for preemergence and postemergence problems (Anonymous, 1974). Although little is known about the mode of action of endothall, it is generally thought to cause rapid membrane disruption and ion leakage (Morrod, 1976). The chemical products used in this study are Aquathol K, the dipotassium salt of endothall, and Hydrothol 47, the di-(N, N-dimethylalkylamine) derivative. Both products are manufactured by the Pennwalt Corporation.

A. Field Study.

A field study was conducted on a man-made lagoon which becomes choked with *Potamogeton crispus* in the late spring. Used as an access channel to West Twin Lake, the lagoon has a long arm and a short arm, which served as the experimental and control areas, respectively (Figure 1) (for a description of the lake, see Cooke et al., 1978). The mean water depth is approximately 1 m

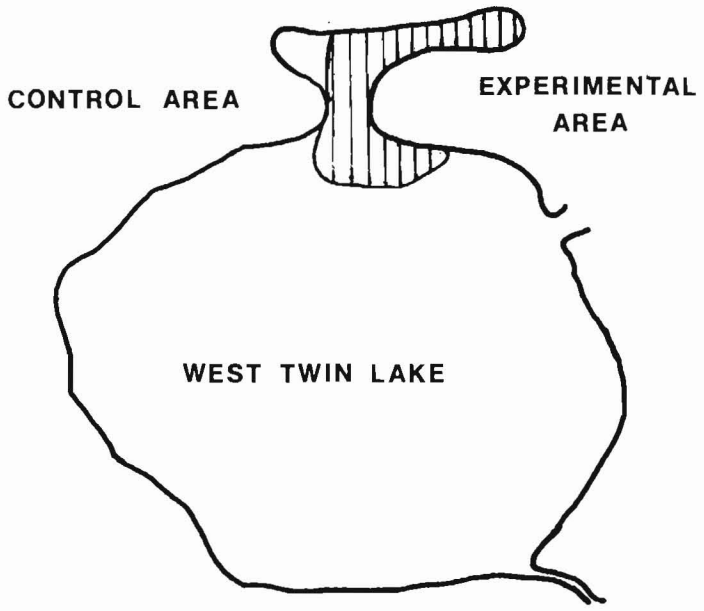


Figure 1. West Twin Lake and lagoon area. Striped area marks the extent of herbicide treatment in experimental site

and 6.3 km^2 in area. The long arm of the channel was treated with herbicide on 3 May 1978, and again on 8 May. Forty-five kilograms of the granular form of endothall (Hydrothol 47, Pennwalt) were applied on the first day and on the second treatment day 19 ℓ of the liquid form (Aquathol K, Pennwalt) was sprayed. The intended concentration on each treatment date was 5 ppm of the active ingredient. Samples were collected in the middle of each area at a 0.5 m depth for dissolved oxygen, total phosphorus, soluble phosphorus, and soluble reactive phosphorus. Three pretreatment observations were made. After herbicide treatment, water was analyzed on the third and fifth days, then daily for 9 days.

B. Microcosm Study.

Two experiments involving microcosms were performed. Experiment I, executed in the fall of 1978, was a pilot study. Two microcosms were used, each containing sediment from the 1 m depth of the lagoon area (Figure 1). Twelve microcosms constructed in the spring of 1979 contained sediment from the 2 m depth of the littoral zone on the southern side of West Twin Lake. For both experiments, aquatic communities were housed in 57-ℓ aquaria (15 gal). The glass tanks were 60 cm long, 30 cm wide, and 30 cm high. Sediment and water were collected from each location and promptly returned to the laboratory. Sediment gathered with an Ekman dredge was sifted through a coarse-meshed wire screen to remove plant material, then thoroughly mixed in a large vat. Ten litres of the sediment was then dispersed evenly across the bottom of each aquarium. Lake water was collected in 18-ℓ carboys, combined, and mixed in a vat, then slowly added to the aquaria. Water lost by evaporation was replaced with tapwater. These procedures were followed for both experiments.

The sediment taken from each area was classified according to its ability to adsorb soluble reactive phosphorus. Adsorption and desorption were surveyed by the Williams et al. (1970) method. A volume in cubic centimetres equivalent to 0.5 g dry weight of each sediment was placed in a 125-ml Erlenmeyer flask. To estimate adsorption, an aliquot of a 0.2 M NaCl solution containing 250 µg KH_2PO_4 per litre was added to each flask, bringing the liquid phase volume up to 25 ml. Twenty-five millilitres of glass-distilled water was then added to make the final liquid volume 50 ml. The flasks were placed on a shaker table for 12 hr, then a portion was removed and centrifuged for

20 min. Twenty-five millilitres of the liquid phase was filtered through glass fiber filters before determining SRP. Desorption was measured by adding 50 ml of a 0.1 M NaCl solution without phosphorus to a sediment aliquot. The flasks were shaken and the same procedure followed to determine SRP. Net adsorption was calculated as adsorption minus desorption. Ten replicates were attempted for each experiment.

Lighting for the microcosms was provided by 1.2-m light banks, each containing two 40-W cool white fluorescent light bulbs. Banks were positioned 127 mm above each aquarium. The bulbs generated 81.0 $\mu E/m^2/sec$ to the surface of each aquarium. Throughout experiment I, lighting was set on a 16-hr-day, 8-hr-night cycle to promote growth. The cycle was changed to a 12-hr-light, 12-hr-dark period for experiment II. Two aquaria were placed under each light bank. In experiment I, one aquarium was designated as the control while the other was the experimental to be treated with herbicide. The 12 microcosms used in experiment II were arranged as shown in Figure 2. Black plastic was draped over the light banks and along the outer sides of each aquarium to prevent ambient light from entering. Cardboard was inserted between aquaria along the inner sides in tables 2 and 3 to prevent light exchange between microcosms. Microcosms were declared control or experimental on the basis of position on the tables.

Potamogeton crispus, a rapidly growing nuisance weed, was chosen as the experimental plant. The plant propagates by runners during the early spring in West Twin Lake, attaining high densities by June. In late June, the population produces asexual turions before senescence. A synchronous die-back then occurs, and the plants decompose and settle to the sediment. Turions were collected from a stand of macrophytes and stored in a refrigerator until needed for experiment I. The turions were germinated in small plastic trays filled with lake water and sediment. A 0.6-m fluorescent light bank producing 81.0 $\mu E/m^2/sec$ was set on an 8-hr-day length which proved successful in initiating germination. Four small macrophytes were then planted in each aquarium for experiment I. Small cuttings collected in the lagoon area were used for experiment II because turions were not available. Runners were trimmed from the plants and nine planted in each aquarium.

A method for determining plant standing crop and tissue phosphorus was attempted to estimate the total amount of macrophyte phosphorus in each microcosm before herbicide treatment. Germinated turions were grown for 3 weeks in

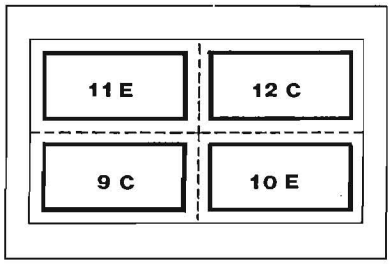


Figure 2. Arrangement of microcosms in experiment II. Heavy lines represent the aquaria, surrounding light lines denote black plastic drapes, and dotted lines are cardboard light shields

microcosms under the lighting conditions of experiment I. The plants characteristically developed runners, resulting in a population of plants of different sizes. The plants were then cut at the sediment surface to exclude runners, dried at 60°C for 5 days, and weighed to the nearest 0.1 mg. The leaves on each plant were counted before drying to compare leaf number with standing crop. Macrophytes were also collected from the lagoon area and littoral zone of West Twin Lake and treated similarly.

The relationship between leaf number and standing crop is shown in Figure 3 for both laboratory- and lagoon-grown macrophytes. It was assumed that plant growth is described by an exponential function. Plants with similar leaf numbers had higher standing crops in the field than in the laboratory.

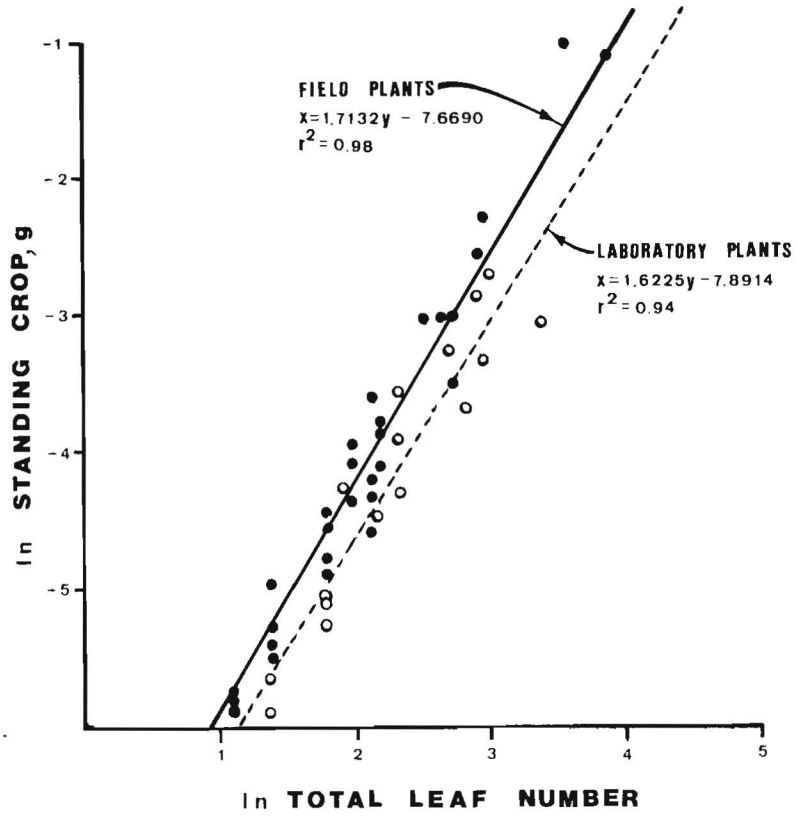


Figure 3. ln total leaf number versus plant standing crop for individual plants

The difference was probably the result of the less intense lighting conditions in the laboratory. It was observed that both germinated plants and cuttings grew similarly under the same lighting conditions although cuttings did not produce runners.

Macrophyte tissue phosphorus was compared with standing crop for both laboratory and field plants (Figure 4). As standing crop increased, the milligrams of phosphorus per gram of plant standing crop and the percent phosphorus per plant standing crop decreased in an exponential manner. Small plants had phosphorus percentages an order of magnitude higher than the values ranging from 0.1 to 1 percent reported in the literature (Caines, 1965; Riemer and Toth, 1969; Boyd, 1970). The percentage decreased to within the commonly reported range as standing crop increased. Figure 5 shows the regression equation between standing crop and milligrams of tissue phosphorus per gram of standing crop. Laboratory plants had slightly higher phosphorus concentrations than field plants, but all data were combined.

The number of leaves on each plant was recorded individually on

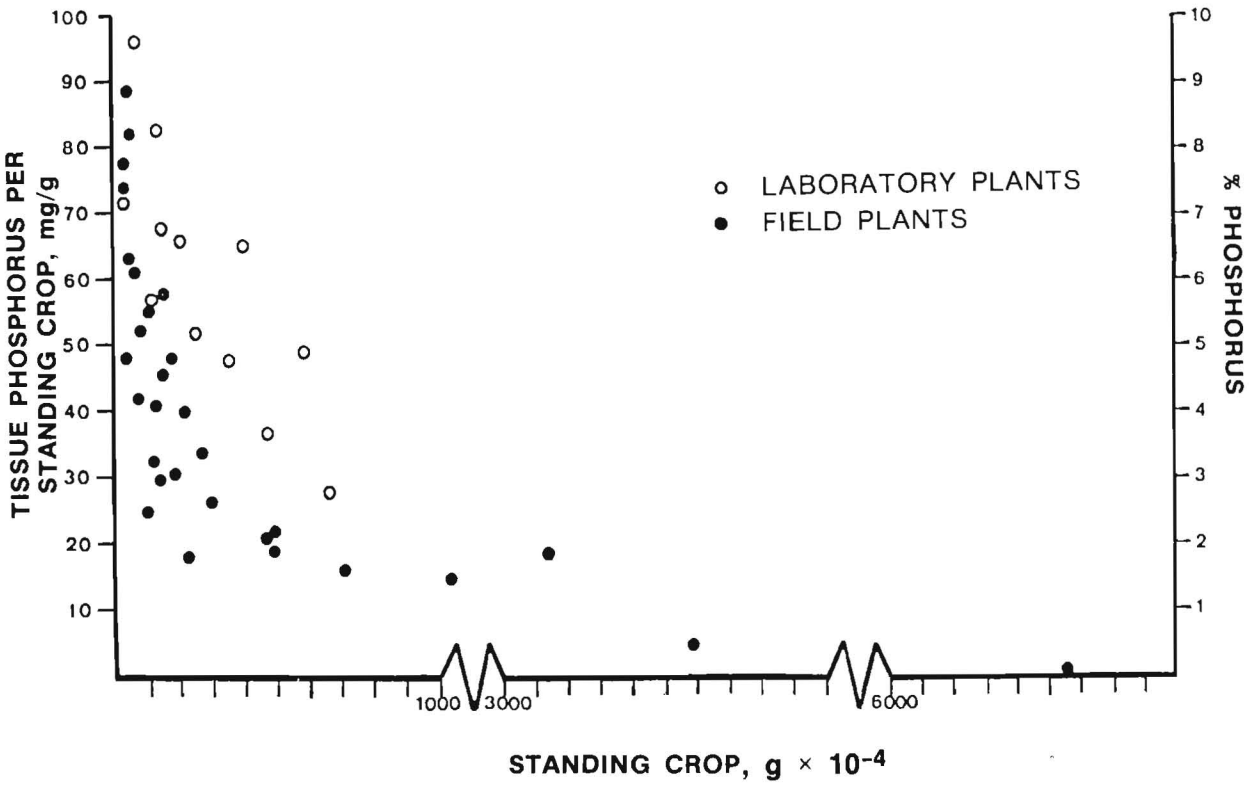


Figure 4. Macrophyte tissue phosphorus and percent tissue phosphorus per plant standing crop versus standing crop

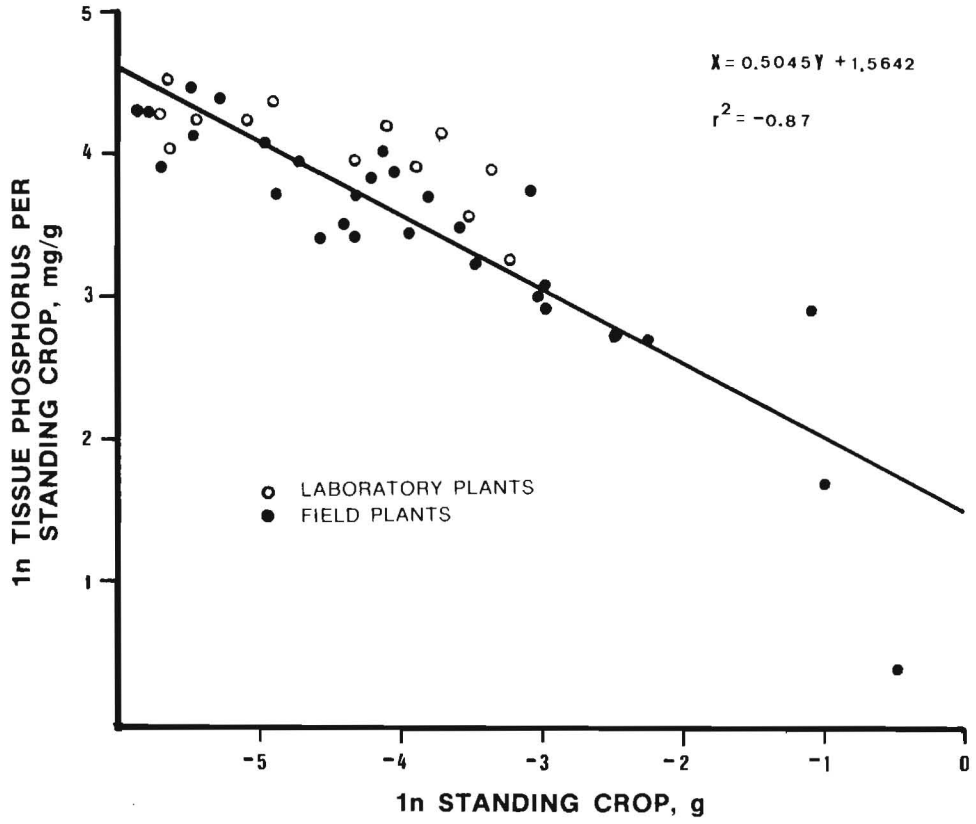


Figure 5. ln standing crop versus ln tissue phosphorus per plant standing crop

scheduled dates for both herbicide perturbation experiments. For each microcosm, the standing crop of individual plants was determined from the equation,

$$\ln \text{ standing crop} = -7.8914 - 1.6225Y \quad (1)$$

where Y = ln leaf number. The milligrams of phosphorus per gram of standing crop (SC) for each plant was obtained from the equation:

$$\ln \text{ mg P/g SC} = 1.5642 - 0.5045Y \quad (2)$$

where Y = ln plant standing crop. The total phosphorus contained in a plant was determined by multiplying standing crop by the calculated mg P/g SC. The total standing crop and macrophyte phosphorus content for each microcosm were determined by summing all values for the individual plants.

1. Experiment I.

Two aquaria were sampled for dissolved oxygen; chlorophyll a; and total, soluble, and soluble reactive phosphorus at weekly intervals for 5 weeks prior to herbicide treatment. Samples were collected in the middle of each aquarium with a polyethylene siphon between 1000 hr and 1500 hr. On 22 November 1978, 173 mg of Hydrothol 47 (Pennwalt), the equivalent of 2.0 ppm of the active ingredient, was applied to the experimental aquarium. The other aquarium served as a control. Phosphorus samples were taken in the morning and the evening for 3 days, then daily thereafter for 2 weeks. Samples for chlorophyll determination were taken once after herbicide treatment.

2. Experiment II.

Six control and six experimental microcosms were sampled twice before herbicide treatment. The analyses performed were dissolved oxygen; total phosphorus; soluble phosphorus; SRP; chlorophyll from the wall, aquarium wall, and sediment; and community metabolism.

Six microcosms were treated with 173 mg of Hydrothol 47 (Pennwalt) on 29 April 1979. Phosphorus was measured on the first, second, third, fifth, and fourteenth day after treatment. Chlorophyll was analyzed once and metabolism five times. Control microcosms were monitored at the same time.

C. Analyses.

1. Chemical.

Total phosphorus (TP), soluble phosphorus (SP), and soluble reactive phosphorus (SRP) were determined by the U. S. Environmental Protection Agency (USEPA) method (1971). Particulate phosphorus (PP) was obtained by subtracting SP from TP. Soluble unreactive phosphorus (SUP) was the difference between SP and SRP. Macrophyte tissue phosphorus was analyzed according to Chapman and Pratt (1961).

The biomass of algal communities which inhabited the water, the aquarium wall, and the sediment was measured by pigment extraction. Water chlorophyll a was determined by filtering 500 ml of microcosm water onto a glass fiber filter, grinding, and extracting in 20 ml of acetone (Long and Cooke, 1971). Microscope slides which had been placed in the aquaria were wiped clean with glass fiber filters to collect algae attached to the wall. The filters were then extracted in 10 ml of acetone after grinding (Long and Cooke, 1971). The top 5 mm of a known area of the sediment was removed by suction. Sediment algae were trapped in tissue paper according to Eaton and Moss (1966), then completely dried before extracting pigments in 10 ml of acetone. All pigment fractions were extracted in 90 percent acetone for 12 hr. The chlorophyll a concentration was calculated with the trichromatic equation of Strickland and Parsons (1968).

2. Community Metabolism.

The metabolic activities of an aquatic community cause diel changes in dissolved oxygen. The work of Odum (1956) and Odum and Hoskin (1958) established the methodology for determining the rates of gross primary productivity (GPP) and respiration (R) using open-water techniques. Odum (1956) described the processes governing the rate of dissolved oxygen change ($g/m^2/hr$) as:

$$Q = GPP - R \pm D + A \qquad (3)$$

where

Q = rate of dissolved oxygen change

GPP = rate of gross primary productivity

R = rate of community respiration

D = rate of diffusion into or out of a system

A = rate of drainage accrual (which will be omitted in this argument)

However, with use of the Odum and Hoskin (1958) method, the effects of diffusion will be omitted from the GPP calculation resulting in an error when daytime diffusion equals nighttime diffusion. The error will occur when the entire oxygen rate-of-change curve is corrected for diffusion.

Before a logical argument for the error can be presented, definitions of the variables estimated in this study by the diel oxygen curve method must be mentioned. Gross primary productivity is the rate of community photosynthesis, some of which is simultaneously catabolized by both autotrophs and heterotrophs. Catabolism of carbon inputs from outside the system will also be reflected in gross primary productivity. Net primary productivity is the rate of community photosynthesis stored in excess of community respiration during the daylight (Odum and Hoskin, 1958; Beyers, 1963). Twenty-four-hour net primary productivity is the net photosynthetic storage during a 24-hr-day/night cycle. Respiration is the rate of organic matter degradation. All values can be expressed as hourly ($g/m^2/hr$; $g/m^3/hr$) or daily ($g/m^2/day$; $g/m^3/day$) rates.

The Odum and Hoskin (1958) Method.

Data collected from microcosm No. 10 were used to present the calculations of gross primary productivity and respiration using the Odum and Hoskin (1958) method (Table 1). Dissolved oxygen and temperature were recorded at 4-hr intervals for 24 hr with a YSI oxygen probe, calibrated by the Winkler

Table 1

Data Used to Calculate Gross Primary Productivity Using the Odum and Hoskin (1958) Method

Time	Oxygen (mg/P)	% O_2 Saturation	Temperature (°C)
2130	8.1	97	23.2
2430	7.3	87	22.8
0430	6.5	75	21.8
0830	6.0	70	21.6
1230	6.5	77	22.4
1630	7.3	88	23.2
2130	7.6	91	23.0

method (USEPA 1971), and a thermister thermometer for periods beginning and ending at the start of the night cycle. The curves are shown in Figures 6A and 6C. An oxygen rate-of-change curve Q was then obtained from the oxygen curve using 2-hr intervals and rate values were plotted in the middle of each extrapolation (Figure 6E).

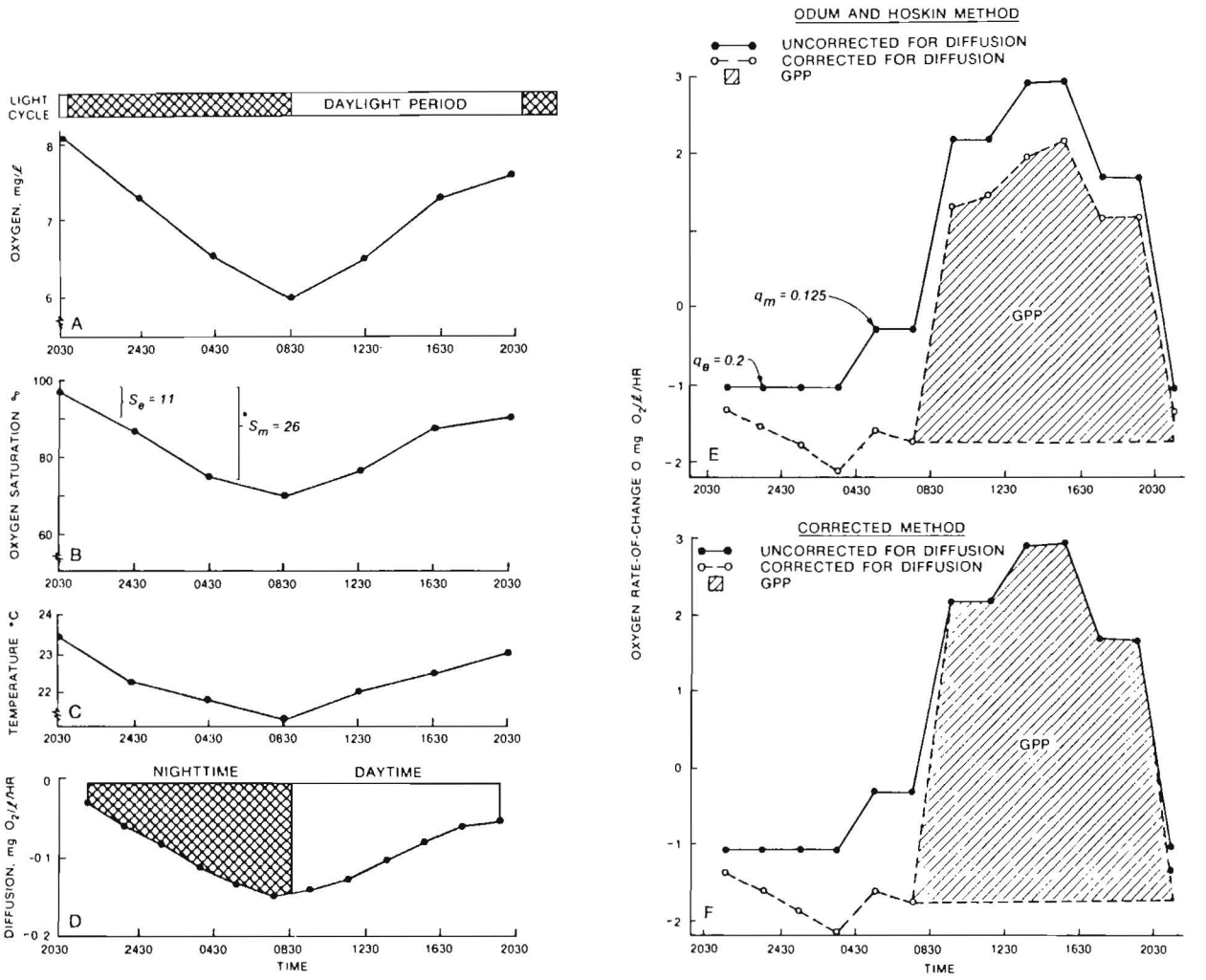


Figure 6. Diel variations in oxygen (A), oxygen saturation (B), temperature (C), and diffusion (D). Also shown is a comparison of the Odum and Hoskin (1958) method (E) and the corrected method (F) for determining community metabolism when nighttime and daytime diffusion are similar. See text for further explanation

A diffusion coefficient k was calculated from the equation:

$$k = 100 \left(\frac{q_m - q_e}{S_m - S_e} \right) \qquad (4)$$

where

k = volume based diffusion coefficient, mg $O_2/\ell/hr$

q_m = rate of oxygen change at a chosen predawn point, mg $O_2/\ell/hr$

q_e = rate of oxygen change at a chosen postsunset point, mg $O_2/\ell/hr$

S_m = percent saturation deficit at the predawn point chosen for q_m

S_e = percent saturation deficit at the postsunset point chosen for q_e

In Table 2, the calculation for k is shown using the data from Figures 6C and 6E.

The rate of diffusion D was obtained from the equation:

$$D = kS/100 \tag{5}$$

where

D = diffusion rate, mg $O_2/\ell/hr$

S = percent saturation deficit for each 2-hr rate interval on the Q curve

then added to or subtracted from each oxygen rate-of-change value for oversaturated or undersaturated conditions, respectively. Diffusion calculations are shown for each oxygen rate-of-change in Table 2 and the diffusion corrected rates are shown in Figure 6E. Total nighttime and daytime diffusion was then obtained from the diffusion rates integrated over the 12-hr-day/night cycle. It is important to note that nighttime diffusion equalled daytime diffusion.

From the corrected curve, respiration (mg $O_2/\ell/day$) was determined by multiplying the average hourly nighttime respiration rate by 24 hr (see Table 2 for calculation). Respiration included the effects of diffusion, according to the equation

$$R = -Q_{night} + Din_{night} \tag{6}$$

where

Q_{night} = nighttime dissolved oxygen rate of change, mg $O_2/\ell/day$

Din_{night} = nighttime dissolved oxygen diffusion into the system, mg $O_2/\ell/day$

in this example.

The error by Odom and Hoskins (1958) which results in the omission of diffusion occurs in the calculation of gross primary productivity. The

Table 2

Calculations of Parameters Used to Determine Community Metabolism Using the Odum and Hoskin (1958) and Corrected Methods

PARAMETER	ODUM AND HOSKIN (1958) METHOD	VALUE	CORRECTED METHOD	VALUE
k	$k = 100\left[\frac{q_m - q_e}{S_m - S_e}\right] = 100\left[\frac{0.125-0.200}{26-11}\right]$	0.50 mg O_2/ℓ/hr into the system	$k = 100\left[\frac{q_m - q_e}{S_m - S_e}\right] = 100\left[\frac{0.125-0.200}{26-11}\right]$	0.50 mg O_2/ℓ/hr into the system
D	D = kS/100		D = kS/100	
	Time 2130 2330 0130 0330 0530 0730 S/100 -0.06 -0.11 -0.16 -0.22 -0.26 -0.29 D 0.03 0.06 0.08 0.11 0.13 0.15		Time 2130 2330 0130 0330 0530 0730 S/100 -0.06 -0.11 -0.16 -0.22 -0.26 -0.29 D 0.03 0.06 0.08 0.11 0.13 0.15	
	Time 0930 1130 1330 1530 1730 1930 S/100 -0.28 -0.25 -0.20 -0.15 -0.11 -0.10 D 0.14 0.13 0.10 0.08 0.06 0.05		Time 0930 1130 1330 1530 1730 1930 S/100 -0.28 -0.25 -0.20 -0.15 -0.11 -0.10 D 0.14 0.13 0.10 0.08 0.06 0.05	
	Nightime D = $\bar{D}_{night}$ mg O_2/ℓ/hr (12 hr)	1.12 mg O_2/ℓ/12 hr	Nightime D = $\bar{D}_{night}$ mg O_2/ℓ/hr (12 hr)	1.12 mg O_2/ℓ/12 hr
	Daytime D = $\bar{D}_{day}$ mg O_2/ℓ/hr (12 hr)	1.12 mg O_2/ℓ/12 hr	Daytime D = $\bar{D}_{day}$ mg O_2/ℓ/hr (12 hr)	1.12 mg O_2/ℓ/12 hr
R	R mg O_2/ℓ/hr = $-Q_{night}$ + Din_{night}		R mg O_2/ℓ/hr = $-Q_{night}$ + Din_{night}	
	Time 2130 2330 0130 0330 0530 0730 -Q 0.20 0.20 0.20 0.20 0.13 0.13 D 0.03 0.06 0.08 0.11 0.13 0.15 R 0.23 0.26 0.28 0.31 0.26 0.28		Time 2130 2330 0130 0330 0530 0730 -Q 0.20 0.20 0.20 0.20 0.13 0.13 D 0.03 0.06 0.08 0.11 0.13 0.15 R 0.23 0.26 0.28 0.31 0.26 0.27	
	R_{Total} = $\bar{R}$ mg O_2/ℓ/hr (24 hr)	6.48 mg O_2/ℓ/day	R_{Total} = $\bar{R}$ mg O_2/ℓ/hr (24 hr)	6.48 mg O_2/ℓ/day
GPP	GPP = (3750 squares) (0.001 mg O_2/ℓ)	3.75 mg O_2/ℓ/day	GPP = (4885 squares) (0.001 mg O_2/ℓ)	4.89 mg O_2/ℓ/day

NOTE: R_{Total} = 24-hr respiration (mg O_2/ℓ/day)

$\bar{R}$ = mean hourly respiration (mg O_2/ℓ/hr)

$\bar{D}$ = mean hourly diffusion (mg O_2/ℓ/hr)

average, diffusion-corrected, nighttime respiration rate was extended across the daytime hump of the corrected curve (Figure 6E), then the squares inside the shaded area were counted. The area of one square was determined by multiplying its vertical axis by its horizontal axis value. This area was multiplied by the total number of squares counted to obtain gross primary productivity (mg O_2/ℓ/day) (see Table 2 for calculations).

The effects of diffusion and simultaneous respiration should then have been included in the calculation of GPP according to the equation,

$$GPP = Q_{day} + R_{night} - Din_{day} \quad (7)$$

However, when Equation 6 was substituted into Equation 7, producing the equation

$$GPP = Q_{day} + (-Q_{night} + Din_{night}) - Din_{day}, \quad (8)$$

diffusion was cancelled from the GPP calculation when Din_{night} equalled Din_{day}.

The omission of diffusion from the GPP calculation occurred when the entire Q curve was corrected for diffusion. According to the Odum and Hoskin (1958) method, GPP became the summation of nighttime Q and daytime Q. To verify this, GPP calculated from values substituted into Equation 8 was compared with the value obtained from the area under the corrected curve. Table 3 shows that the sum of 3.70 mg O_2/ℓ/day closely corresponded to the value of 3.75 mg O_2/ℓ/day obtained from the area under the curve.

The Corrected Method.

Their error was corrected by using the same data from Table 1. The same oxygen, percent oxygen saturation, temperature, diffusion, and oxygen rate-of-change curves used in the Odum and Hoskin calculations were used for the correction. On the rate-of-change curve, only nighttime rates were corrected for diffusion; daytime rates were not corrected (Figure 6F). The average, diffusion corrected, nighttime rate was then extended across the daytime period hump, and the bounded area was determined to obtain GPP. Equation 7 describes the GPP calculation using the corrected method as

Table 3

Comparison of Calculations of GPP from an Oxygen Mass Balance Equation (Equation 8) and the Area Under the Curve Method of Odum and Hoskin (1958)

Mass balance equation (data taken from Table 1):

$$GPP = Q_{day} + (-Q_{night} + Din_{day}) - Din_{day}$$

where

$Q_{day} = 7.6 \text{ mg}/\ell - 6.0 \text{ mg}/\ell$ = 1.6 mg O_2/ℓ/daylight

$-Q_{night} = -(6.0 \text{ mg}/\ell - 8.1 \text{ mg}/\ell)$ = 2.1 mg O_2/ℓ/night

$-Din_{day} = (\bar{D}in_{day})$ (12 hr) = -(0.093 mg/ℓ/hr) (12 hr) = -1.12 mg O_2/ℓ/night

$Din_{night} = (\bar{D}in_{night})$ (12 hr) = (0.093 mg/ℓ /hr) (12 hr) = 1.12 mg O_2/ℓ/daylight

thus

$GPP = 1.6 + 2.1 + 1.1 - 1.1$ = 3.70 mg O_2/ℓ/day

Area under the curve:

GPP = (3750 squares) (0.001 mg O_2/ℓ/square) = 3.75 mg O_2/ℓ/day

$$GPP = Q_{day} + R_{night} = Q_{day} + (-Q_{night} + Din_{day}) \quad (9)$$

which now includes the effects of diffusion.

The corrected method only applies to situations where nighttime diffusion equals daytime diffusion. In highly productive systems, the inverse of this situation often occurs. At night, community respiration depletes the oxygen concentration below its saturation value causing oxygen to diffuse into the system. Daytime net primary productivity results in oxygen oversaturation and diffusion out of the system. The net effect is described by the equation:

$$Din_{night} = -(Dout_{day}) \quad (10)$$

In this case, the entire curve should be corrected for diffusion following the procedure of Odum and Hoskin (1958) to obtain correct gross primary productivity and respiration rates.

3. Calculation of GPP, NPP, 24-hr NPP, and diffusion.

In this study, nighttime and daytime diffusion were usually equal for all determinations, so gross primary productivity and respiration were calculated with the corrected method. Net primary productivity was computed as the difference between gross primary productivity and nighttime respiration. Gross primary productivity was subtracted from the 24-hr respiration rate to obtain 24-hr net primary productivity.

The value for the diffusion coefficient k was obtained from the Velz (1939) nomograph. The nighttime respiration rates, used to calculate k according to the Odum and Hoskin (1958) method, produced extremely variable coefficients, ranging from -1.50 to +1.75 mg $O_2/\ell/hr$ or -0.34 to +0.399 g $O_2/m^2/hr$. Areal-based literature values ranged from 0.03 to 0.08 g $O_2/m^2/hr$ for still-water aquaria which are substantially lower than the range reported here (review Odum, 1956). To determine the coefficient, a mixing time was required. Dye was carefully injected into an aquarium filled with enough tap water to approximate the volume contained in the experimental aquaria. Complete visual dispersion took approximately 12 hr. Based on the mixing time, water depth (0.2 m), and mean temperature (23°C), diffusion rates (g $O_2/m^2/hr$) were determined for saturation deficits ranging from 0.1 to 0.6 percent. The relationship between the saturation deficit and the diffusion

rate were then graphed, and the slope of the line was calculated to determine K , the areal-based diffusion constant, which was 0.086 g $O_2/m^2/hr$. The value for K was within the range of values reported for still-water aquaria, and was used for all diffusion calculations. The volume-based diffusion coefficient k (mg $O_2/\ell/hr$) was obtained by dividing 0.086 by 0.228 m, the depth of the systems.

4. Autotrophic Components.

The effects of herbicide treatment on community metabolism were ascertained for three autotrophic components using diel oxygen changes and light/dark bottle changes. These components were: macrophyte-epiphyte, benthic, and planktonic.

Planktonic metabolism was measured with light and dark bottles incubated in each aquarium for 4 hr, the time period overlapping the middle of the day. Planktonic GPP was determined as:

$$\text{GPP}(\text{mg } O_2/\ell/\text{day}) = \frac{\text{Light bottle (mg/}\ell\text{) - dark bottle (mg/}\ell\text{)}}{\text{4-hr incubation}} \quad 12 \text{ daylight hours} \qquad (11)$$

and planktonic respiration (R) as:

$$R \text{ (mg } O_2/\ell/\text{day)} = \frac{\text{initial bottle - dark bottle (mg/}\ell\text{)}}{\text{4-hr incubation}} \quad 24 \text{ hr} \qquad (12)$$

To be consistent with the calculation of the net primary productivity (NPP) values using the Odum and Hoskin (1958) method (see Section 3), planktonic NPP was computed as the difference betwen GPP and daytime R (mg $O_2/\ell/\text{day}$ ' 2). The difference between GPP and total R was computed to obtain 24-hr net primary productivity (24-hr NPP).

Benthic metabolism was determined using the corrected diel oxygen change method. A plexiglass cylinder measuring 0.055 m in diameter and 0.304 m in height was carefully pushed into the sediment to the bottom of each aquarium. The thickness of each plexiglass cylinder was 6.35 mm, which stopped diffusion between the enclosed water column and aquarium water. A column of water, covering a sediment area of 0.002 m^2 and open to the atmosphere, was isolated from the rest of the aquarium. *Potamogeton crispus* cuttings were not included

in the cylinders; thus, only the planktonic and epipelic algal communities were represented. Changes in oxygen over a 24-hr period were then used to obtain daily GPP and R rates for the communities enclosed by each cylinder. Since the metabolism in each cylinder represented both planktonic and benthic inputs, the daily planktonic GPP and R rates measured from light and dark bottles were subtracted from the cylinder rates to obtain the benthic rates. The difference between the benthic GPP and nighttime benthic R was then used to obtain benthic net productivity (see Section 3 for a description of the equation). Total benthic R (24-hr R) was subtracted from benthic GPP to calculate 24-hr NPP.

To estimate macrophyte-epiphyte metabolism, the daily cylinder GPP and R rates were subtracted from the total microcosm GPP and R rates. Net primary productivity was determined using the macrophyte-epiphyte GPP and R rates, according to Section 3.

III. RESULTS

A. Field Study.

Before herbicide treatment, particulate phosphorus (PP) was lower in the experimental water than the control site on two occasions (Figure 7). Both values were nearly similar 2 hr before the first herbicide application. After herbicide treatment PP increased in the experimental area to 27 µg/ℓ on day 4, descended to 8 µg/ℓ on day 9, then increased to 29 µg/ℓ on day 10. In the control area, PP fluctuated between 15 and 23 µg/ℓ for 10 days, then fell to 7 µg/ℓ on day 11. PP was similar in the experimental and control areas on days 13 and 15.

Soluble unreactive phosphorus (SUP) was consistently higher in the experimental water than the control area before herbicide treatment. After the first treatment date the concentration decreased slightly, remained constant until day 7, then increased to a maximum peak of 33 µg/ℓ one day after the second herbicide application. The concentration decreased to 16 µg/ℓ on day 11, then another peak of 24 µg/ℓ was noted on day 12. In the control area, SUP fluctuated less, maintaining a concentration above 15 µg/ℓ on days 1 and 4. The concentration dropped to slightly lower values on days 6 and 11, then increased to 16 µg/ℓ on day 12, remaining steady thereafter.

Soluble reactive phosphorus (SRP) in both sites was low before herbicide treatment. SRP increased slightly to 11 µg/ℓ on day 11 in the control area; however, the values fluctuated around 5 µg/ℓ on all other dates. In the experimental area, SRP increased to 12 µg/ℓ on day 6, one day before the second herbicide application, then reached 22 and 29 µg/ℓ on days 7 and 11. The values in the experimental and control areas were similar after day 12.

After herbicide treatment, dissolved oxygen (DO) was lower in the experimental area than the control section for ten days (Figure 7). The lowest recorded oxygen concentration of 2.6 ppm occurred on day 4. Oxygen began increasing steadily from day 7 to day 10, then fell to 3.4 ppm on day 12. In the control area, oxygen decreased to 4.9 ppm on day 4, increased to 8.5 ppm on day 10, then descended to 4.7 ppm. Oxygen was similar in both areas on day 12.

The macrophytes did not begin to settle until day 4, but were noticeable on the sediment surface by the second herbicide application. The short-term

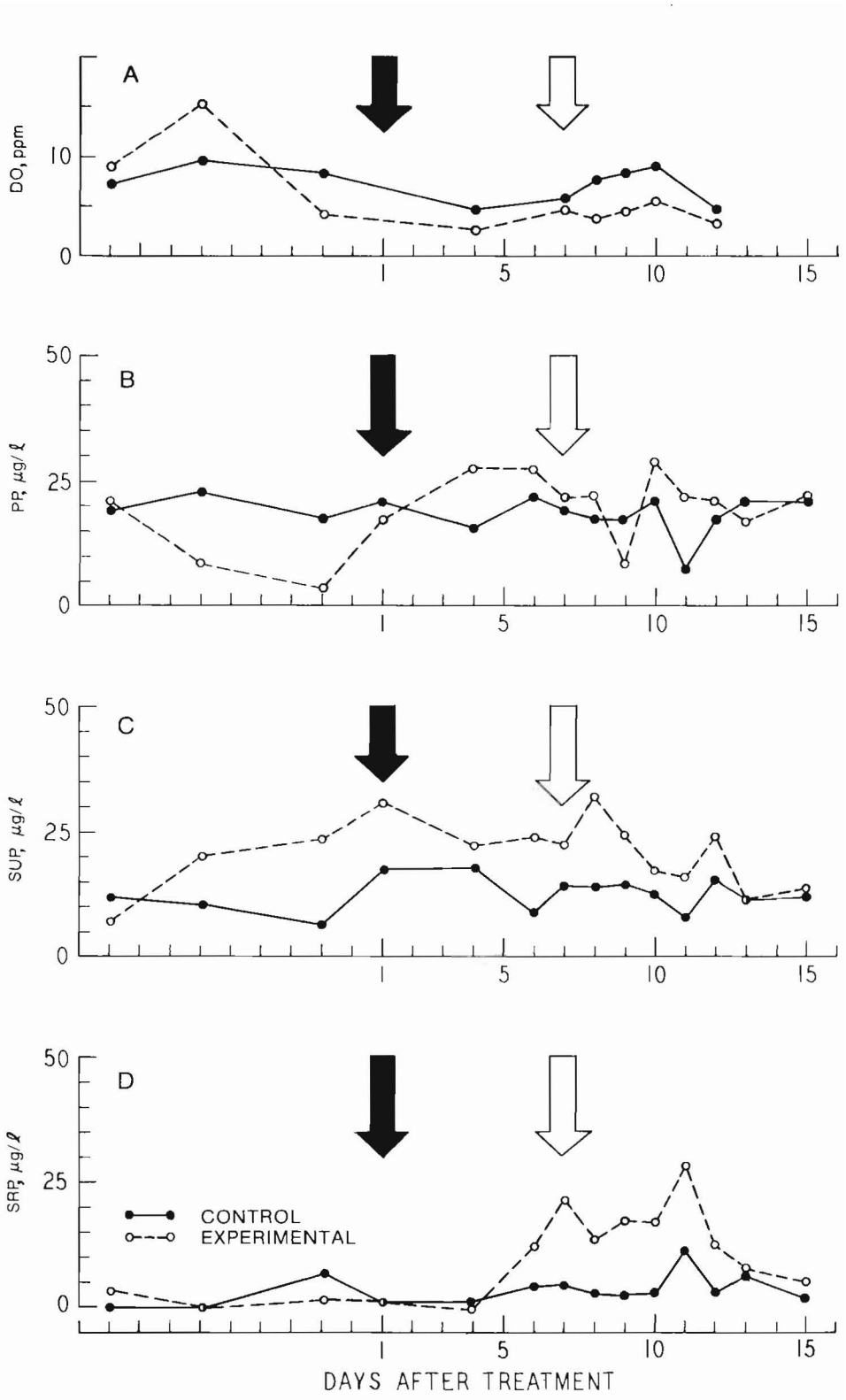


Figure 7. Dissolved oxygen and phosphorus data from the field experiment. The solid arrow indicates first herbicide treatment, the clear arrow indicates the second treatment date

changes in phosphorus in experimental and control areas on each day of herbicide application are listed in Table 4. On the first treatment day, no obvious differences in PP and SUP were apparent between the two sites. A slight increase in SRP occurred in the experimental site 2 hr after herbicide application, which persisted until 2000 hr. One observation was taken in each area for the entire study so significant differences could not be ascertained.

Table 4

Changes in Phosphorus (µg/ℓ) Before and Immediately After Herbicide Treatment in the Lagoon

Time	Particulate Phosphorus		Soluble Unreactive Phosphorus		Soluble Reactive Phosphorus	
	Control	Experimental	Control	Experimental	Control	Experimental
			Day 1			
0830	20.9	17.4	17.5	26.2	1.7	1.7
1200	14.0	29.7	22.7	29.2	1.7	1.7
1400	8.7	12.3	24.2	19.2	1.7	8.7
1600	--	--	--	--	0.0	7.0
2000	--	--	--	--	1.7	8.7
			Day 6			
1130	3.5	22.7	14.0	22.7	5.2	22.7
1530	19.2	29.7	15.7	13.9	3.5	17.5
1930	36.6	3.5	17.5	27.9	0.0	19.2

On the second treatment date (Table 4), day 6, PP increased in the control area and decreased in the experimental site. Strong westerly winds may have mixed PP and possibly endothall from the experimental site into the control site, thus contaminating the control site. However, higher SRP concentrations were recorded in the experimental area, but a similar trend was not apparent in the control site. Plants in the control area did not seem to be affected by the herbicide and remained firmly standing in the water for the duration of the study, indicating that herbicide contamination in the control area may have been minimal.

B. Microcosm Experiment I.

The number of plants in the experimental and the control microcosm increased by the formation of runners. During the early stages of population increase, young plants grew well, producing a tuft of leaves at the apex as plant heights reached the water surface. As microcosms became crowded, some of the newly sprouted plants ceased to grow in height and instead became thick stemmed. Turions developed on the plants 1-1/2 months after germination. Prior to herbicide treatment, the control microcosm total standing crop was 17.2 g/m^2 and the experimental microcosm had a standing crop of 13.3 g/m^2. The control microcosm contained 326.3 mg/m^2 of macrophyte tissue phosphorus whereas the experimental system had a macrophyte tissue phosphorus content of 287 mg/m^2.

The granular form of endothall (Hydrothol 47) was applied to the experimental microcosm, the intended concentration being 2.0 ppm of the active ingredient. One day after herbicide treatment, experimental plants began wilting and were noticeably discolored. Within 1 week, the macrophytes had settled to the sediment surface. Leaf deterioration began during the next 2 weeks and by day 20 epiphytes had colonized the decaying plants, giving them a green, feltlike appearance. Although data were not taken, epipelic algal growth appeared to be extensive in the experimental microcosm when compared to the control microcosm on day 20.

The phosphorus status of the microcosms is depicted in Figure 8, each data point representing one observation. Before herbicide treatment, phosphorus fractions were similar in each microcosm. After the application of herbicide, PP fluctuated for 2 weeks, reaching 37 and 41 µg/ℓ on days 3 and 9, respectively, before descending to the control concentration on day 10. In the control microcosm, PP wavered slightly for 8 days, decreasing to lower concentrations after day 8. Soluble unreactive phosphorus was slightly elevated in the experimental microcosm for 6 days, then a pulse of short duration was observed on days 8 through 10. The accumulation of soluble reactive phosphorus was rapid for the first 4 days, reaching 47 µg/ℓ on day 6, then declined steadily. SRP in the control microcosm was not detectable throughout the experiment.

The expanded short-term changes in phosphorus before and immediately after herbicide treatment are depicted in Table 5. PP levels gradually

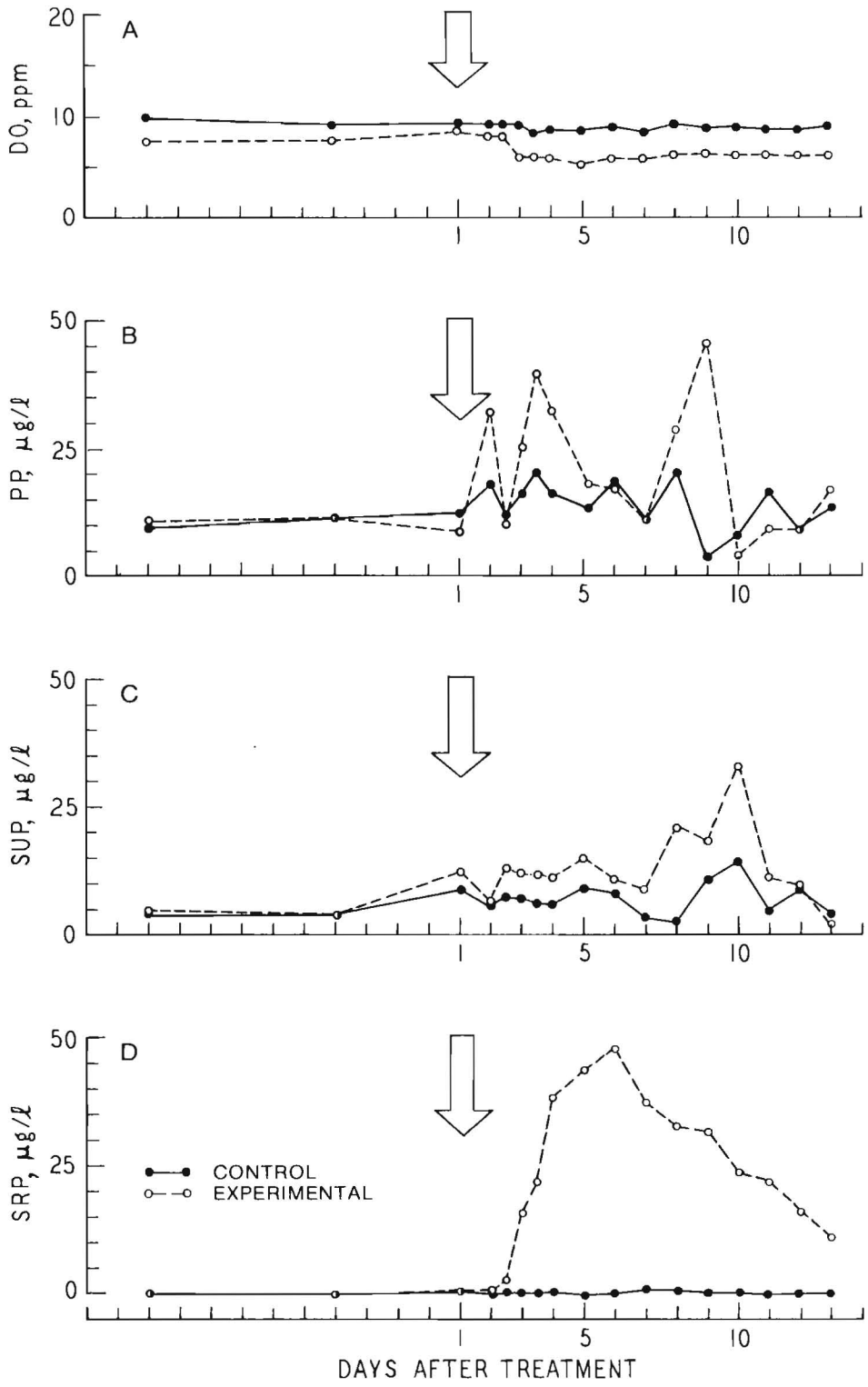


Figure 8. Dissolved oxygen and phosphorus data from microcosm experiment I. The arrow represents the date of herbicide treatment

Table 5

Changes in Phosphorus (μg/ℓ) Before and Immediately After Herbicide Treatment in Microcosm Experiment I*

Time	Particulate Phosphorus		Soluble Unreactive Phosphorus		Soluble Reactive Phosphorus	
	Control	Experimental	Control	Experimental	Control	Experimental
			Day 1			
0700	7.0	10.5	10.8	11.5	1.4	0.7
1800	23.2	7.1	8.7	13.9	0.0	0.0
2330	10.5	19.2	8.3	8.7	0.0	0.0
			Day 2			
1000	17.8	32.2	5.6	6.9	0.0	0.0
2100	12.2	10.3	7.5	13.2	0.0	2.8
			Day 3			
1100	16.0	25.4	7.3	12.2	0.0	16.0
1930	20.7	39.5	6.6	12.2	0.0	22.8

* Each value represents one observation.

increased in concentration by 1000 hr on day 2, fell, then increased again by 1930 hr on day 3 in the experimental microcosm. PP increases were less extensive in the control microcosms. The SUP concentration began to increase by 2100 hr on day 2, remaining slightly higher than the control level, until larger pulses occurred on days 8 and 10 (Figure 8). SRP also began increasing by 2100 hr on day 2.

Dissolved oxygen decreased after herbicide treatment, although the concentration never descended below 5 ppm (Figure 8). The sediment microzone was light in color and appeared to be oxidized throughout the experiment.

The results of the measurements of chlorophyll *a* fractions are presented in Figure 9. Note that the scale changes between the fractions. Water chlorophyll *a* remained very low after treatment, and water and wall chlorophyll *a* were similar in both systems during the experiment. The observed particulate phosphorus increase on day 9 was not reflected in the water chlorophyll *a* concentration. Sediment chlorophyll *a* increased in the experimental microcosms to a mean of 4.9 mg/m^2 9 days after herbicide treatment. Although data were

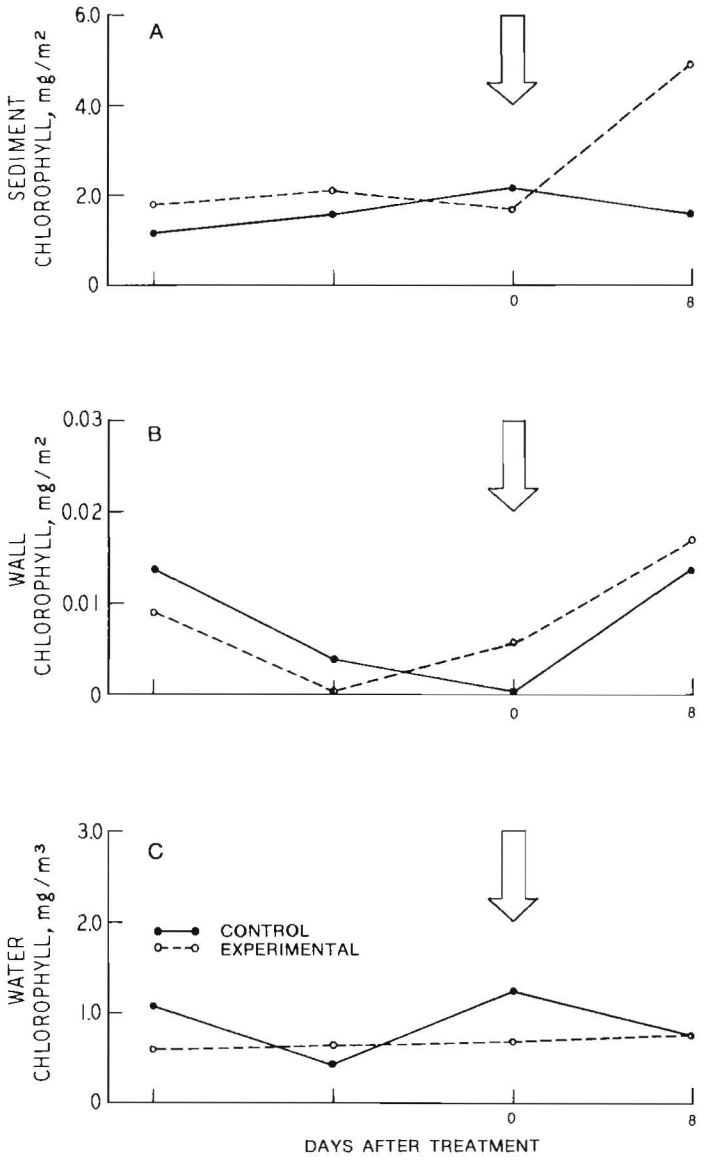


Figure 9. The response of sediment, wall, and water chlorophyll a to herbicide treatment in microcosm experiment I. Arrow indicates herbicide treatment

not collected, the experimental microcosm had a dense mat of epipelic algae on the sediment surface and epiphytic algae colonizing the decomposing plants 20 days after herbicide treatment.

C. Microcosm Experiment II.

Microcosm experiment II was conducted to reexamine the effects of Hydrothol 47 on phosphorus concentrations in the water, but differed from

experiment I in a number of respects. Twelve microcosms were used in experiment II to replicate phosphorus observations, and community metabolism was monitored to assess the response of autotrophic components to an endothall perturbation. The sediment used in experiment II came from a different location, the West Twin Lake littoral zone. Lagoon sediment was used for experiment I. This proved to be an important difference in that the sediments from the two locations had dissimilar phosphorus-adsorbing capacities. Cuttings of *Potamogeton crispus* were used in experiment II rather than turions, which were germinated for experiment I.

The cuttings grew in length throughout the experiment but formed few runners, in contrast to the plants in microcosm experiment I. Many plants grew to the water surface, possibly releasing oxygen directly to the atmosphere and thus some of the metabolism measurements may be in error. Toward the end of the experiment many lower leaves and some entire plants died in the control microcosms. Turions also developed as the plants matured.

After treating the experimental microcosms with a dose of 2.0 ppm Hydrothol 47, the macrophytes began to discolor and wilt within 1 day. On day 5, the plants had fallen to the sediment surface, and by day 24, many of the leaves had disintegrated. Algae were noticeable on the plants and the sediment surface as decomposition progressed. The autotrophic groups in the control microcosm were *Potamogeton crispus*, *Chara* sp., and other algae, mainly inhabiting the sediment.

The six control and six experimental tanks had comparable macrophyte standing crops and tissue phosphorus contents 1 day before herbicide treatment (Table 6). Standing crop and tissue phosphorus were lower in each microcosm compared to experiment I.

Table 6

Standing Crop and Macrophyte Phosphorus in the Control and Experimental Microcosms (± S.E.)

	$\bar{X}$ Standing Crop g/m^2		$\bar{X}$ Tissue P mg/m^2	
Control	3.36	(0.35)	89.93	(9.17)
Experimental	3.70	(0.19)	95.55	(7.19)

Before herbicide treatment, the mean PP, SUP, and SRP values in the water were not statistically distinguishable in the control and experimental microcosms (Figure 10). In the experimental systems, mean PP increased to a maximum peak of 25 μg/ℓ (±0.86 S.E.) on day 3, fell to 22 μg/ℓ (±2.03 S.E.) on day 4, then decreased to control levels by day 7. Mean SUP slightly increased ($p < 0.05$, t-test) after herbicide treatment on day 7, then descended to control concentrations on day 14. However, unlike experiment I, the mean SRP value did not deviate significantly from the control mean throughout the early stages of plant decomposition. In the control microcosms, all mean phosphorus fractions fluctuated slightly during the experiment.

The absence of an SRP pulse in microcosm experiment II suggested that the sediment adsorbed SRP more strongly than the sediment used in microcosm experiment I. Table 7 displays the sediment adsorption capacity for KH_2PO_4. The microcosm experiment I sediment adsorbed a net mean total of 93 percent of the introduced phosphorus compared to a mean of 31 percent adsorbed by the experiment II sediment. Thus, an SRP pulse was observed in microcosm experiment I, containing sediment which adsorbed phosphorus strongly. No pulse occurred in microcosm experiment II which contained sediment weakly adsorbing phosphorus.

A chlorophyll a increase was recorded in the experimental microcosms (Figure 11). Although mean water and wall chlorophyll a did not increase, mean sediment chlorophyll a was higher in experimentally treated tanks 15 days after herbicide treatment ($p < 0.05$, t-test). A one-tailed Student's t-test was chosen, assuming that endothall killed only aquatic plants. The mean sediment chlorophyll a value also increased to a lesser extent in the control microcosms.

Herbicide treatment caused an abrupt short-term change in the diel oxygen pattern of the experimental microcosms. An example of a typical oxygen curve before and 6 days after herbicide treatment is shown in Figures 12a and 12b for microcosm No. 7. Before the perturbation, a distinct diel oxygen change was recorded, and the oxygen saturation did not descend below 70 percent. Herbicide treatment caused a drastic decrease in oxygen concentrations, the saturation value remaining nearly constant at 52 percent.

No diel oxygen change was detected throughout the day shortly after endothall application (Figure 12b). Oxygen changes were small in all experimentally treated systems. As shown in Table 8, the nighttime diffusion rate

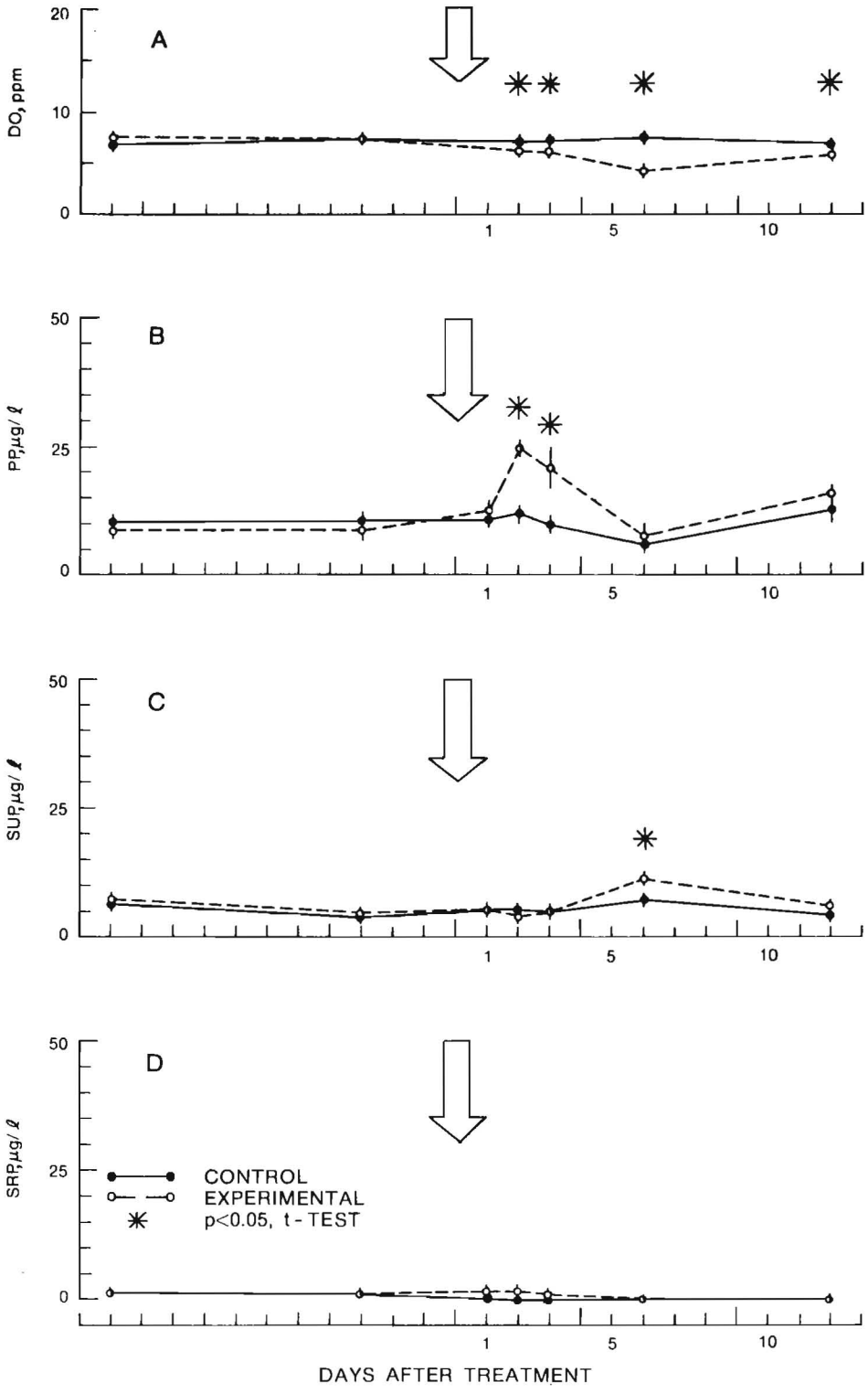


Figure 10. Dissolved oxygen and phosphorus data from microcosm experiment II. Points represent mean values; bars indicate the standard error; and asterisks indicate significant differences ($p < 0.05$, t-test). Arrow marks the date of herbicide treatment

Table 7

Sediment Adsorption - Desorption

Experiment I Sediment (µg/ℓ)			Experiment II Sediment (µg/ℓ)		
Trial	Adsorbed	Desorbed	Trial	Adsorbed	Desorbed
1	219.2		1	100.1	11.1
2	219.2		2	80.9	12.2
3	219.8		3	83.6	12.3
4	218.3		4	83.2	13.1
5	218.7	4.5	5	81.6	12.9
6	218.7	4.7	6	89.2	17.1
7	212.1	5.3	7	83.2	14.1
8	219.6	4.7	8	78.7	13.3
9	218.1	4.2	9	81.9	14.3
10	217.8	4.2	10	84.5	12.9
$\bar{X}$	218.1	4.8	$\bar{X}$	84.7	13.3

	Initial KH_2PO_4 introduced (µg/ℓ)	Net adsorbed (µg/ℓ)	%
Experiment I	229.4	213.5	93
Experiment II	229.4	71.4	31

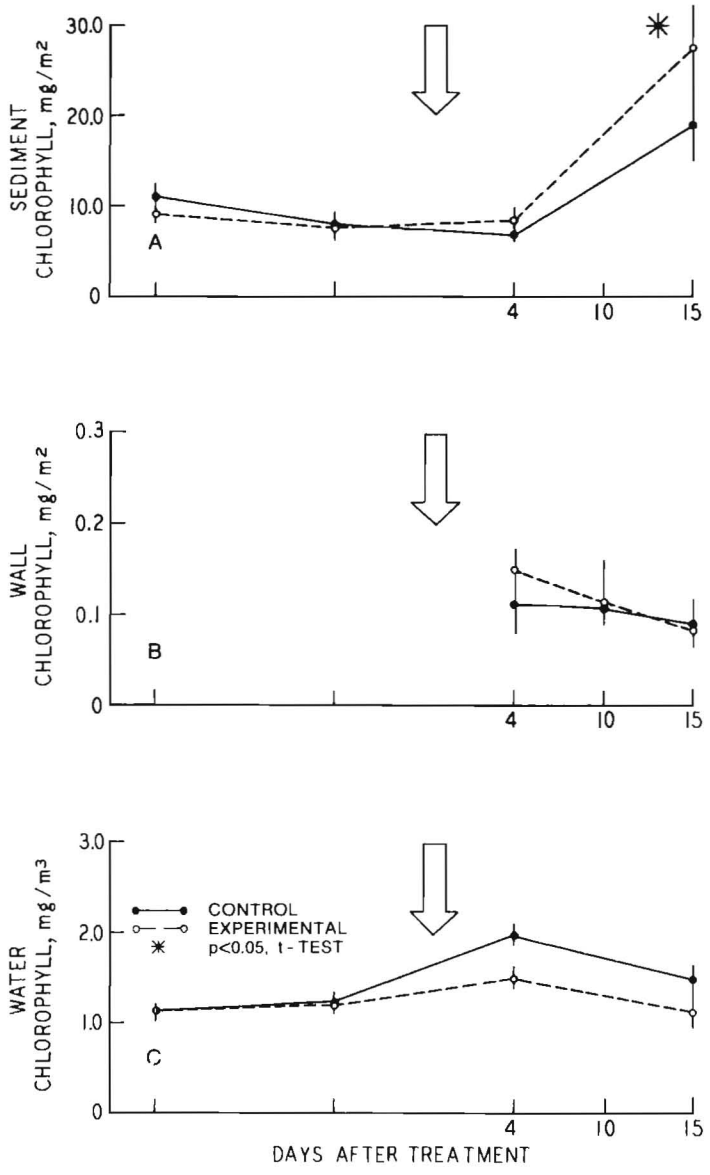
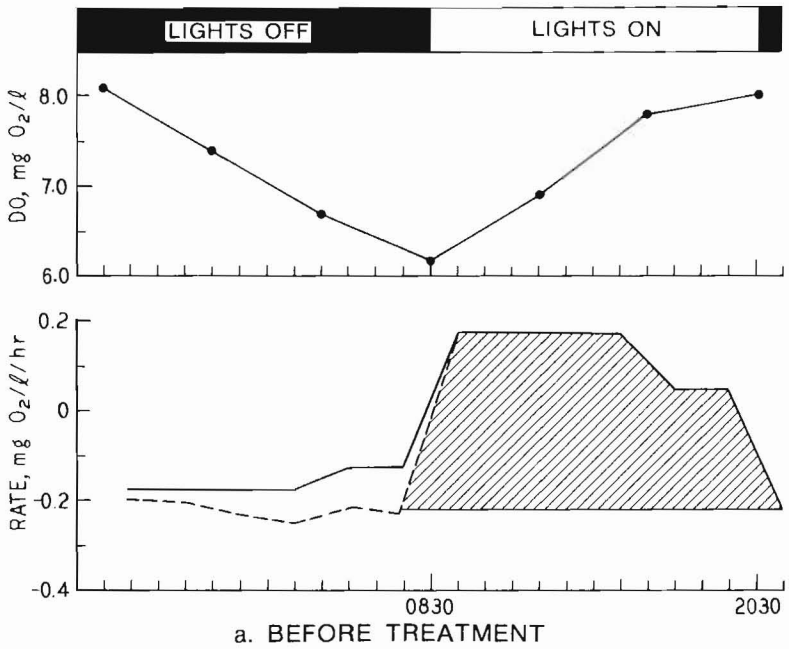
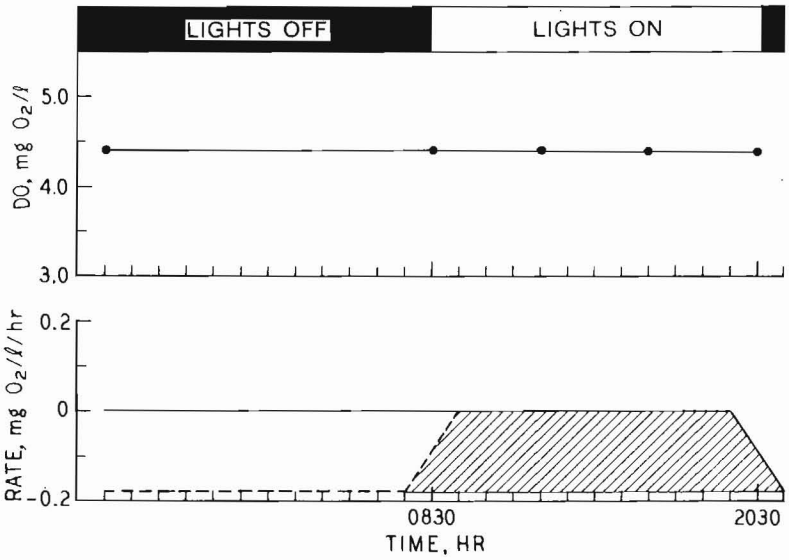


Figure 11. Mean sediment, wall, and water chlorophyll a concentrations from microcosm experiment II. Bars represent standard errors and asterisks indicate significant differences ($p < 0.05$, t-test)



a. BEFORE TREATMENT



b. SIX DAYS AFTER TREATMENT

Figure 12. Diel oxygen changes and rate-of-change curves from microcosm No. 7. Dotted line is rate-of-change curve corrected for diffusion

Table 8

Metabolic Data from Microcosm No. 7 Six Days After Herbicide Treatment (mg O_2/ℓ/day)

R_{night}	R_{total}	GPP	NPP	24-hr NPP	D_{night}	D_{day}
2.19	4.39	2.16	-0.03	-2.23	+2.19	+2.19

R_{night} = Nighttime respiration (12-hr period)
R_{total} = Total respiration (24-hr period)
GPP = Gross primary productivity
NPP = Net primary productivity during the daylight
24-hr NPP = Net primary productivity during 24 hr
D_{night} = Nighttime diffusion into the system
D_{day} = Daytime diffusion into the system

equalled the nighttime respiration rate, indicating a steady-state situation which accounted for the lack of an oxygen change. Since net primary productivity was negligible, gross primary productivity was also similar to nighttime respiration, which is assumed to be the same as daytime respiration. The experimental microcosms were therefore heterotrophic.

As shown in Figure 13, 1 day prior to herbicide treatment, the mean microcosm GPP rate was slightly greater in the experimental microcosms, although this was not statistically significant. The macrophyte-epiphyte component was the dominant producer in both experimental and control microcosms, contributing a mean of 51 percent, while the planktonic component was the next dominant in both sets, followed by the benthic component. The mean macrophyte-epiphyte and benthic GPP were slightly higher in the experimental microcosms, but not to a statistically significant extent.

Six days after herbicide treatment, microcosm GPP decreased to a mean rate of 2.56 mg O_2/ℓ/day in the experimental microcosms, which was significantly lower than the control microcosms ($p < 0.05$, t-test). Mean microcosm GPP also decreased in the control microcosms to a smaller extent, the decreases in both sets being caused, in part, by a drop in the planktonic rates.

The mean macrophyte-epiphyte and benthic components had opposing responses 6 days after herbicide treatment. The mean macrophyte-epiphyte GPP decreased to 0.51 mg O_2/ℓ/day, significantly lower than the mean control rate

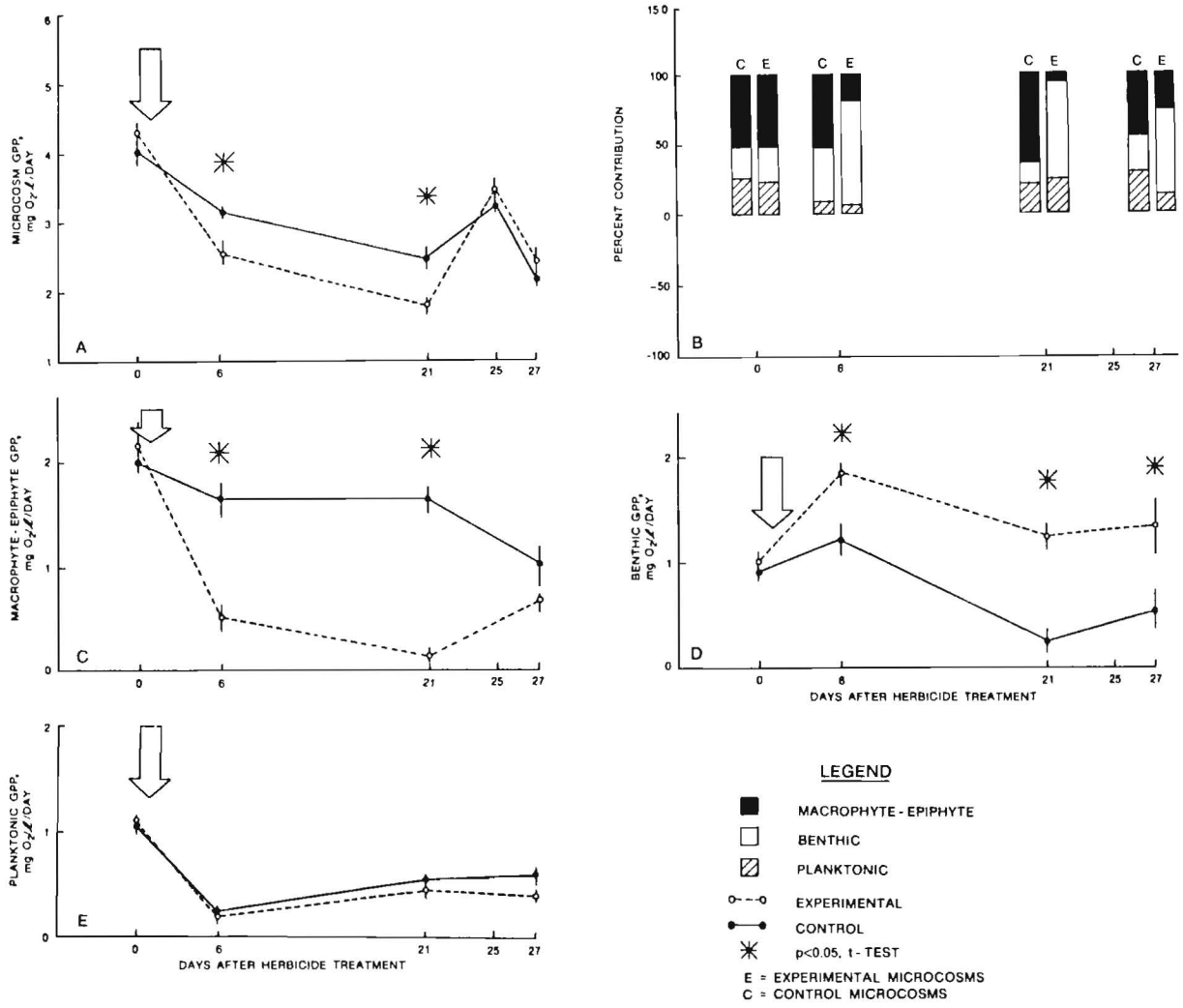


Figure 13. Changes in mean gross primary productivity after herbicide treatment: total microcosm GPP (A), percent productivity contribution by each component (B), macrophyte-epiphyte GPP (C), benthic GPP (D), and planktonic GPP (E). Bars represent standard errors and asterisks indicate significant differences ($p < 0.05$, t-test)

of 1.65 mg O_2/ℓ/day ($p < 0.05$, t-test), while the mean benthic GPP increased from 1.02 mg O_2/ℓ/day, one day before herbicide application, to 1.84 mg O_2/ℓ/day on day 6. Although the mean benthic GPP rate also increased in the control microcosms, it was significantly lower than the treated microcosm rates ($p < 0.05$, t-test).

A change in the component dominating microcosm GPP occurred in the experimental microcosms on day 6. While the macrophyte-epiphyte component was responsible for a mean of 53 percent of the production in the control set,

72 percent of the mean microcosm GPP was contributed by the benthic component in the experimental microcosms and the macrophyte-epiphyte component contributed only 20 percent.

Twenty-one days after herbicide treatment, the mean microcosm GPP continued to decrease in the control and experimental microcosms, the experimental mean GPP remaining significantly lower ($p < 0.05$, t-test). Mean macrophyte-epiphyte GPP did not change from day 6 in the control microcosms, but decreased further in the experimental set to a mean 0.11 mg O_2/ℓ/day. The mean benthic component rate decreased from day 6 but maintained a higher rate than the mean control benthic GPP ($p < 0.05$, t-test).

The macrophyte-epiphyte component continued to dominate microcosm GPP in the control microcosms, whereas the benthic component remained the dominant producer in the experimental microcosms. The mean percent macrophyte-epiphyte GPP contribution increased from 53 to 67 percent in the control set, but the mean percent benthic input declined slightly to 69 from 72 percent on day 6 in experimental systems. The macrophyte-epiphyte component contributed only 11 percent to the microcosm GPP in the experimental units.

On day 25, microcosm GPP had recovered to control levels. In both experimental and control units, mean microcosm GPP increased. Component measurements were not taken on this day.

Twenty-seven days after herbicide treatment, the benthic component remained the dominant producer in the experimental systems; however, the next dominant producer was the macrophyte-epiphyte component. Since the macrophyte tissue was deteriorating on the sediment surface, and no significant regrowth had occurred, this metabolism was by epiphytes and wall algae. The macrophyte-epiphyte component continued to dominate production in the control microcosms.

Mean net primary productivity followed a similar pattern to GPP after herbicide treatment (Figure 14). In the control microcosms, microcosm NPP fluctuated between a mean of 1.06 mg O_2/ℓ/day and 1.76 mg O_2/ℓ/day. While both experimental and control sets had similar mean microcosm NPP rates one day before herbicide treatment, endothall caused total NPP to decline to 0.13 mg O_2/ℓ/day in the experimental systems on day 6. On day 25, microcosm NPP rebounded to a mean of 1.83 mg O_2/ℓ/day, which was similar to the control rate of 1.74 mg O_2/ℓ/day.

The endothall caused a decrease in the macrophyte-epiphyte NPP rate to a mean of -0.06 mg O_2/ℓ/day by day 6, and on day 21, the mean rate was

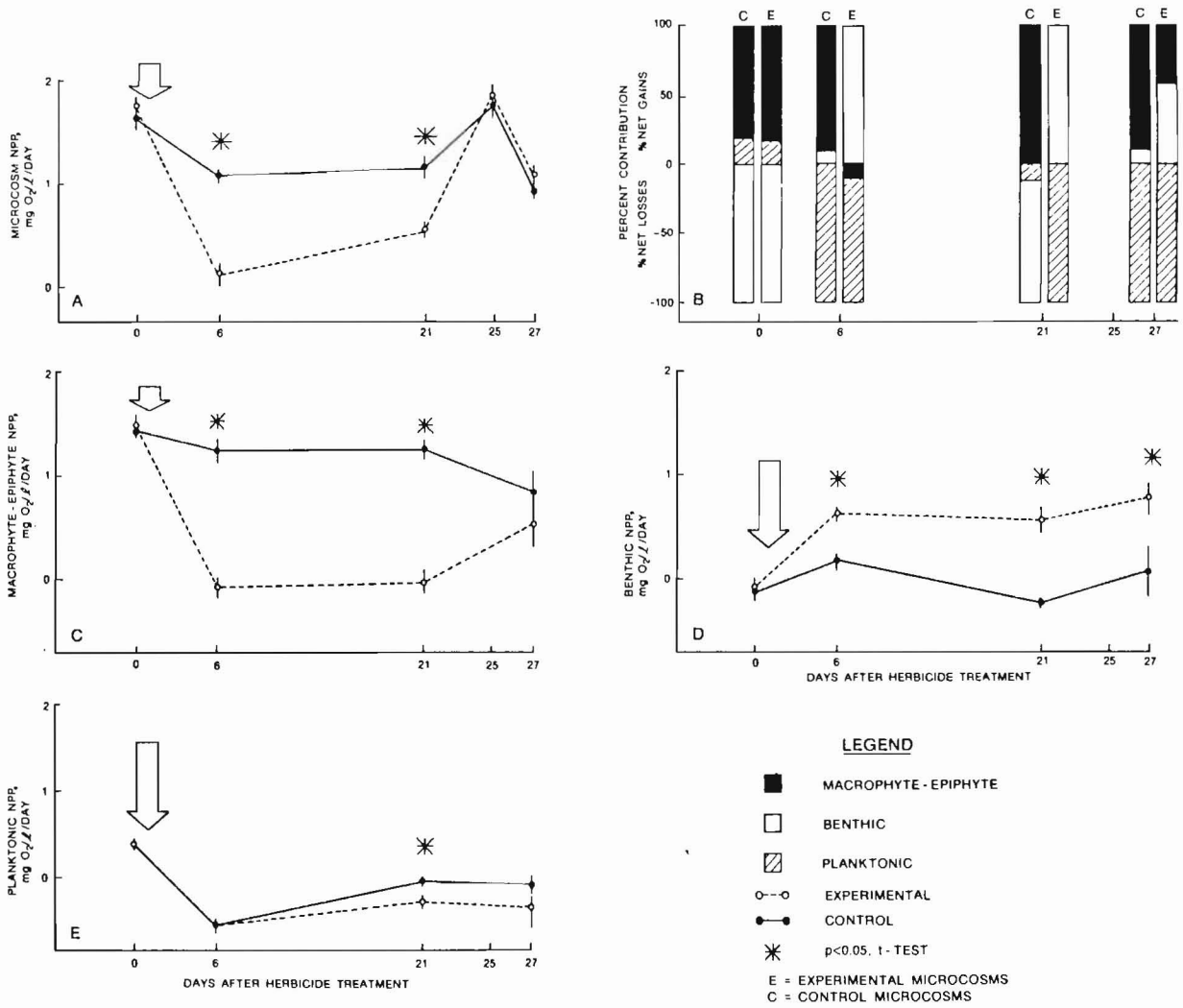


Figure 14. Changes in mean net primary productivity: total microcosm NPP (A), percent productivity contribution by each component (B), macrophyte-epiphyte NPP (C), benthic NPP (D), and planktonic NPP (E). Bars represent standard errors and asterisks indicate significant differences ($p < 0.05$, t-test)

0 mg O_2/ℓ/day, while in the control set, the macrophyte-epiphyte component maintained a mean rate of 1.26 mg O_2/ℓ/day. By day 27, net primary productivity was apparent in this component of the experimental systems, the increase probably being caused by the epiphytic algae.

While macrophyte-epiphyte NPP declined initially after the herbicide perturbation, mean benthic NPP increased in the experimental units. By day 27, mean benthic NPP had increased further to 0.78 mg O_2/ℓ/day while in the control units, benthic NPP remained low, fluctuating around 0 mg O_2/ℓ/day.

The mean planktonic NPP was similar in both sets throughout the experiment. Before herbicide application, small net photosynthetic gains were recorded, but by day 6 both sets had similar mean planktonic net loss rates of -0.45 mg O_2/ℓ/day. The rates then fluctuated slightly, remaining low by day 27.

As shown in Figure 15, mean microcosm 24-hr net primary productivity rates were negative in both systems prior to herbicide treatment. In the control systems, mean 24-hr NPP rates steadily increased to a positive 0.47 mg O_2/ℓ/day by day 25, then dropped to -0.35 mg O_2/ℓ/day on day 27. The

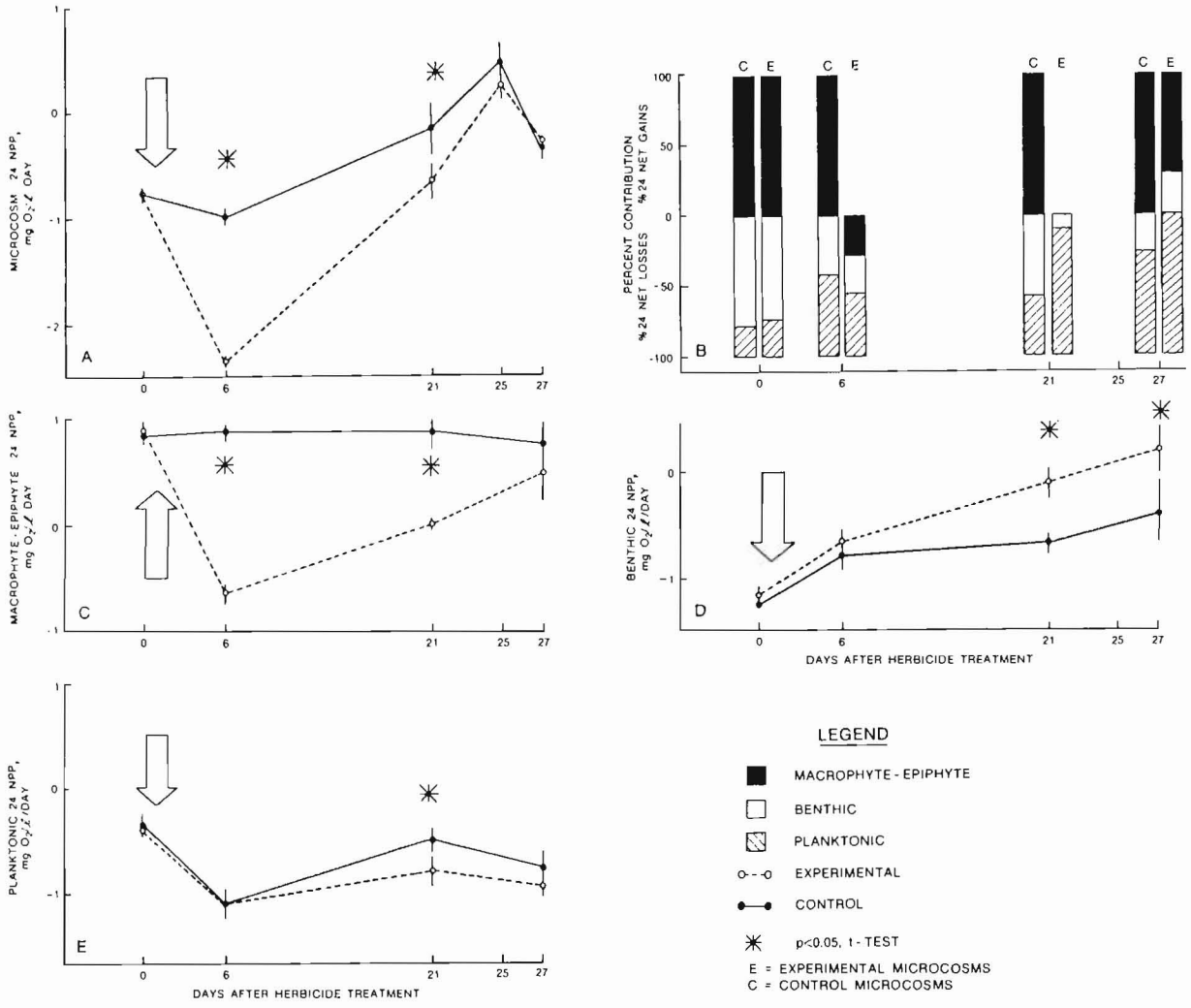


Figure 15. Changes in mean 24-hr net primary productivity (24 NPP): total microcosm 24-hr NPP (A), percent productivity contribution by each component (B), macrophyte-epiphyte 24-hr NPP (C), benthic 24-hr NPP (D), and planktonic 24-hr NPP (E). Bars represent standard errors and asterisks indicate significant differences ($p < 0.05$, t-test)

herbicide caused a greater mean net carbon loss from the experimental systems of -2.35 mg O_2/ℓ/day on day 6, but by day 25, the mean 24-hr NPP rebounded to a positive 0.25 mg O_2/ℓ/day, similar to the control rate.

Before herbicide application, 24-hr NPP varied between the three components. The macrophyte-epiphyte component was responsible for net photosynthetic gains to the systems 1 day before herbicide treatment while the highest net losses were from the benthic component.

On day 6 mean 24-hr NPP in the experimental systems dropped from a positive 0.89 mg O_2/ℓ/day to -0.63 mg O_2/ℓ/day in the macrophyte-epiphyte component. The rates recovered by day 27, presumably because of epiphytic production. In the control systems, the mean macrophyte-epiphyte 24-hr NPP remained positive and fluctuated between 0.75 and 0.88 mg O_2/ℓ/day.

While a small benthic net primary productivity gain was recorded in the experimental aquaria on day 6, the mean 24-hr NPP rate was -0.62 mg O_2/ℓ/day. The mean 24-hr NPP rate then increased steadily to 0.22 mg O_2/ℓ/day by day 27. The control microcosms had similar trends but lower values, and remained negative by day 27.

The planktonic 24-hr NPP rates were similar for both experimental and control microcosms throughout the experiment. The rates were always negative, but fluctuated.

The mean microcosm and component respiration rates are depicted in Figure 16. Before treatment, mean microcosm R rates were similar in control and experimental units. The benthic component dominated respiration while the macrophyte-epiphyte and planktonic had similar but smaller contributions.

In the experimental systems, the mean microcosm R remained constant by day 6 of herbicide treatment while the control mean rate steadily declined from day 0. The sustained microcosm R in the experimental systems was caused by slight increases in the mean macrophyte-epiphyte and benthic component R rates. However, these component increases were not statistically different from the control macrophyte-epiphyte and benthic component R rates.

Twenty-one days after herbicide treatment, the experimental microcosm R had declined to control levels. An R value of -0.14 mg O_2/ℓ/day occurred in the macrophyte-epiphyte component because the cylinder rates were slightly higher than total microcosm rates in many cases. There were similar cases in the control units on this day also. Although benthic R was only slightly higher compared to the control rates, this component remained the dominant

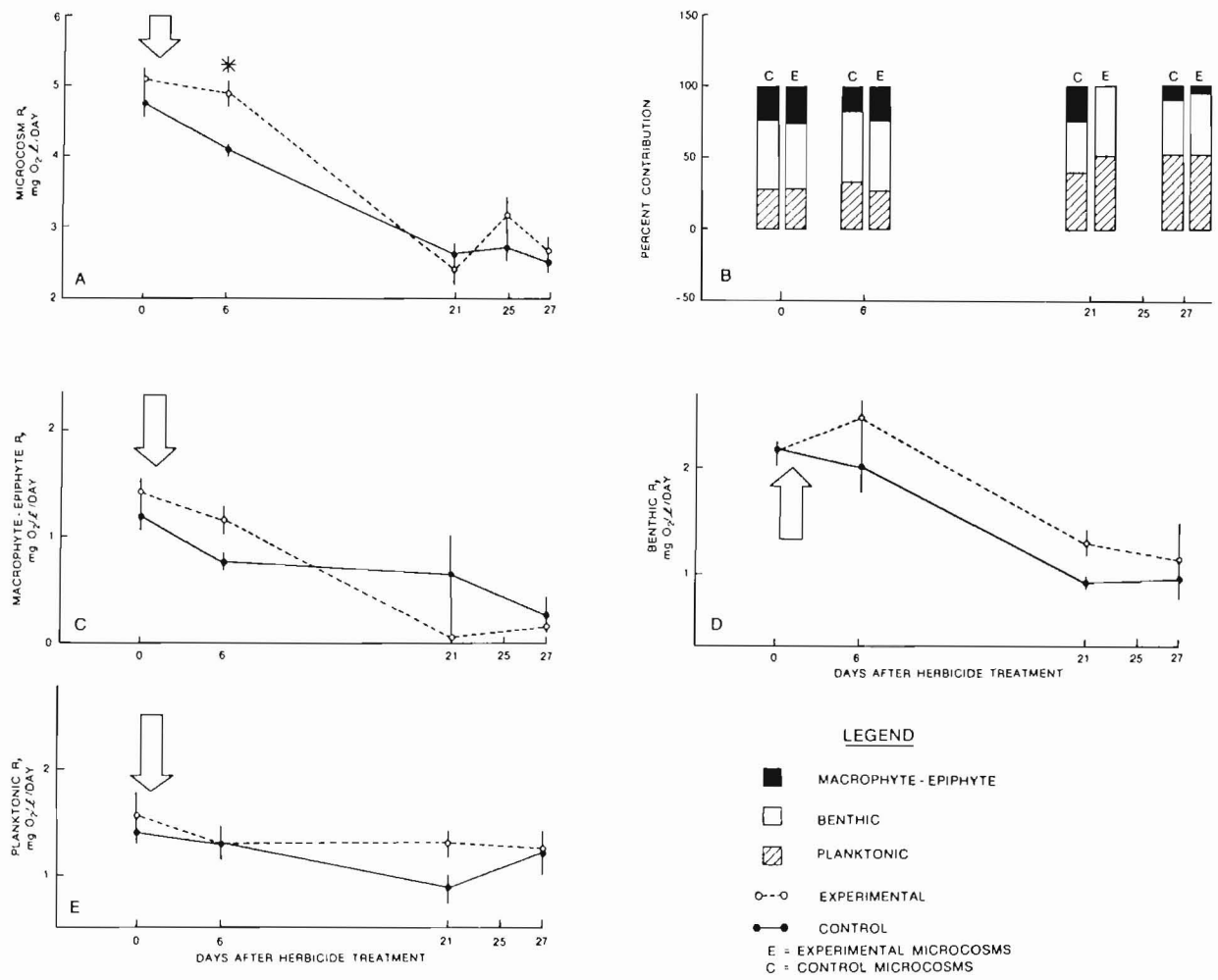


Figure 16. Changes in mean respiration: total microcosm (A), percent respiration contribution by each component (B), macrophyte-epiphyte (C), benthic (D), and planktonic (E). Bars represent standard errors and asterisks indicate significant differences ($p < 0.05$, t-test)

contributor with 85 percent of the total R in the experimental systems.

The gross primary productivity/respiration (P/R) ratio was similar and below 1.0 prior to herbicide treatment in both sets (Figure 17). By day 6, a decline occurred in the experimental systems to 0.51, which then increased steadily to 1.09 by day 25. The mean P/R also increased in the control microcosms because the respiration rate gradually decreased.

D. Summary.

Table 9 summarizes the events that happened in microcosm experiments I and II after herbicide treatment.

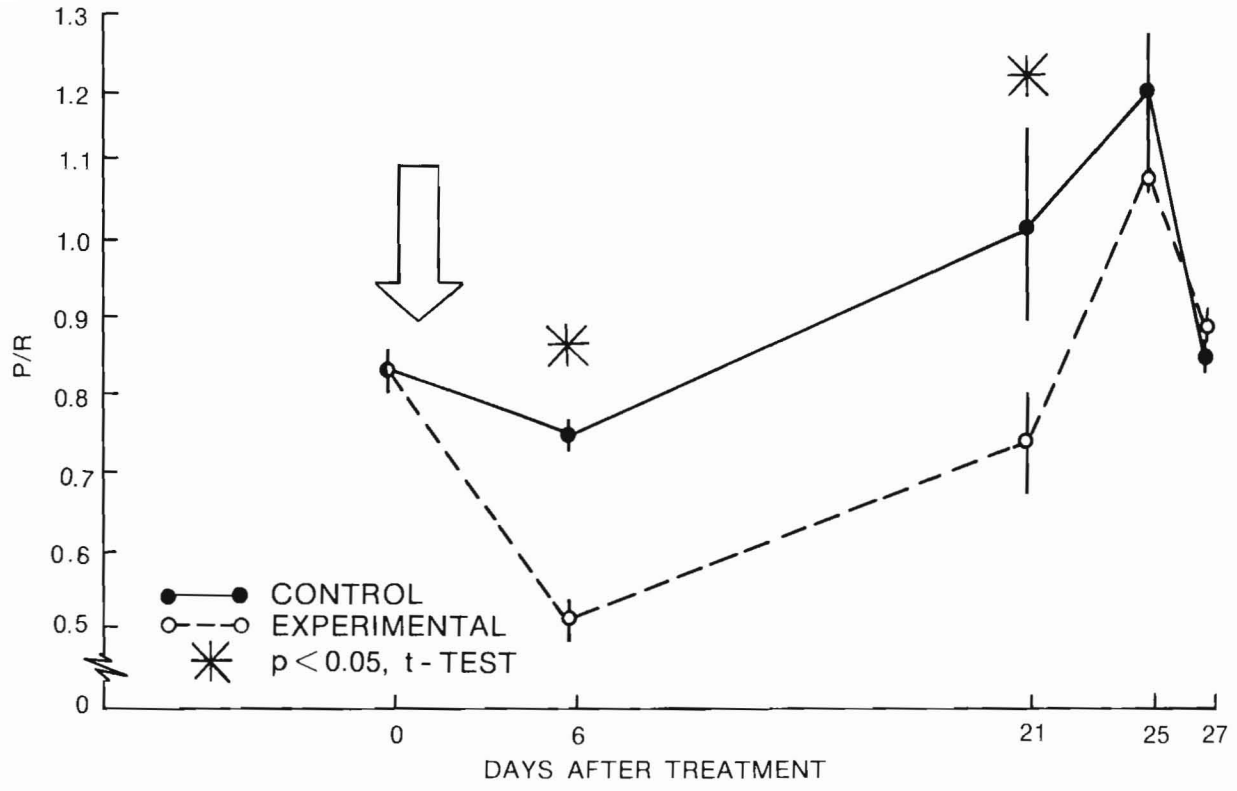


Figure 17. Changes in mean microcosm P/R

Table 9

Changes Occurring After Herbicide Treatment in Microcosm Experiments I and II

Characteristic	Experiment I	Experiment II
Macrophyte standing crop and phosphorus	Higher	Lower than experiment I
Changes in PP	Higher pulse	Lower pulse than experiment I
Changes in SUP	Higher pulse	Lower pulse than experiment I
Changes in SRP	Rapid pulse	No pulse
Water chlorophyll	No increase	No increase
Wall chlorophyll	No increase	No increase
Sediment chlorophyll	Increase	Increase
Microcosm GPP	--	Decreased relative to controls, then rebounded
Microcosm NPP	--	Decreased relative to controls, then rebounded
Microcosm 24-hr NPP	--	Decreased relative to controls, then rebounded
Microcosm R	--	Decreased relative to controls, then fell
Microcosm P/R	--	Decreased relative to controls, then rebounded
Sediment adsorption capacity for KH_2PO_4	Strong	Weak
Observed color of sediment	Light-colored microzone	Light-colored microzone
Dissolved oxygen status	5 ppm	3.5 ppm

IV. DISCUSSION

The impact of herbicide treatment and macrophyte decomposition on the phosphorus concentration in the water needs further clarification. Laboratory studies have shown that phosphorus is rapidly leached from the tissue of herbicide-killed (Nichols and Keeney, 1973) and naturally dying aquatic plants (Jewell, 1971; Carpenter and Adams, 1978; Howard-Williams and Davies, 1979). The leached phosphorus may become adsorbed to the sediment and assimilated by algae, resulting in blooms. This may be particularly true when macrophyte stands contain large amounts of tissue phosphorus at the time of herbicide treatment. For instance, in the laboratory, Rho and Gunner (1978) demonstrated that soluble phosphorus, accumulated in sterile lake water from decomposing *Myriophyllum heterophyllum*, enhanced the growth of various inoculated algal monocultures. However, Nichols and Keeney (1973) showed that soluble phosphorus increased less in the water during plant decomposition in the presence of oxidized sediment. Field studies have recorded chlorophyll (Fish, 1966; Newbold, 1975; Brooker, 1976), algal productivity (Walsh et al., 1971), and community productivity (Brooker and Edwards, 1973) increases after herbicide treatment, but these experimenters did not observe an increase in soluble reactive phosphorus unless the aquatic system became deoxygenated. This suggested that leached phosphorus may not be immediately available to algae because the sediment may act as a sink in the oxidized state for phosphorus (Mortimer, 1971).

None of the field studies investigated the rapid phosphorus leaching process of plant decomposition, which may cause an SRP increase immediately after the plants are killed. A study of this type could provide evidence that phosphorus from herbicide-killed plants is directly available for algal assimilation and growth. The objective of the present study was to examine the phosphorus status of the water immediately after killing *Potamogeton crispus* with endothall in laboratory microcosms, and to monitor subsequent changes in algal chlorophyll a and community metabolism. In addition, phosphorus changes were monitored in the field after an endothall application to a community of *Potamogeton crispus*.

There were several shortcomings to the microcosm experiment. The oxygen changes used to determine community metabolism were small throughout the entire study, and it was also evident from light/dark bottle changes and wall

and water chlorophyll a data that the planktonic component was negligible and probably not a realistic representation of field conditions. Two possible factors may have caused this: (1) the glass substrate was not favorable for wall colonization, and (2) the initial low SRP concentration in the water may have limited algal growth. The findings of Barko et al. (1977) suggested that some benthic algae were of pelagic origin. With little water mixing and initial low SRP concentrations in the microcosms, algae probably sedimented and inhabited the more phosphorus-rich sediment; hence, this was the primary algal group in the microcosm studies.

These deficiencies might be improved with higher intensity lighting and continuous stirring. Special lighting which simulates more accurately the irradiance of the sun may increase the productivity of all photosynthetic components. Most plankton rely on water mixing to maintain flotation. Maintaining a constant current which would not disturb the sediment might improve this problem. With stirring, oxygen gradients would be eliminated and one or several diffusion coefficients could be used, depending on the mixing time, to calculate community metabolism more accurately. Diffusion was probably the most critical element in the metabolic calculations, and its precise determination is essential for any diel study.

In both microcosm studies, phosphorus was assumed to be the element limiting algal productivity, but no attempt was made to confirm this with, for instance, an algal assay. Determining the limiting nutrient would have greatly substantiated the results of this study. However, it is widely accepted that phosphorus is an important limiting nutrient to algal growth and lake eutrophication (see Powers et al., 1972).

A. Phosphorus.

Soluble reactive phosphorus accumulation in the water varied between the three experiments. In the field study, an SRP pulse did not occur until day 6 of the first application date, but SRP accumulated in the water within 3 days in microcosm experiment I. These differences could have been the result of problems in scaling the microcosm experiment with the field study. Differences in the sediment surface to water volume ratio, and the herbicide concentrations between the two systems, probably affected the diffusion of herbicide and its impact on killing the plants. Although the intended concentration of 5.0 ppm

of endothall in the field study was higher than the concentrations of 2.0 ppm used for both microcosm studies, the surface to volume ratio was three times higher in the microcosms. The microcosms were not physically scaled to the lagoon, and it is likely that the endothall spread more rapidly in the smaller volume microcosms than in the lagoon, having a faster effect on the plants.

However, the field and the microcosm experiment I studies showed that SRP can accumulate in the water for a short period of time after herbicide treatment. The SRP pulse was presumably the result of leaching from decaying *Potamogeton crispus*. That SRP is a product of plant decomposition is supported by the Sephadex fractionation study of leachate from dried macrophytes done by Carpenter and Adams (1978). They found that phosphorus leached from *Myriophyllum spicatum* was entirely in the fine particulate and orthophosphate forms. However, the phosphorus contribution of the sediment may have also been important in each study. Mortimer (1971) and Hargrave (1972) have shown that an Eh change affecting phosphorus at the sediment-water interface occurs only when the oxygen is less than 1 ppm. In the present study, the oxygen concentration was not determined at the interface, and oxygen depletion could have occurred in this zone, particularly in the lagoon. However, oxygen never descended below 3 ppm, 0.5 m above the sediment surface in the lagoon, and remained higher than 4 ppm, 125 mm above the sediment surface in both microcosm studies.

Other studies following oxygen and phosphorus changes in herbicide-treated ponds and aquaria have found SRP increases occurring only during oxygen depletion of the water and sediment Eh reduction (Walker, 1963; Simsiman et al., 1972), implying that SRP pulses caused by plant decomposition could be the result of the reducing environment, phosphorus not being immediately adsorbed to the sediment. On the other hand, these experimenters did not follow daily changes in SRP after herbicide treatment. The SRP pulse in microcosm experiment I was very distinct and declined rapidly after the fourth day. Since the microcosms were static, phosphorus movement out of the sediment would probably be through molecular diffusion, which might not account for the rapid pulse. A 1-cm-thick, light-colored microzone above darker sediment was also apparent throughout the decay process of *Potamogeton crispus* in the experiment I microcosm, suggesting that the sediment may have been oxidized, and had the capacity to adsorb SRP. The sediment-adsorbing capacity was high (93 percent of the introduced KH_2PO_4) in both the field study and the

microcosm experiment I. If this were the case, the present results also suggested that if the sediments were oxidized, SRP leachate was still temporarily available for algal assimilation and did not quickly become adsorbed to the sediment.

In contrast to the above studies, no SRP pulse was recorded within the first 2 weeks of plant decomposition in microcosm experiment II. The leached phosphorus could have been taken up by other autotrophs or adsorbed to the sediment. The sediment used in each microcosm experiment came from different locations suggesting that the differences in the SRP responses between the two experiments might be accounted for by differences in the sediment-adsorbing capacity for phosphorus. It was hypothesized that the sediment used in microcosm experiment II was responsible for adsorbing a substantial portion of the phosphorus leached from killed *Potamogeton crispus*, resulting in no pulse, whereas, the microcosm experiment I sediment was thought to adsorb phosphorus less strongly, explaining the SRP pulse recorded. However, the opposite was noted (Table 7). Microcosm experiment I sediment adsorbed phosphorus strongly, but microcosm experiment II sediment did not (31 percent of the introduced KH_2PO_4).

In both microcosm experiments I and II, an increase in the sediment chlorophyll a was observed, suggesting phosphorus uptake by algae. It is apparent that SRP was available for sediment algal assimilation after herbicide treatment in microcosm experiment I. Furthermore, the absence of an SRP pulse in experiment II suggested that a rapid transfer of phosphorus from one autotrophic component to another occurred, resulting in the assimilation of a portion of the leached phosphorus by the sediment algae and a consequent chlorophyll a increase.

Laboratory experiments employing ^{32}P have discerned the movement of orthophosphate to algae or bacteria and the sediment. For instance, Hayes and Phillips (1958) found phosphorus accumulation in the mud was decreased by the presence of bacteria in the water. Moreover, Sebetich (1975) and Whitaker (1975) demonstrated that ^{32}P added to microcosms was immediately taken up by algae, the sediment accumulating the majority of the phosphorus later. The ^{32}P studies suggested that no SRP pulse occurred in microcosm experiment II because the sediment algae quickly accumulated the rapidly leached phosphorus. From the findings of experiment I, it is presumed that the sediment algae assimilated phosphorus lost from herbicide-killed *Potamogeton crispus* as well,

which resulted in a chlorophyll increase. However, the macrophyte tissue phosphorus concentration of 287.0 mg/m^2 present in the herbicide-treated microcosm of experiment I was much higher than the mean concentration of 95.6 mg/m^2 estimated in the experiment II microcosms. This was the result of the greater *Potamogeton crispus* standing crop in the first experiment. Excessive phosphorus leaching from the macrophytes in experiment I probably caused the SRP pulse during the early stages of plant decomposition.

Particulate phosphorus also increased during the first 2 weeks of plant decomposition. The pulses were more evident and distinct in the microcosm experiments than the field study, the static conditions of the microcosms possibly magnifying the fluctuations. Particulate phosphorus also appears to be a product of the rapid leaching process, as demonstrated by Carpenter and Adams (1978) in vitro. Water chlorophyll a did not increase, which would suggest that the leached phosphorus was being taken up by the planktonic algae and incorporated into a particulate form. Rather, the PP may have represented fine particulate matter leached from the macrophytes or phosphorus accumulation in bacteria.

B. Community Metabolism.

It appears that before herbicide treatment, the macrophytes made a substantial contribution to the microcosm gross primary productivity, net primary productivity, and 24-hr net primary productivity. That the epiphytic contribution was small is inferred from the significant decrease in the GPP, NPP, and 24-hr NPP rates of the macrophyte-epiphyte component 5 days after the perturbation. Had the epiphytes been more productive, the decrease in these rates might not have been as large. Endothall may have been toxic to the epiphytes also; however, no differences were apparent between the control and experimental wall chlorophyll a concentrations after the perturbation.

The benthic component made the smallest GPP contribution and had net carbon losses before herbicide treatment. As a result, microcosm P/R ratios were below 1.0 in both sets. A high organic content in the sediment may have stimulated benthic respiration, although this was not determined. The low light intensity was also probably a factor because all components had low productivity rates throughout the entire experiment.

Herbicide treatment caused a reduction in the microcosm productivity

rates and an increase in the microcosm total respiration rate relative to the controls. Thus, the herbicide represented a stress to community productivity but a subsidy to total respiration, using the formal definitions of Odum et al. (1979), as the variables of microcosm metabolism exhibited opposite deflection patterns immediately after the herbicide perturbation before recovering to control levels.

The extent of the impact of *Potamogeton crispus* loss was not reflected to a large degree in the microcosm GPP on day 6. Since microcosm GPP was the summation of R and NPP, the stimulation of respiration offset the deflated NPP values, thereby buffering the effect of endothall on GPP. Benthic GPP increased, further offsetting the impact of the *Potamogeton crispus* loss.

The destruction of *Potamogeton crispus* was reflected by a decrease in the microcosm NPP and 24-hr NPP rates. Endothall toxicity probably indirectly impaired the photosynthetic activities of the macrophytes through cell membrane disruption as no net productivity was observed 5 days after herbicide treatment in the macrophyte-epiphyte component. The 24-hr NPP rate indicated a net carbon loss to this component which corresponded to the slightly higher macrophyte-epiphyte respiration rate when compared to the control set.

Other workers have found that microcosm metabolism responded to a perturbation by a decrease in both GPP and R (Beyers, 1963; Copeland, 1965). Beyers attributed this phenomenon, occurring in overgrazed microcosms, to the elimination of an autotrophic component which contributed to both productivity and respiration. These results suggest that oxygen decreases in the water after a herbicide perturbation may not be the result of a biological oxygen demand but, rather, the destruction of the photosynthetic and respiration capabilities of a component in the community. Brooker and Edwards (1973) also found that a decrease in both gross primary productivity and respiration occurred immediately after paraquat was added to Barry Reservoir. They noted that the respiration decrease corresponded with the calculated respiration rate of the macrophytes present before herbicide application.

In microcosm experiment II, while microcosm GPP, NPP, and 24-hr NPP decreased with the death of *Potamogeton crispus*, microcosm respiration increased initially before descending to the control values. Although the photosynthetic activities of the macrophytes were destroyed, an abundance of labile organic material in the form of leached dissolved molecules and coarse particulate matter may have become immediately available for microorganism

consumption. Such an organic subsidy should stimulate heterotrophic growth and community respiration. The stress may have disrupted the feedback between microcosm GPP and R, temporarily causing the accelerated output of carbon. The more negative microcosm 24-hr NPP value also suggested increased carbon degradation by heterotrophs.

The diel oxygen changes were small in the experimental microcosms 5 days after herbicide treatment. The possibility that the plants continued to respire after herbicide treatment (Newbold, 1975) does not explain the increased microcosm R when compared with the mean control rate. The observed increase does correspond to the time bacterial populations increase during plant decomposition (Kistritz, 1978). The occurrence of a soluble unreactive phosphorus pulse 7 days after herbicide application suggested that bacteria were present and may have been responsible for the high rate of microcosm respiration. Soluble unreactive phosphorus is usually a measurement of organic phosphorus forms. Organic phosphorus is not a major product of the early stages of plant decomposition (Carpenter and Adams, 1978), but rather bacteria convert orthophosphate to organic forms (Barsdate et al., 1974). In other studies, bacterial populations inhabiting the mud and water, and epiphytic bacteria have increased in number after aquatic macrophytes were killed with paraquat (Fry et al., 1973; Ramsay and Fry, 1976). Rho and Gunner (1978) also noted rapid bacterial growth following decomposition of *Myriophyllum heterophyllum* in the laboratory. These authors suggested that the bacterial increases were caused by the utilization of leached organic carbon and nutrients from the dead plants. Thus, a soluble unreactive phosphorus pulse might be an indicator of the presence of bacteria, which would explain the higher microcosm R and more negative 24-hr NPP recorded shortly after herbicide treatment. The recovery to control values presumably followed the depletion of labile organic matter.

The microcosm respiration increase deflection relative to the controls was not large. This was probably due to the lower macrophyte standing crop in each experiment II microcosm compared to experiment I. However, herbicide is usually applied when macrophytic growth becomes excessive. Treatment may stimulate respiration causing rapid oxygen depletion which can have devastating aesthetic and financial effects on the recreational use of a lake. Owens and Maris never published, but their results were cited in Brooker and Edwards (1973) who described their findings. They found that in a herbicide-treated pond, plant death resulted in total deoxygenation in 4 days. Jewell (1971)

reported that aquatic weed decomposition can be more harmful to the water quality than decaying phytoplankton because a larger percent of the tissue is potentially decomposable and decay is faster.

The ecological significance of this study is that the metabolic recovery depended on a shift from macrophyte to algal dominance in light energy fixation. Microcosm GPP, NPP, and 24-hr NPP rebounded after the herbicide perturbation because benthic, and later epiphytic, metabolism was stimulated. Walsh et al. (1971) also noted that phytoplankton productivity increased significantly after the destruction of macrophytes. Furthermore, the recovery of community metabolism and increase in benthic chlorophyll a were apparently associated with the movement of phosphorus from *Potamogeton crispus* to the epipelic algae. Although not shown in this study, phosphorus was implied to be the nutrient which limited algal growth. The recovery of community productivity to control rates was probably related to the rapid reproductive ability of the algae.

The recovery of microcosm metabolism indirectly supports the hypothesis reported by Menzel et al. (1970) and Rhyther (1970). Menzel et al. (1970) found that chlorinated hydrocarbons did not have the same effect on different diatom species, and concluded that poisons would more likely affect the dominance of species rather than destroy the entire functioning of a community. Resistant species would adapt to the stressful situation, becoming opportunistic invaders. Rhyther expanded on this idea and addressed the problem concerning the fate of the global oxygen balance with continued toxic waste dumping into the ocean. He also asserted that a stress imposed on a system would result in a change in the dominance of species, and implied a shift in the flow of energy to resistant autotrophs. Copeland (1965) reported similar findings from light-stressed marine microcosms. A reduction in light intensity caused a shift from turtlegrass to blue-green algal metabolic dominance. The blue-green algae had a competitive advantage and were able to adapt to the light stress.

In this study the herbicide perturbation stimulated further succession as productivity became dominated by a bloom of quickly growing, short-lived algae. *Potamogeton crispus*, *Chara* spp., and algae were responsible for microcosm productivity in the control microcosms. Odum (1969) has pointed out that ecosystem development can be pushed back by allogenic forces to a less mature or "bloom" state by, for instance, the addition of phosphorus or of other

nutrients which subsidize productivity. Endothall disrupted the development and nutrient cycling in the microcosms by destroying *Potamogeton crispus*, which caused the accelerated release of phosphorus from the tissue, thereby promoting a sediment algal bloom.

While microcosm metabolism did recover, the herbicide stress imposed on the macrophyte-epiphyte component caused significant deflections in total microcosm metabolism, and oxygen concentrations decreased temporarily. This response was probably one of several possible responses that could occur in a system, depending on the importance of the contribution made to the community metabolism by the stressed component. For instance, stressing the algal component may not have caused as large a deflection in community productivity. With the use of herbicides, management plans must consider that stressing a component important to the metabolism of a community can lead to a temporary decrease in the water quality followed by the invasion of opportunistic species which may then become a further nuisance. Plans must weigh the economic and recreational importance of macrophyte control against the possibility of having other pest species and fish kills on their hands.

The use of microcosms to study the ecological effects of an herbicide had several advantages. The most important point was the ability to control and manipulate certain factors which could not be feasibly done in a field study. The amount of *Potamogeton crispus* biomass could be controlled to a certain extent using cuttings. Although the plants grew after transplantation, runners were rarely produced. In future studies with aquatic macrophyte communities, the ecological effects of an herbicide on different plant biomass levels might be investigated using microcosms. Partial answers to the questions of oxygen demand and the extent of phosphorus release following herbicide treatment could be used to predict these events in the field. The importance of the sediment as a source or a sink for phosphorus could be assessed with microcosms. Different plant biomass levels could be combined with sediments of different phosphorus-adsorbing capacities. Questions could be asked about the metabolic and biomass responses of algae to herbicide treatment when the sediment has a high phosphorus-adsorbing capacity.

The microcosms are replicable and easy to monitor, allowing the investigator to examine a range of possibilities. Studies on the effect of different herbicides on microcosm replicates could be performed. Several parameters can be sampled easily, more accurately, and at closer time intervals. Microcosms

could be scaled to study the influence of volume and mixing on herbicide diffusion, activity on plants, and phosphorus accumulation in the water. The use of microcosms for control and manipulation may enhance field study results and direct the experimenter to solutions of field problems.

Excessive aquatic weed growth is a serious problem which demands immediate attention for recreational, aesthetic, and commercial reasons; herbicide application presently appears to be a feasible method for macrophyte control. However, the indirect effects of herbicide treatment act to both stress and subsidize community productivity and respiration, respectively, which can lead to decreases in oxygen and other water quality parameters. In general, it appears that community metabolism recovers from a herbicide perturbation. However, this recovery depends on a temporary shift in the flow of energy to nontarget species, i.e. algae. The shift, in part, is caused by the scavenging of nutrients, particularly phosphorus, which formerly limited their growth. From a lake management standpoint, the stimulation of resistant species growth can cause equally dissatisfying problems.

Some inferences from the present study can be applied to lake management plans using herbicides to control aquatic weed problems. Endothall treatment can indirectly cause temporary phosphorus increases in the water because the element is leached from the plant material during senescence. The magnitude of phosphorus increases probably depends, in part, on the standing crop of macrophyte tissue phosphorus at the time of treatment, and the assimilation efficiencies of the nontarget species and sediment for the leached macrophyte phosphorus during macrophyte death. Caution should be used with the evidence from the field study because the rapidity of the effects of endothall were not determined. The diffusion of endothall may have been slower in the field, retarding its toxic effects.

Management plans should consider the life cycle of the nuisance plants. *Potamogeton crispus* grows rapidly in the spring and fall but has a short peak growing season of approximately 3 months in northeastern Ohio. Treatment of this species at peak densities could stimulate the oxygen demand and aggravate the water quality more so than its natural dieback. Windy spring days could mix the leached phosphorus into the open water, stimulating algal blooms. If treatment is necessary, small dosages at intervals during the early stages of the *Potamogeton crispus* growing season might curtail the possibly devastating effects of a massive diedown. Other means of macrophyte control that do not

have toxic effects, such as biological control with fish or harvesting techniques, might be a safer alternative until more research is done on the biochemical degradation of endothall. The chemical may become tied up in the anoxic deeper sediment layers of the littoral zone for longer periods of time. Although the safety factor is believed to be high for humans, its long-term effects on health are not known.

Further questions in the field of herbicides should be pursued to gain a better understanding of the indirect effects on other biota and of the fate of phosphorus after macrophyte destruction. For example, the role of the sediment in adsorbing phosphorus lost from decomposing macrophytes should be studied. Of the three major algal communities, epiphytic, epipelic, and planktonic, which is most stimulated by herbicide treatment? Can the extent of oxygen demand and appearance of algal blooms be predicted for use as a management tool? Finally, the rate of respiration and gross primary productivity should be closely investigated in conjunction with bacterial responses after herbicide treatment for macrophyte communities of differing biomass.

V. SUMMARY

1. Research was directed toward assessing the effects of endothall on (a) phosphorus leaching from *Potamogeton crispus*, and (b) the chlorophyll a and metabolic responses of autotrophic components.

2. A field study was conducted to observe changes in phosphorus species (particulate phosphorus, soluable unreactive phosphorus, and soluable reactive phosphorus) and oxygen concentrations after the application of Hydrothol 47 and Aquathol K (5.0 ppm). Further investigations with endothall were conducted with microcosms. A pilot microcosm study (microcosm experiment I) was performed to measure changes in phosphorus, oxygen, and algal chlorophyll a after the application of Hydrothol 47 (2.0 ppm). Changes in phosphorus concentrations and the metabolism of three autotrophic components, macrophyte-epiphyte, benthic, and planktonic, were assessed in replicate microcosms after treatment with 2.0 ppm Hydrothol 47 (microcosm experiment II).

3. The application of endothall to *Potamogeton crispus* communities caused increases in the SRP concentration of the water for short periods of time in the field and microcosm experiment I. It appeared that phosphorus leached from macrophyte tissue was responsible for these increases. However, the input of phosphorus from the sediment was not investigated.

4. In microcosm experiment I, the elevated SRP concentration appeared to be directly available for algal uptake because epipelic chlorophyll a increases were observed after herbicide application.

5. An SRP increase was not observed in microcosm experiment II. However, benthic chlorophyll a increased after herbicide treatment, suggesting that phosphorus leached from *Potamogeton crispus* was immmediately taken up for growth.

6. The apparent differences in the SRP responses between the two microcosm experiments may be attributed to differences in the standing crop of macrophyte phosphorus. The macrophyte tissue phosphorus concentration was three times higher in microcosm experiment I. Therefore, the occurrence of elevated SRP concentrations in the water may depend on the macrophyte standing crop of phosphorus at the time of treatment, and phosphorus assimilation efficiencies by algae.

7. Particulate phosphorus also increased in the water after herbicide treatment and appeared to be material leached from *Potamogeton crispus*.

8. Soluble unreactive phosphorus increased in the water several days after herbicide treatment in microcosms, possibly as a product of bacterial decomposition.

9. Endothall caused an initial significant decrease in mean microcosm gross primary productivity, net primary productivity, and 24-hr net primary productivity, but an increase in mean microcosm respiration rates relative to controls. The opposite deflection patterns observed in these metabolic rates caused an oxygen decrease in the water.

10. The endothall stress directly affected the mean gross primary productivity, net primary productivity, and 24-hr net primary productivity rates of the macrophyte-epiphyte component in herbicide-treated microcosms, causing the significant decrease in total microcosm productivity rates. Stressing an autotrophic component which makes a substantial contribution to the metabolism of a system can therefore cause temporary decreases in water quality and indirectly affect other biota through oxygen decreases.

11. The mean primary productivity/respiration ratio dropped after the perturbation, indicating that treated microcosms were temporarily more heterotrophic than control microcosms. The decrease suggested that labile organic material which leached from senescing *Potamogeton crispus* sustained heterotrophic metabolism.

12. Microcosm metabolism recovered from herbicide treatment, and this response depended on a shift from *Potamogeton crispus* to benthic algal dominance in photosynthesis and respiration.

13. The shift in photosynthetic dominance appeared to be stimulated by the uptake of leached macrophyte phosphorus by epiphytic and epipelic algae, which were resistant to the herbicide perturbation.

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APPENDIX 1

Values Used to Calculate Plant Standing Crop and Milligrams of Tissue Phosphorus Per Standing Crop (mg P/g SC) for Microcosm Experiments I & II

Leaf No.	ln Leaf No.	Weight g	ln Weight	mg P/g SC	ln P mg/g
		Laboratory Grown Plants			
4	1.3863	0.0032	-5.7446	72.57	4.2845
6	1.7917	0.0034	-5.6839	96.54	4.5699
6	1.7917	0.0042	-5.4726	68.74	4.2303
6	1.7917	0.0027	-5.9145	71.73	4.2729
9	2.1972	0.0035	-5.6549	57.57	4.0530
10	2.3025	0.0199	-3.9170	--	--
10	2.3025	0.0072	-4.9336	83.49	4.4247
10	2.3025	0.0164	-4.1105	66.79	4.2005
15	2.6390	0.0241	-3.7255	65.79	4.1864
17	2.8332	0.0127	-4.3661	52.31	3.9571
18	2.8903	0.0341	-3.3781	49.20	3.8958
19	2.9444	0.0198	-3.9221	48.42	3.8789
21	3.0445	0.0467	-3.0640	27.06	3.2980
26	3.2580	0.0279	-3.5791	37.53	3.6251
		Field Plants			
3	1.0986	0.0032	-5.7446	48.03	3.8718
3	1.0986	0.0027	-5.9145	74.41	4.3095
3	1.0986	0.0030	-5.8091	77.50	4.3502
4	1.3863	0.0050	-5.2983	82.40	4.4115
4	1.3863	0.0040	-5.5214	89.10	4.4897
4	1.3863	0.0069	-4.9762	61.01	4.1110
4	1.3863	0.0042	-5.4726	63.78	4.1554
6	1.7917	0.0105	-4.5563	55.59	4.0180
6	1.7917	0.0075	-4.8928	42.77	3.7558
6	1.7917	0.0119	-4.4312	33.82	3.5210
6	1.7917	0.0084	-4.7795	52.41	3.9591

(Continued)

APPENDIX 1 (Concluded)

Leaf No.	ln Leaf No.	Weight g	ln Weight	mg P/g SC	ln P mg/g
		Field Plants (Continued)			
7	1.9459	0.0169	-4.0804	48.68	3.8852
7	1.9459	0.0126	-4.3740	41.59	3.7278
7	1.9453	0.0188	-3.9738	31.68	3.4557
8	2.0794	0.0272	-3.6045	34.55	3.5424
8	2.0794	0.0147	-4.2199	46.71	3.8439
8	2.0794	0.0131	-4.3351	30.67	3.4233
8	2.0794	0.0100	-4.6051	25.85	3.4232
9	2.1972	0.0224	-3.7987	17.94	2.8870
9	2.1972	0.0155	-4.1669	57.59	4.0533
9	2.1972	0.0216	-3.8350	40.60	3.7037
13	2.5649	0.0473	-3.0512	21.23	3.0554
14	2.6390	0.0494	-3.0078	19.25	2.9575
15	2.7080	0.0496	-3.0037	22.26	3.1028
16	2.7726	0.0300	-3.5065	26.58	3.2801
18	2.8903	0.0775	-2.5574	16.16	2.7825
19	2.9444	0.1036	-2.2672	15.93	2.7682
36	3.5835	0.3480	-1.0555	5.49	1.7029
47	3.8501	0.3140	-1.1583	19.03	2.9460
87	4.4659	0.6583	-0.4181	1.62	0.4861

APPENDIX 2

Calculated Total Standing Crops and Total mg Tissue Phosphorus for Microcosm Experiments I & II

Date*	Standing Crop	Tissue P
	Experiment I	
Oct. 15		
E	1.0655	32.9660
C	1.2123	35.3178
Oct. 30		
E	2.3973	59.3746
C	1.6559	46.0191
Nov. 20		
E	2.4013	53.3873
C	3.0864	67.1326
	Experiment II	
April 9		
1C	0.2159	7.6263
2E	0.1894	6.5225
3C	0.1569	5.8237
4E	0.2115	7.1688
5C	0.1875	7.0610
6E	0.1741	6.3411
7E	0.2147	7.3795
8C	0.2116	7.4107
9C	0.2478	8.4207
10E	0.2081	7.0871
11E	0.2159	7.2679
12C	0.1884	7.3116
April 15		
1C	0.3919	12.1095
2E	0.3735	11.7305
3C	0.2847	7.9659
4E	0.4211	11.8472
5C	0.3771	11.0500
6E	0.3784	9.8248

(Continued)

* E = experimental; C = control.

APPENDIX 2 (Concluded)

Date	Standing Crop	Tissue P
	Experiment II (Continued)	
7E	0.4023	11.6878
8C	0.3529	10.2816
9C	0.4344	12.9803
10E	0.2967	8.2955
11E	0.3842	11.2937
12C	0.3092	9.5010
April 27		
1C	0.7087	19.2000
2E	0.6718	17.7789
3C	0.4571	12.6588
4E	0.7309	19.6584
5C	0.5911	15.0598
6E	0.6278	15.4324
7E	0.7322	19.2603
8C	0.5755	16.1968
9C	0.6598	17.3113
10E	0.6119	14.6946
11E	0.6350	16.5683
12C	0.6219	16.2914

APPENDIX 3

Total Phosphorus (TP), Soluble Phosphorus (SP), Soluble Reactive Phosphorus (SRP), Particulate Phosphorus (PP), Soluble Unreactive Phosphorus (SUP), and Oxygen in Experimental (E) and Control (C) Areas - Field Study

Date	TP, µg/ℓ		SP, µg/ℓ		SRP, µg/ℓ		PP, µg/ℓ		SUP, µg/ℓ		Oxygen, ppm	
	E	C	E	C	E	C	E	C	E	C	E	C
5/25	31.4	31.4	10.5	12.2	3.5	0.0	20.9	19.2	7.0	12.2	9.1	7.3
5/28	29.7	33.2	20.9	10.5	0.0	0.0	8.8	22.7	20.9	10.5	15.5	9.7
6/1	29.7	31.4	26.2	13.9	1.7	7.0	3.5	17.5	24.5	6.9	4.2	8.4
Herbicide Treatment - 6/3												
6/3	50.6	40.2	33.2	19.2	1.7	1.7	17.4	21.0	31.5	17.5	--	--
6/6	50.6	34.9	22.7	19.2	0.0	1.7	27.9	15.7	22.7	17.5	2.6	4.9
6/8	64.6	36.7	36.7	13.9	12.2	5.2	27.9	22.8	24.5	8.7	--	--
Herbicide Treatment - 6/9												
6/9	68.1	38.4	45.4	19.2	22.7	5.2	22.7	19.2	22.7	14.0	4.9	6.4
6/10	69.8	34.9	47.2	17.5	14.0	3.5	22.6	17.4	33.2	14.0	3.6	7.6
6/11	50.6	34.9	41.9	17.5	17.5	3.5	8.7	17.4	24.4	14.0	4.4	8.4
6/12	64.6	36.7	34.9	15.7	17.5	3.5	29.7	21.6	17.4	12.2	5.5	8.5
6/13	68.1	36.7	45.4	19.2	29.6	12.2	22.7	7.1	15.8	7.0	--	--
6/14	57.6	36.7	36.6	19.2	12.2	3.5	21.0	17.5	24.4	15.7	3.4	4.7
6/15	38.4	40.2	20.9	19.2	8.7	7.0	17.5	21.0	12.2	12.2	--	--

APPENDIX 4

Total Phosphorus (TP), Soluble Phosphorus (SP), Soluble Reactive Phosphorus (SRP), Particulate Phosphorus (PP), Soluble Unreactive Phosphorus (SUP), and Oxygen in Microcosm Experiment I

Date	TP, µg/ℓ		SP, µg/ℓ		SRP, µg/ℓ		PP, µg/ℓ		SUP, µg/ℓ		Oxygen, ppm	
	E	C	E	C	E	C	E	C	E	C	E	C
11/11	15.9	15.0	5.6	5.6	0.0	0.75	10.3	9.4	5.4	4.9	7.7	9.9
11/18	16.2	16.2	4.9	4.9	0.0	0.0	11.3	11.3	4.9	4.9	7.6	9.0
Herbicide Treatment - 11/22												
11/22	21.6	21.6	13.2	9.4	0.37	0.37	8.5	12.2	12.8	9.0	8.2	9.2
11/23	32.9	23.5	7.5	5.6	0.56	0.0	32.2	17.9	6.9	5.6	7.8	8.6
11/23	26.3	20.7	15.9	8.5	2.8	0.0	10.3	12.2	13.2	7.5	7.2	8.6
11/24	53.6	23.5	28.2	7.5	16.0	0.0	25.4	16.0	12.2	7.5	5.8	8.9
11/24	74.3	27.3	34.8	6.6	22.6	0.0	39.5	20.7	12.2	6.6	5.9	8.1
11/25	80.9	22.6	49.9	6.6	38.4	0.37	31.0	16.0	11.5	6.2	5.7	8.4
11/26	79.0	22.6	60.2	9.4	43.8	0.0	18.8	13.2	15.6	9.2	5.2	8.5
11/27	76.2	27.3	59.3	8.5	47.8	0.0	16.9	18.5	11.5	8.5	5.7	8.7
11/28	58.3	15.0	47.0	3.8	37.6	0.37	11.3	11.3	9.4	3.4	5.6	8.1
11/29	82.8	26.3	54.8	3.8	33.5	1.1	28.2	20.7	21.1	2.6	6.2	9.0
11/30	96.0	16.9	50.8	13.2	32.0	0.75	45.2	3.7	18.2	11.3	5.6	8.9
12/1	62.1	24.5	58.3	15.0	24.5	0.75	3.8	7.5	33.9	14.3	5.4	8.9
12/2	43.3	22.6	33.9	5.6	21.8	0.75	9.4	16.9	11.3	4.9	5.4	8.7
12/3	35.7	18.8	26.3	9.4	16.6	0.75	9.4	9.4	9.8	8.6	5.8	8.6
12/4	33.9	20.7	13.2	3.8	11.3	0.0	16.9	13.2	1.8	3.6	5.4	8.8

APPENDIX 5

Total Phosphorus (TP), Soluble Phosphorus (SP), Soluble Reactive Phosphorus (SRP), Particulate Phosphorus (PP), Soluble Unreactive Phosphorus (SUP), and Oxygen in Microcosm Experiment II

Microcosm No.	TP, µg/ℓ	SP, µg/ℓ	SRP, µg/ℓ	PP, µg/ℓ	SUP, µg/ℓ	Oxygen, ppm
			April 17			
1C	19.2	8.7	2.1	10.5	6.6	6.6
2E	17.5	8.7	1.7	8.8	7.0	6.6
3C	19.2	8.4	2.1	10.8	6.3	6.6
4E	19.2	7.3	1.4	11.9	5.9	7.4
5C	17.5	8.0	1.0	9.5	7.0	7.4
6E	17.5	8.7	1.4	8.8	7.3	8.0
7E	17.5	9.1	1.4	8.4	7.7	7.8
8C	19.2	9.1	1.7	10.1	7.4	7.5
9C	17.5	6.6	1.4	10.9	5.2	7.4
10E	17.5	9.4	1.7	8.1	7.7	7.3
11E	17.5	10.1	1.4	7.4	8.7	7.6
12C	19.2	7.3	1.0	11.9	6.3	7.4
			April 25			
1C	13.9	5.9	2.1	8.0	3.8	6.8
2E	13.9	8.4	1.4	5.5	7.0	6.8
3C	19.2	5.5	0.7	13.7	4.8	7.4
4E	15.7	5.5	0.7	10.2	4.8	7.6
5C	15.7	6.2	0.7	9.5	5.5	7.8
6E	15.7	6.6	0.7	9.1	5.9	7.2
7E	13.9	4.9	0.7	9.0	4.2	7.5
8C	15.7	4.9	0.7	10.8	4.2	7.0
9C	15.7	5.2	0.7	10.5	4.5	7.1
10E	15.7	4.5	0.7	11.2	3.8	6.6
11E	15.7	7.0	0.7	8.7	6.3	7.4
12C	13.9	4.5	0.7	9.4	4.8	7.4
			April 29			
1C	17.5	5.9	0.7	11.6	5.2	--
2E	19.2	7.3	2.4	11.9	4.9	--
3C	19.2	6.6	0.7	12.6	5.9	--
4E	20.9	8.0	2.4	12.9	5.6	--
5C	15.7	5.9	0.0	9.8	5.9	--
6E	19.2	8.7	1.7	10.5	7.0	--

(Continued)

APPENDIX 5 (Continued)

Microcosm No.	TP, µg/ℓ	SP, µg/ℓ	SRP, µg/ℓ	PP, µg/ℓ	SUP, µg/ℓ	Oxygen, ppm
			April 29 (Continued)			
7E	20.9	5.9	1.0	15.0	6.9	--
8C	17.5	5.9	0.0	11.6	5.9	--
9C	17.5	6.2	0.0	11.3	6.2	--
10E	19.2	7.3	1.0	11.9	6.3	--
11E	20.9	5.5	0.7	15.4	4.8	--
12C	17.5	5.5	0.7	12.0	4.8	--
			April 30			
1C	17.5	5.9	0.0	11.6	5.9	6.6
2E	33.2	8.0	2.8	25.2	5.2	5.8
3C	19.2	5.9	0.0	13.3	5.9	6.9
4E	33.2	8.0	5.2	25.2	2.8	6.1
5C	15.7	4.5	0.0	11.2	4.5	7.8
6E	29.7	8.0	3.5	21.7	4.5	6.3
7E	33.2	5.5	2.8	27.7	2.7	6.2
8C	17.5	7.3	0.7	10.2	6.6	7.2
9C	19.2	5.5	0.0	13.7	4.8	7.3
10E	29.7	5.2	0.0	24.5	5.2	6.5
11E	33.2	5.2	0.0	28.5	5.2	6.0
12C	19.2	5.5	0.0	13.7	5.5	7.1
			May 1			
1C	17.5	5.5	0.7	12.0	4.8	6.7
2E	31.4	5.5	0.7	25.9	4.8	5.8
3C	17.5	7.0	1.0	8.0	6.0	7.1
4E	26.2	5.5	1.0	20.7	4.5	6.5
5C	15.7	5.5	0.7	10.2	4.8	7.6
6E	26.3	7.0	2.8	19.2	4.2	6.5
7E	31.4	5.5	0.7	25.9	4.8	6.1
8C	13.9	6.2	0.7	7.7	5.5	7.1
9C	15.7	5.5	1.0	10.2	4.0	6.9
10E	17.5	4.9	0.7	12.6	4.2	5.7
11E	34.9	8.4	0.7	26.5	7.7	5.8
12C	17.5	5.5	0.7	12.0	4.8	7.0

(Continued)

APPENDIX 5 (Concluded)

Microcosm No.	TP, µg/ℓ	SP, µg/ℓ	SRP, µg/ℓ	PP, µg/ℓ	SUP, µg/ℓ	Oxygen, ppm
			May 4			
1C	13.9	7.3	0.0	6.6	7.3	6.8
2E	16.2	11.5	0.0	4.7	11.5	3.5
3C	12.2	9.1	0.0	3.1	9.1	7.4
4E	16.2	12.9	0.0	3.3	12.9	4.2
5C	13.9	5.9	0.0	8.0	5.9	8.2
6E	15.7	9.1	0.0	6.6	9.1	4.4
7E	26.2	11.2	0.0	15.0	11.2	4.4
8C	13.9	5.5	0.0	8.4	5.5	7.5
9C	15.7	8.0	0.0	7.7	8.0	7.6
10E	20.9	10.1	0.0	10.8	10.1	4.4
11E	19.2	12.6	0.0	6.6	12.6	4.2
12E	12.2	6.2	0.0	6.0	6.2	7.1
			May 10			
1C	22.7	7.0	1.4	15.7	5.6	6.1
2E	29.7	11.9	2.1	17.8	9.8	5.5
3C	26.2	7.0	0.0	19.0	7.0	6.4
4E	22.7	5.2	0.0	15.7	7.0	5.8
5C	13.9	5.2	1.4	8.7	3.8	7.0
6E	20.9	3.8	0.0	17.1	3.8	5.6
7E	24.4	5.2	0.0	19.2	5.2	5.7
8C	17.5	5.2	0.0	12.3	5.2	6.8
9C	19.2	6.6	0.0	12.6	6.6	6.8
10E	19.2	4.9	0.0	14.3	4.9	5.4
11E	22.7	7.0	0.0	15.7	7.0	5.8
12C	19.2	8.0	1.4	11.2	6.6	6.2

APPENDIX 6

Water, Wall, and Sediment Chlorophyll Values,

Microcosm Experiment I

Microcosm	NOV. 4	NOV. 18	NOV. 22	DEC. 1
		Water Chlorophyll, mg/m^3		
C	1.098	0.446	1.301	0.771
E	0.613	0.609	0.753	0.773
		Wall Chlorophyll, mg/m^3		
C	0.0136	0.0039	0.0	0.0136
E	0.0090	0.0	0.0059	0.0173
		Sediment Chlorophyll, mg/m^2		
C	0.0266	0.0257	0.0927	0.0518
	0.0402	0.0567	0.0251	0.0288
E	0.0315	0.0551	0.0446	0.1320
	0.0747	0.0531	0.0441	0.1163

APPENDIX 7

Water, Wall, and Sediment Chlorophyll Values,

Microcosm Experiment II

Microcosm No.	APRIL 7	APRIL 21	MAY 2	MAY 13
		Water Chlorophyll, mg/m^3		
1C	1.126	1.579	1.951	1.996
2E	1.068	1.197	0.893	0.638
3C	0.975	1.510	2.668	2.263
4E	1.161	1.531	1.046	0.278
5C	1.068	0.873	1.615	1.090
6E	1.219	1.336	0.894	0.905
7E	1.035	0.986	1.708	1.090
8C	1.670	1.508	1.949	1.090
9C	1.416	1.266	1.754	1.647
10E	1.138	1.067	1.369	0.731
11E	1.833	1.438	1.581	1.740
12C	1.069	1.171	1.952	1.276

	MAY 2	MAY 8	MAY 13
	Wall Chlorophyll, mg/m^3		
1C	0.027	0.060	0.068
2E	0.011	0.049	0.033
3C	0.054	0.034	0.029
4E	0.114	0.140	0.075
5C	0.194	0.366	0.228
6E	0.263	0.191	0.075
7E	0.152	0.114	0.049
8C	0.121	0.111	0.061
9C	0.109	0.056	0.033
10E	0.226	0.143	0.058
11E	0.138	0.060	0.136
12C	0.170	0.015	0.118

(Continued)

APPENDIX 7 (Concluded)

Microcosm No.	APRIL 17	APRIL 21	MAY 2	MAY 13
	Sediment Chlorophyll, mg/m^2			
1C	14.898	14.954	7.597	11.254
	4.547	11.001	8.010	8.723
2E	4.338	4.363	4.018	40.931
	8.470	10.630	7.224	29.584
3C	7.890	6.918	8.653	10.172
	10.335	4.756	10.208	16.495
4E	14.538	14.694	6.776	16.961
	9.330	5.869	10.469	49.326
5C	9.006	6.960	7.597	17.661
	15.787	10.518	4.410	48.976
6E	12.722	14.694	8.685	11.221
	7.899	6.405	9.864	23.554
7E	7.391	5.660	19.387	30.623
	10.894	7.076	8.683	19.960
8C	12.334	9.425	6.403	21.171
	9.801	8.841	9.377	28.969
9C	5.579	7.384	7.145	15.896
	15.840	5.707	4.039	15.295
10E	7.472	4.593	8.154	14.177
	9.748	7.171	5.131	30.312
11E	7.384	11.397	11.026	23.473
	10.444	6.776	5.152	42.828
12C	15.629	5.498	6.217	20.538
	12.028	9.059	5.479	13.551

APPENDIX 8

Dissolved Oxygen and Temperature Changes for Calculation of Microcosm Metabolism, O_2(°C)

April 19

Time	1C	2E	3C	4E	5C	6E
8:30 PM	7.3 (23.9)	7.5 (23.8)	7.3 (24.1)	8.1 (23.3)	8.2 (24.3)	8.4 (24.1)
12:30	6.7 (22.2)	6.8 (22.0)	6.9 (23.0)	7.7 (22.8)	7.6 (23.0)	7.5 (23.0)
4:30	6.3 (21.9)	6.3 (21.9)	6.2 (22.7)	7.0 (22.3)	7.0 (22.6)	6.7 (22.6)
8:30 AM	5.9 (21.6)	5.9 (21.6)	5.8 (22.5)	6.5 (22.0)	6.5 (22.2)	6.2 (22.0)
12:30	6.4 (22.4)	6.4 (22.3)	6.4 (23.7)	6.9 (23.0)	6.8 (23.2)	6.7 (23.2)
4:30	6.6 (23.0)	6.6 (22.8)	6.6 (23.8)	7.4 (23.2)	7.4 (23.4)	8.0 (23.6)
8:30	6.7 (23.0)	7.3 (23.0)	7.2 (24.0)	7.8 (23.7)	8.0 (23.9)	8.2 (23.9)
	7E	8C	9C	10E	11E	12C
8:30 PM	8.1 (23.5)	8.1 (24.0)	8.5 (23.3)	8.1 (23.5)	8.1 (23.2)	8.1 (24.0)
12:30	7.4 (22.9)	7.5 (23.0)	7.8 (22.5)	7.3 (22.8)	7.4 (22.3)	7.2 (22.6)
4:30	6.7 (22.4)	6.7 (22.6)	6.9 (22.0)	6.5 (21.8)	6.8 (21.7)	6.4 (22.0)
8:30 AM	6.2 (22.1)	6.2 (22.1)	6.4 (21.7)	6.0 (21.6)	6.4 (21.3)	6.2 (21.6)
12:30	6.9 (23.0)	6.6 (23.2)	6.9 (22.4)	6.5 (22.4)	6.9 (22.0)	6.8 (22.5)
4:30	7.8 (23.6)	7.5 (23.9)	7.4 (23.9)	7.3 (23.0)	7.6 (22.5)	7.4 (22.7)
8:30	8.0 (23.4)	8.0 (24.0)	7.6 (23.0)	7.6 (23.0)	8.0 (23.0)	7.7 (23.0)

April 27

	1C	2E	3C	4E	5C	6E
8:30 PM	6.8 (23.2)	7.2 (23.0)	7.7 (23.5)	8.2 (23.5)	8.3 (23.8)	8.3 (24.2)
12:30	6.6 (22.0)	6.8 (22.9)	7.4 (22.9)	7.7 (22.5)	7.7 (23.0)	7.4 (23.0)
4:30	6.4 (21.0)	6.4 (20.9)	6.9 (21.5)	7.1 (21.0)	7.1 (21.5)	6.8 (21.7)
8:30 AM	6.0 (19.8)	5.9 (19.5)	6.2 (20.2)	6.2 (19.9)	6.3 (20.2)	6.2 (20.2)
12:30	6.2 (22.2)	6.4 (19.8)	6.7 (20.5)	7.2 (21.1)	7.0 (21.9)	6.8 (21.5)
4:30	6.8 (22.5)	6.8 (20.8)	7.4 (21.2)	7.6 (22.3)	7.8 (22.5)	7.2 (22.3)
8:30	7.4 (23.5)	7.5 (23.0)	8.1 (23.0)	8.4 (23.2)	8.2 (23.3)	8.2 (23.3)

(Continued)

April 27 (Continued)

Time	7E	8C	9C	10E	11E	12C
8:30 PM	7.7 (23.5)	7.4 (23.8)	7.7 (24.8)	7.3 (23.5)	7.9 (22.9)	7.7 (22.9)
12:30	7.2 (22.8)	6.9 (22.8)	7.2 (23.0)	6.9 (22.5)	7.4 (22.0)	7.2 (22.0)
4:30	6.8 (21.5)	6.6 (21.5)	6.8 (21.5)	6.4 (21.1)	7.0 (21.0)	6.8 (20.5)
8:30 AM	6.2 (19.9)	6.0 (19.9)	6.1 (20.2)	5.7 (19.9)	6.3 (19.6)	6.0 (19.4)
12:30	6.8 (21.0)	6.6 (21.6)	6.7 (21.9)	6.4 (21.6)	7.1 (21.1)	6.7 (21.1)
4:30	7.5 (22.2)	7.0 (22.7)	7.1 (23.1)	6.6 (22.5)	7.4 (22.7)	7.4 (22.5)
8:30	7.9 (23.0)	7.5 (23.5)	7.5 (24.2)	7.2 (23.3)	8.0 (23.0)	7.6 (23.0)

May 3

	1C	2E	3C	4E	5C	6E
9:30 AM	6.2 (22.2)	3.6 (22.0)	6.6 (22.6)	3.9 (22.5)	7.4 (22.8)	4.2 (22.6)
1:30	6.4 (23.2)	3.4 (22.8)	7.0 (24.0)	4.0 (23.6)	7.8 (23.5)	4.3 (23.6)
5:30	6.8 (23.8)	3.5 (22.8)	7.4 (24.5)	4.2 (23.8)	8.2 (23.8)	4.4 (23.8)
9:30 AM	7.3 (23.6)	3.2 (23.0)	7.8 (24.5)	4.4 (24.0)	8.6 (23.5)	4.4 (24.0)
9:30	6.1 (22.0)	4.0 (22.1)	6.4 (22.2)	3.9 (22.8)	7.0 (22.2)	4.2 (22.8)

	7E	8C	9C	10E	11E	12C
9:30 AM	4.4 (22.6)	6.6 (22.5)	6.6 (22.0)	4.3 (22.0)	4.3 (22.0)	6.4 (21.8)
1:30	4.4 (23.8)	7.1 (23.8)	7.2 (22.8)	4.4 (22.8)	4.4 (22.8)	6.7 (23.0)
5:30	4.4 (23.8)	7.5 (23.8)	7.6 (23.2)	4.4 (22.8)	4.2 (22.5)	7.1 (23.1)
9:30 PM	4.4 (24.0)	7.6 (24.2)	7.7 (23.7)	4.4 (22.8)	4.5 (22.8)	7.2 (23.5)
9:30	4.4 (23.0)	6.3 (22.3)	6.2 (22.0)	4.2 (22.1)	4.2 (22.0)	6.2 (22.0)

(Continued)

May 18

Time	1C	2E	3C	4E	5C	6E
9:30 PM	8.2 (23.3)	8.6 (22.8)	8.4 (23.2)	8.5 (22.9)	9.4 (23.5)	8.8 (23.2)
3:30	8.0 (22.0)	7.9 (22.0)	7.8 (22.2)	8.4 (21.9)	8.6 (22.0)	8.4 (22.0)
9:30 AM	7.3 (21.3)	7.5 (21.2)	7.3 (21.2)	7.7 (21.2)	8.1 (21.2)	7.9 (21.3)
1:30	7.5 (23.0)	7.8 (22.3)	7.8 (23.0)	8.4 (22.0)	8.6 (22.5)	8.2 (22.5)
5:30	7.8 (23.0)	8.0 (22.8)	8.0 (23.5)	8.6 (22.5)	8.7 (22.8)	8.2 (23.0)
9:30	8.0 (24.0)	8.0 (23.5)	8.4 (24.0)	8.8 (23.4)	9.1 (23.4)	8.3 (23.9)

	7E	8C	9C	10E	11E	12C
9:30 PM	8.4 (23.2)	9.4 (23.9)	9.3 (23.3)	7.8 (22.8)	8.7 (23.2)	8.6 (23.2)
3:30	8.0 (22.0)	8.7 (22.3)	9.0 (22.0)	7.4 (21.9)	8.1 (22.0)	7.6 (22.3)
9:30 AM	7.5 (21.7)	8.0 (21.9)	8.3 (21.4)	7.0 (21.2)	7.6 (21.3)	7.4 (21.2)
1:30	8.0 (22.7)	8.7 (23.0)	9.0 (22.8)	7.3 (22.5)	8.2 (22.0)	7.7 (23.0)
5:30	8.0 (23.0)	8.9 (23.0)	9.2 (23.2)	7.4 (22.8)	8.3 (23.0)	8.0 (23.2)
9:30	8.0 (24.0)	9.4 (23.5)	9.7 (23.5)	7.3 (22.8)	8.3 (23.5)	8.3 (23.6)

May 22

	1C	2E	3C	4E	5C	6E
9:30 PM	7.7 (22.5)	8.0 (22.1)	7.7 (23.2)	9.6 (22.4)	8.4 (22.0)	8.5 (21.9)
3:30	7.5 (21.2)	7.8 (21.2)	7.3 (22.0)	8.8 (22.3)	7.8 (22.0)	7.8 (21.9)
9:30 AM	6.8 (20.9)	7.3 (20.8)	6.9 (21.0)	8.1 (21.2)	7.3 (21.0)	7.4 (21.0)
1:30	7.1 (21.9)	7.5 (21.6)	7.2 (22.9)	8.7 (22.5)	7.7 (22.8)	7.9 (22.0)
5:30	7.5 (22.4)	8.2 (22.0)	8.2 (23.1)	9.8 (22.0)	8.8 (23.0)	8.9 (22.5)
9:30	8.0 (22.9)	8.6 (22.7)	8.5 (23.3)	10.4 (22.2)	9.4 (23.2)	9.4 (23.0)

(Continued)

May 22 (Continued)

Time	7E	8C	9C	10E	11E	12C
9:30 PM	8.4 (22.8)	9.1 (23.0)	9.6 (22.2)	7.9 (22.4)	9.0 (22.2)	8.0 (22.2)
3:30	7.9 (21.9)	8.8 (22.0)	9.1 (21.1)	7.5 (21.4)	8.5 (21.0)	7.6 (21.0)
9:30 AM	7.4 (21.0)	8.0 (21.3)	8.3 (20.7)	7.1 (20.9)	7.6 (20.7)	7.1 (21.0)
1:30	7.9 (22.0)	8.6 (22.1)	9.0 (21.2)	7.6 (21.9)	8.1 (21.5)	7.8 (21.9)
5:30	8.7 (22.5)	9.3 (23.0)	10.0 (22.3)	8.1 (22.2)	9.2 (22.0)	8.6 (22.1)
9:30	9.4 (23.0)	9.8 (24.0)	10.4 (23.0)	8.4 (23.0)	9.6 (22.6)	8.9 (23.0)

May 24

	1C	2E	3C	4E	5C	6E
9:30 PM	8.7 (22.2)	9.6 (22.2)	8.8 (22.5)	10.5 (22.0)	8.3 (22.0)	9.8 (22.2)
9:30 AM	7.8 (22.0)	8.1 (21.7)	7.7 (22.0)	9.0 (21.7)	7.2 (21.7)	8.0 (21.7)
1:30	8.0 (22.9)	8.6 (22.4)	8.0 (22.7)	9.3 (22.3)	7.7 (22.2)	8.8 (22.4)
5:30	8.1 (23.0)	9.0 (22.9)	8.8 (23.0)	10.1 (22.9)	8.0 (23.0)	9.2 (23.2)
9:30	8.4 (23.7)	9.4 (23.2)	8.8 (23.0)	10.2 (23.0)	8.1 (23.0)	9.5 (23.2)

	7E	8C	9C	10E	11E	12C
9:30 PM	9.9 (22.0)	9.8 (22.8)	9.7 (22.0)	9.2 (22.2)	9.4 (21.3)	9.2 (22.2)
9:30 AM	8.4 (21.7)	8.4 (21.7)	8.3 (21.3)	7.8 (21.7)	8.0 (21.2)	7.8 (21.2)
1:30	8.9 (22.4)	9.0 (23.3)	8.9 (22.7)	8.4 (22.3)	8.6 (21.7)	8.4 (22.2)
5:30	9.4 (23.2)	9.4 (23.2)	9.1 (23.2)	8.6 (22.4)	8.9 (22.1)	8.7 (23.0)
9:30	9.7 (23.6)	9.5 (24.0)	9.3 (23.0)	8.8 (23.0)	9.0 (22.5)	8.8 (23.4)

APPENDIX 9

Dissolved Oxygen and Temperature Changes for Calculation of Cylinder Metabolism, O_2(°C)

April 19

Time	1C	2E	3C	4E	5C	6C
8:30 PM	7.3 (23.9)	7.5 (23.8)	7.3 (24.1)	8.1 (23.3)	8.2 (24.3)	8.4 (24.1)
12:30	6.8 (22.2)	7.1 (22.1)	7.0 (23.0)	7.9 (22.8)	7.8 (23.0)	7.7 (23.0)
4:30	6.4 (21.8)	6.4 (21.9)	6.5 (22.7)	7.4 (22.3)	7.2 (22.6)	7.2 (22.6)
8:30 AM	6.2 (21.6)	6.2 (21.6)	6.3 (22.5)	7.0 (22.0)	6.7 (22.2)	6.9 (22.0)
12:30	6.4 (22.0)	6.2 (22.0)	6.5 (23.0)	7.2 (22.8)	6.8 (22.8)	7.0 (22.9)
4:30	6.4 (23.0)	6.2 (22.4)	6.6 (23.8)	7.3 (23.0)	6.9 (23.2)	7.2 (23.6)
8:30	6.4 (22.8)	6.1 (22.8)	6.6 (24.0)	7.2 (23.4)	6.8 (24.0)	7.1 (23.5)
	7E	8C	9C	10E	11E	12C
8:30 PM	8.1 (23.5)	8.1 (24.0)	8.5 (23.3)	8.1 (23.6)	8.1 (23.2)	8.1 (24.0)
12:30	7.5 (22.6)	7.5 (23.0)	8.0 (22.3)	7.4 (22.8)	7.7 (22.0)	7.5 (22.6)
4:30	7.0 (22.4)	6.8 (22.6)	7.4 (22.0)	6.9 (21.8)	7.2 (21.7)	7.0 (22.0)
8:30 AM	6.8 (22.1)	6.5 (22.1)	6.9 (21.7)	6.6 (21.6)	6.9 (21.3)	6.7 (21.6)
12:30	6.9 (22.8)	6.6 (23.0)	7.1 (22.1)	6.8 (22.1)	7.2 (21.8)	6.9 (22.0)
4:30	6.9 (23.2)	6.7 (23.8)	7.2 (22.8)	6.9 (22.8)	7.4 (22.5)	7.1 (22.5)
8:30	6.8 (23.2)	6.7 (24.0)	6.8 (23.0)	6.9 (23.0)	7.2 (23.0)	6.8 (23.0)

April 27

	1C	2E	3C	4E	5C	6E
8:30 PM	7.1 (22.8)	6.8 (22.8)	7.7 (23.8)	8.4 (23.5)	8.4 (23.8)	7.7 (24.0)
12:30	7.0 (22.2)	6.5 (21.9)	7.4 (22.9)	8.2 (22.5)	8.0 (23.0)	7.4 (23.0)
4:30	6.8 (21.0)	6.4 (20.9)	7.2 (21.5)	7.8 (21.0)	7.8 (21.5)	7.1 (21.7)
8:30 AM	6.5 (19.8)	5.9 (19.5)	6.6 (20.2)	7.2 (19.9)	7.1 (20.2)	6.6 (20.2)
12:30	6.6 (22.2)	6.0 (19.8)	6.7 (20.5)	7.0 (21.1)	7.0 (21.9)	6.5 (21.5)

(Continued)

April 27 (Continued)

Time	1C	2E	3C	4E	5C	6C
4:30	6.6 (22.5)	6.2 (20.8)	6.8 (21.1)	7.0 (22.3)	7.0 (22.5)	6.6 (22.3)
8:30	6.7 (23.5)	6.5 (23.0)	7.1 (23.0)	7.2 (23.2)	7.4 (23.3)	7.1 (23.3)
	7E	8C	9C	10E	11E	12C
8:30 PM	7.9 (23.8)	7.4 (23.5)	7.7 (23.2)	7.0 (23.2)	7.9 (22.5)	7.8 (22.9)
12:30	7.7 (22.8)	7.1 (22.8)	7.5 (23.0)	6.8 (22.5)	7.6 (22.0)	7.5 (22.0)
4:30	7.2 (21.8)	7.0 (21.5)	7.1 (21.5)	6.6 (21.1)	7.1 (21.0)	7.2 (20.5)
8:30 AM	6.8 (19.9)	6.6 (19.9)	6.5 (20.2)	6.1 (19.9)	6.7 (19.6)	6.4 (19.9)

May 18

	1C	2E	3C	4E	5C	6E
9:30 PM	8.0 (22.8)	7.9 (22.2)	8.3 (23.0)	8.3 (22.7)	8.5 (22.7)	8.4 (23.0)
3:30	7.9 (22.0)	7.4 (22.0)	7.7 (22.0)	8.1 (21.9)	8.0 (21.9)	8.0 (22.0)
9:30 AM	7.5 (21.3)	6.8 (21.3)	7.3 (21.5)	7.4 (21.2)	7.8 (21.1)	7.4 (21.3)
1:30	7.5 (22.2)	7.4 (22.0)	7.6 (22.8)	7.9 (22.2)	7.8 (22.0)	8.0 (22.3)
5:30	7.2 (23.0)	6.8 (22.8)	7.6 (23.2)	7.7 (22.5)	7.6 (22.8)	8.0 (23.0)
9:30	7.1 (23.3)	6.8 (23.2)	7.6 (23.8)	7.7 (23.0)	7.5 (23.2)	7.9 (23.9)
	7E	8C	9C	10E	11E	12C
9:30 PM	8.7 (22.9)	9.1 (23.0)	8.8 (22.4)	7.4 (22.2)	9.1 (22.9)	8.0 (22.8)
3:30	8.3 (22.0)	8.4 (22.0)	8.4 (22.0)	7.0 (21.9)	8.3 (22.0)	7.7 (22.0)
9:30 AM	8.0 (21.7)	8.0 (21.9)	7.8 (21.3)	6.8 (21.2)	7.8 (21.3)	7.4 (21.2)
1:30	8.4 (22.4)	8.1 (22.8)	8.0 (22.0)	7.1 (22.2)	8.3 (22.0)	7.5 (22.0)
5:30	8.3 (25.0)	8.0 (23.0)	7.6 (23.0)	6.9 (22.5)	8.4 (23.0)	7.5 (22.8)
9:30	8.3 (23.5)	8.0 (23.6)	7.4 (23.2)	6.9 (23.0)	8.5 (23.2)	7.2 (23.3)

(Continued)

May 24

Time	1C	2E	3C	4E	5C	6E
9:30 PM	9.1 (22.2)	9.9 (22.2)	9.2 (22.3)	10.9 (22.0)	8.6 (22.0)	8.9 (22.2)
9:30 AM	7.8 (22.0)	8.6 (21.7)	8.2 (22.0)	9.0 (21.7)	7.6 (21.7)	8.3 (21.7)
1:30	7.8 (22.5)	8.9 (22.7)	8.4 (22.4)	9.3 (22.2)	7.5 (22.2)	8.5 (22.2)
5:30	7.7 (23.0)	9.2 (22.9)	8.4 (23.0)	9.7 (22.9)	7.0 (23.0)	8.4 (23.2)
9:30	7.6 (23.7)	9.6 (23.2)	8.7 (23.2)	9.7 (23.0)	7.0 (23.0)	8.3 (23.2)
	7E	8C	9C	10E	11E	12C
9:30 PM	8.6 (22.3)	10.2 (22.3)	10.3 (22.2)	9.3 (22.2)	9.4 (21.3)	9.2 (21.8)
9:30 AM	8.2 (21.7)	8.7 (21.7)	8.9 (21.7)	8.2 (21.7)	7.6 (21.2)	8.0 (20.9)
1:30	8.4 (22.4)	9.0 (22.7)	9.1 (22.0)	8.7 (22.0)	8.1 (21.7)	8.0 (22.0)
5:30	8.2 (23.2)	8.8 (23.2)	8.8 (22.4)	8.6 (22.4)	8.3 (22.1)	8.2 (22.9)
9:30	8.0 (23.6)	8.9 (23.9)	8.6 (23.0)	8.6 (23.0)	8.5 (22.5)	8.2 (23.5)

APPENDIX 10

Daily Metabolic Rates (mg O_2/ℓ/day) for the Macrophyte-Epiphyte, Benthic, and Planktonic Components

Microcosm No.	Macrophyte-Epiphyte GPP	NPP	24NPP	R	Benthic GPP	NPP	24NPP	R	Planktonic GPP	NPP	24NPP	R
					April 19							
1C	0.92	0.60	0.28	0.64	0.38	-0.29	-0.97	1.35	1.90	0.50	-0.91	2.81
2E	2.12	1.51	0.89	1.23	0.00	-0.58	-0.89	1.17	1.94	0.50	-0.94	2.88
3C	1.90	1.24	0.58	1.32	0.30	-0.24	-0.78	1.08	1.72	0.40	-0.92	2.64
4E	1.71	1.10	0.49	1.22	1.48	0.60	-0.28	1.76	0.27	-0.40	-1.07	1.34
5C	1.85	1.53	1.21	0.64	0.97	-0.13	-1.22	2.19	0.96	0.10	-0.77	1.73
6E	2.92	2.01	1.11	1.81	1.10	0.28	-0.55	1.65	1.06	-0.09	-1.24	2.30
7E	2.46	1.74	1.03	1.43	0.48	-0.14	-0.76	1.24	1.30	0.00	-1.29	2.59
8C	1.71	1.38	1.04	0.67	1.50	0.22	-1.07	2.57	0.94	-0.02	-0.98	1.92
9C	1.92	1.30	0.68	1.24	0.99	-0.24	-1.47	2.46	0.93	0.14	-0.65	1.58
10E	2.02	1.26	0.49	1.53	1.01	0.28	-0.45	1.46	1.45	0.02	-1.41	2.86
11E	1.72	1.30	0.88	0.84	1.20	0.45	-0.31	1.51	0.90	-0.15	-1.19	2.09
12C	2.13	1.42	0.70	1.43	1.20	0.25	-0.70	1.90	0.92	-0.16	-1.24	2.16
					April 27							
1C	1.66	1.20	0.74	0.92	0.80	0.45	-0.70	1.50	0.90	0.15	-0.60	1.50
2E	1.35	1.10	0.65	0.70	1.45	0.30	-0.85	2.30	1.20	0.30	-0.60	1.80
3C	1.88	1.58	0.92	0.96	1.10	-0.10	-1.30	2.40	1.20	0.60	0.00	1.20
4E	3.23	2.20	1.17	2.06	1.01	-0.15	-1.31	2.32	0.60	0.15	-0.30	0.90
5C	2.52	1.59	0.67	1.85	1.08	-0.15	-1.38	2.46	0.90	0.45	0.00	0.90
6E	2.49	1.51	0.52	1.97	1.13	0.05	-1.04	2.17	1.20	0.45	-0.30	1.50
7E	1.99	1.41	1.32	1.17	0.84	-0.15	-1.14	1.98	1.20	0.45	-0.30	1.50
8C	2.30	1.51	0.72	1.58	0.74	-0.16	-1.05	1.79	0.90	0.45	-0.60	1.50
9C	1.98	1.49	1.00	0.98	0.62	-0.55	-1.72	2.34	1.20	0.30	-0.60	1.80

(Continued)

APPENDIX 10 (Continued)

Microcosm No.	Macrophyte-Epiphyte GPP	NPP	24NPP	R	Benthic GPP	NPP	24NPP	R	Planktonic GPP	NPP	24NPP	R
					April 27 (Continued)							
10E	1.86	1.21	0.55	1.31	1.07	0.00	-1.07	2.14	1.20	0.30	-0.60	1.80
11E	2.06	1.61	1.15	0.91	0.64	-0.41	-1.45	2.09	1.20	0.30	-0.60	1.80
12C	1.83	1.40	0.96	0.87	1.17	-0.10	-1.37	2.54	1.20	0.30	-0.60	1.80
					May 3							
1C	1.38	1.00	0.61	0.77	1.53	0.41	-0.72	2.25	0.30	-0.30	-0.90	1.20
2E	0.04	-0.47	-0.98	1.02	1.43	0.67	-0.09	1.52	0.30	-0.60	-1.50	1.80
3C	1.35	1.01	0.67	0.68	1.60	0.24	-0.62	2.22	0.30	-0.30	-0.90	1.20
4E	0.96	0.19	-0.58	1.54	2.08	0.91	-0.26	2.35	0.30	-0.60	-1.50	1.80
5C	2.35	1.81	1.27	1.08	0.49	0.09	-0.31	0.80	0.30	-0.70	-1.70	2.00
6E	0.42	0.11	-0.20	0.62	2.28	0.70	-0.89	3.17	0.00	-0.60	-1.20	1.20
7E	0.36	-0.10	-0.56	0.92	1.84	0.41	-1.03	2.87	0.00	-0.30	-0.60	0.60
8C	1.51	1.30	1.09	0.42	1.54	0.30	-0.94	2.48	0.00	-0.60	-1.20	1.20
9C	2.03	1.48	0.92	1.11	1.06	0.23	-0.61	1.67	0.30	-0.60	-1.50	1.80
10E	0.53	-0.10	-0.72	1.25	1.73	0.50	-0.74	2.47	0.30	-0.30	-0.60	1.20
11E	0.76	0.02	-0.72	1.48	1.71	0.50	-0.71	2.42	0.30	-0.30	-0.60	1.20
12C	1.25	0.98	0.71	0.54	1.11	-0.20	-1.51	2.62	0.30	0.00	-0.30	0.60
					May 18							
1C	1.44	1.05	0.66	0.78	-0.12	-0.65	-1.19	1.07	0.60	0.30	0.00	0.60
2E	0.03	0.00	0.00	-0.65	1.44	0.33	-0.49	1.93	0.30	-0.45	-1.20	1.50
3C	1.16	1.07	0.98	0.18	0.86	0.11	-0.34	1.20	0.60	-0.15	-0.90	1.50
4E	0.40	0.40	0.00	-0.59	1.02	0.40	0.08	0.94	0.60	-0.15	-0.90	1.50
5C	2.07	1.53	0.99	1.08	0.16	-0.20	-0.56	0.72	0.30	0.00	-0.30	0.60
6E	-0.09	0.00	0.00	-0.79	1.13	0.40	-0.33	1.46	0.30	-0.30	-0.90	1.20

(Continued)

APPENDIX 10 (Concluded)

Micro-cosm No.	Macrophyte-Epiphyte GPP	NPP	24NPP	R	Benthic GPP	NPP	24NPP	R	Planktonic GPP	NPP	24NPP	R
					May 18 (Continued)							
7E	0.42	0.09	-0.23	0.65	0.60	0.25	-0.41	1.01	0.60	0.30	0.00	0.60
8C	1.67	1.49	1.30	0.37	0.32	-0.29	-0.60	0.92	0.60	0.00	-0.60	1.20
9C	1.32	1.32	1.32	-0.90	0.08	-0.52	-0.82	0.90	0.60	0.00	-0.60	1.20
10E	0.16	-0.01	-0.19	0.35	1.22	0.62	0.01	1.21	0.30	-0.30	-0.60	1.20
11E	-0.28	-0.38	0.00	0.19	1.88	1.08	0.57	1.31	0.60	-0.15	-0.90	1.50
12C	2.22	1.12	0.01	2.12	0.19	-0.22	-0.62	0.81	0.60	0.00	-0.60	1.20
					May 24							
1C	0.38	0.75	1.12	-0.74	0.59	-0.20	-0.99	1.58	0.60	0.00	-0.60	1.20
2E	0.49	-0.01	-0.51	1.00	1.46	1.18	0.90	0.56	0.60	0.00	-0.60	1.20
3C	0.32	-0.08	-0.48	0.80	1.27	1.06	0.85	0.42	0.60	0.00	-0.60	1.20
4E	0.06	0.06	0.05	0.01	1.16	0.94	0.71	0.45	0.30	-0.30	-0.90	1.20
5C	1.49	1.27	1.05	0.44	-0.14	-0.82	-1.50	1.36	0.90	0.30	-0.30	1.20
6E	1.31	1.41	1.50	-0.19	1.63	0.45	-0.73	2.36	0.30	-0.45	-1.20	1.50
7E	1.63	1.29	0.94	0.69	0.39	0.10	-0.19	0.58	0.30	-0.30	-0.90	1.20
8C	1.09	1.02	0.94	0.15	0.56	0.20	-0.17	0.73	0.60	-0.15	-0.90	1.50
9C	1.68	1.46	1.23	0.45	0.22	-0.12	-0.45	0.67	0.30	-0.30	-0.90	1.20
10E	0.88	0.51	0.13	0.75	0.96	0.65	0.33	0.63	0.60	-0.15	-0.90	1.50
11E	-0.49	0.14	0.76	-1.25	2.54	1.35	0.15	2.39	0.30	-0.45	-1.20	1.50
12C	0.99	0.82	0.64	0.35	0.86	0.35	-0.16	1.02	0.60	-0.15	-0.90	1.50