*CHAPTER 4*

# EXPERIMENTAL AND MODELING RESULTS

This chapter presents the field and modeling results, including the prediction of the cause of the fish kills. Results of the flow and runoff calibration are shown. The results of the calibration and validation of water quality parameters are justified in terms of the goodness of fit. The calibrated and validated model was then used to simulate scenarios to find out the cause of the fish kills. The predictive capability of the model was also tested to ensure that the model prediction of *Chl a* before and during the fish kills was valid. The final section of this chapter proposes the engineering-based management solutions to prevent the fish kills.

## 4.1 RESULTS FROM FIELD COLLECTION

This section presents field data of $PO_4$-P in the runoff, $NH_3$-N loading from aquaculture, cyanobacteria enumeration and phenols measurement in the river water and its sediment.

### 4.1.1 Non-Point Source Loading of $PO_4$-P from Paddy Fields

From the field survey around Lake Sua Ten during the fish kill in 2003, $PO_4$-P were measured to be 2.01 mg/L in water from the regular paddy field, and 5.02 mg/L in water from the paddy field near a sugarcane plantation. The water was clear and soapy-looking with some debris.

### 4.1.2 Point Source Loading of $NH_3$-N from Aquaculture

$NH_3$-N from the CT, ST, and KP/BN aquacultures were added to the model as the point source loadings in segments 7, 9 and 10, respectively. As it turned out, after the model

calibration and validation, $NH_3$-N point source loadings from aquaculture had to be adjusted to zero. It meant that $NH_3$-N from aquaculture had no direct impact on $NH_3$-N in the river. This might be due to the fact that as soon as $NH_3$-N was released from aquaculture, it was immediately taken up by algae in the river. The field survey as shown in Table 4.2 confirmed that there was a large amount of green algae at the CT aquaculture.

4.1.3 Experimental Tilapia Aquaculture

By the beginning of June, 2002, fish in both the pond (location C in Figure 3.8), and the CT aquaculture (location A in Figure 3.8) started to die. On June 15$^{th}$, 2002, all fish in the pond died completely, while only 8 out of 50 experimental tilapias died at location A. From June-July, approximately 60 kilograms of commercial tilapia at the CT aquaculture died everyday. A new batch of 50 experimental tilapias was placed in the pond again on a cloudy day of July 7$^{th}$, 2002 and they died almost completely overnight. The experiment with tilapia was stopped when the purpose of the experiment which was to find out whether or not the fish kills in the river and the pond occurred during the same period, was achieved.

As mentioned in the previous chapter, if there were any chemicals that killed the fish at the CT aquaculture, they must also have been present in the pond because the fish kills occurred during the same period. The possibility that these chemicals could be detected in the pond was higher than at the CT aquaculture, because the percentage of fish death was higher in the pond and the chemicals in the pond could not be easily diluted. Therefore, during the fish kills of July, the water samples from locations A, B, C and D were analyzed by GC/MS, as discussed in the next section.

During the fish kills in July of 2002, the water sample from the pond was clear and greenish. Water in the inner area of this pond, where circulation was poor, looked soapy (Figure 4.1) after the surface was perturbed from sampling. The soapy-looking water was subsequently analyzed for its phosphorus and nitrogen contents. Their results are discussed in another section.

Figure 4.1 Water in the pond looked soapy after being perturbed by a sampling container.

4.1.4 GC/MS Analysis Results of Possible Pesticides and Other Toxic Compounds

Pesticides

One-gallon water samples from locations NS, NJ, NP, KB, PS, CT, ST, and KP/BN were collected from March - August of 2002, and analyzed by GC/MS for pesticides and other toxic organic chemicals. During the fish kills, water samples from locations A, B, C and D as shown in Figure 3.8 were also analyzed for comparison with samples from the river. The GC/MS analysis was set up under an incremental time-step temperature function to run at a temperature as high as 350 $^{0}$C to ensure that chemicals with high-boiling points were not missed. From the GC/MS analysis of water samples from the river from March-August of 2002, there was no significant peak in the GC chromatogram. All MS fragmentation patterns of small unknown peaks in the GC chromatograms were checked against the library database

of known pesticides, and none could be matched against known pesticides. If the cause of fish kills in July of 2002 was the pesticide in the river, the chance of detecting the pesticides in this month was much better than any other times; but still none was detected. This finding was not unusual. According to the pesticides data in this river from PCD in Table 3.3, they were reported as below the detection limits.

From the GC/MS analysis, the cause of the fish kills from pesticides could not yet be ruled out because the fish kills of 2002 was very mild, and pesticides below non-detectable levels could have been the cause. At very low concentrations, the background noise in the MS fragmentation patterns of unknown peaks could interfere in the matching process with the GC/MS library database. The best way to identify these small peaks in the GC chromatogram was to do co-injection with pesticide standards. However, these standards were not available during the time of the experiment. Due to time and budget constraint, and availability of pesticide data from PCD (Table 3.3) in this river from 1999 - 2001, the identification of these small peaks was discontinued in order to allow time for pursuing the large peak of heptadecane detected by GC/MS in the fish pond. Eventually, if the peak of heptadecane did not provide any lead to the cause of the fish kills, the identification of these small peaks would have been resumed.

In this study, a field survey on the type of pesticides commonly used around the Pong River was also conducted. Glyphosate, Dimathoate and Lannate were the most commonly used pesticides. Glyphosate is herbicide used to kill weeds in the rice field, where as Dimathoate and Lannate are insecticides. 96-hr $LC_{50}$ values of glyphosate, Dimathoate and Lannate are 10-197 mg/L (US EPA, 1993), 6.2 mg/L (Johnson and Finley, 1980) with rainbow trout, and 0.25 mg/L (El-Refai *et al.*, 1976) with Nile tilapia. Among these three pesticides,

Lannate might be the most toxic to fish. According to EPA(1993), glyphosate is "practically non-toxic to fish." Dimethoate is readily degraded by hydrolysis, and lost through evaporation by 23 to 40% and through biodegradation by 77% in a nonsterile clay loam soil after 2 weeks (Howard, 1991). Lannate is "moderate to highly toxic" for fish (Kaplan and Sherman, 1977; Howard, 1989) and has long half-live in water (USDA, 1990). Its 96-hr $LC_{50}$ is the lowest. Despite its high toxicity, Lannate is not expected to remain in the rice field after the season in which it was applied (NRC, 1977).

No conclusion on whether the pesticides were the cause of the fish kills in 1999 could be made without more scientific evidence. "No detection" of pesticides did not simply imply "no existence." Pesticides at undetectable quantities could contribute to the fish kills, even though they could not cause low DO as observed on May 10$^{th}$, 1999. If no pesticides were detected in the river, there should have been more study of the pesticides in the runoff, and the GC co-injection of the pesticide standards with river samples during the fish kills.

Other Toxic Compounds

From the GC/MS analysis, the predominant chemical detected in the pond water was heptadecane (Figure 4.2). The identification of heptadecane was later confirmed by the instrumentation laboratory at Chulalongkorn University since the heptadecane standard was not available during the GC/MS analysis. Heptadecane slowly disappeared from the pond within three to four weeks. Another minor chemical found in the fish pond was palmitic acid. It was confirmed by co-injection with the palmitic acid standard, obtained as a gift from a Malaysian factory. Water samples from locations A, B, and D did not show any significant peaks, perhaps due to the dilution from the Pong River.

The presence of heptadecane oil in the pond was unusual. If a case of a petroleum spill was to be considered, then other hydrocarbons besides heptadecane should have also been detected, as petroleum was composed of various hydrocarbons with different carbon chain lengths. Moreover, heptadecane was not used in any process in the pulp mill or as domestic use. The only source that heptadecane could have come from was the cell wall of cyanobacteria. Heptadecane has been known as a signature of cyanobacteria, as discussed in Chapter 2. The pond and its vicinities in the river were thus checked for the presence of cyanobacteria and the eutrophic states in 2003. The results in the next section show that cyanobacteria indeed existed in the pond. The presence of cyanobacteria explained why most of heptadecane came out of the filter paper during the sample extraction. Cyanobacteria were bigger than regular algae of 15 μm in diameter (Madigan *et al.*, 1984); therefore, all of cyanobacteria should have been trapped in the filter paper of 1.2 μm in pore size.

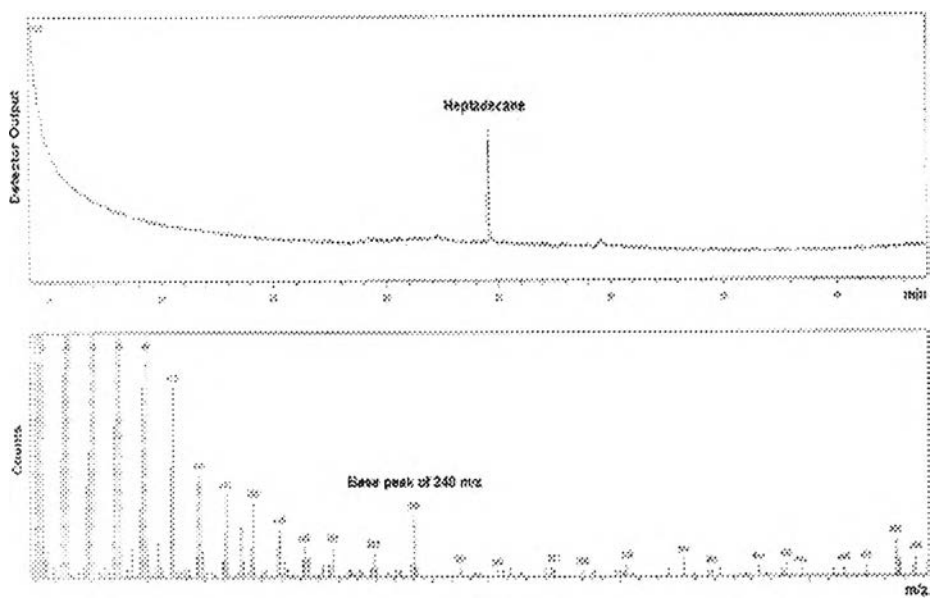



Figure 4.2. GC/MS Spectrum of heptadecane with the base peak of 240 m/z and retention time of 24.57 min. When the fish kills became severe with the experimental tilapia (98% death), heptadecane reduced sharply.

As for palmitic acid which was also detected, this compound was not pursued any further because it was a natural-occurring compound and it could have come from any living sources. After heptadecane disappeared, a new set of compounds as shown in Figure 4.3, was detected by GC/MS in the pond where the circulation was poor. These compounds appeared to have long alkyl chains from analyzing their fragmentation patterns. They were thus co-injected with other toxic long-chained compounds which might have been used by the mill, e.g., Arquad $[(C10)_2N(CH_3)_2^+Cl^-]$, cetrimonium chloride $(C16N(CH_3)_2^+Cl^-)$, and alkyl ketene dimmer (AKD-C16). Arquad and cetrimonium chloride might have been used as surfactants in any process. AKD was used by the mill for sizing effects to prevent water penetration (Lindström, 1989; Roberts, 1997). The effluent from the mill was also analyzed by GC/MS to determine if there were any peaks that would match with the unknown peaks in Figure 4.3. From the analysis, the unknown peaks did not match with the co-injection standards and mill's effluent. Therefore, it was concluded that these unknown compounds were not from the effluent.

Since these compounds were not from the mill's effluent and appeared after heptadecane and thus cyanobacteria, disappeared, these compounds could be the cell components of dead *Microcystis* sp. The pattern of the GC spectrum in Figure 4.3 was very similar to that of the algal fossil found by Silliman and Schelske (2003). The identification process of these unknown peaks by the GC/MS method was not continued because the presence of *Microcystis* sp. could have been easily confirmed by algal enumeration. Moreover, the time required for the procurement of standard chemicals for co-injection would not allow this project to complete in due time.

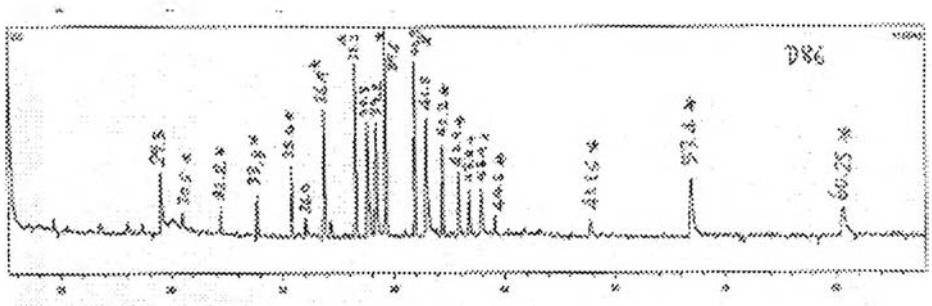


Figure 4.3 GC spectrum of unknown compounds found in the fish pond after heptadecane disappeared. The number on top of the peak indicated the specific retention time for each compound.

### 4.1.5 Phosphorus and Nitrogen Contents in the Fish Pond

As mentioned in the previous section, the unusual soapy-looking water sampled from the fish pond, was analyzed for phosphorus and nitrogen in comparison with water from other locations, and the results are shown in Table 4.1. The water sample from the CT aquaculture contained very small amounts of phosphorus and nitrogen in all forms. The water sample from the fish pond showed total phosphorus (TP) of 0.15 mg/L which was considered quite high. According to Thomann and Mueller (1987), a pond with TP greater than 0.020 mg/L, could be considered eutrophic. The ratio of TN/TP in the pond was determined to be 28.7. According to Chapra (1997), if TN/TP was greater than 7.2, the pond was considered phosphorus-limiting. There were other parameters, such as chlorophyll *a* and Secchi depth, used in this study for determining the eutrophic state of the pond, and they will be discussed next.

Table 4.1 Phosphorus and nitrogen contents at different locations in May of 2003.

| Location | $PO_4$-P (mg/L) | TP (mg/L) | $NO_3$ (mg/L) | TKN (mg/L) | $NH_3$-N (mg/L) |
|---|---|---|---|---|---|
| CT aquaculture (A) | <0.05 | <0.05 | <0.06 | <0.5 | <0.2 |
| Outer Chot Lake (B) | 0.25 | 0.28 | 0.19 | 0.6 | <0.2 |
| Fish pond (C) | <0.05 | 0.15 | 0.12 | 4.3 | <0.2 |
| Creek Chot(D) | 0.11 | 0.15 | 0.05 | 0.7 | <0.2 |

4.1.6 Cyanobacteria Enumeration and Secchi Depth Measurement

Tables 4.2, 4.3 and 4.4 show the temperatures, Secchi depths, total plankton counts, *Microcystis* sp. populations, and chlorophyll *a* at the CT aquaculture (location A in Figure 3.8), Outer Chot Lake (location B in Figure 3.8), and fish pond (location C in Figure 3.8), respectively, in 2003. The Secchi depth is the measurement of how far deep the Secchi disc can be seen under the water surface; thus it can indirectly give information on the quantity of chlorophyll *a* in the river which obstructs the view of the Secchi disc. Large Secchi depth means there is low chlorophyll *a*.

According to Thomann and Mueller (1997), the waterbody with TP above 20 μg/L (discussed in the previous section), Secchi depths below 200 cm, and chlorophyll *a* above 10 μg/L, is considered eutrophic. Locations A, B, and C were therefore considered eutrophic. The Secchi depths, total plankton and *Microcystis* sp. populations at all locations reduced as rainy season progressed, starting from July 8$^{th}$, 2003.

From Tables 4.2, 4.3 and 4.4, *Microcystis* sp. peaked in May of 2003. It should also be pointed out that *Microcystis* sp. of 17,273 cells/mL in the fish pond on May 24$^{th}$, 2003, almost put this pond into a category of relatively mild adverse health effects, as classified by the World Health Organization (WHO, 1998). According to WHO, water with 20,000 cyanobacterial cells per mL is considered under a classification of "relatively mild and/or low probabilities of adverse health effects" to human.

Table 4.2 *Microcystis* sp. count and other variables at the CT aquaculture (location A in Figure 3.8).

| Date | Temp (air) | Temp (water) | Secchi Depth (cm) (SDV) | Total Plankton (cells/mL) | *Microcystis* sp. (cells/mL) | *Chl* a (μg/L) |
|---|---|---|---|---|---|---|
| May 24th, 2003 | 33.5 | 31.7 | 100 | 9,743 | 8,288 | 3.9 |
| June 14th, 2003 | 35.4 | 31.3 | 95 | 7,297 | 4,462 | 2.3 |
| June 28th, 2003 | 32.5 | 31.6 | 94 | 8,745 | 4,514 | 2.5 |
| July 8th, 2003 | 32.5 | 32.1 | 98 | 4,939 | 1,181 | 4.5 |
| July 26th, 2003 | 32.1 | 31.2 | 92 | 6,229 | 1,579 | 3.5 |
| August 12th, 2003 | 32.7 | 31 | 75 | 5,238 | 1,421 | 3.2 |
| August 30th, 2003 | 27.6 | 29.6 | 73 | 6,469 | 2,130 | 4 |
| September 13th, 2003 | 31.8 | 29.3 | 45 | 4,942 | 1,155 | 2.4 |
| September 27th,2003 | 32 | 29.6 | 62 | 4,872 | 975 | 1.7 |

Table 4.3 *Microcystis* sp. count and other variables in the Outer Chot Lake (location B in Figure 3.8).

| Date | Temp (air) | Temp (water) | Secchi Depth (cm) (SDV) | Total Plankton (cells/mL) | *Microcystis* sp. (cells/mL) | *Chl* a (μg/L) |
|---|---|---|---|---|---|---|
| May 24th, 2003 | 32.3 | 31.5 | 116 | 16,035 | 13,396 | 10.6 |
| June 14th, 2003 | 34.8 | 31.6 | 96 | 9,877 | 4,226 | 2.1 |
| June 28th, 2003 | 31.8 | 31.1 | 105 | 10,465 | 7,964 | 3.8 |
| July 8th, 2003 | 32.6 | 31.2 | 98 | 6,877 | 1,898 | 3.7 |
| July 26th, 2003 | 31.6 | 31.6 | 81 | 6,338 | 2,141 | 3.7 |
| August 12th, 2003 | 32.3 | 31.2 | 77 | 5,643 | 1,563 | 3.6 |
| August 30th, 2003 | 27.9 | 29.7 | 75 | 6,682 | 1,950 | 4.1 |
| September 13th, 2003 | 32.4 | 30.2 | 42 | 4,560 | 945 | 3.0 |
| September 27th,2003 | 32.2 | 30.4 | 58 | 4,642 | 1,089 | 1.6 |

Table 4.4 *Microcystis* sp. count and other variables in the fish pond (location C in Figure 3.8).

| Date | Temp (air) | Temp (water) | Secchi Depth (cm) (SDV) | Total Plankton (cells/mL) | *Microcystis* sp. (cells/mL) | *Chl* a (μg/L) |
|---|---|---|---|---|---|---|
| May 24th, 2003 | 34.1 | 31.6 | 111 | 19,388 | 17,273 | 14.4 |
| June 14th, 2003 | 36.1 | 31.7 | 93 | 15,510 | 5,246 | 2.9 |
| June 28th, 2003 | 31.2 | 30.5 | 92 | 13,562 | 9,802 | 4.4 |
| July 8th, 2003 | 32.5 | 30.9 | 102 | 5,531 | 1,009 | 3.1 |
| July 26th, 2003 | 32.8 | 31.3 | 118 | 6,394 | 1,931 | 3.1 |
| August 12th, 2003 | 33.7 | 31.4 | 76 | 6,078 | 1,702 | 3.8 |
| August 30th, 2003 | 27.6 | 29.7 | 74 | 8,366 | 2,122 | 4.9 |
| September 13th, 2003 | 32.1 | 30.2 | 40 | 4,271 | 1,001 | 2.6 |
| September 27th,2003 | 32.5 | 31 | 73 | 4,564 | 995 | 1.6 |

The analytical findings of *Microcystis* sp., heptadecane, and high TP concentration in the fish pond in 2003, indicated that the possibility of an algal bloom in the pond existed, and the bloom could have been the cause of the fish kills in the experimental aquaculture of 2002. The bloom could have also been the cause of the fish kills in the river. In the next section, the phenols were analyzed.

### 4.1.7 Phenols Analysis

Phenols, as toxic chemicals, were measured in the river water from July of 2002- April of 2003 at the same sampling locations as the 1999 and 2000 studies because the same transport model was planned to be used. The standard calibration curve of phenols and the results of phenol measurement are shown in Figure 4.4 and Table 4.5, respectively. Phenols found in the ppb range were consistent with those found in other rivers of Thailand

(Boonyatumanond, 2001). The locations where phenols were found most was around and below the Chot lagoon. There were only two segments, CT and ST in February of 2003 that phenols exceeded the standard of 5 μg/L. No phenols above the detection limit of 2 □g/L were found in the sediments in March and April of 2003. Therefore, no further measurement in the sediment was performed.

Phenol data from July - December of 2002 were planned for model calibration, and phenol data from January - April of 2003 for model validation. There was no source of phenols from the Dam, similar to the sediment. Through modeling, it was found that the flow from Creek Chot, particularly during the dry period, was not enough to generate high phenols in segments downstream. The model calibration of phenols thus could not be performed as planned.

Low phenols from July of 2002 and April of 2003, except at CT and ST in February, did not seem to be caused by the spill from the pulp mill. If the presence of phenols was due to the spill from the mill, there should have been no phenols detected in upstream segments above the Chot lagoon. It was observed during the study that whenever the water sample looked brownish, its phenol measurement seemed to show some value. This might be because brownish water contained natural compounds such as lignin, tannins, hemicelluloses, and flavonoids which could undergo degradation to produce phenols, as mentioned in Chapter 2. Also, whenever the water samples were picked up from locations, e.g., CT, ST and NW, near the river banks and aquatic weeds, there were phenols at the level of 1.1 - 7.8 ppb. This might be because phenols were released from parts of the plant, as phenols are ubiquitous in the plant kingdom (Kevin Robards, 2003). A model calibration of phenols could not be performed if the loading of phenols from each plant and natural compounds are not identified precisely.

From the phenol measurement and its modeling attempt, it could be concluded that phenols were not contributed from the mill's effluent. This conclusion was confirmed because the mill stopped releasing its effluent to Creek Chot since 1998. Moreover, the fish kills were not caused by phenols because, according to Table 4.5, there were no phenols at all on July 1st, 2002 when fish kills started. And on February 1st, 2003, there were some phenols at CT, ST and KP/BN, but no fish kills occurred.

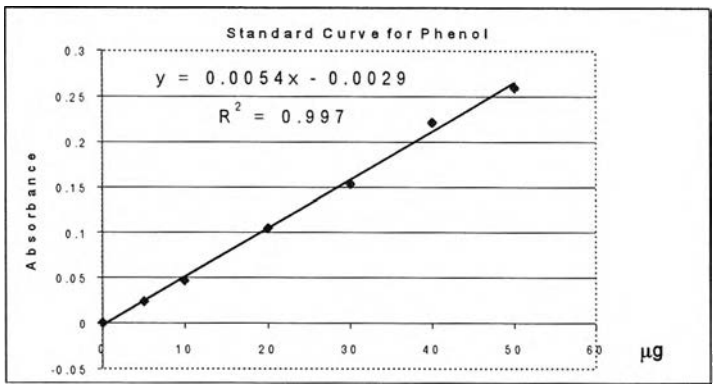



Figure 4.4 Standard calibration curve of phenols.

Table 4.5. Phenolic data in water at various locations, including locations B, C and D in Figure 3.8 from July of 2002 and July of 2003.

| Location | Jul 1st | Aug 3st | Sept 1st | Oct 5st | Dec 2st | Jan 1st | Feb 2st | Mar 3st | Apr 4st |
|---|---|---|---|---|---|---|---|---|---|
| Dam | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 |
| NS | 0 | 0 | 0 | 0 | 0 | 0 | 1.1 | 0 | 0 |
| NJ | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 |
| NP | 0 | 0 | 0 | 0 | 0 | <1 | 1.1 | 0 | 0 |
| KB | 0 | 0 | 0 | 0 | 0 | 2.6 | 1.1 | 0 | 0 |
| PS | 0 | 0 | 0 | 0 | 0 | 2.6 | 1.1 | 0 | 0 |
| CT | 0 | 0 | 0 | <1 | 0 | <1 | 4.8 | <1 | 0 |
| ST | 0 | 1.1 | 0 | <1 | 0 | 3.7 | 7.8 | <1 | 0 |
| KP/BN | 0 | 1.1 | 0 | <1 | 0 | 1.8 | 1.5 | <1 | 0 |

| | | | | | | | | | |
|---|---|---|---|---|---|---|---|---|---|
| NW | 0 | <1 | 0 | <1 | 0 | 3.3 | 2.6 | <1 | 0 |
| B (Outer Chot) | 0 | 0 | 0 | <1 | 0 | 1.8 | 3.3 | <1 | 0 |
| C (Pond) | 0 | 0 | 0 | <1 | 0 | 3.3 | 1.9 | <1 | 0 |
| D (behind Lake) | 0 | 0 | 0 | <1 | 0 | 11.9 | 3.7 | <1 | 0 |

## 4.2 MODELING RESULTS

This section begins with the modeling results of conventional nutrients. The model of phenols could not be performed for the reasons already explained. ON was not calibrated in this study because this data were derived from three other variables, namely TKN, $NH_3$-N and $NO_3$-N, and the error of ON would thus be the result of the addictive effect from these variables. As mentioned, high *Microcystis* sp. population, TP and a trace of heptadecane were found in the fish pond, suggesting that the possibility of an algal bloom killed fish in the pond, and the pond nearby existed. One way to find out whether the bloom could be the cause of the fish kills in the river, was to sample the water as often as possible, preferably every few days, and count the *Microcystis* sp. population as a function of the number of fish death.

The other method was to use modelling to simulate algae (measured as *Chl a*) under the conditions before and during the fish kill in the past from available monitoring studies. The first method would be the most convincing way to prove that the bloom was the cause of fish kill - if the bloom was significant enough with the subsequent fish kills in the year of monitoring. However, the first method was quite risky because it required open-ended effort and time in algal monitoring before the optimal condition for the bloom with the subsequently fish kill re-occurred by itself – assuming the bloom was the cause of the fish kill. If the bloom during monitoring was small and only a small number of fish died, it would have been very

difficult to trace the fish kill to the bloom in this river. Due to the time and budget constraint, and the availability of the 1999 and 2000 monitoring data in which the fish kill occurred in 1999, a mathematical modelling became the choice of this study, and the bloom was studied by simulating *Chl a*.

4.2.1 Results of the Calibration of Flow with Lignin/tannin

After the dynamic condition of the model was decided for studying the fish kill, the model was constructed step by step as described in Chapter 3. The transport model was first calibrated. Table 4.6 shows the result of the segment volumes after they have been adjusted during the hydrodynamic calibration. It should be pointed out that the volumes starting from segment PS became smaller than the previous segment. This was probably due to the pumping station of the pulp and paper mill which pumped about 44,000 $m^3$ of water from the river daily. The pull from the pumping station made the water travel faster than usual, and thus the segment volume seem smaller. Also, the sampling points were not exactly in the middle of the segment as desired. Some sampling points were located either above or below the middle of the segments because the 1999 and 2000 monitoring studies were not intended for the modeling purposes. Therefore, different segment volumes were inevitable.

Table 4.6 Segment Volumes after Calibration with Lignin/tannin.

| Segment No. | Name | Abbreviated Name | Volume after Calibration ($m^3$) | Remarks |
|---|---|---|---|---|
| 1 | Known Soong | NS | 900,000 | Upstream segment |
| 2 | Known Jik | NJ | 900,000 | |
| 3 | Segment 3 | Sgmt3 | 700,000 | |
| 4 | Nong Pur | NP | 700,000 | |
| 5 | Kum Bon | KB | 600,000 | |

| | | | | |
|---|---|---|---|---|
| 6 | Pumping Station | PS | 500,000 | |
| 7 | Chot (200 above) | CT | 500,000 | |
| 8 | Segment 8 | Sgmt8 | 400,000 | Chot Lake meets here |
| 9 | Sua Ten | ST | 450,000 | Lake Sua Ten meets here |
| 10 | KumPae/Bua Noi | KP/BN | 650,000 | |
| 11 | Segment 11 | Sgmt11 | 700,000 | Downstream segment |

After the calibration of the transport model, the regression analysis between the observed and predicted lignin/tannin yielded $R^2$ of 0.73, as shown in Figure 4.5. The comparison between the predicted and observed lignin/tannin at each segment is shown in Figure 4.6, with RMSE of 0.11 mg/L. Past studies used salinity to calibrate the flow of the estuaries, which could not be applied with the freshwater Pong River. In the model of Tha Chin River and Snohomish River, RMSEs were 0.03 (Simachaya, 1999) and 2.18 ppt (USEPA, 1995). The salinity RMSEs were in the ppt unit as the unit of salinity was ppt. If the RMSEs were compared without considering the unit, RMSE in this model fell within the range (0.11 vs. 0.03 and 2.18).

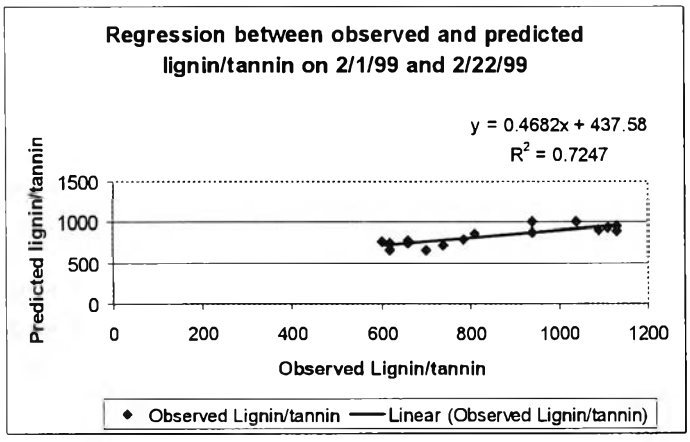



Figure 4.5 Regression between Observed and Predicted Lignin/tannin on February 1st and 22nd, 1999.

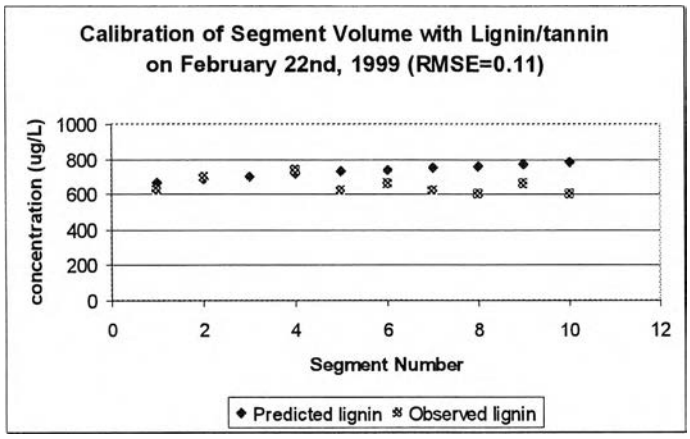



Figure 4.6 Comparison between predicted and observed lignin/tannin at each segment.

4.2.2 Results of Calibration of Runoff with Lignin/Tannin

After the Dam flow was calibrated in the model, the runoff was calibrated next with all lignin/tannin data in 1999. Figures 4.7 to 4.14 show the complete calibration with the 1999 lignin/tannin data, resulting in runoff for each segment as shown in Appendix M The multiple regression analysis of the lignin/tannin calibration yielded $R^2$ values of 0.75, 0.75, 0.70, 0.69, 0.73, 0.80, 0.75 and 0.64 for segments NS, NJ, NP, KB, PS, CT, ST and KP/BN, respectively. RMSEs were 0.12, 0.12, 0.14, 0.14, 0.13, 0.10, 0.12 and 0.20 mg/L for segments NS, NJ, NP, KB, PS, CT, ST and KP/BN, respectively, giving the average RMSE of 0.13 mg/L for the whole river.

Then the model was subsequently calibrated with the lignin/tannin data of 2000 to obtain the runoff in 2000. The same kinetic constant and coefficient values which were set at zero, in the 1999 model were used for the 2000 analysis. Exogenous variables such as river flows and temperature were changed in accordance to 2000 conditions. The results of the runoff calibration for each segment with the lignin/tannin data of 2000 are shown in Figures 4.15 - 4.22, and the runoff data for each segment are shown in Appendix N. The multiple

regression analysis of the validation results gave $R^2$ values of 0.88, 0.84, 0.88, 0.83, 0.85, 0.83, 0.62 and 0.64 for segments NS, NJ, NP, KB, PS, CT, ST and KP/BN, respectively. RMSEs were 0.02, 0.03, 0.02, 0.03, 0.03, 0.04, 0.05 and 0.05 mg/L for segment NS, NJ, NP, KB, PS, CT, ST and KP/BN, respectively, giving the average RMSE of 0.03 mg/L for the whole river.

After the transport model and the runoff were calibrated, it seemed to reproduce temporal and spatial distributions of lignin/tannin quite well. If the goodness of fit was judged by the profile as mentioned in Chapter 3, then this model demonstrated the goodness of fit because the predicted and observed lignin/tannin values showed similar profiles. Since this study introduced a novel method of calibrating the runoff from a conservative trace, RMSE in this study could not be compared with other studies. The difference between the predicted and observed values arose from the possible laboratory errors, and temporal difference between various measurements of lignin/tannin.

Calibration of the Transport Model with the 1999 Lignin/Tannin Data

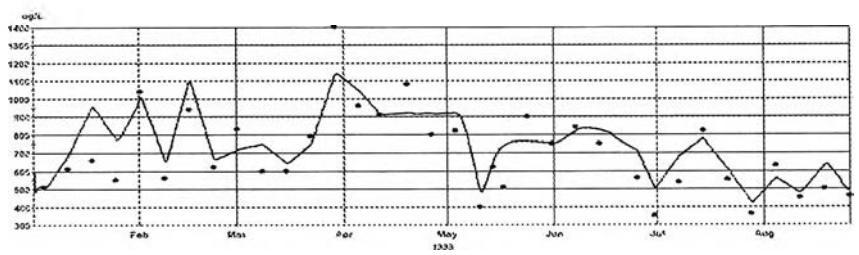


Figure 4.7 Model Calibration of Lignin/tannin at Segment NS. (line: predicted, dotted: observed).

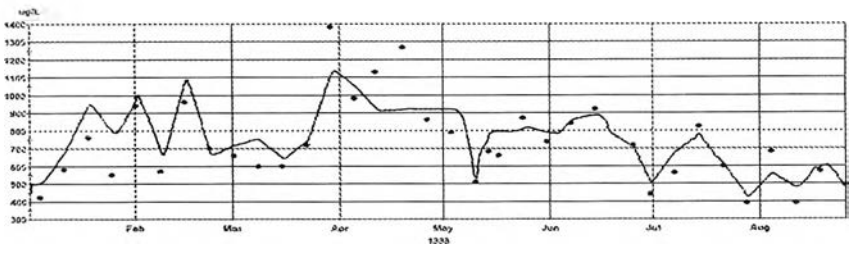


Figure 4.8 Model Calibration of Lignin/tannin at Segment NJ. (line: predicted, dotted: observed).

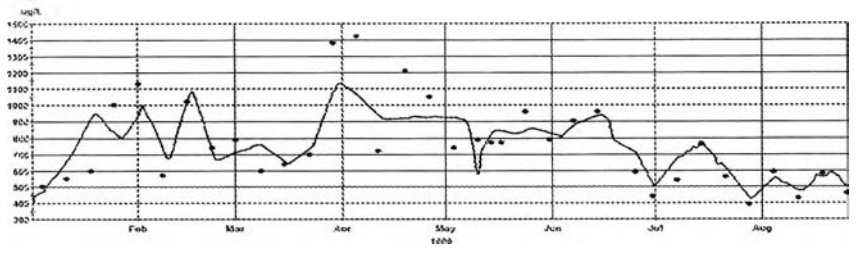


Figure 4.9 Model Calibration of Lignin/tannin at Segment NP. (line: predicted, dotted: observed).

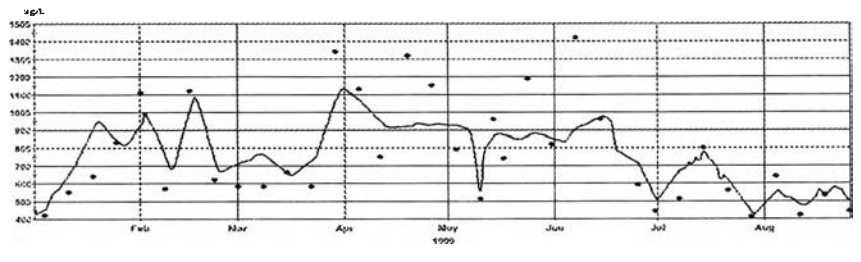


Figure 4.10 Model Calibration of Lignin/tannin at Segment KB (line: predicted, dotted: observed).

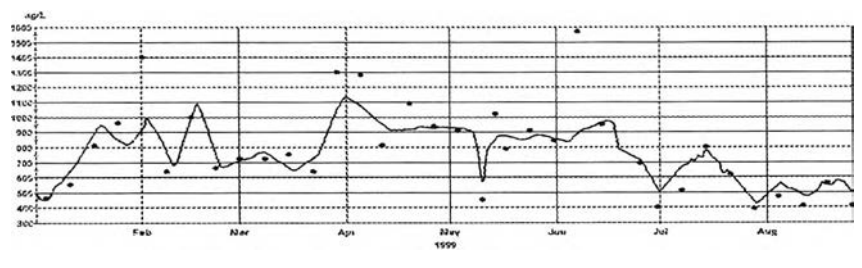


Figure 4.11 Model Calibration of Lignin/tannin at Segment PS. (line: predicted, dotted: observed).

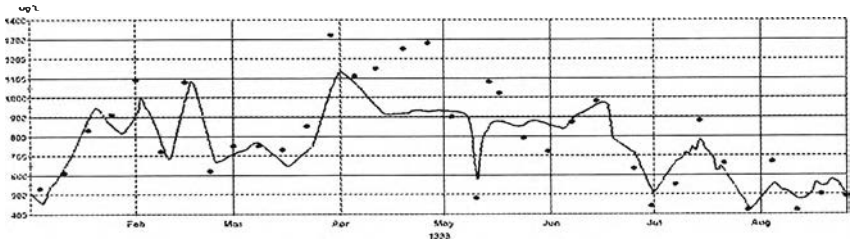



Figure 4.12 Model Calibration of Lignin/tannin at Segment CT. (line: predicted, dotted: observed).

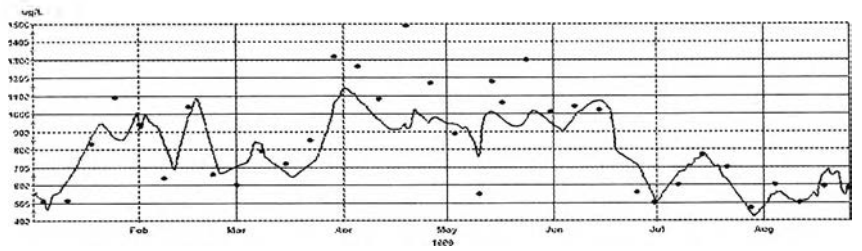



Figure 4.13 Model Calibration of Lignin/tannin at Segment ST. (line: predicted, dotted: observed).

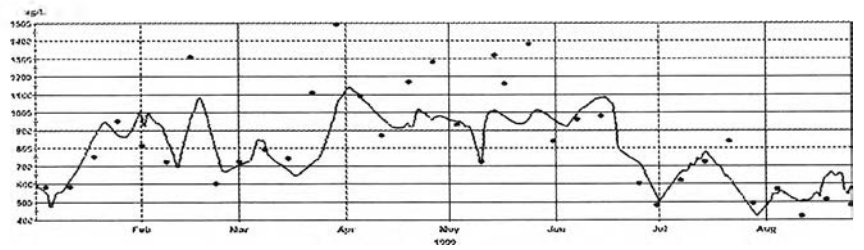



Figure 4.14 Model Calibration of Lignin/tannin at Segment KP/BN (line: predicted, dotted: observed).

## Calibration of the Transport Model with the 2000 Lignin/Tannin Data

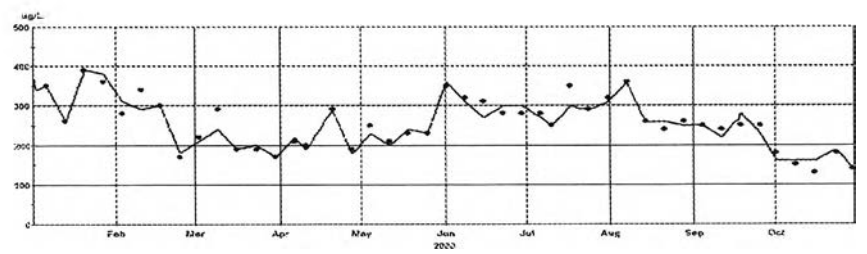



Figure 4.15 Model Calibration of Lignin/tannin at Segment NS (line: predicted, dotted: observed).

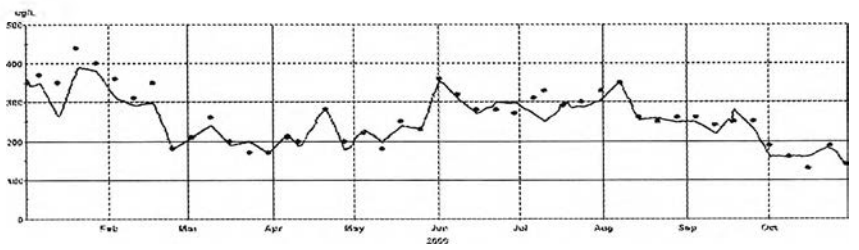


Figure 4.16 Model Calibration of Lignin/tannin at Segment NJ (line: predicted, dotted: observed).

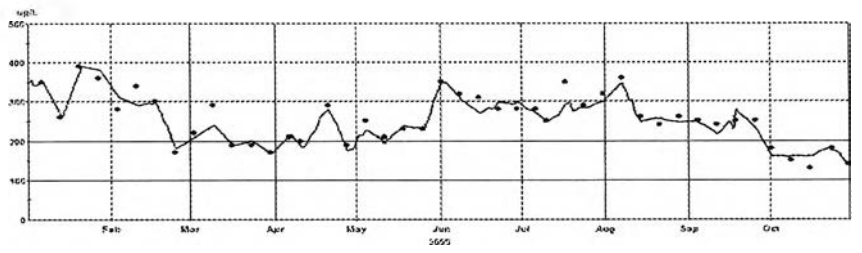


Figure 4.17 Model Calibration of Lignin/tannin at Segment NP (line: predicted, dotted: observed).

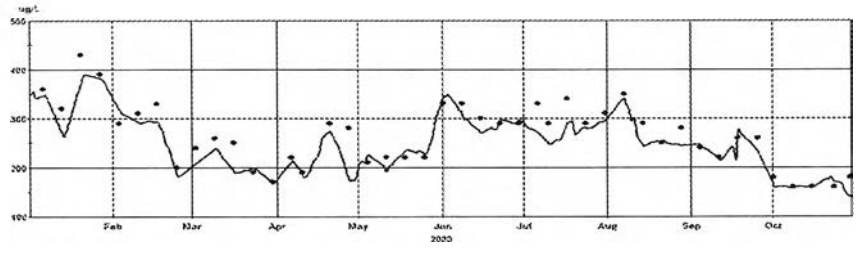


Figure 4.18 Model Calibration of Lignin/tannin at Segment KB (line: predicted, dotted: observed).

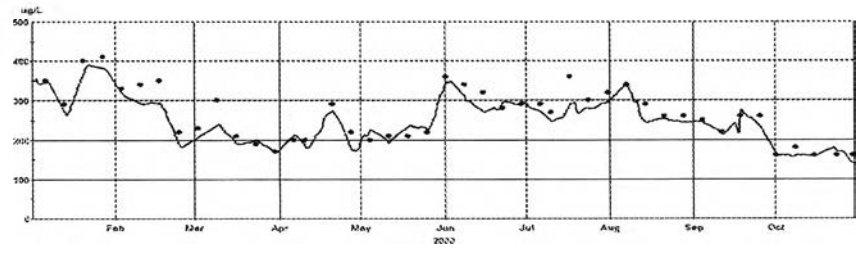


Figure 4.19 Model Calibration of Lignin/tannin at Segment PS (line: predicted, dotted: observed).

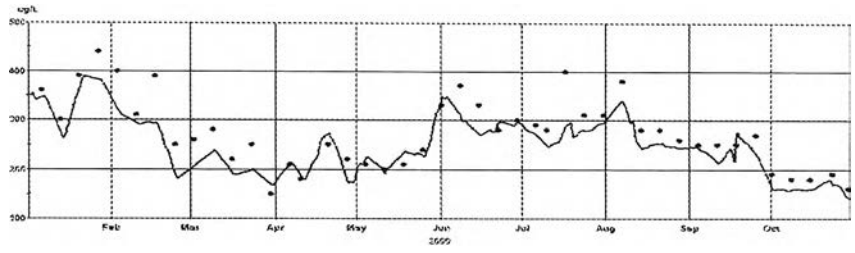


Figure 4.20 Model Calibration of Lignin/tannin at Segment CT (line: predicted, dotted: observed).

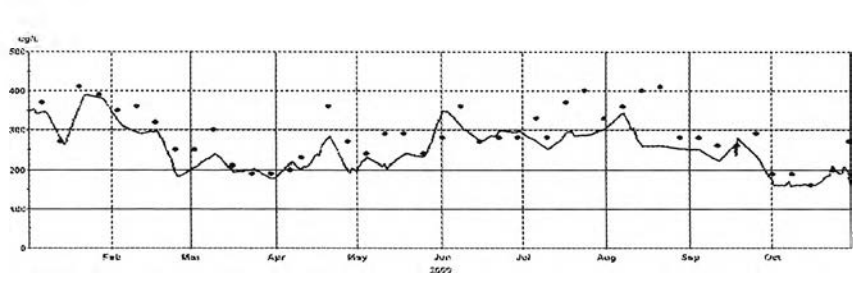


Figure 4.21 Model Calibration of Lignin/tannin at Segment ST(line: predicted, dotted: observed).

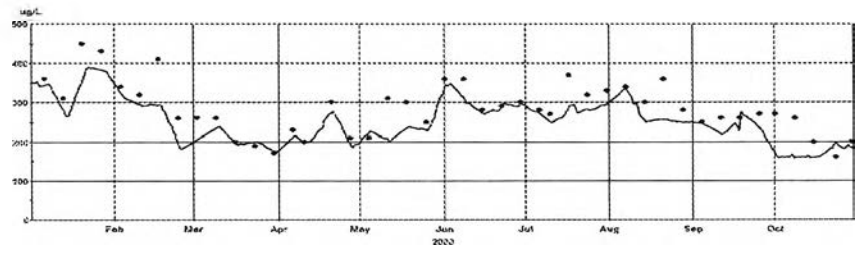


Figure 4.22 Model Calibration of Lignin/tannin at Segment KP/BN (line: predicted, dotted: observed).

### 4.2.3 Calibration and Validation of Conventional Nutrients

In model calibration, the chemical constants and coefficients were adjusted until the predicted and observed water quality data were in the closest proximity. The comparison of kinetic constants and coefficients between this model and other studies are shown in Table 4.7. This model's constants and coefficients fell within the ranges of past studies. The differences of the reaeration and deoxygenation rates between this model and Simachaya's model were

quite large, even though both models were constructed for rivers in the same country. These differences might be due to the fact that this model was simulated under dynamic conditions, while Simachaya's model was simulated under steady-state conditions.

Table 4.7 Comparison of Kinetic Constants between This Model with Others.

| Description | Units | Values in This Study | Values in Other Models | Reference |
|---|---|---|---|---|
| Reaeration Rate at $20^0$C | $Day^{-1}$ | 0.08 | 0.22 | Simachaya (1999) |
| Deoxygenation Rate at $20^0$C | $Day^{-1}$ | 0.08 | 0.21-0.61 | Ambrose *et a.l* (1993b) |
| | | | 0.18 | Bowie *et al.* (1985) |
| | | | 0.02 | Martin *et al.* (1996) |
| | | | 0.152 | Cusimano (1997) |
| | | | 0.6 | Suarez *et al.* (1995) |
| | | | 0.2 | Tetra Tech, Inc. (1995) |
| | | | 0.1-0.5 | Thomann and Muller (1987) |
| | | | 0.08 | Lung and Larson (1995) |
| | | | 0.30 | Simachaya (1999) |
| Temperature Coefficient of Deoxygenation | - | 1.047 | 1.047 | Ambrose *et al.* (1993b) |
| | | | 1.047 | Cusimano (1997) |
| | | | 1.02-1.08 | Bowie *et al.* (1985) |
| | | | 1.08 | Martin *et al.* (1996) |
| | | | 1.05 | Suarez *et al.* (1995) |
| | | | 1.05 | Simachaya (1999) |
| Nitrification Rate at $20^0$C | $Day^{-1}$ | 0.09 | 0.02-0.20 | Bowie *et al.* (1985) |
| | | | 0.08 | Martin *et al.* (1996) |
| | | | 0.20 | Cusimano (1997) |
| | | | 0.10 | Tetra Tech, Inc. (1995) |
| | | | 0.05 | Simachaya (1999) |
| Temperature Coefficient of | - | 1.08 | 1.02-1.08 | Bowie *et al.* (1985) |
| | | | 1.08 | Ambrose *et a.l* (1993b) |

| | | | | |
|---|---|---|---|---|
| Nitrification | | | 1.05 | and Martin *et al.* (1996)<br>Suarez *et al.* (1995)<br>Simachaya (1999) |
| Denitrification Rate at $20^0$C | $Day^{-1}$ | 0.16 | 0.09 | Wool *et al.*, (2000) |
| Temperature Coefficient of Denitrification | - | 1.045 | 1.045<br>1.045<br>1.045<br>1.05 | Cusimano (1997)<br>Martin *et al.* (1996)<br>Bowie *et al.* (1985)<br>Simachaya (1999) |
| Mineralization Rate at $20^0$C | $Day^{-1}$ | 0.075 | 0.10<br>0.10<br>0.01-0.4<br>0.20<br>0.05<br>0.30 | Cusimano (1997)<br>Martin *et al.* (1996)<br>Bowie *et al.* (1985)<br>Suarez *et al.* (1995)<br>Tetra Tech, Inc. (1995)<br>Simachaya (1999) |
| Temperature Coefficient of Mineralization | - | 1.08 | 1.045<br>1.07<br>1.08<br>1.05 | Cusimano (1997)<br>Martin *et al.* (1996)<br>Bowie *et al.* (1985)<br>Simachaya (1999) |

It should be pointed out that CBOD and DO from the beginning of April and end of June were not considered in the model calibration. The omission of data during this period was justified because there were fish kills on May $7^{th}$ -$8^{th}$, 1999, and if the algal bloom was the cause, the presence of live algae could affect the observed CBOD and DO. The algorithm of CBOD simulation did not account for live algae. Although the DO simulation accounted for increased oxygen from the algal photosynthesis, the amount of *Chl a* were unknown, and therefore could not be simulated to produce the desired level of DO. Similar to DO, unknown *Chl a* during the bloom and its die-off also affected $NH_3$ and $NO_3$.

Model Calibration and Validation of CBOD

The results of the CBOD calibration are shown in Figures 4.23-4.30. The multiple regression analysis of the CBOD calibration results gave $R^2$ values of 0.63, 0.7, 0.65, 0.72, 0.72, 0.69, 0.49 and 0.37 for segments NS, NJ, NP, KB, PS, CT, ST and KP/BN, respectively. The model calibrations of CBOD at segments ST and KP/BN gave $R^2$ values below 0.60. The carbonaceous biological oxygen demand RMSEs were 0.88, 0.69, 0.82, 0.66, 0.66, 0.69, 0.79 and 0.88 for segments NS, NJ, NP, KB, PS, CT, ST and KP/BN, respectively, giving the average RMSE of 0.76 mg/L for the whole river.

The results of the CBOD validation are shown in Figures 4.31-4.38. The multiple regression analysis of the CBOD validation results gave $R^2$ values of 0.60, 0.64, 0.64, 0.62, 0.56, 0.58, 0.40 and 0.44 for segments NS, NJ, NP, KB, PS, CT, ST and KP/BN, respectively. The $R^2$ values at segments PS, CT, ST and KP/BN were below 0.60. The carbonaceous biological oxygen demand RMSEs were 0.44, 0.35, 0.35, 0.43, 0.18, 0.44, 0.47 and 0.61 mg/L for segments NS, NJ, NP, KB, PS, CT, ST and KP/BN, respectively, giving the average RMSE of 0.41 mg/L for the whole river. It is worth noting that although $R^2$ of the validation models were not as high as those of the calibration models, the average RMSE of the validation models was lower than the calibration model.

With respect to the criteria for the goodness of fit, all CBOD calibration and validation results showed similar profiles between the predicted and observed CBOD. This model's average carbonaceous biological oxygen demand RMSEs were 0.76 and 0.41 mg/L, while the RMSE of the Tha Chin River model was reported to be 0.58 mg/L (Simachaya, 1999). Although this model was simulated under dynamic conditions, it still exhibited the "generality" in its prediction with RMSEs close to that of the steady-state model of the Tha Chin River.

Although some CBOD data were already eliminated during calibration, some CBOD data might still include the contribution from live algae. Considering that this model was dynamic and there was some contamination of live algae in CBOD, the RMSE of this model should be considered quite reasonable.

Factors for the discrepancy between the observed and predicted CBOD were probably due to the unavailable CBOD data inside Lake Sua Ten and the algal contribution on CBOD, which was discussed earlier. In the model validation, CBOD from the second sampling location, just below the Dam, on June 1st, 2000, was used as the boundary concentration. And, the CBOD concentrations calculated from the average values at the Dam and the second sampling location on January 6th, February 17th, May 25th, and June 15th were used as boundary concentrations. Although the distance between the Dam and the second location was very close to each other (200 m.), CBOD were however much different. It was definitely due to the algal concentrations. The boundary concentrations of CBOD thus must to be adjusted to the average between the two points.

Model Calibration of CBOD

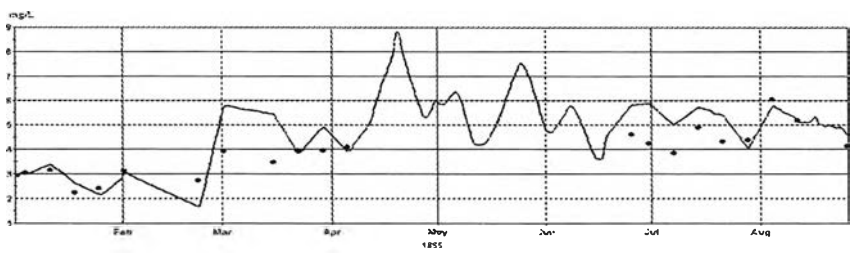


Figure 4.23 Model Calibration of CBOD at Segment NS (line: predicted, dotted: observed).

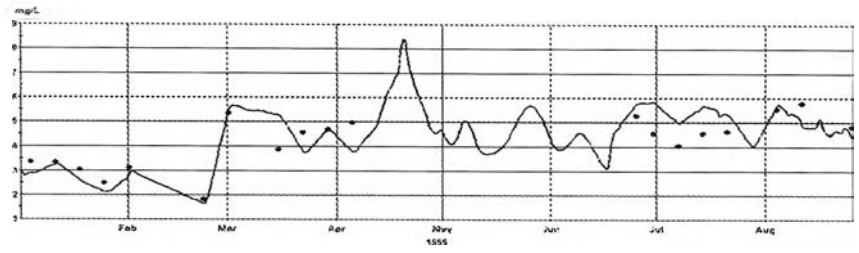


Figure 4.24 Model Calibration of CBOD at Segment NJ (line: predicted, dotted: observed).

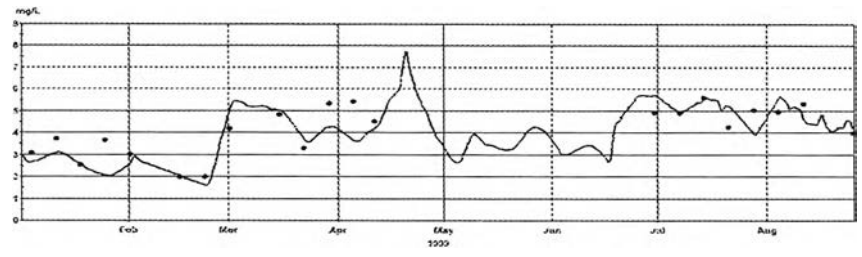


Figure 4.25 Model Calibration of CBOD at Segment NP (line: predicted, dotted: observed).

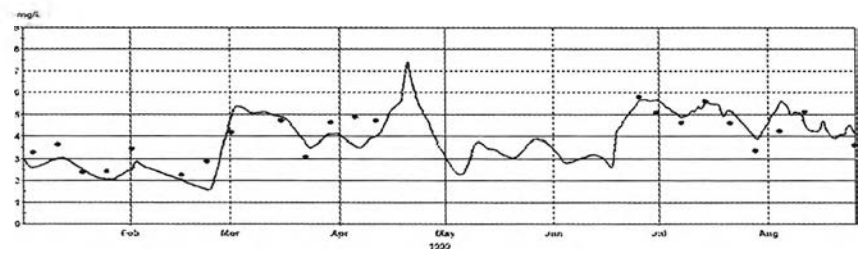


Figure 4.26 Model Calibration of CBOD at Segment KB (line: predicted, dotted: observed).

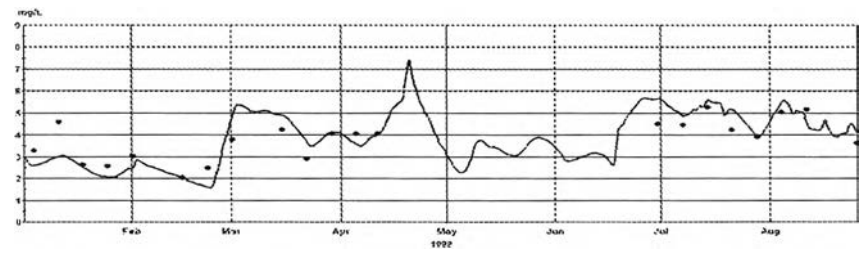


Figure 4.27 Model Calibration of CBOD at Segment PS (line: predicted, dotted: observed).

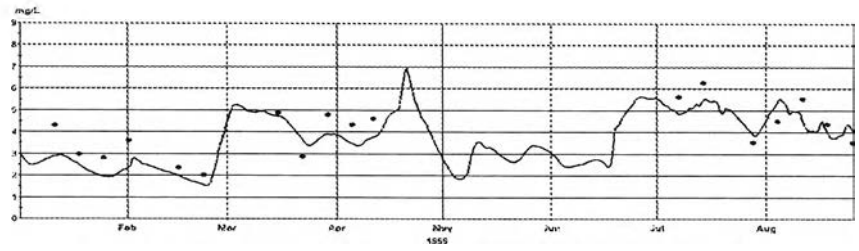



Figure 4.28 Model Calibration of CBOD at Segment CT (line: predicted, dotted: observed).

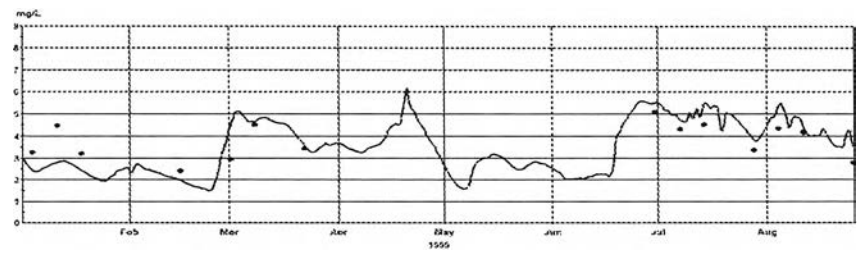



Figure 4.29 Model Calibration of CBOD at Segment ST (line: predicted, dotted: observed).

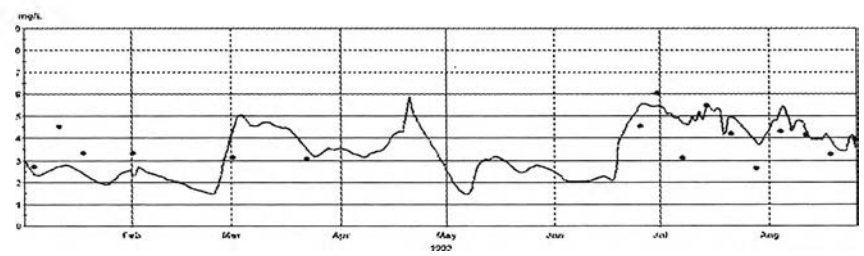



Figure 4.30 Model Calibration of CBOD at Segment KP/BN (line: predicted, dotted: observed).

## Model Validation of CBOD

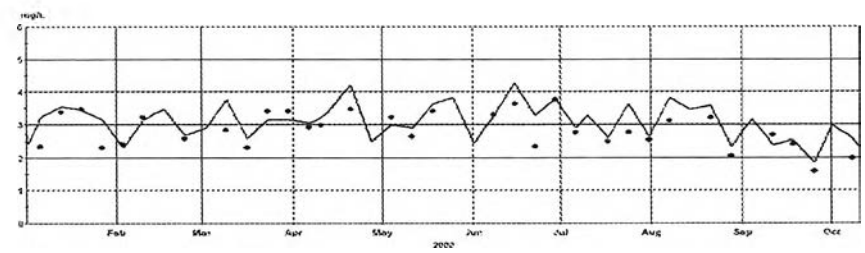



Figure 4.31 Model Validation of CBOD at Segment NS (line: predicted, dotted: observed).

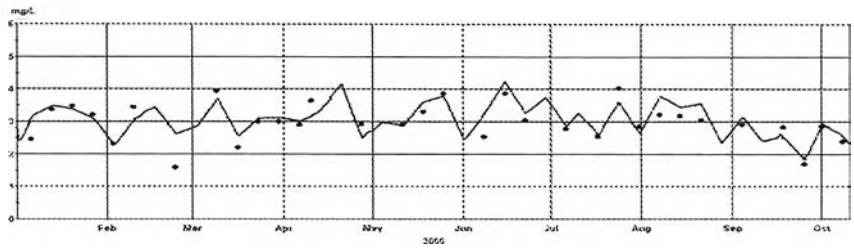



Figure 4.32 Model Validation of CBOD at Segment NJ (line: predicted, dotted: observed).

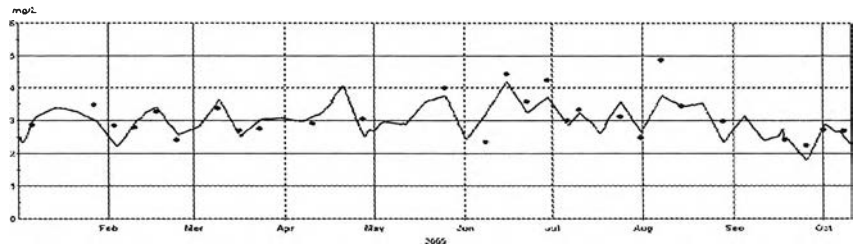



Figure 4.33 Model Validation of CBOD at Segment NP(line: predicted, dotted: observed).

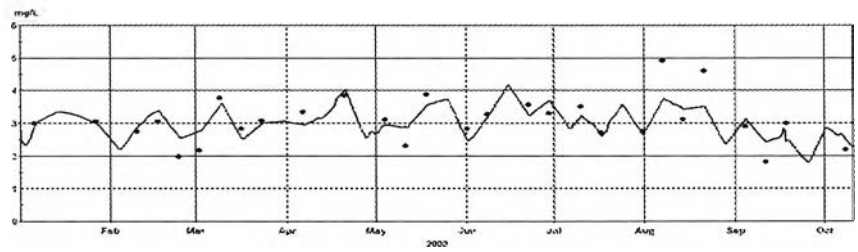



Figure 4.34 Model Validation of CBOD at Segment KB (line: predicted, dotted: observed).

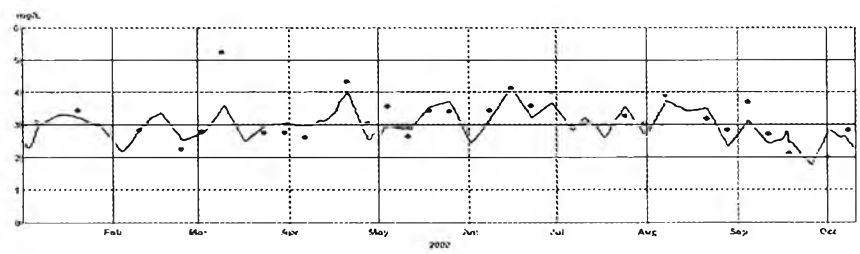



Figure 4.35 Model Validation of CBOD at Segment PS (line: predicted, dotted: observed).

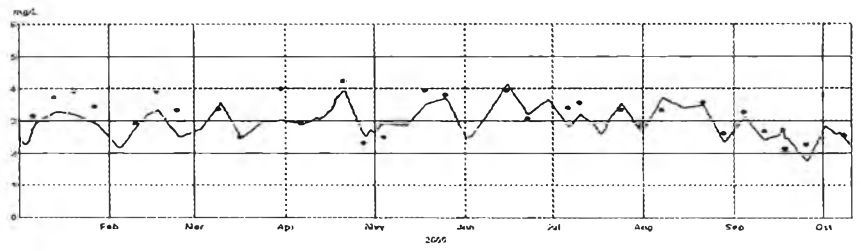


Figure 4.36 Model Validation of CBOD at Segment CT (line: predicted, dotted: observed).

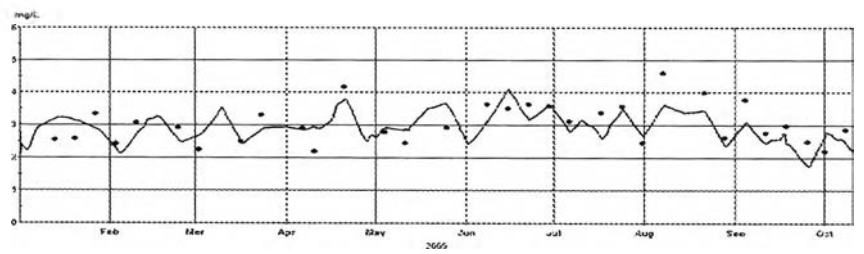


Figure 4.37 Model Validation of CBOD at Segment ST (line: predicted, dotted: observed).

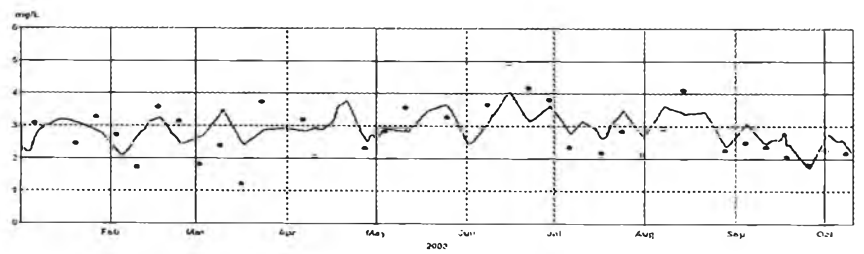


Figure 4.38 Model Validation of CBOD at Segment KP/BN.

## Model Calibration and Validation of DO

The results of the DO calibration are shown in Figures 4.38 - 4.46. The multiple regression analysis of the DO calibration gave $R^2$ values of 0.86, 0.72, 0.74, 0.77, 0.68, 0.6, 0.65, and 0.55 for segment NS, NJ, NP, KB, PS, CT, ST and KP/BN, respectively. The dissolved oxygen RMSEs were 0.65, 0.73, 0.73, 0.80, 0.83, 1.06, 0.86, and 1.04 mg/L for segment NS, NJ, NP, KB, PS, CT, ST and KP/BN, respectively, giving the average RMSE of 0.84 mg/L for the whole river.

The results of the DO validation are shown in Figures 4.47 - 4.54. The multiple regression analysis of the DO validation gave $R^2$ values of 0.94, 0.87, 0.83, 0.78, 0.78, 0.68, 0.61, and 0.64 for segments NS, NJ, NP, KB, PS, CT, ST and KP/BN, respectively. The dissolved oxygen RMSEs were 0.35, 0.49, 0.53, 0.62, 0.62, 0.73, 0.87, and 0.86 for segments NS, NJ, NP, KB, PS, CT, ST and KP/BN, respectively, giving the average RMSE of 0.63 mg/L for the whole river under study.

With respect to the criteria for the goodness of fit, all DO calibration and validation in this model showed similar profiles between the observed and predicted values. The average dissolved oxygen RMSEs (0.84 and 0.63 mg/L) in this model were also comparable to other models, considering that this model was simulated under dynamic conditions. In the model of Lake Vegoritis, the dissolve oxygen RMSEs ranged between 0.24 and 1.38, with an average value of 0.82 mg/L in 1991, and between 0.34 and 1.58, with an average of 1.02 mg/L in 1993 (Antonopoulos and Gianniou, 2003). In the models of the Puyallup, Spokane, and Tha Chin Rivers, the dissolved oxygen RMSEs were reported to be 0.20, 0.38, and 0.52 mg/L, respectively (Pelletier 1993, 1994; Simachaya, 1999). Given the fact that the Pong River was eutrophic, the average RMSEs of 0.84 and 0.63 mg/L were within a reasonable reported diurnal variation which could fluctuate as much as 8 and 7 mg/L in the Grand River and Snohomish River (O'Connor and Di Toro, 1970; US EPA, 1995).

Factors for the discrepancy between the observed and predicted DO were probably due to unavailable DO data inside Lake Sua Ten, and the contribution of the algal photosynthesis on the observed DO.

## Model Calibration of DO

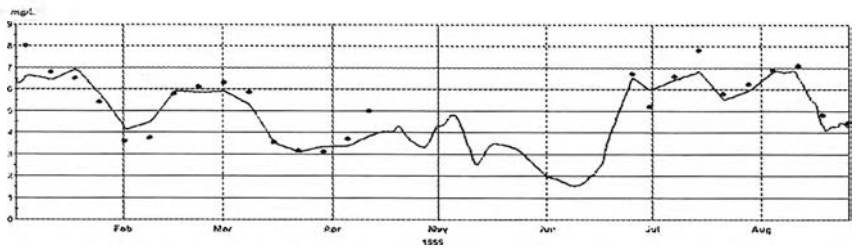



Figure 4.39 Model Calibration of DO at Segment NS (line: predicted, dotted: observed).

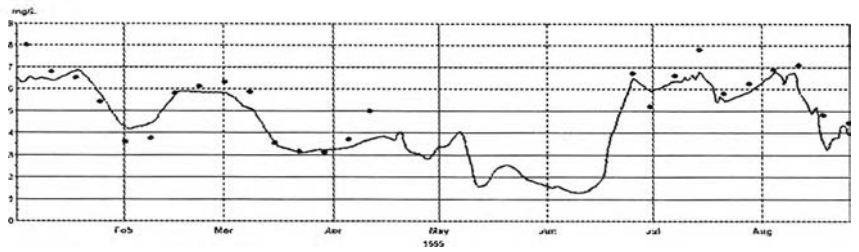



Figure 4.40 Model Calibration of DO at Segment NJ (line: predicted, dotted: observed).

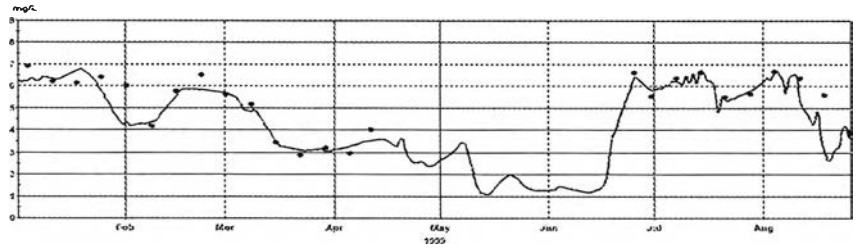



Figure 4.41 Model Calibration of DO at Segment NP (line: predicted, dotted: observed).

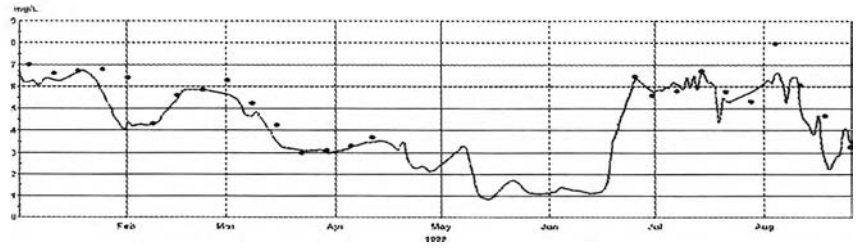



Figure 4.42 Model Calibration of DO at Segment KB (line: predicted, dotted: observed).

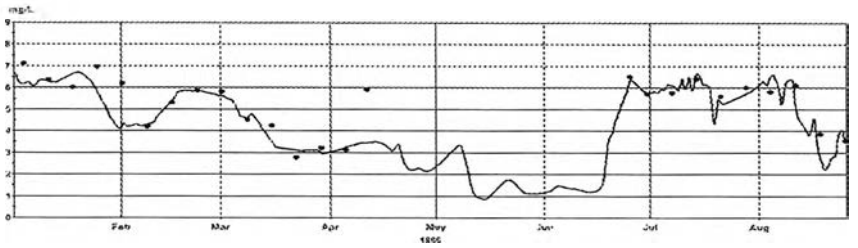


Figure 4.43 Model Calibration of DO at Segment PS (line: predicted, dotted: observed).

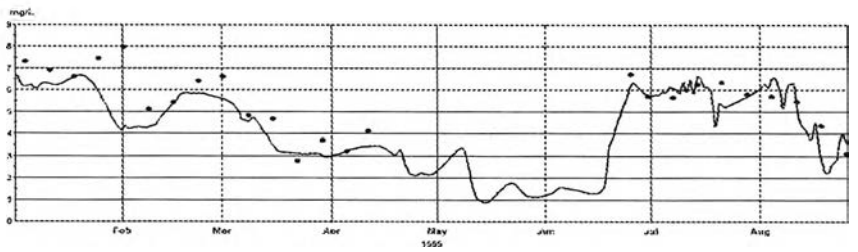


Figure 4.44 Model Calibration of DO at Segment CT (line: predicted, dotted: observed).

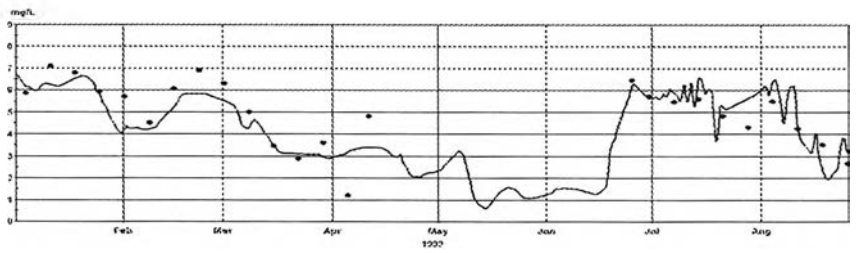


Figure 4.45 Model Calibration of DO at Segment ST (line: predicted, dotted: observed).

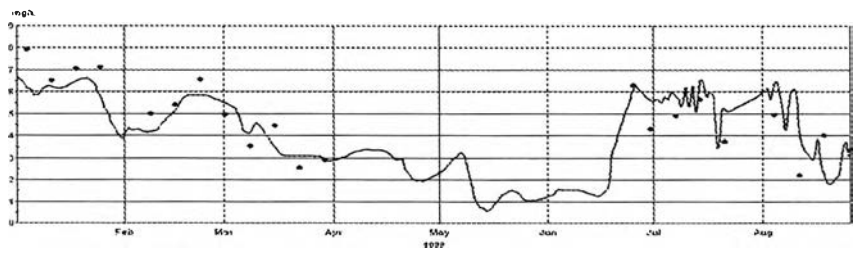


Figure 4.46 Model Calibration of DO at Segment KP/BN (line: predicted, dotted: observed).

## Model Validation of DO

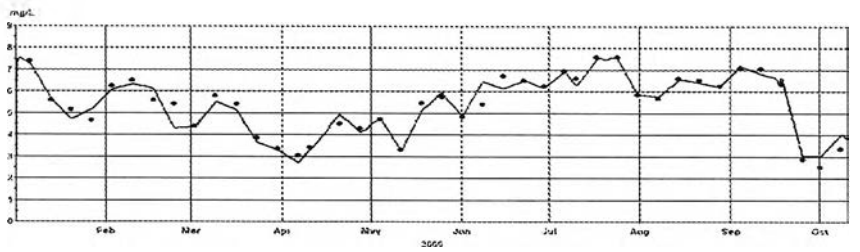



Figure 4.47 Model Validation of DO at Segment NS (line: predicted, dotted: observed).

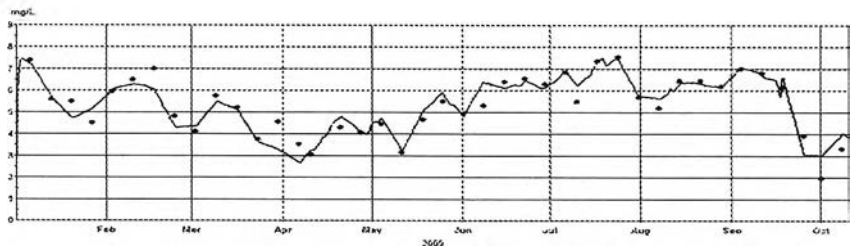



Figure 4.48 Model Validation of DO at Segment NJ (line: predicted, dotted: observed).

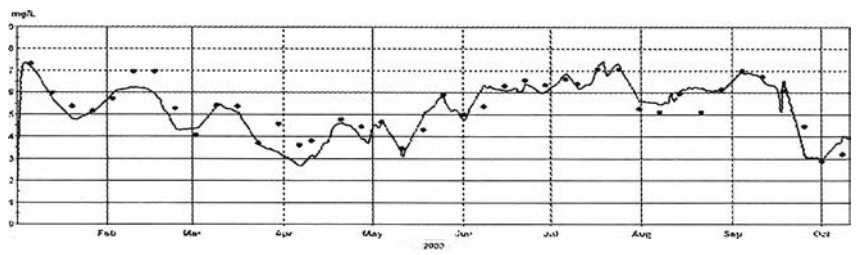



Figure 4.49 Model Validation of DO at Segment NP (line: predicted, dotted: observed).

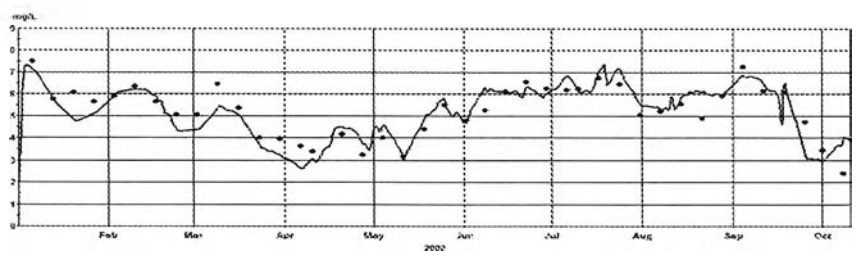



Figure 4.50 Model Validation of DO at Segment KB (line: predicted, dotted: observed).

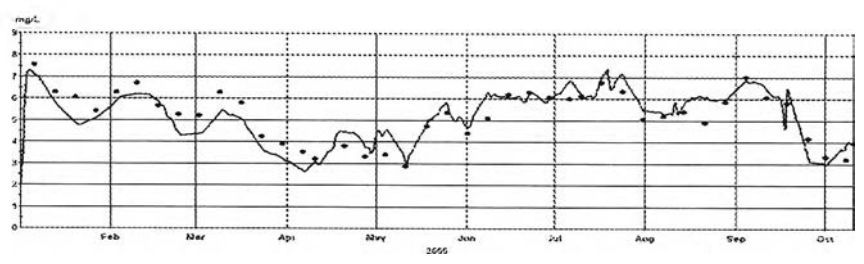


Figure 4.51 Model Validation of DO at Segment PS (line: predicted, dotted: observed).

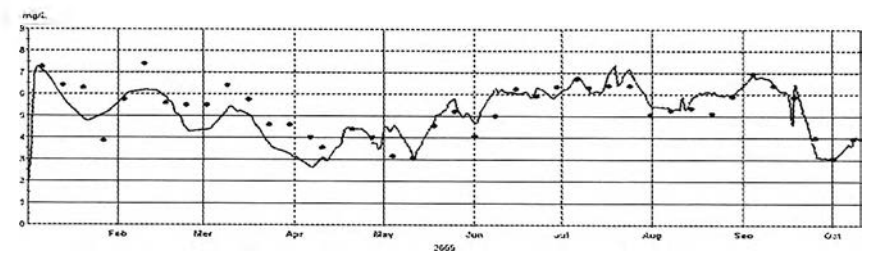


Figure 4.52 Model Validation of DO at Segment CT (line: predicted, dotted: observed).

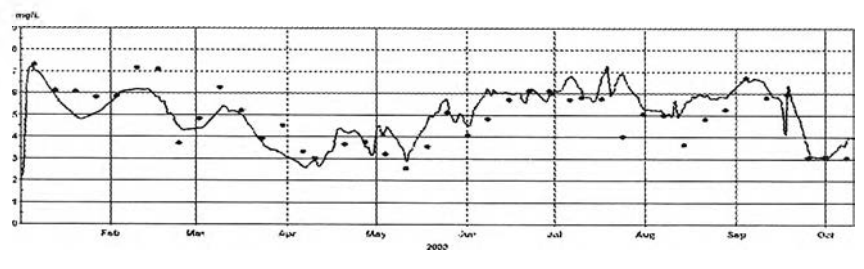


Figure 4.53 Model Validation of DO at Segment ST (line: predicted, dotted: observed).

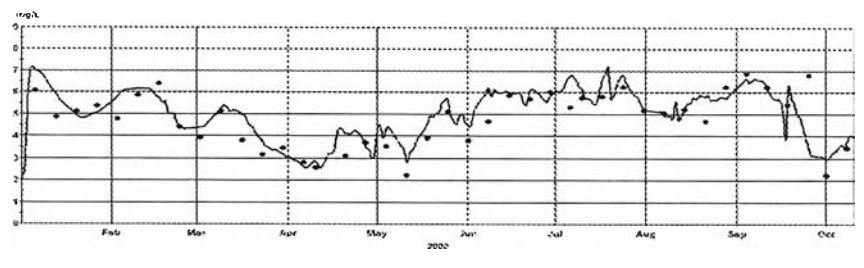


Figure 4.54 Model Validation of DO at Segment KP/BN (line: predicted, dotted: observed).

Model Calibration and Validation of $NH_3$-N

The results of the $NH_3$ calibration are shown in Figures 4.55-4.62. The multiple regression analysis of the $NH_3$ calibration gave $R^2$ values of 0.65, 0.71, 0.67, 0.63, 0.63, 0.64, 0.64, and 0.64 for segment NS, NJ, NP, KB, PS, CT, ST and KP/BN, respectively. The ammonia RMSEs were 0.06, 0.05, 0.06, 0.06, 0.06, 0.06, 0.06, and 0.06 mg/L for segment NS, NJ, NP, KB, PS, CT, ST and KP/BN, respectively, giving the average RMSE of 0.06 mg/L for the whole river.

The results of the $NH_3$ validation are shown in Figures 4.63-4.70. The multiple regression analysis of the $NH_3$ validation gave $R^2$ values of 0.83, 0.76, 0.66, 0.64, 0.66, 0.60, 0.53, and 0.51 for segments NS, NJ, NP, KB, PS, CT, ST and KP/BN, respectively. The ammonia RMSEs were 0.005, 0.01, 0.007, 0.01, 0.007, 0.008, 0.009, and 0.009 mg/L for segment NS, NJ, NP, KB, PS, CT, ST and KP/BN, respectively, yielding the average RMSE of 0.008 mg/L.

With respect to the criteria for goodness of fit, this model showed similar profiles between observed and predicted $NH_3$-N. When compared with other models, this model's average ammonia RMSEs of 0.06 and 0.008 mg/L were within the range. The ammonia RMSEs for the Spokane and Tha Chin Rivers model were reported to be 0.01 and 0.37 mg/L, respectively (Pelletier, 1994; Simachaya, 1999). The detection limit of the Nesslerization Method for measuring $NH_3$-N is 0.02 mg/L (APHA, 1975). As the standard calibration curve of this method was not published in the 1999 and 2000 studies, it could not be determined if the average ammonia RMSEs were within the error.

Factors for the discrepancy between the observed and predicted $NH_3$-N were probably due to unavailable $NH_3$ data inside Lake Sua Ten, the algal uptake of $NH_3$, and the re-suspension of nitrogen from the sediment.

## Model Calibration of $NH_3$-N

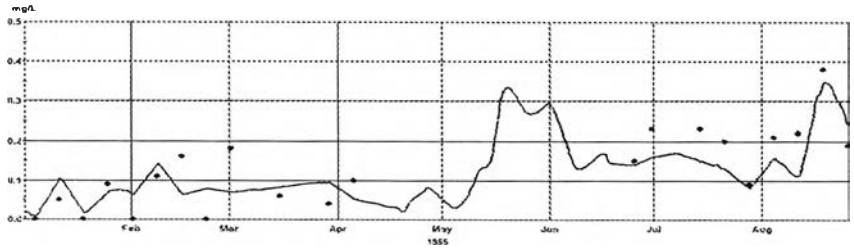


Figure 4.55 Model Calibration of $NH_3$-N at Segment NS (line: predicted, dotted: observed).

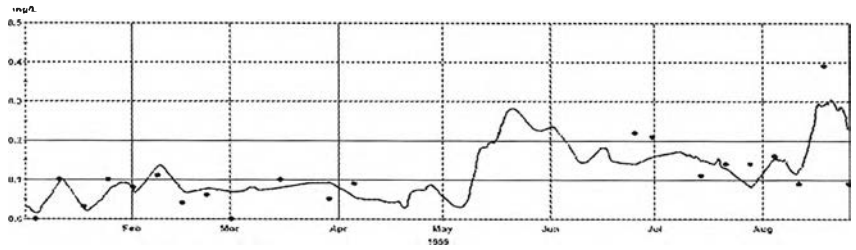


Figure 4.56 Model Calibration of $NH_3$-N at Segment NJ (line: predicted, dotted: observed).

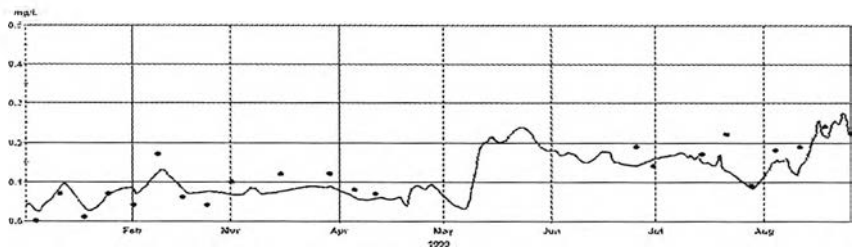


Figure 4.57 Model Calibration of $NH_3$-N at Segment NP (line: predicted, dotted: observed).

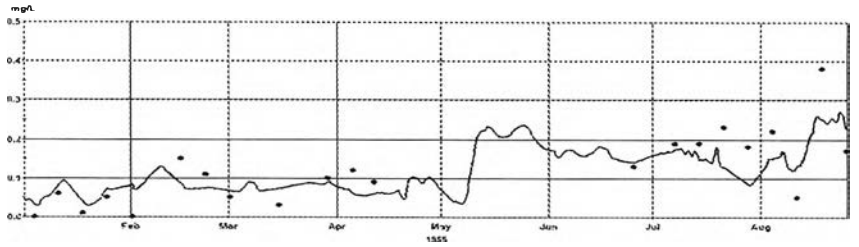


Figure 4.58 Model Calibration of $NH_3$-N at Segment KB (line: predicted, dotted: observed).

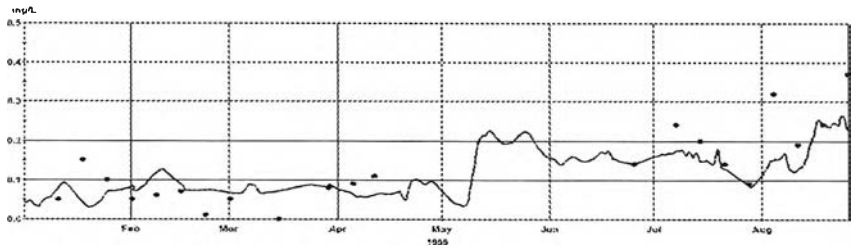


Figure 4.59 Model Calibration of $NH_3$-N at Segment PS (line: predicted, dotted: observed).

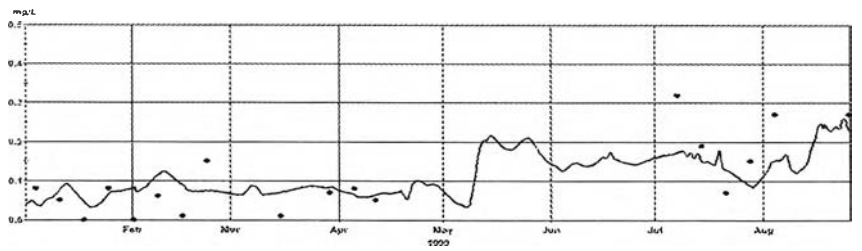


Figure 4.60 Model Calibration of $NH_3$-N at Segment CT (line: predicted, dotted: observed).

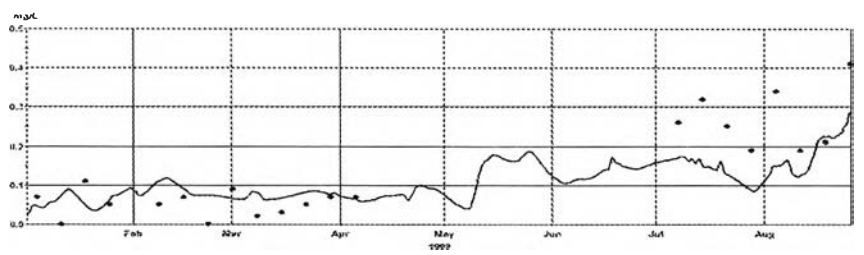


Figure 4.61 Model Calibration of $NH_3$-N at Segment ST (line: predicted, dotted: observed).

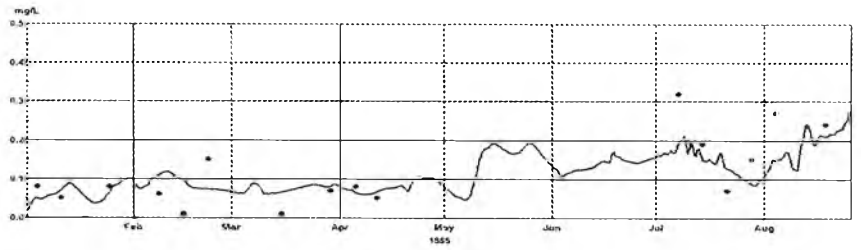


Figure 4.62 Model Calibration of $NH_3$-N at Segment KP/BN (line: predicted, dotted: observed).

## Model Validation of $NH_3$-N

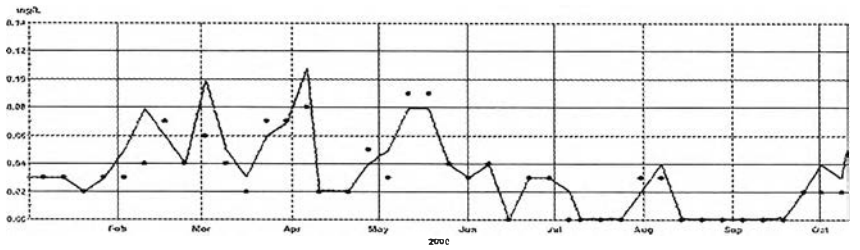


Figure 4.63 Model Validation of $NH_3$-N at Segment NS (line: predicted, dotted: observed).

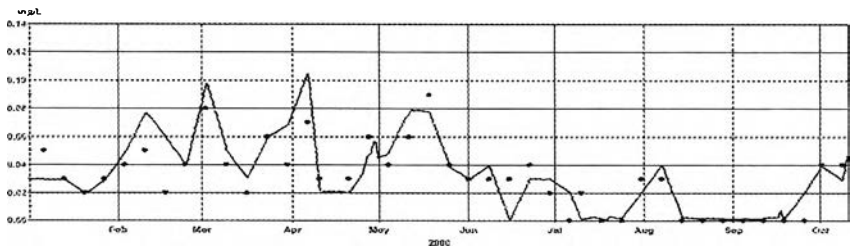


Figure 4.64 Model Validation of $NH_3$-N at Segment NJ (line: predicted, dotted: observed).

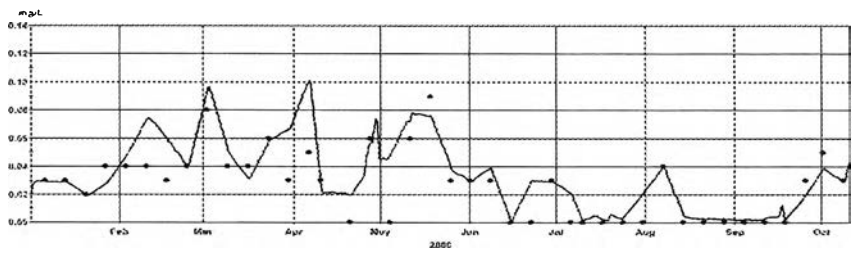


Figure 4.65 Model Validation of $NH_3$-N at Segment NP (line: predicted, dotted: observed).

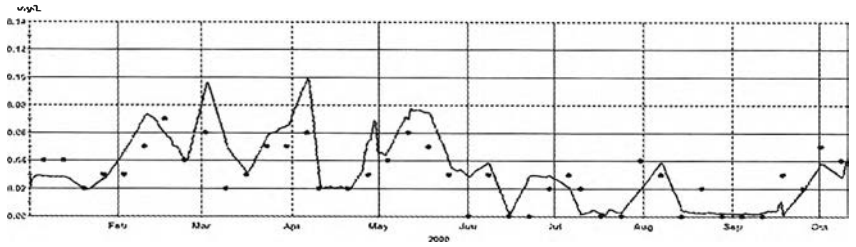


Figure 4.66 Model Validation of $NH_3$-N at Segment KB (line: predicted, dotted: observed).

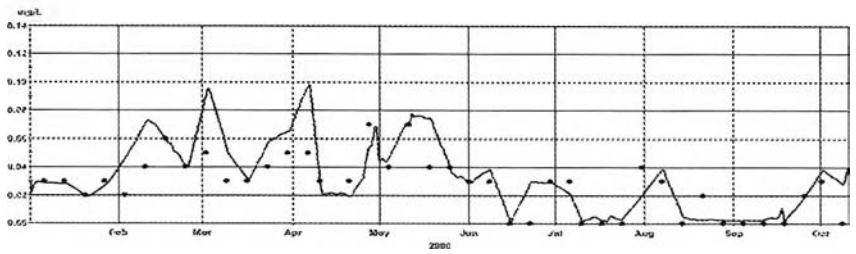


Figure 4.67 Model Validation of $NH_3$-N at Segment PS (line: predicted, dotted: observed).

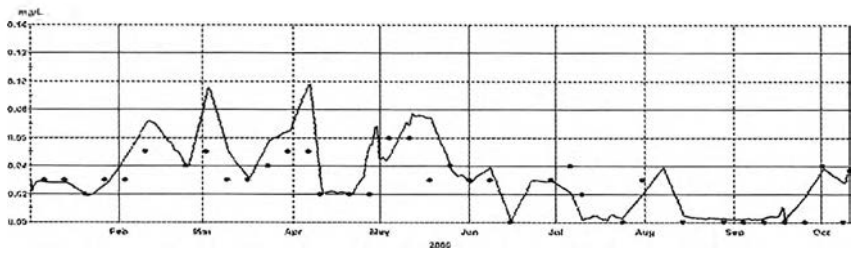


Figure 4.68 Model Validation of $NH_3$-N at Segment CT (line: predicted, dotted: observed).

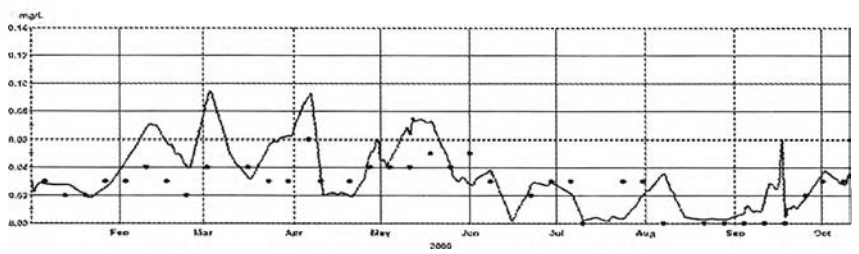


Figure 4.69 Model Validation of $NH_3$-N at Segment ST (line: predicted, dotted: observed).

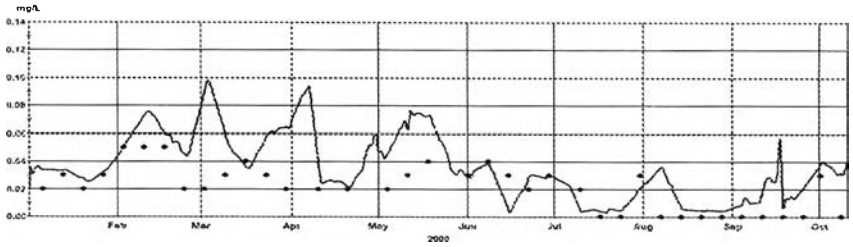


Figure 4.70 Model Validation of $NH_3$-N at Segment KP/BN (line: predicted, dotted: observed).

Model Calibration and Validation of $NO_3$-N

The results of the $NO_3$-N calibration are shown in Figures 4.71 - 4.78. The multiple regression analysis of the $NO_3$-N calibration gave $R^2$ values of 0.65, 0.71, 0.77, 0.63, 0.70, 0.71, 0.66, and 0.51 for segment NS, NJ, NP, KB, PS, CT, ST and KP/BN, respectively. The nitrate RMSEs were 0.05, 0.04, 0.03, 0.05, 0.04, 0.04, 0.05, and 0.06 for segments NS, NJ, NP, KB, PS, CT, ST and KP/BN, respectively, giving the average nitrate RMSE of 0.04 mg/L for the whole river.

The results of the $NO_3$-N validation are shown in Figures 4.79 - 4.86. The multiple regression analysis of the $NO_3$-N validation gave $R^2$ values of 0.94, 0.91, 0.70, 0.66, 0.70, 0.82, 0.66, and 0.58 for segment NS, NJ, NP, KB, PS, CT, ST and KP/BN, respectively. The nitrate RMSEs were 0.004, 0.004, 0.013, 0.015, 0.013, 0.006, 0.013 and 0.009 mg/L, respectively, giving the average RMSE of 0.01 mg/L for the whole river.

With respect to the criteria for goodness of fit, this model exhibited similar profiles between the observed and predicted $NO_3$-N. This model's average nitrate RMSEs of 0.04 and 0.01 mg/L were less than the reported RMSE of 0.16 mg/L at the Tha Chin River (Simachaya, 1999). There was no other published nitrate RMSE for comparison with this model. Similar to $NH_3$-N, it could not be determined if the average nitrate RMSEs were within the experimental error because of unpublished calibration curves in the 1999 and 2000 studies.

Factors for the discrepancy between the observed and predicted $NO_3$-N were probably due to unavailable $NO_3$ data inside Lake Sua Ten and possible algal uptake of $NO_3$ as its nutrient.

Model Calibration $NO_3$-N

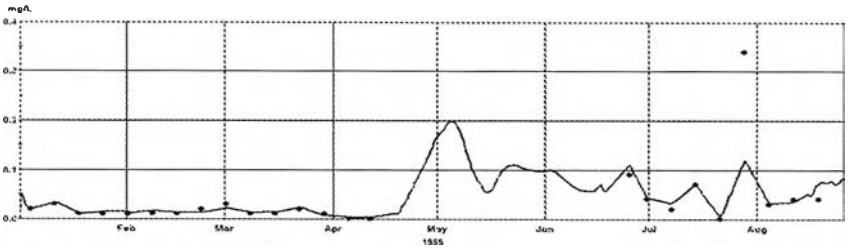


Figure 4.71 Model Calibration of $NO_3$-N at Segment NS (line: predicted, dotted: observed).

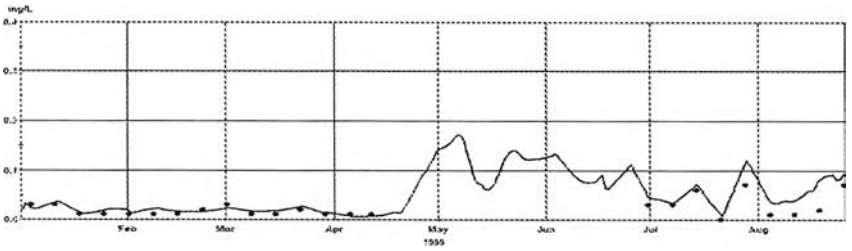


Figure 4.72 Model Calibration of $NO_3$-N at Segment NJ (line: predicted, dotted: observed).

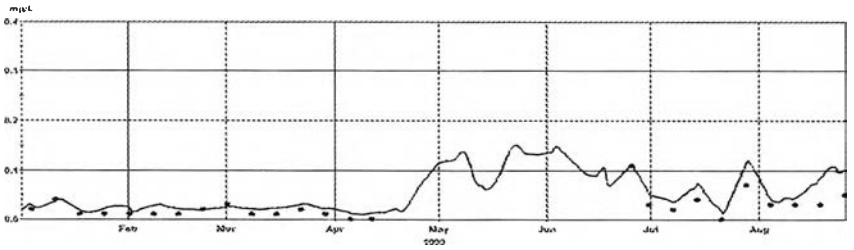


Figure 4.73 Model Calibration of $NO_3$-N at Segment NP (line: predicted, dotted: observed).

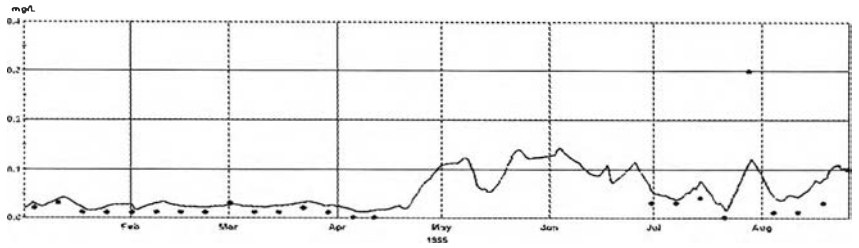


Figure 4.74 Model Calibration of $NO_3$-N at Segment KB (line: predicted, dotted: observed).

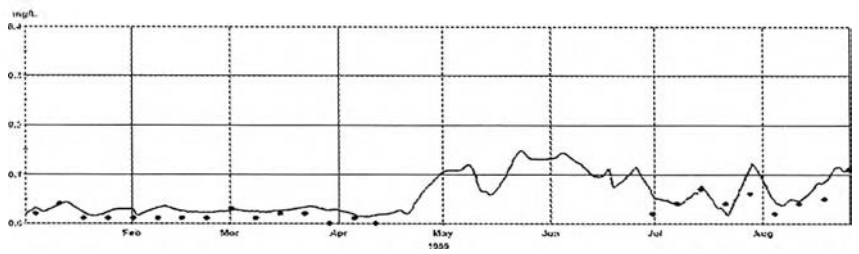


Figure 4.75 Model Calibration of $NO_3$-N at Segment PS (line: predicted, dotted: observed).

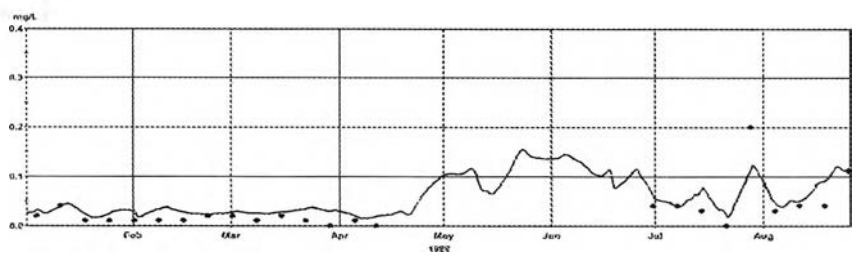


Figure 4.76 Model Calibration of $NO_3$-N at Segment CT (line: predicted, dotted: observed).

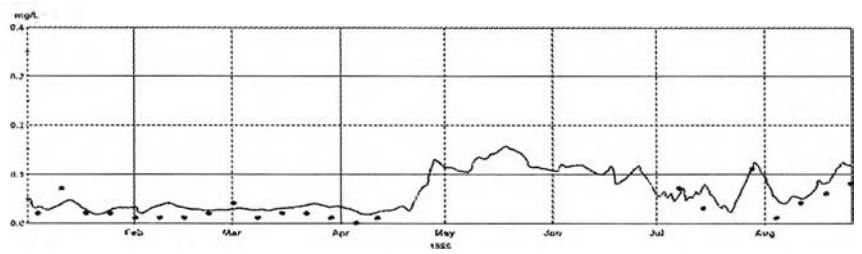


Figure 4.77 Model Calibration of $NO_3$-N at Segment ST (line: predicted, dotted: observed).

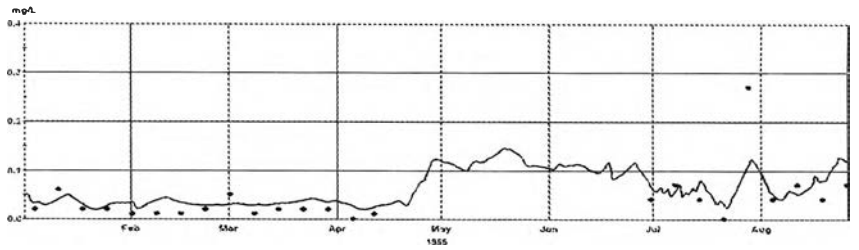


Figure 4.78 Model Calibration of $NO_3$-N at Segment KP/BN (line: predicted, dotted: observed).

## Validation of $NO_3$-N

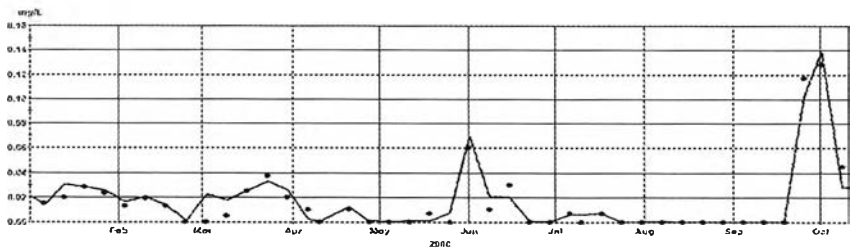


Figure 4.79 Model Validation of $NO_3$-N at Segment NS (line: predicted, dotted: observed).

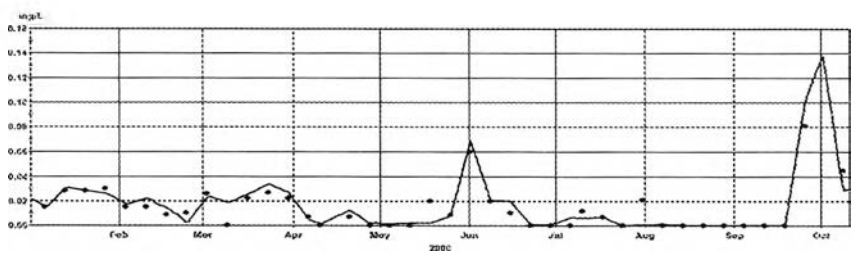


Figure 4.80 Model Validation of $NO_3$-N at Segment NJ (line: predicted, dotted: observed).

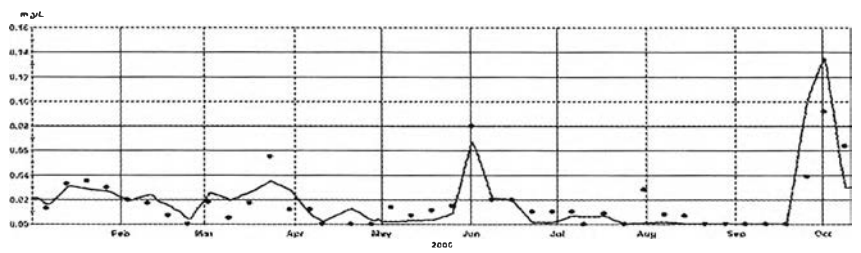


Figure 4.81 Model Validation of $NO_3$-N at Segment NP (line: predicted, dotted: observed).

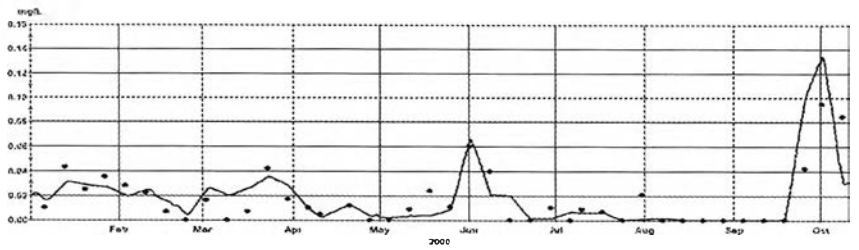


Figure 4.82 Model Validation of $NO_3$-N at Segment KB (line: predicted, dotted: observed).

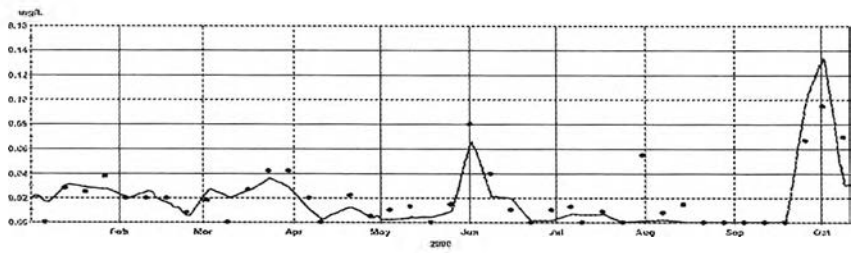


Figure 4.83 Model Validation of $NO_3$-N at Segment PS (line: predicted, dotted: observed).

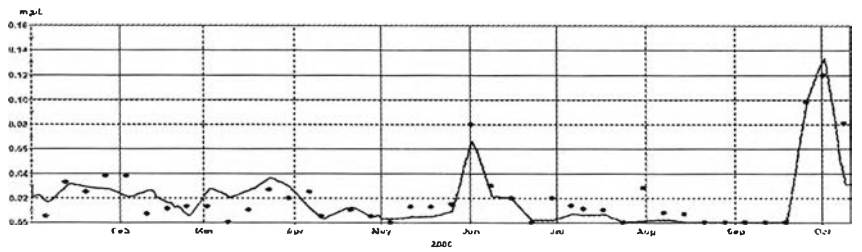


Figure 4.84 Model Validation of $NO_3$-N at Segment CT (line: predicted, dotted: observed).

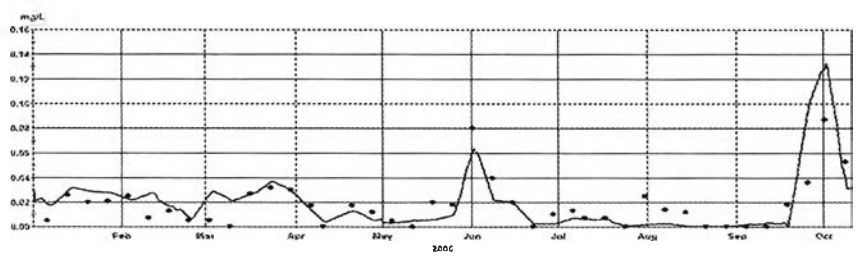


Figure 4.85 Model Validation of $NO_3$-N at Segment ST (line: predicted, dotted: observed).

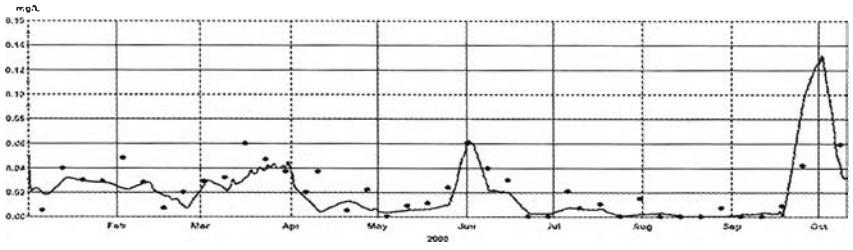


Figure 4.86 Model Validation of $NO_3$-N at Segment KP/BN (line: predicted, dotted: observed).

Overall, this model demonstrated the predictive capability of conventional nutrients for the future because its predictions "curve-fit" reasonably well with observed values. This model used more criteria for determining the goodness of fit than other models. This explained why there were limited RMSEs from other models to be compared with this model's. In the models of the Lower Neuse River (Lung and Paerl, 1988,) and Balatuin River (McAvoy *et al.*, 2003), no error analysis was performed to calibrate and validate their models. In the models of the Upper Mississippi River (Lung and Larson, 1995), the reservoir in the South Carolina, USA (Tufford and McKellar, 1999), Nadong River, Korea (Park and Lee, 2002), UK catchments (Boorman, 2003), Dender River (Demuynck *et al.*, 1997), and Duhernal Lake (Park and Uchrin, 1997; Park et al., 2003), the goodness of fit was judged by comparing only the profiles between the observed and predicted values.

## 4.3 MODEL PREDICTION

After calibration and validation, the model could be used to:

1) Investigate the cause-effect relationship of the fish kills on May 10$^{th}$, 1999.
2) Predict - through sensitivity analysis - which rate constants, coefficients and other variables merit particular attention for better environmental management of the river, and fish-kill prevention.

3) Recommend management solutions to prevent future fish kills.

4.3.1 Cause Analysis of Fish Kills

The causes of fish kills discussed in Chapter 2 were investigated by modeling in this section. The scenarios of the algal bloom, low DO, high BOD and high $NH_3$ were simulated and analyzed. Phenols, pesticides and heavy metals from experimental and existing data were qualitatively examined.

Cause Analysis of Fish Kills on the Algal Bloom

With the calibrated-validated dynamic model, real-time simulation of the condition before and during the fish-kill period of May $9^{th}$-$10^{th}$, 1999 was performed to study the cause of the fish kills. Since *Chl a* was not monitored in the river in 1999 and 2000 and could not thus be calibrated, the model could only predict the relative magnitude of *Chl a* from the typical phytoplankton constants and coefficients as shown in Table 4.8. The relative magnitude of uncalibrated *Chl a* could only be used for the comparison of the blooms in 1999 and 2000. As mentioned in Chapter 3, there were fish kills in 1999 and none in 2000; if the cause of the fish kills was due to the algal bloom, then the relative *Chl a* in 1999 should be predicted higher in 1999 than in 2000. In this analysis, different scenarios were simulated for the *Chl a* comparison, and all comparisons were made against the baseline scenario. The baseline scenario in this study, if not specified, was simulated with the runoff $PO_4$-P at 2.01 mg/L, under no existing *Chl a* in the river and no additional *Chl a* from the Dam and runoff. Figure 4.87 shows the baseline prediction of the relative *Chl a* in 1999.

Table 4.8 Phytoplankton Conditions for This Study with References.

| Phytoplankton Condition | This Model | Other Models | References |
|---|---|---|---|
| Phytoplankton Maximum Growth Rate @20°C | 2 | 2 | Jones and Federico,1984<br>Wool *et al.*, 2000 |
| Phytoplankton Growth Temperature Coefficient | 1.068 | 1.07<br>1.068 | Ambrose *et al.*, 1988<br>Wool *et al.*, 2000 |
| Phytoplankton Light Formulation Switch | 1 | 1 | Di Toro *et al.*, 1978 |
| Maximum Quantum Yield Constant | 720 | 720 | Wool *et al.*, 2000 |
| Self-Shading Extinction | 0.017 | 0.017 | Wool *et al.*, 2000 |
| Carbon:Chlorophyll Ratio | 50 | 50<br>20-50 | Bowie *et al.*,1985<br>Wool *et al.*, 2000 |
| Optimal Light Saturation | 300 | 200-500 | Wool *et al.*, 2000 |
| Half-Saturation Constant for Nitrogen | .005 | 0.015 | Bowie *et al.*,1985 |
| Half-Saturation Constant for Phosphorus | .003 | 0.003 | Bowie *et al.*,1985 |
| Endogenous Respiration Rate @20°C | 0.125 | 0.15<br>0.125 | Bowie *et al.*,1985<br>Wool *et al.*, 2000 |
| Respiration Temperature Coefficient | 1.045 | 1.045 | Wool *et al.*, 2000 |
| Death Rate Non-Zooplankton Predation | 0.02 | 0.5<br>0.02 | Bowie *et al.*,1985<br>Wool *et al.*, 2000 |
| Zooplankton Grazing Rate | 0.02 | 1<br>0 | Bowie *et al.*,1985<br>Wool *et al.*, 2000 |
| Phosphorus:Carbon Ratio | .025 | .025 | Wool *et al.*, 2000 |
| Nitrogen:Carbon Ratio | 0.25 | 0.25 | Wool *et al.*, 2000 |
| Half-Saturation for Recycle of Nitrogen and Phosphorus | 0.5 | 0.5<br>0.5 | Ambrose *et al.*, 1988<br>Wool *et al.*, 2000 |
| Fraction Daily Light | 0.58 | 0.3-0.7 | Wool *et al.*, 2000 |

Scenario 1: Baseline Scenario

In the simulation result of the baseline scenario (Scenario 1), as shown in Figure 4.87, there were two *Chl a* peaks on May 4$^{th}$ and June 4$^{th}$, 1999. Both algal blooms gradually

increased from segments NS to CT in the direction from upstream to downstream, but started to drop off at ST and KP/BN, due to dilution from the Chot lagoon and Lake Sua Ten where no algae was assumed to previously exist. Figure 4.87 only shows simulations at segments CT, ST and KP/BN for easy identification. When the same model was simulated with conditions of 2000, no *Chl a* was observed at all locations (Figure not shown).

The comparison of simulated *Chl a* between 1999 and 2000 demonstrated that conditions of the Dam flows, temperature and runoff from approximately April 27$^{th}$ - June 17$^{th}$, 1999 were enough to cause two potential algal blooms as shown by two peaks - without any existing algae in the river. The first bloom started on April 27$^{th}$, 1999, peaked on May 4$^{th}$, 1999 and rapidly disappeared on May 10$^{th}$, 1999, possibly because of lack of nutrients or low light extinction. The second bloom peaked on June 3$^{rd}$, 1999 and disappeared on June 18$^{th}$, 1999, possibly because high Dam flows flushed out the bloom. Coincidently, during April 27$^{th}$ - June 18$^{th}$, 1999, the Dam reduced its water release to less than 1 MCM, and the temperature was high. In addition, there was agricultural runoff with ample supply of nutrients for algal growth.

The notable growth of algae from April 27$^{th}$-June 18$^{th}$, 1999 was detected as high observed CBOD, which caused a large difference between the predicted and observed CBOD at all sampling sites in the river. Figure 4.88 shows an example of the mentioned large difference between the predicted and observed CBOD at CT from April 27$^{th}$ - June 18$^{th}$, 1999. The predicted CBOD was lower than the observed CBOD because the predicted CBOD had no contribution from live algae.

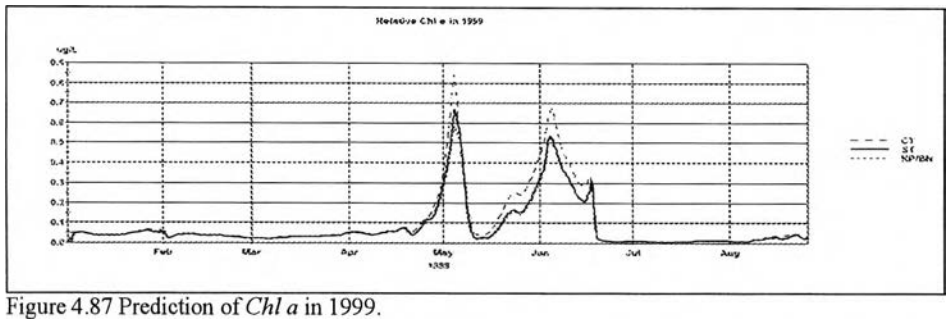


Figure 4.87 Prediction of *Chl a* in 1999.

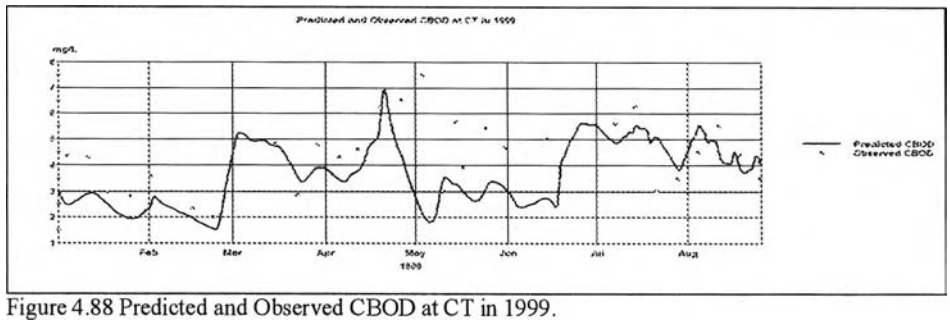


Figure 4.88 Predicted and Observed CBOD at CT in 1999.

Scenario 2: What if there was an existing algae of 4 μg/L in the river on April 26th, 1999? (The concentration of 4 μg/L was arbitrarily chosen from a range of 2.4-4.5 μg/L measured at the CT aquaculture in 2003)

In this scenario, the initial *Chl a* in each segment was set at 4 □g/L, which was within the range of the observed *Chl a* at the CT aquaculture in Table 4.2. The simulation was set to start on April 26th, 1999, not January 1st, 1999, because if the run started earlier, *Chl a* would have reduced by itself due to the cellular respiration (aging) and lack of runoff nutrients. Usually, dead algae from the benthic layer of the river could recycle its nitrogen and phosphorus components to the water column for continual algal growth. However, this model could not simulate this recycle process because the benthic nitrogen and phosphorus data in the 1999 and 2000 studies were not available for modeling. Runs with different starting dates were also tried to examine the potential magnitude of *Chl a* as a function of the starting dates.

The simulation result of Scenario 2 is shown in Figure 4.89. With existing concentration of 4 μg/L, *Chl a* at NP, CT, ST and KP/BN on May 4th, 1999 increased from 0.35, 0.84, 0.65 and 0.57 μg/L in the baseline scenario to 1.77 (406%), 4.25 (406%), 4.12 (534%), and 4.35 μg/L (1339%), respectively. Algae in the Chot lagoon also increased from zero to 7.65 μg/L on May 4th, 1999. Contrary to the baseline scenario, *Chl a* at ST and KP/BN was as larger as at CT due to the addition of algae from the Chot lagoon.

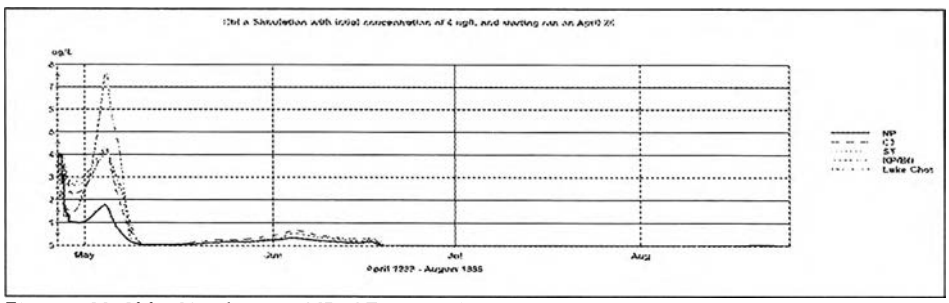



Figure 4.89 *Chl a* Simulation at NP, CT, ST, and KP/BN with initial *Chl a* of 4 μg/L and starting run on April 26th, 1999.

The best starting date for the simulation which yielded the highest percent increase of the bloom was April 28th, 1999; *Chl a* increased by 540, 462, 594, and 704% at NP, CT, ST and KP/BN, respectively. Algae in the Chot lagoon increased as high as 7.31 μg/L. The second bloom in Figure 4.89, on the other hand, did not change in magnitude. An interpretation of the second bloom must be made with precaution. When the first bloom die-off occurred, *Chl a* dipped to approximately zero; thus the simulation of the second bloom started from the initial *Chl a* of zero, not 4 μg/L. The sharp *Chl a* dip to zero after the first bloom was due to the imperfection of the model to sustain the algal population by recycling nitrogen and phosphorus of dead algae.

Scenario 3: What if there was an algal addition of 5 μg/L from the Dam to the river with the existing algae of 4 □g/L from April 19th - May 3rd, 1999?

This scenario was very likely to happen because high CBOD and DO were observed in the reservoir from April 19th - May 3rd, 1999, as shown in Figure 4.90. Possibly, high CBOD in the reservoir from April 19th - May 3rd, 1999, was due to live algae from a bloom. The high nitrate in the reservoir which peaked on May 3rd, 1999, must have caused this bloom (Figure 3.4). Nitrate on April 19th, 1999 was not high in the reservoir, possibly because, as soon as nitrate was added to the reservoir, it was immediately and rapidly taken up by the existing non-$N_2$ fixing cyanobacterial genus, *Microcystis* sp. If nitrate in the reservoir was due to the runoff or leaching, nitrate could possibly add to the reservoir on April 19th, 1999 because from January 1st - April 19th, 1999, there was an average rainfall of 0.20 m, and on a single day of April 18th, 1999, there was as much as 0.06 m of rainfall.

The initial *Chl a* at the Dam was set at 5 μg/L because this concentration was usually found in the river, around the CT aquaculture when there was no fish kill. The result of the simulation (Figure 4.91), shows that *Chl a* at NP, CT, ST and KP/BN on May 4th, 1999 increased from 0.35, 0.84, 0.65 and 0.57 μg/L in the baseline scenario to 13.7 (3814%), 14.4 (1614%), 11.6 (1685%), and 10.3 μg/L (1707%), respectively. *Chl a* in the Chot lagoon also increased from 0 to 9.6 μg/L on May 4th, 1999.

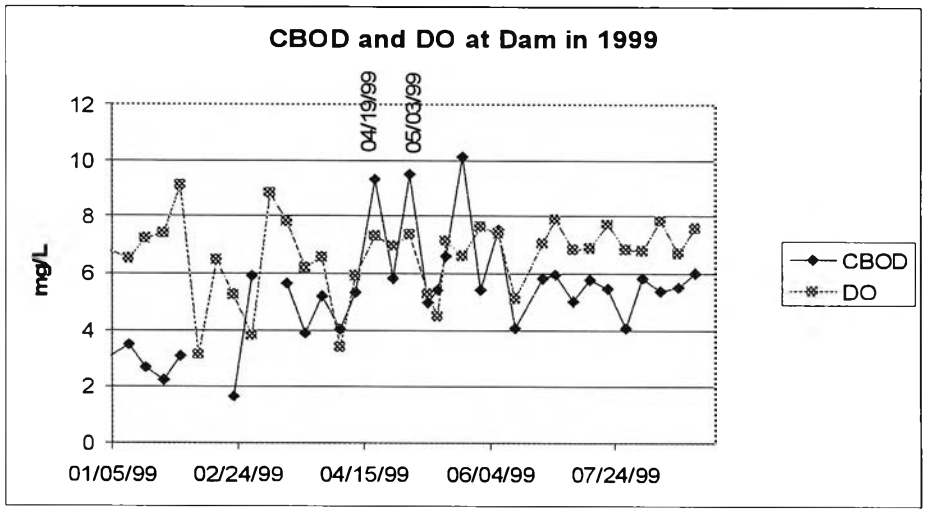



Figure 4.90 Contradicting high CBOD and DO in the Dam from April 19th - May 3rd, 1999.

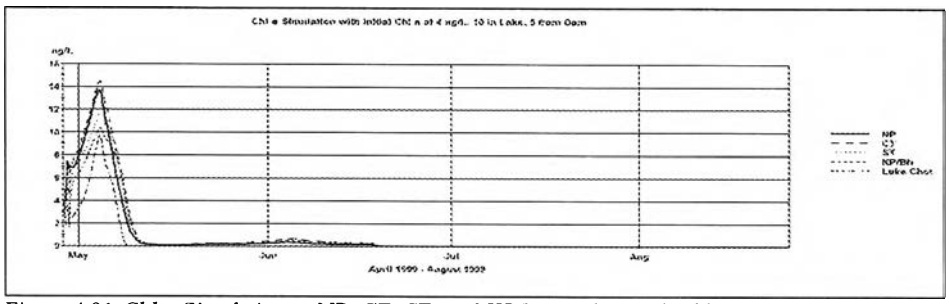


Figure 4.91 *Chl a* Simulation at NP, CT, ST, and KP/BN with initial *Chl a* of 4 μg/L, 10 μg/L in the Chot lagoon and 5 μg/L from Dam and starting run on April 26th, 1999.

Scenario 4: How much algae must there be to produce the observed DO on May 3rd and May 10th, 1999?

With uncalibrated *Chl a*, it was impossible to know how much algae actually existed in the first bloom on May 4th, 1999. The simulation of DO against observed DO on May 3rd and May10th, 1999, however, could be used to determine the approximate amount of algae needed to generate the observed DO. Figure 4.92 shows the result of DO prediction to achieve observed DO at CT on May 3rd and May 10th, 1999. To achieve the predicted DO, as shown in Figure 4.91, at least 84 μg/L of algae must exist in the river (Figure 4.92). On May 1st, 1999 there were as much as 95 μg/L of algae.

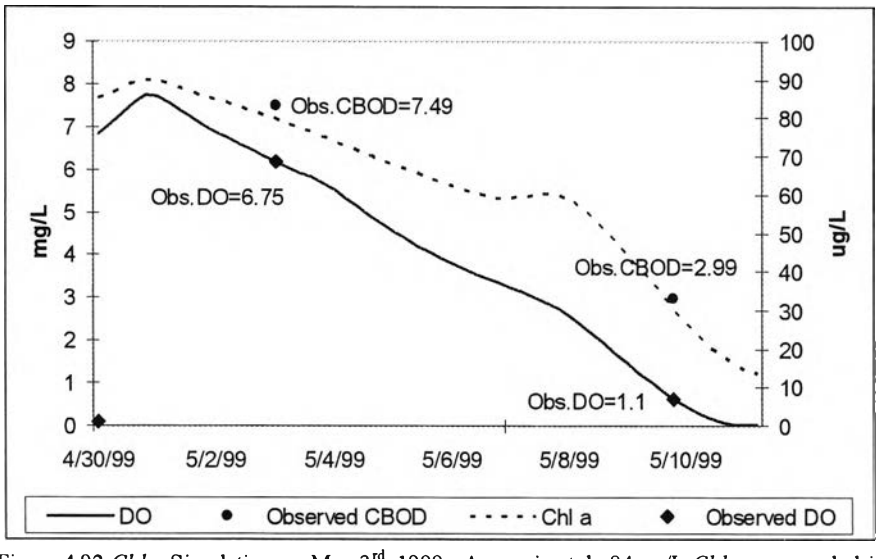



Figure 4.92 *Chl a* Simulation on May 3rd, 1999. Approximately 84 μg/L *Chl a* was needed in order to reduce DO to the observed value on May 10th, 1999.

Predictive Capability of *Chl a*

Although the algal bloom could be predicted by modeling, the predictive capability of the model on *Chl a* had to be analyzed before a conclusion could be made. The predictive capability of the model was determined by exposing the weakness of the model with unlikely but possible scenarios. One weakness in this model was the linear interpolation between observed data of temperatures and boundary nutrients, in order to obtain the missing data. The problem with data interpolation was that it might not reflect reality.

In this study, during the first bloom from April 19th – May 10th, 1999, the lowest possible water temperatures, and Dam $NH_3$-N, $NO_3$-N, and ON were used in place of the interpolated values to simulate the bloom. If the bloom persisted with the least possible temperature and nutrients, then the conclusion that the bloom existed was legitimate. Since high water temperature accelerates the bloom, the lowest possible values of air temperature were used for the *Chl a* simulation. According to Figure 4.93, the mean air temperature was

always lower than the water temperatures at the time of sampling; therefore, it was reasonable to fill in the gaps of observed water temperatures with air temperatures.

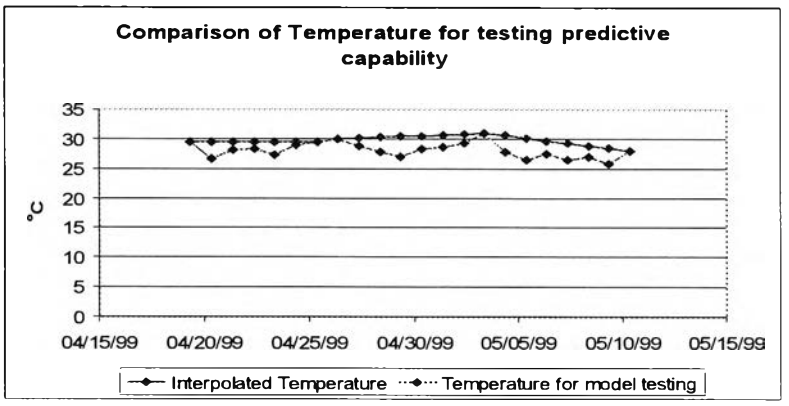



Figure 4.93 Comparison between Interpolated Temperatures and Temperatures Selected for testing the model's predictive capability.

Figures 4.94, 4.95 and 4.96 show the comparison between the interpolated Dam $NH_3$-N, $NO_3$-N and ON and the new set of Dam $NH_3$-N, $NO_3$-N and ON for testing the model's predictive capability of *Chl a*. The new set of Dam values was chosen from the lower values between adjacent observed values. With the new set of temperature and Dam nutrients shown in Figures 4.94-4.96 and Table 4.9, the simulation of *Chl a* was performed against the baseline scenario. The simulation result (Figure 4.97) show decreased *Chl a* at NP, CT, ST, and KP/BN by 34, 27, 29 and 30%, respectively. The algal bloom still existed, after the conditions had been substituted with the least favorable data.

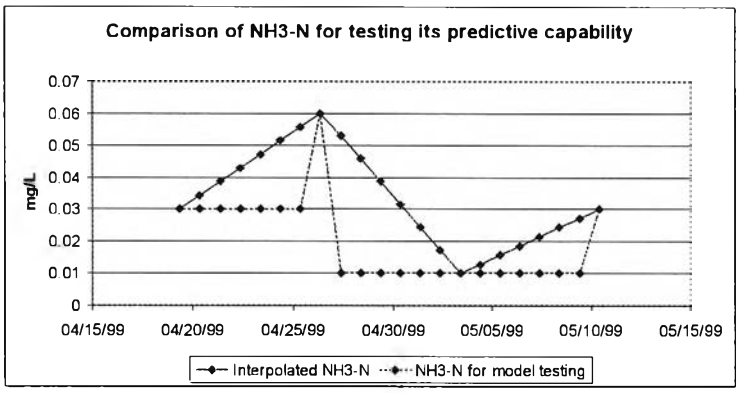



Figure 4.94 Comparison between Interpolated $NH_3$-N and selected $NH_3$-N for testing the model's predictive capability.

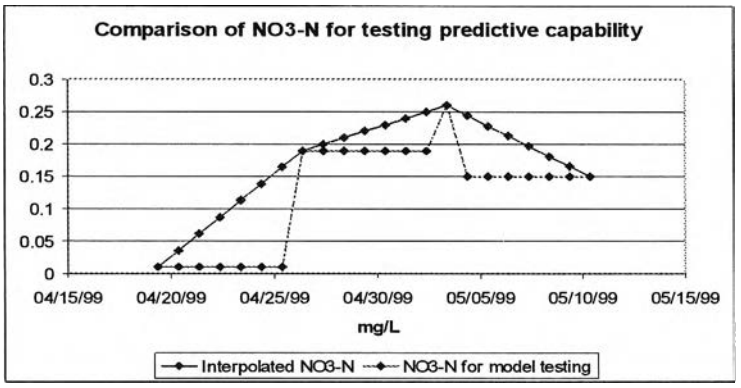



Figure 4.95 Comparison between Interpolated $NO_3$-N and selected $NO_3$-N for testing the model's predictive capability.

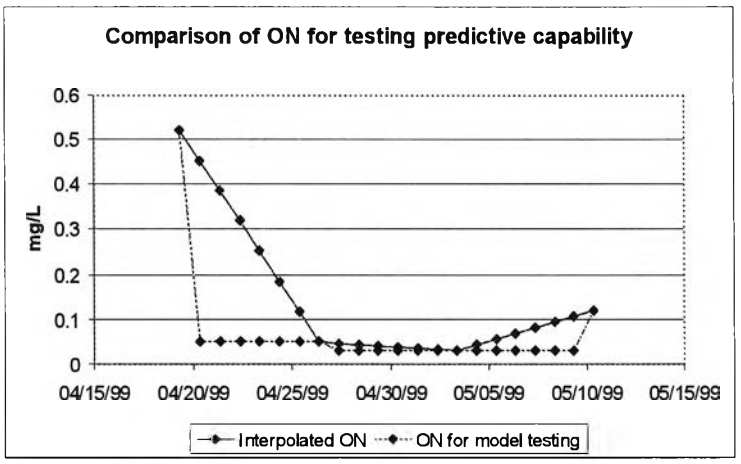



Figure 4.96 Comparison between Interpolated ON and Selected ON for testing the model's predictive capability.

Table 4.9 Temperature and Dam Nutrients for Algal Bloom Simulation.

| Date | Temperature (a*2,$^0$C) | Temperature (w*3,$^0$C) | $NH_3$-N (mg/L) | $NO_3$-N (mg/L) | ON (mg/L) |
|---|---|---|---|---|---|
| April 19th, 1999*1 | 27.7 | 29.5 | 0.03 | 0.01 | 0.52 |
| April 20th, 1999 | 26.6 | 26.6 | 0.03 | 0.01 | 0.05 |
| April 21st, 1999 | 28.1 | 28.1 | 0.03 | 0.01 | 0.05 |
| April 22nd, 1999 | 28.3 | 28.3 | 0.03 | 0.01 | 0.05 |
| April 23rd, 1999 | 27.3 | 27.3 | 0.03 | 0.01 | 0.05 |
| April 24th, 1999 | 29 | 29 | 0.03 | 0.01 | 0.05 |
| April 25th, 1999 | 29.7 | 29.7 | 0.03 | 0.01 | 0.05 |
| April 26th, 1999*1 | 30.3 | 30 | 0.06 | 0.19 | 0.05 |
| April 27th, 1999 | 28.8 | 29.7 | 0.01 | 0.19 | 0.03 |
| April 28th, 1999 | 27.8 | 27.8 | 0.01 | 0.19 | 0.03 |
| April 29th, 1999 | 27 | 27 | 0.01 | 0.19 | 0.03 |
| April 30th, 1999 | 28.3 | 28.3 | 0.01 | 0.19 | 0.03 |
| May 1st, 1999 | 28.7 | 28.7 | 0.01 | 0.19 | 0.03 |
| May 2nd,1999 | 29.3 | 29.3 | 0.01 | 0.19 | 0.03 |
| May 3rd, 1999*1 | 29.9 | 31 | 0.01 | 0.26 | 0.03 |
| May 4th, 1999 | 27.8 | 27.8 | 0.01 | 0.15 | 0.03 |
| May 5th, 1999 | 26.4 | 26.4 | 0.01 | 0.15 | 0.03 |
| May 6th, 1999 | 27.4 | 27.4 | 0.01 | 0.15 | 0.03 |
| May 7th,1999 | 26.5 | 26.5 | 0.01 | 0.15 | 0.03 |
| May 8th, 1999 | 27 | 27 | 0.01 | 0.15 | 0.03 |
| May 9th, 1999 | 25.8 | 25.8 | 0.01 | 0.15 | 0.03 |
| May 10th, 1999*1 | 23.8 | 28 | 0.03 | 0.15 | 0.12 |

*1 Observed data
*2 mean air temperature
*3 water temperature during sampling

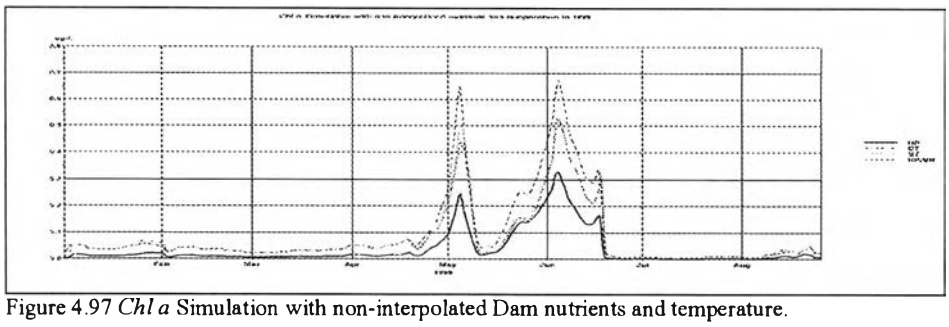


Figure 4.97 *Chl a* Simulation with non-interpolated Dam nutrients and temperature.

The predictive capability of the model was also tested by setting all nutrients from the Dam equal to zero. If the bloom still existed, then it meant that the bloom was caused by nutrients in the runoff and the Dam flow. If such was the case, then the interpolation of Dam nutrients in the model really had no effect on this model. Figure 4.98 shows that the bloom could exist with no Dam $NH_3$-N, $NO_3$-N and ON. The nitrogen and phosphorus nutrients in the runoff, temperature, and Dam flows were enough to cause the bloom, but the bloom decreased by 71, 45, 43 and 42% at NP, CT, ST and KP/BN, respectively.

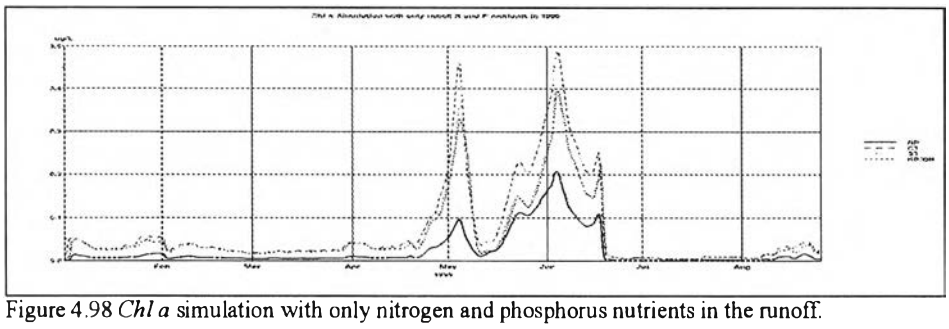


Figure 4.98 *Chl a* simulation with only nitrogen and phosphorus nutrients in the runoff.

In summary, the model predicted the algal bloom with subsequent low DO before the fish kills. Through scenarios, it was determined that the runoff nutrients by themselves were enough to generate the algal growth without any existing algae in the river. In 1999, the bloom could grow much higher with some initial algae (1.7 to 4.5 μg/L in Table 4.2) in the river. The

bloom became even more intense when algae were added from the Dam to the river (Figure 4.91). If DO was used to estimate the amount of algae on May 3rd, 1999, there could be at least 84 μg/L algae in segment CT before the fish kills on May 9th-10th, 1999.

During the field survey in this study, the runoff with scum-like appearance of algae was observed in segments NP and KB. Algae were able to grow in the runoff because there were enough nutrients and time for algae to grow before the runoff combined with the river. If *Chl a* was simulated with algae in the runoff, the bloom would unquestionably grow even bigger.

Cause Analysis of Fish Kills on DO

The fish kills occurred from May 9th-10th, 1999; therefore, it was better to use the observed DO on May 10th for the analysis of the fish kills. In Figures 4.99-4.101, DO abruptly dropped to approximately 1 mg/L at all three aquaculture sites on May 10th, 1999 which coincided with the fish kill incident. As mentioned in Chapter 2, low DO of approximately 1 mg/l could kill fish. The effect of low DO on May 14th, 1999 on the fish was unknown because aquaculturalists moved their fish to the nursery pond on land since the morning of May 10th. On May 17th, 1999, the fish were put back into the net pens. No fish death was reported on this date, even though DO at CT was only 1.6 mg/L, a little higher than on May 10th. At ST and KP/BN, DO was higher than at CT on May 17th, 1999.

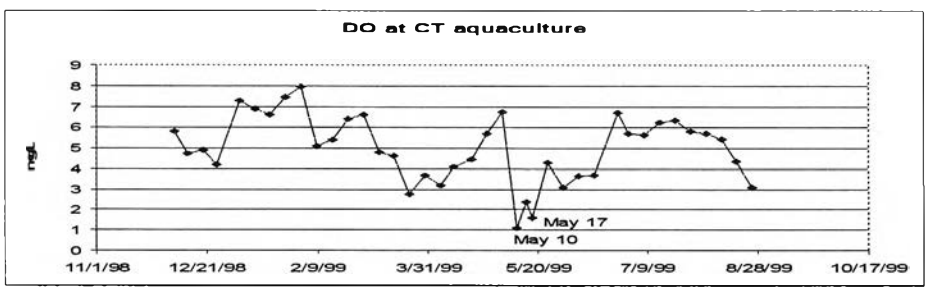


Figure 4.99 Observed DO at the CT Aquaculture

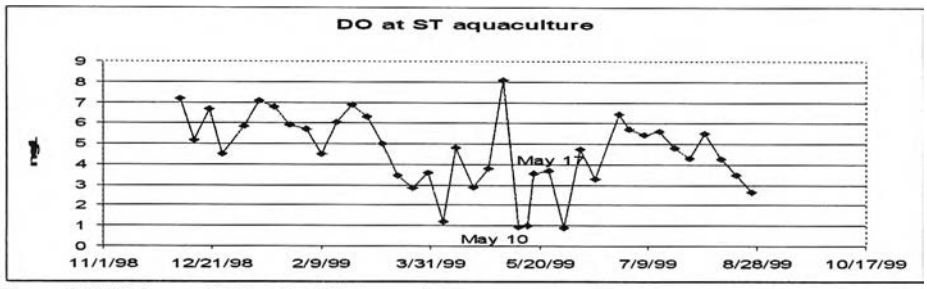



Figure 4.100 Observed DO at the ST Aquaculture

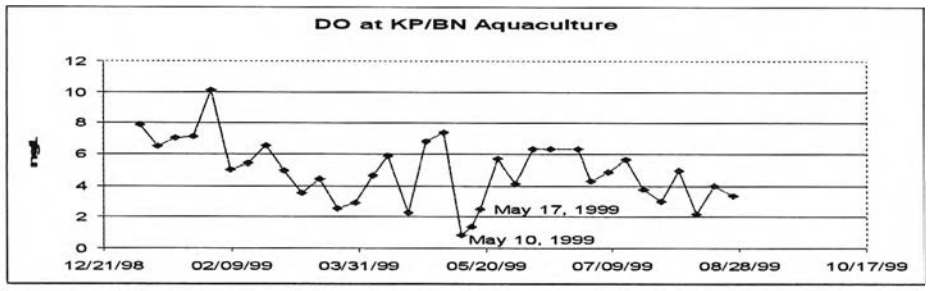



Figure 4.101 Observed DO at the KP/BN Aquaculture

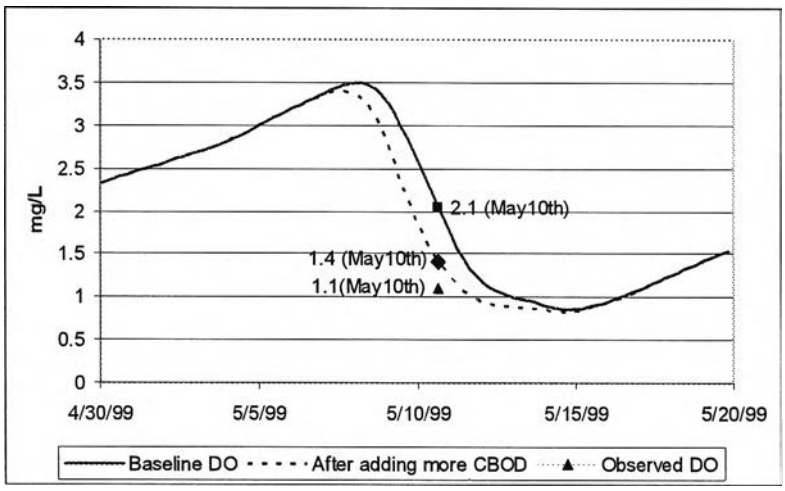
Since low DO was accompanied by fish kills on the same day, it was very likely that DO was the reason for the fish kills. However, it could not be concluded that DO was the only reason for the fish kills. As mentioned in Chapter 2, most fish kills are caused by more than one reason. The cause of the low DO could come from three main factors, namely algal bloom, $NH_3$ and BOD. The simulation of algal bloom, as previously mentioned, suggested that an algal bloom was very possible. The proof of the algal bloom, however, could not exclude other factors of high $NH_3$ and BOD until it could be proven otherwise by modeling.

Cause Analysis of Fish Kills on BOD

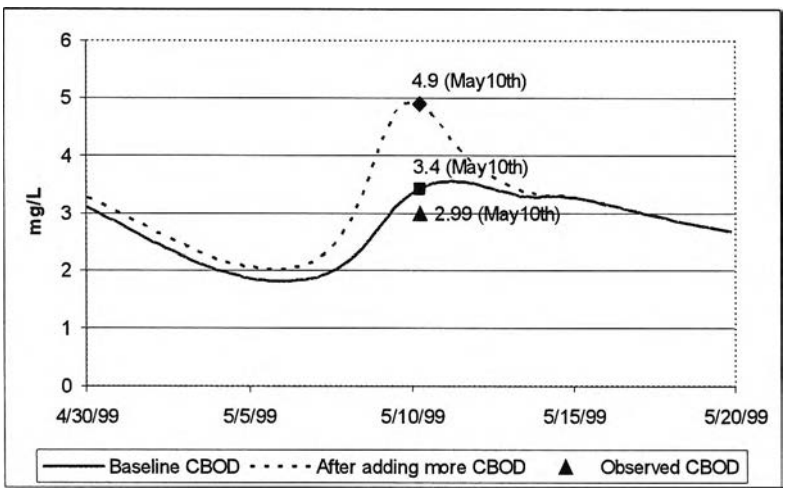
Since low DO was suspected of killing the fish, BOD which could cause low DO was analyzed in this study. BOD could have been added to the river from various sources such as the Dam water, river sediment and other point and non-point sources from May $4^{th}$-$9^{th}$, 1999, a period

which no data were collected. The possibility that high BOD came from these sources were thus investigated through scenario simulations. The scenario of BOD from other point and non-point sources was included in the BOD from the sediment since this BOD was added as a point source to each segment in the model and it could include BOD from the sediment or other sources. First, CBOD was simulated under a scenario of high Dam BOD from May 4$^{th}$-9$^{th}$, 1999 to see how high Dam CBOD must be, in order to reduce DO to the observed DO in all segments. Second, CBOD was simulated under a scenario that BOD from re-suspension of benthic layer, and other sources was added to the water column from April 26$^{th}$-June 18$^{th}$, 1999. Segment CT was used as an example for analyzing the scenario simulation.

To investigate how high Dam BOD must be from May 4$^{th}$-9$^{th}$, 1999 in order to cause low DO on May 10$^{th}$, 1999, the simulation was run by assuming that there was no algal bloom and algal die-off that caused low DO. The simulation of *Chl a* was thus bypassed. After test runs were conducted within a certain range, it was found that 20 mg/L of CBOD had to be added from the Dam to the river from May 4$^{th}$-6$^{th}$, 1999 to cause DO at CT to reduce to approximately 1 mg/L on May 10$^{th}$, 1999. Without the additional CBOD, DO would have been at 2.1 mg/L (Figure 4.102a). The results of DO and CBOD simulation after high CBOD of 20 mg/L was added during May 4$^{th}$–6$^{th}$, 1999 are shown in Figure 4.102b. CBOD which used to be at 3.4 mg/L on May 10$^{th}$, 1999 in a baseline scenario was increased to 4.9 mg/L with the additional Dam CBOD. The addition of 20 mg/L CBOD from the Dam was unlikely to happen in 1999 because 1) the highest CBOD in the Dam in 1999 and 2000 were only 9.3 and 5.6 mg/L, respectively; 2) the addition of high CBOD from the Dam caused CBOD at CT to shift in the wrong direction, specifically from 3.4 to 4.9 mg/L.




(a)



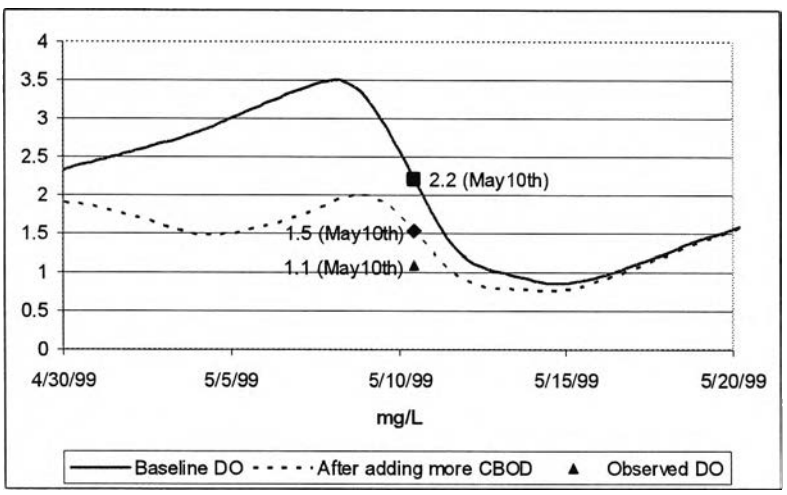

(b)

Figure 4.102. DO (a) and CBOD (b) simulation with additional 20 mg/L CBOD from Dam on May 4th-6th, 1999. Notice that CBOD shifted away from the observed value in order to lower DO.

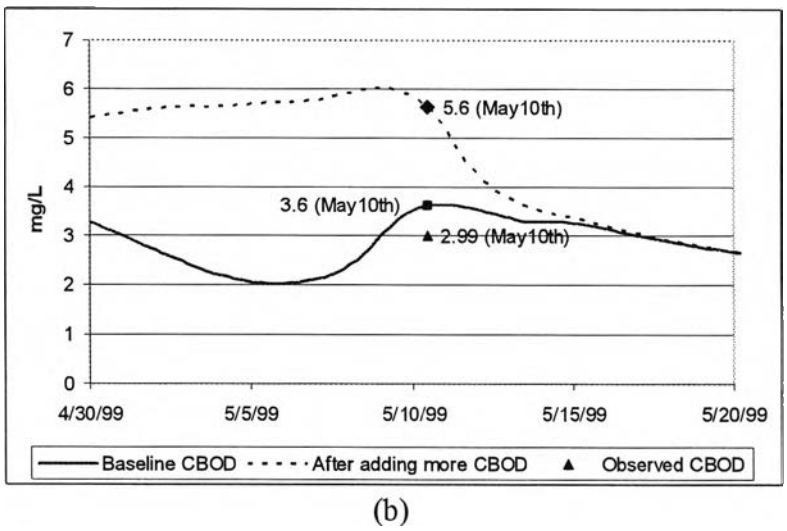
The next scenario was to simulate the addition of CBOD from the sediment and other sources to reduce predicted DO to the observed DO on May 10th, 1999. After test runs were conducted within a certain range, it was found that if 400 kg/day of CBOD was added from the sediment and other sources from April 26th - May 10th, 1999, the predicted DO could be reduced from 2.2 to 1.5 mg/L on May 10th (Figure 4.103a). The CBOD re-suspension was simulated by adding CBOD as a point source to each segment. The scenario of CBOD re-

suspension was however unlikely to happen because, as shown in Figure 4.103b, when CBOD was re-suspended from the sediment at 400 kg/day, the predicted CBOD at CT on May 10th, 1999 shifted away further from the observed CBOD of 2.99 to 5.6 mg/L. If CBOD was added from the sediment, CBOD and DO would only shift in the opposite directions.

In summary, the simulations of high CBOD from the Dam and sediment scouring demonstrated that these scenarios were unlikely to happen in 1999. The addition of high CBOD only reduced DO in the water column. But, the observed CBOD and DO indicated otherwise; specifically, both observed CBOD and DO increased or decreased in the same direction during the low flows.




(a)




(b)

Figure 4.103. DO (a) and CBOD (b) simulation with additional 400 mg/L CBOD from sediment scouring during April 26$^{th}$-May 10$^{th}$, 1999. Notice that CBOD shifted away from the observed value in order to lower DO.

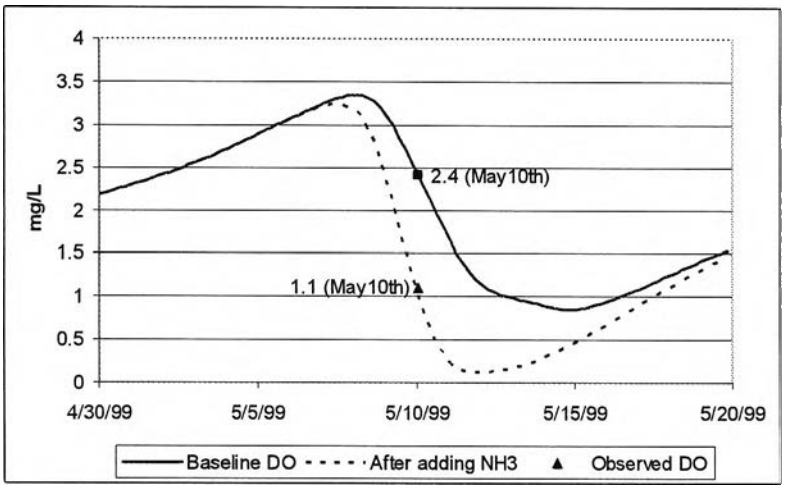
Cause Analysis of Fish Kills on $NH_3$

$NH_3$ could kill fish directly at a toxic dose, or indirectly by lowering DO through nitrification. The lethal ammonia concentration on fish depends on temperature and pH. Similar to BOD, the source of $NH_3$-N could have been from the Dam water, sediment flux/scouring, and point/non-point sources. The mineralization of organic matter during the summer has been well documented (Therkildsen and Lomstein, 1993; van Luinj *et al.*, 1999). In the sediment, $NH_3$ was one of the main compounds released from microbial degradation of organic compounds (Stief *et al.*, 2003). In the tilapia pond of Thailand, the benthic flux of ammonia averaged 13.2 mM $NH_4^+$ $m^{-2}$ $day^{-1}$ (Riise and Roos, 1997).

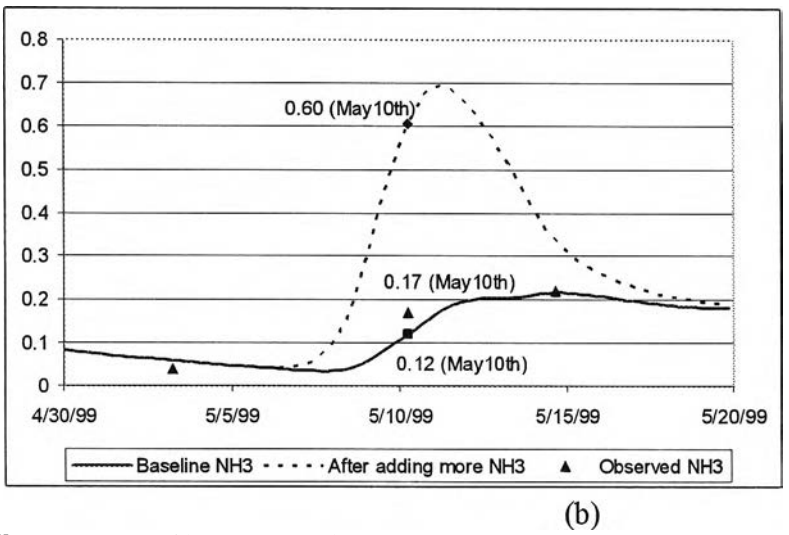
The baseline simulation results of $NH_3$-N from the Dam at CT, ST and KP/BN in Figures 4.60-4.62 were examined to see whether $NH_3$-N existed in high concentrations before or during the fish kills. $NH_3$-N on May 3$^{rd}$ and 10$^{th}$, 1999 were 0.05 and 0.21 mg/L at CT, 0.08 and 0.03 mg/L at ST and 0.02 and 0.01 mg/L at KP/BN, respectively. These $NH_3$

concentrations could not have killed the fish for the following reasons. First of all, as mentioned in Chapter 2, $NH_3$-N in the range between 0.14 - 0.43 mg/L could not kill Nile tilapia, and all $NH_3$-N found on May 3rd and 10th, 1999 were not beyond this range. Secondly, according to the quality criteria for water (USEPA, 1986), the 1-hr lethal ammonia concentration for fish is between 1.3 – 3.5 mg/L at a temperature of 30 ° C and pH between 8.0 – 8.5, which was the temperature and range of pH found on May 3rd and 10th, 1999. Thirdly, $NH_3$-N concentrations after May 10th was as high as 0.57 mg/L, but there were no fish kills reported. It was thus concluded that $NH_3$ on May 3rd and 10th, 1999 could not kill Nile tilapia.

To eliminate the possibility that there might have been extremely high Dam $NH_3$-N from May 4th-9th, 1999 which reduced DO on May 10th, 1999, a simulation was run to determine how high $NH_3$-N must have been present in the Dam, in order to reduce DO to 1.1 mg/L at CT. As before, the simulation of *Chl a* was bypassed to ensure that the drop in DO was not caused by the algal death. After test runs were conducted with a certain range, it was found that there must have been 3 mg/L of $NH_3$-N in the Dam from May 4th - 9th, 1999. Figures 4.104a shows the DO simulations before and after the addition of 3 mg/L of $NH_3$-N from the Dam. On May 10th, 1999 DO dropped from 2.4 to 1.1 mg/L after 3 mg/L $NH_3$-N was added from the Dam.




(a)




(b)

Figure 4.104. DO (a) and $NH_3$-N (b) simulation with additional 3 mg/L $NH_3$-N from the Dam during May 4$^{th}$-May 9$^{th}$, 1999 assuming there were no algae involved. Notice that $NH_3$-N shifted away from the observed value in order to reduce DO to the observed DO.

It was very unlikely that the reservoir could have contained 3 mg/L $NH_3$-N from May 4$^{th}$-9$^{th}$, 1999 because this concentration was not close to the range of $NH_3$-N in the Dam in 1999 (Figure 4.105) or 2000 (Figure 4.106) which was at most 0.4 mg/L. Since there were also fish in the reservoir, $NH_3$-N at 3 mg/L should have killed the fish in the reservoir during May 4$^{th}$-9$^{th}$, 1999; but there was no fish-kill report. Moreover, 3 mg/L of $NH_3$-N from the Dam would have produced approximately 0.60 mg/L of $NH_3$-N at CT on May 10$^{th}$, 1999 (Figure

4.104b), but only 0.17 mg/L of $NH_3$-N was detected on this date. High $NH_3$-N on May 17th, 1999 in the reservoir should not be due to the runoff $NH_3$. When $NH_3$ was applied to soil as a fertilizer, it was usually converted through the nitrification process to $NO_3$ (Chandler, 1985; Nishio, 1994). Therefore, when nitrogen was leached or washed into the reservoir, it should have been in the form of $NO_3$. The cause of high $NH_3$-N in the Dam on May 17th, 1999 should have been due to the biodegradation of the algal die-off. In summary, it was concluded that $NH_3$-N was not present in the Dam at 3 mg/L to cause low DO and fish kills.

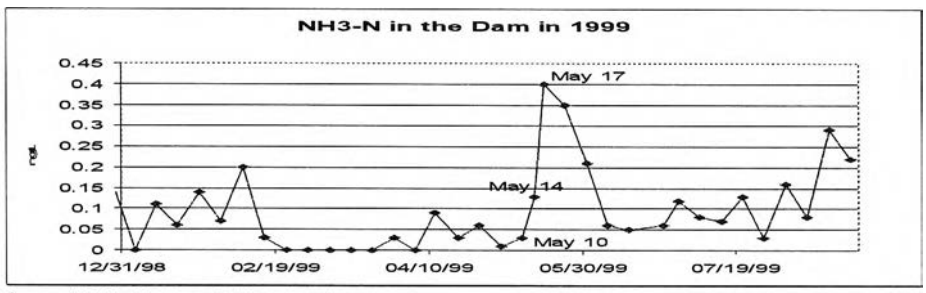


Figure 4.105. Observed $NH_3$-N in the Dam in 1999.

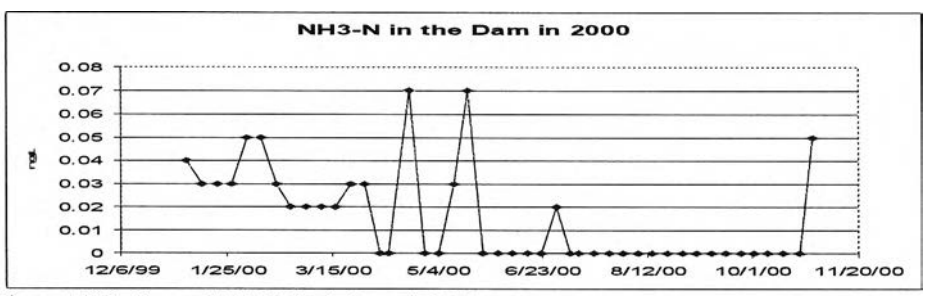


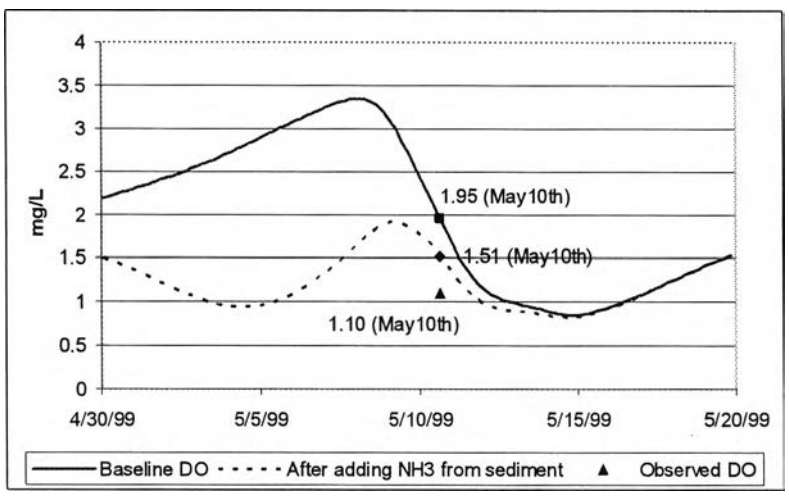
Figure 4.106. Observed $NH_3$-N in the Dam in 2000.

The scenario of $NH_3$-N from the sediment and point/nonpoint sources could be simulated together because $NH_3$-N from these sources were added together to the river as the total "loading." After test runs were conducted with a certain range of $NH_3$-N loadings, it was found that the total of 100 kg/day $NH_3$-N from the sediment and point/nonpoint sources had to be added to each segment of the river in order to achieve the observed DO of 1.1 mg/L at the

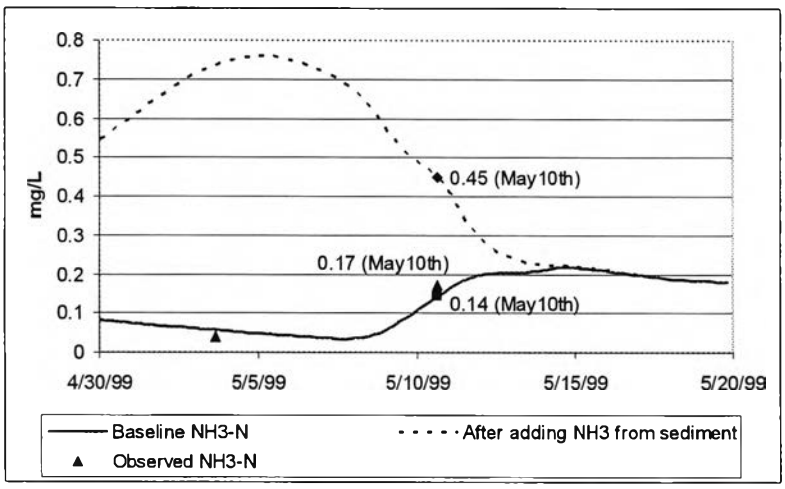
CT aquaculture on May 10th, 1999 (Figures 107a and 107b). This scenario was, however, unlikely because, if it happened, $NH_3$-N should have been detected at 0.45 mg/L instead of 0.14 mg/L on May 10th, 1999.

Cause Analysis of Fish Kills on Nitrite

As mentioned in Chapter 2, 96-hr $LC_{50}$ of $NO_2$-N was between 8 - 81 mg /L for Nile tilapia. Observed $NH_3$-N, which was the precursor of nitrite, were not more than 0.21 mg/L at all three aquaculture sites from May 3rd-10th, 1999, as shown in Figures 4.60-4.62, and the $NH_3$ conversion to nitrite was reported to be around 28% and 35% (Nenov and Kamenski, 2003). Therefore, it was very unlikely that the reactive $NO_2$-N would accumulate in the river as high as 8 mg/L, in order to kill the fish.




(a)




(b)

Figure 4.107. DO (a) and $NH_3$-N (b) simulation with additional 100 mg/L $NH_3$-N from sediment scouring during May $4^{th}$-May $9^{th}$, 1999 assuming there was no algae involved. Notice that $NH_3$-N shifted away from the observed value, in order to reduce DO from 1.95 to 1.51 mg/L.

The analysis of all possible causes of fish kills could be summarized as shown in Table 4.10. The most likely scenario that could happen before the fish kills on May 9th-10th, 1999 was the algal bloom. Before April 19th, 1999, there was enough rainfall that could cause high $NO_3$ loading from the draw-down zone into the reservoir, and produce the subsequent algal bloom in the reservoir. The presence of *Microcystis* sp. in this reservoir, had been confirmed by Teerasak (2003). High CBOD and DO in the reservoir on April 19th and May 3rd, 1999 were indicative of the algal bloom and its photosynthesis. This bloom in the Dam could die on May 10th, 1999 as high $NH_3$ from algal die-off, started to increase (Figure 4.106). When the Dam started to reduce the flows to below 1 MCM per day from April 22nd, 1999 onward, the river's water quality was dominated by large runoff. The release of the algal bloom from the reservoir, coupled with existing algae in the eutrophic river, ample supplies of nutrients from the runoff and high summer temperature could cause a larger bloom in the river than the reservoir. The bloom in the river could have peaked around May 2nd-4th, 1999 as CBOD and DO were high at all sampling sites on this date.

The bloom in the river could die by May 10th, 1999 from two reasons. First, when the runoff nutrients were completely taken up with algae, the bloom would die by itself through cellular respiration. Second, low light extinction from cloudy sky and heavy rain from May 4th - May 9th, 1999 (Appendix D) could cause an accelerated effect on the algal die-off and even lower DO on May 10th, 1999. The model simulation with no light extinction from May 4th-May 9th, 1999 proved this point by showing that DO reduced from 3.7 mg/L in the baseline scenario to 1.9 mg/L on May 10th, 1999 (Figure 108). High $NH_3$ which peaked on May 17th, 1999 at all sampling sites in the river suggested the biodegradation of dead algae.

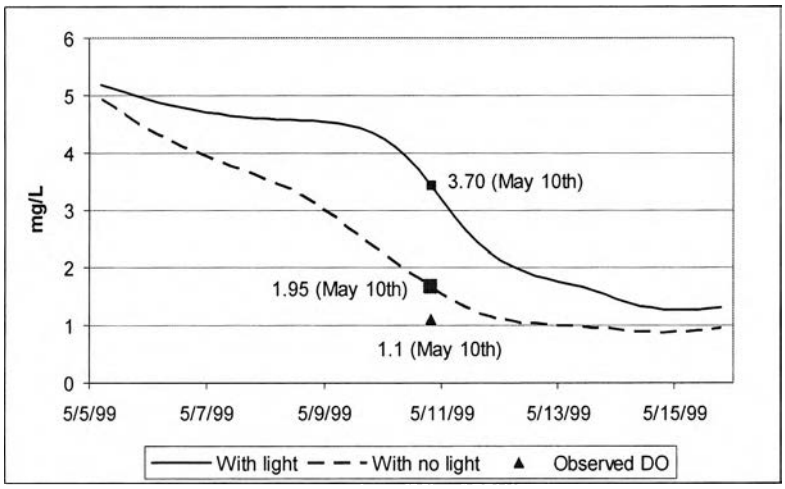



Figure 4.108. DO simulation under normal light and without light from May 4th-11th, 1999. In this scenario, high chlorophyll *a* of 62 □g/L on May 3rd, 1999 was used to show the difference.

The experimental aquaculture of Nile tilapia, algal enumeration, and GC/MS analysis in 2002-2003 also indicated that the fish kills and cyanobacterial proliferation were related. The release of the cyanobacterial toxin after the bloom might occur around May 10th, 1999. The role of the toxin from the bloom might explain why low DO of 1.8 mg/L on May 10th, 1999 killed the fish at CT, while low DO of 2.0 mg/L on May 17th at CT did not. According to Figure 4.87, there were two algal blooms in 1999; the second bloom did not kill the fish because it was flushed out of the river. Had it died due to lack of nutrients or sunlight, it could have caused another fish kill.

The scenario of high BOD from the Dam and sediment flux/scouring which caused low DO and the subsequent fish kills was ruled out through modeling. High BOD could lower DO on May 10th, 1999, but BOD could not reduce itself fast enough to match the observed value in the river. Thus, the predicted and observed BOD contradicted each other.

By the same reasoning as the Dam BOD, the scenario of high Dam $NH_3$ was also ruled out. The scenario of high $NH_3$ from the benthic nitrogen re-suspension and point/nonpoint

sources, was also ruled out. Whenever $NH_3$ was released from the sediment or added as point/non-point sources to the river, it was more likely that it would be taken up by algae as $NH_3$ was one of the primary nutrients for its uptake; and if there was a large amount of $NH_3$, it could cause a bloom. Although data on how much freshwater phytoplankton utilized the benthic nitrogen were unknown, marine phytoplankton consumed about 26-100% of the benthic nitrogen (Kristensen, 1988). Nutrients in the sediment around the coast supply up to 100% of phytoplankton requirements for growth (Rowe *et al.*, 1975; Fisher *et al.*, 1982; Blackburn and Henrikse, 1983; Balzer, 1984; Billen and Lancelot, 1988). The scenario of high $NO_2$ was ruled out because there was never enough $NH_3$ to generate $NO_2$ at the concentration which could kill Nile tilapia.

The scenario of pesticides causing fish kills in 1999 was not ruled out, although they were not found during the mild fish kills of 2002. In 1999, there could have been some pesticides in the runoff, which produced the additive effect on the fish kills. But pesticides by themselves could not be the cause of low DO at CT on May 10$^{th}$, 1999 because pesticides at ppb levels could not provide enough organics for biodegradation. In fact, there were studies suggesting that when the pesticide was added to the water, it reduced the zooplankton's respiration rate and thereby increased DO (Kreutzweiser and Thomas, 1995; Kreutzweiser *et al.*, 2004). If some pesticides were detected in the river and the runoff in 1999 and 2000, a better conclusion could have been made.

The scenario of the phenols causing fish kills was ruled out because the fish did not die when phenols were detected; but when no phenols were detected, the fish died. The scenario of high heavy metals in the runoff was not ruled out, although PCD had found no metals at high concentrations from 1999-2001. In addition, there was no mining operation near this river

segment. This scenario was not ruled out, however, because the heavy metals might not have been measured during the fish kills. But this scenario should be the last resort to explore after the algal bloom was prevented, and the fish kills persisted.

By the same reasoning as the heavy metals and pesticides, this study could not preclude any other toxic chemicals which were not studied, particularly during the fish kills. Any one of these could have an additive effect, if not a direct effect, on the fish kills. However, whatever they were, they must be able to explain how they could cause low DO on May 10$^{th}$, 1999.

From modeling, the algal bloom could explain all physical data thoroughly (high DO on May 3$^{rd}$, 1999 and low DO on May 10$^{th}$, 1999); it was thus reasonable to recommend that the problem of the algal bloom should be solved before searching for other toxic compounds. After the problem of the algal bloom was prevented, and the fish kills continued, then it would be time to allocate resources to search for other chemicals.

Table 4.10 Summary of Causes of Fish Kills and Their Arguments

| Possible Cause | Possible/ Impossible | Explanation |
|---|---|---|
| Algal Bloom & Toxins | Possible | The simulation of *Chl a* demonstrated that the algal bloom could occur. |
| Low DO from Algal Die-Off | Possible | High DO on May 3$^{rd}$, 1999 could be due to algal photosynthesis, and low DO on May 10$^{th}$, 1999 could be due to algal die-off. |
| Low DO from high Dam BOD | Impossible | To cause low DO on May 10$^{th}$, 1999, BOD would shift from the observed BOD of 3.0 to 5.5 mg/L. |
| Low DO from high benthic BOD | Impossible | To cause low DO on May 10$^{th}$, 1999, BOD would end up shifting away from the observed BOD. |
| High $NH_3$ from Dam | Impossible | To cause low DO on May 10$^{th}$, 1999, the Dam $NH_3$ had to be 3 mg/L which was impossible. |

| | | |
|---|---|---|
| High benthic $NH_3$ | Impossible | If $NH_3$ was released from the sediment, it would add its impact to the algal bloom; therefore, the algal bloom was considered instead. |
| High $NO_2$ | Impossible | Not enough $NH_3$ to cause $NO_2$ |
| Pesticides | Possible, but not urgent | Although no pesticides were found in the river during the mild fish kills of 2002, the pesticides could not be ruled out yet until more research was performed. If pesticides were the cause, they must explain how they could not cause low DO. |
| Phenols spill | Impossible | Phenols were not found on the days of the fish kills, but they were instead found on other days. |
| Heavy Metals | Possible but not urgent | Even though there was no mining operation, and the PCD data confirmed low heavy metals in the river, it was still dangerous to conclude that there were no heavy metals in the runoff. "No evidence" did not simply imply "no existence." They might contribute to the fish kills to a small extent. |
| Other Chemicals A - Z | Possible but not urgent | Similar to heavy metals, "No evidence" does not simply imply "no existence." But they must explain how they could cause low DO. |

### 4.3.2 Sensitivity Analysis for Environmental Management of Fish Kills

The sensitivity analysis provides a sensitivity index (SI) which determines the order of impact of all constants, coefficients and other input variables on the water quality parameters – for better environmental management of the river. In this model, the impact of the Dam flow was also analyzed because the Dam flow could be easily managed through the Dam gates. As this study recommended that the algal bloom should be solved first to prevent the fish kills, SIs of all associated constants, coefficients and Dam flows for *Chl a* were determined. For the completeness of this study, SIs for other parameters such as CBOD, DO, $NH_3$-N and $NO_3$-N were also determined for future environmental management.

Since this model was under dynamic conditions and the changes were not constant, the percent changes of parameters must be compared on the spatial and temporal basis. Segments NP and CT were selected randomly for simulation and comparison. SIs for *Chl a* were determined during the bloom on May 4$^{th}$, 1999. SIs for conventional nutrients were determined on February 22$^{nd}$ and March 1$^{st}$, 1999. The comparison of SIs of each parameter was graphically shown below.

*Chl a* Sensitivity Analysis

Figures 4.109-4.110 show the graphic comparison of SIs from the sensitivity analysis of *Chl a* in segments NP and CT on May 4$^{th}$, 1999, respectively. The same trend was observed in both results of *Chl a* sensitivity analysis. Specifically, the change in *Chl a* was proportionally affected by the change in the temperature the most and then the maximum growth rate. Inversely, the change in the Dam flow affected the change in chlorophyll *a* the most, and next was the algal respiration rate. This finding was not unusual. Paerl (1987) states that the flow is one the key factors in determining the algal bloom. In order to prevent the algal bloom, the most obvious and easiest choice was to control the Dam flows, as controlling the algal respiration rate was not possible.

Even though modeling could suggest that the flow should have been increased in order to prevent the bloom, the decision on the appropriate amount of water release and the economic impact must be considered. The economic impact was beyond the scope of this study. The appropriate amount of water that the Dam should release will be discussed in the next section.

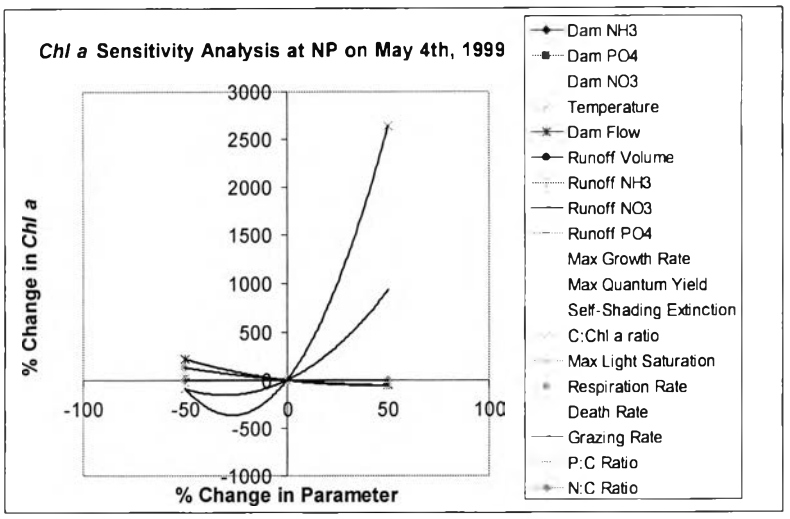



Figure 4.109. Chl a Sensitivity Analysis at NP on May 4th, 1999.

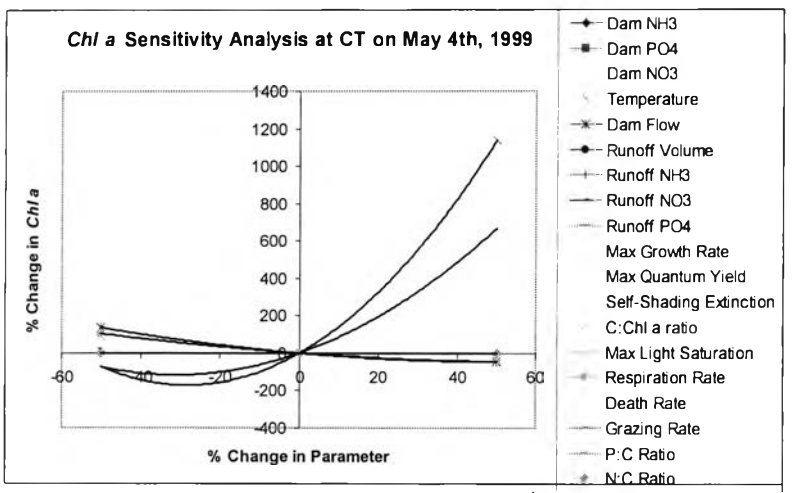



Figure 4.110. *Chl a* Sensitivity Analysis at CT on May 4th, 1999.

## CBOD Sensitivity Analysis

Figures 4.111-4.112 show the results of the sensitivity analysis of CBOD in segment NP on February 22nd, 1999 and March 1st, 1999. Figures 4.113-4.114 show the results of the sensitivity analysis of CBOD in segment CT on February 22nd, 1999 and March 1st, 1999. The same trend was observed in all results of the CBOD sensitivity analysis. In particular, the change in CBOD was mainly affected by the change in the CBOD deoxygenation rate, and the

effect was in the inverse relationship. If the CBOD deoxygenation rate was increased, CBOD in the segment would be reduced. The CBOD deoxygenation rate is normally controlled by the bacterial and fauna population in the river.

The change in CBOD was, on the other hand, directly affected by the flow rate. If the flow rate was increased or decreased, CBOD would also change accordingly because the flow carried CBOD from boundary. The flow in this study could be easily manipulated, if desired, by controlling the Dam gates.

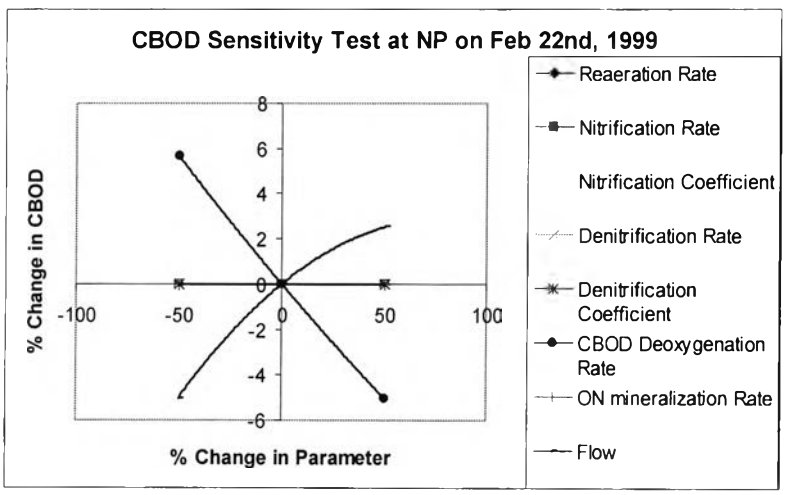



Figure 4.111. CBOD Sensitivity Analysis in segment NP on February 22nd, 1999

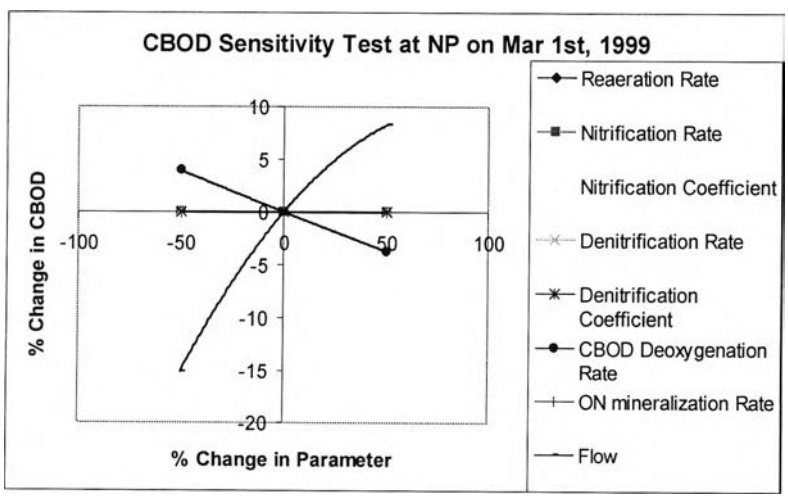



Figure 4.112. CBOD Sensitivity Analysis in segment NP on March 1st, 1999

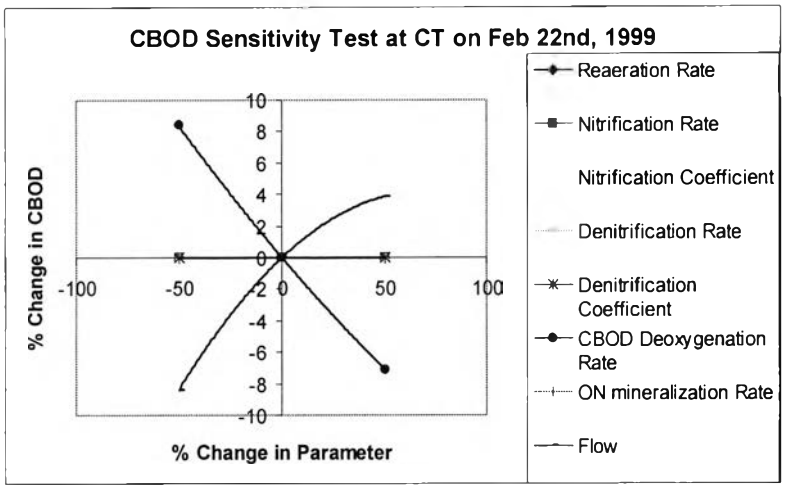



Figure 4.113. CBOD Sensitivity Analysis in segment CT on February 22nd, 1999

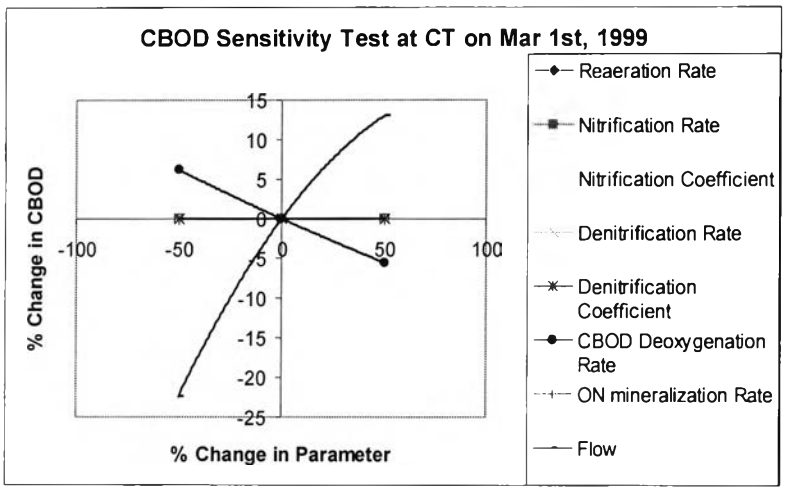



Figure 4.114. CBOD Sensitivity Analysis in segment CT on March 1st, 1999

## DO Sensitivity Analysis

Figures 4.115-4.116 show the results of the sensitivity analysis of DO in segment NP on February 22nd, 1999 and March 1st, 1999. Figures 4.117-4.118 show the results of the sensitivity analysis of DO in segment CT on February 22nd, 1999 and March 1st, 1999. A similar trend was observed in all DO sensitivity analysis results. The nitrification coefficient and CBOD deoxygenation rate mainly affected the DO, and in the inverse proportion. If these two variables were increased, DO would decrease. Manipulating the nitrification coefficient is

not easy because it involves the biochemical change in the nitrifying bacteria; therefore, only the manipulation of the CBOD deoxygenation rate is discussed. The factors which affect the CBOD deoxygenation rate, as already mentioned, are controlled by the bacterial and fauna population in the river.

The change in DO was mainly and directly affected by the reaeration and flow rates. If the reaeration and flow rates were increased, DO would also increase. Factors which affect the reaeration rate are the flow rate, temperature, wind, and river depth. These factors must be manipulated if the decision to control the reaeration rate is desired. The increase in flow on February 22nd, 1999 did not significantly affect DO because the original flow (before the increase or decrease by 50%) was already too small to begin with.

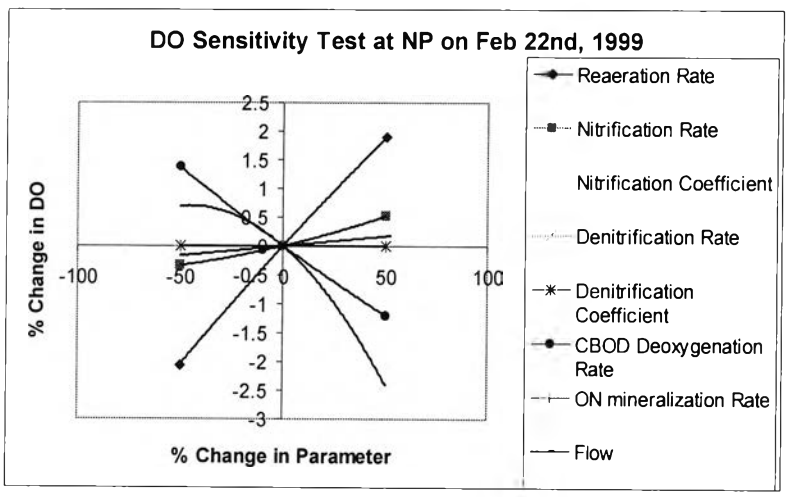



Figure 4.115. DO Sensitivity Analysis in segment NP on February 22nd, 1999

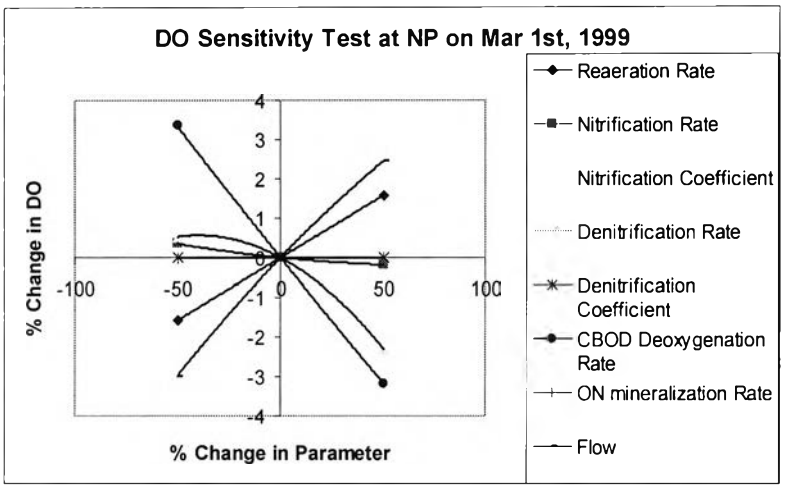



Figure 4.116. DO Sensitivity Analysis in segment NP on March 1st, 1999

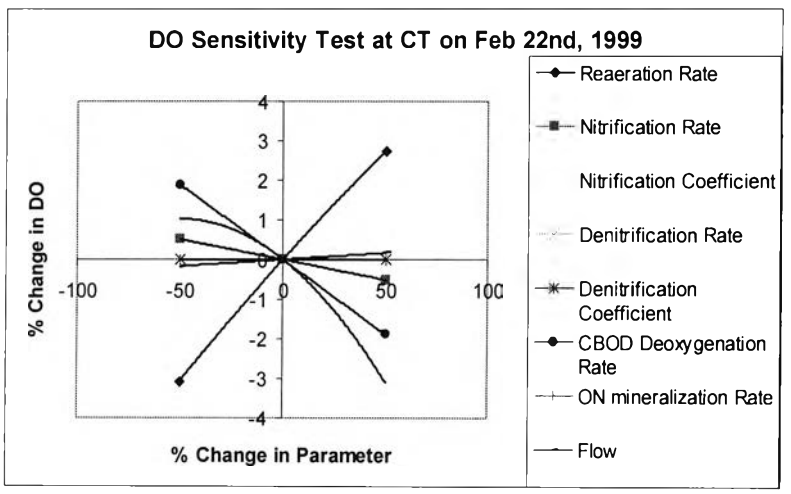



Figure 4.117. DO Sensitivity Analysis in segment CT on February 22nd, 1999

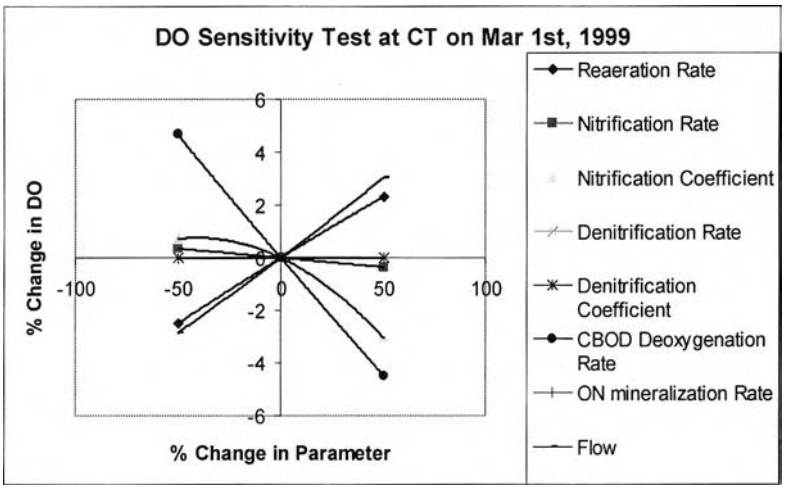



Figure 4.118. DO Sensitivity Analysis in segment CT on March 1st, 1999

$NH_3$–N Sensitivity Analysis

Figures 4.119-4.120 show the results of the sensitivity analysis of $NH_3$ in segment NP on February 22nd, 1999 and March 1st, 1999. Figures 4.121-4.122 show the results of the sensitivity analysis of $NH_3$ in segment CT on February 22nd, 1999 and March 1st, 1999. A similar trend was observed in all $NH_3$ sensitivity analysis results. The nitrification coefficient and nitrification rate significantly affected $NH_3$, in the inverse proportion. If these two variables were increased, $NH_3$ would decrease. The nitrification rate is controlled by the nitrifying bacteria in the river.

The only variable which affected $NH_3$ in the same direction was the mineralization rate of organic nitrogen. When the mineralization rate was increased, $NH_3$ also increased, but to a very small extent. Manipulating this rate may not be cost-effective due to its small impact.

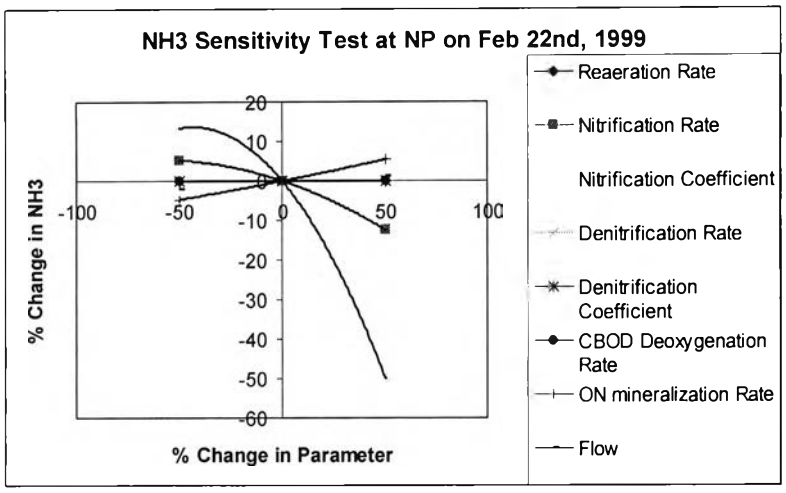



Figure 4.119. $NH_3$–N Sensitivity Analysis in segment NP on February 22nd, 1999

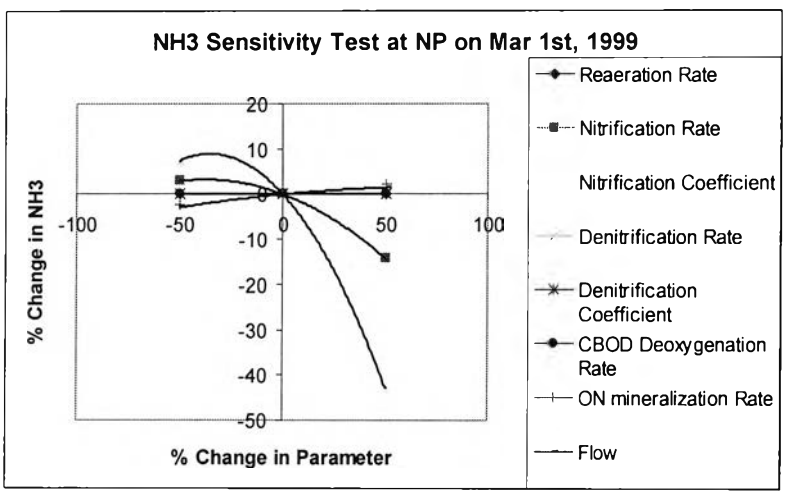



Figure 4.120. $NH_3$–N Sensitivity Analysis in segment NP on March 1st, 1999

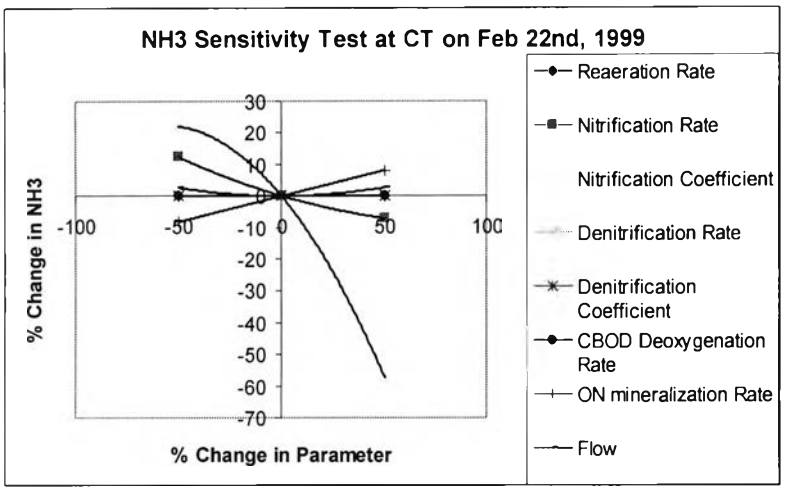



Figure 4.121. $NH_3$–N Sensitivity Analysis in segment CT on February 22nd, 1999

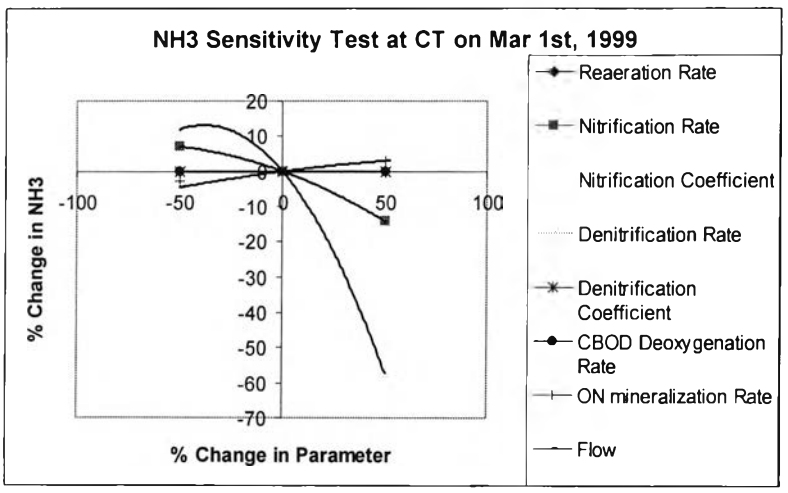



Figure 4.122. $NH_3$–N Sensitivity Analysis in segment CT on March 1st, 1999

## $NO_3$–N Sensitivity Analysis

Figures 4.123-4.124 show the results of the sensitivity analysis of $NO_3$-N in segment NP on February 22nd, 1999 and March 1st, 1999. Figures 4.125-4.126 show the results of the sensitivity analysis of $NO_3$ in segment CT on February 22nd, 1999 and March 1st, 1999. A similar trend was observed in all $NO_3$ sensitivity analysis results. The nitrification coefficient and nitrification rate affected $NO_3$ correspondingly. If these two variables were increased, $NO_3$ also increased because the nitrification process increased $NH_3$, the precursor of $NO_3$. The flow

rate inversely affected $NO_3$, but to a very small extent. When the flow rate was decreased, $NO_3$ increased because $NO_3$ stayed in the segment longer.

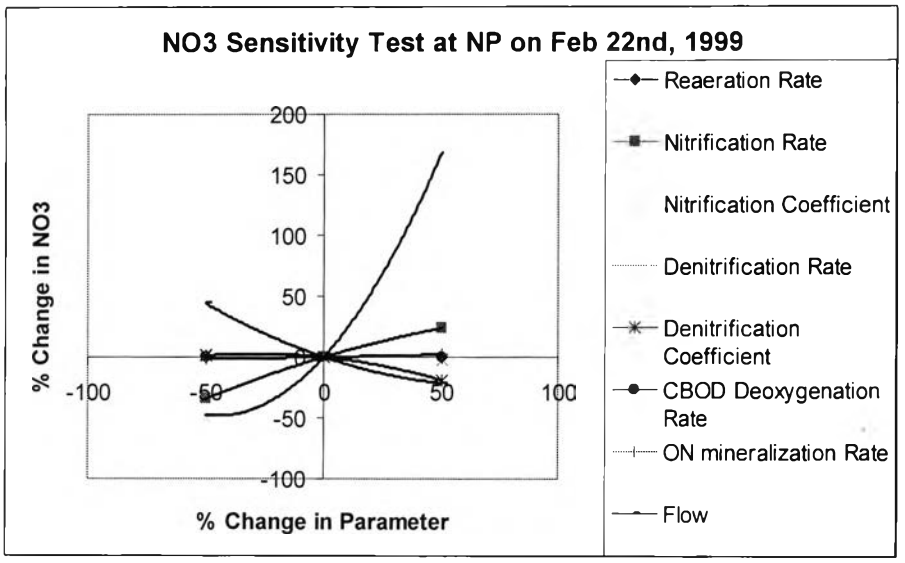



Figure 4.123. $NO_3$-N Sensitivity Analysis in segment NP on February 22nd, 1999

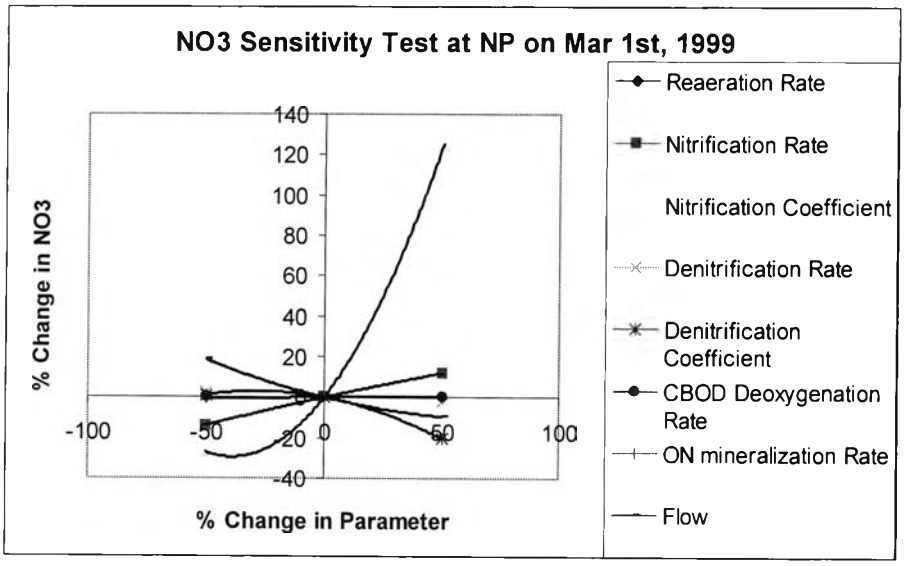



Figure 4.124. $NO_3$–N Sensitivity Analysis in segment NP on March 1st, 1999

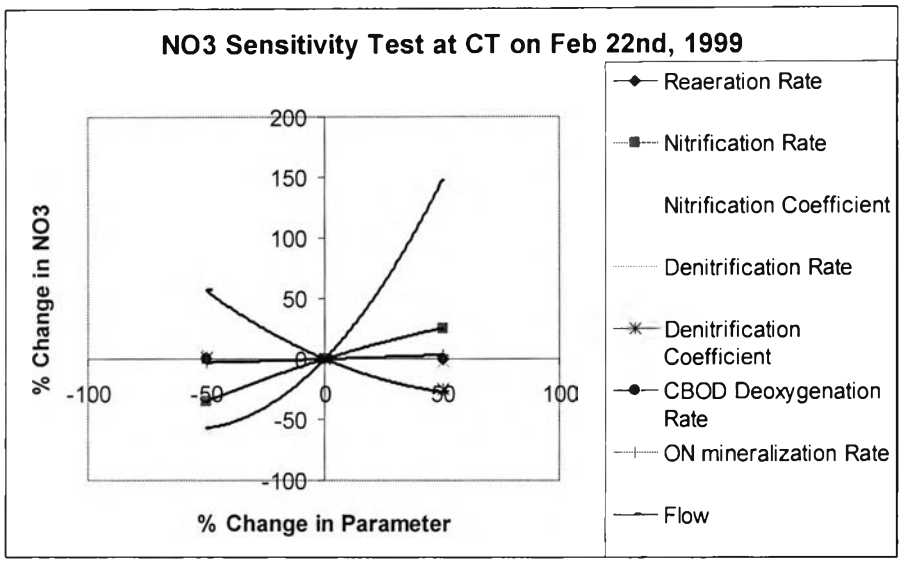



Figure 4.125. $NO_3$-N Sensitivity Analysis in segment CT on February 22$^{nd}$, 1999

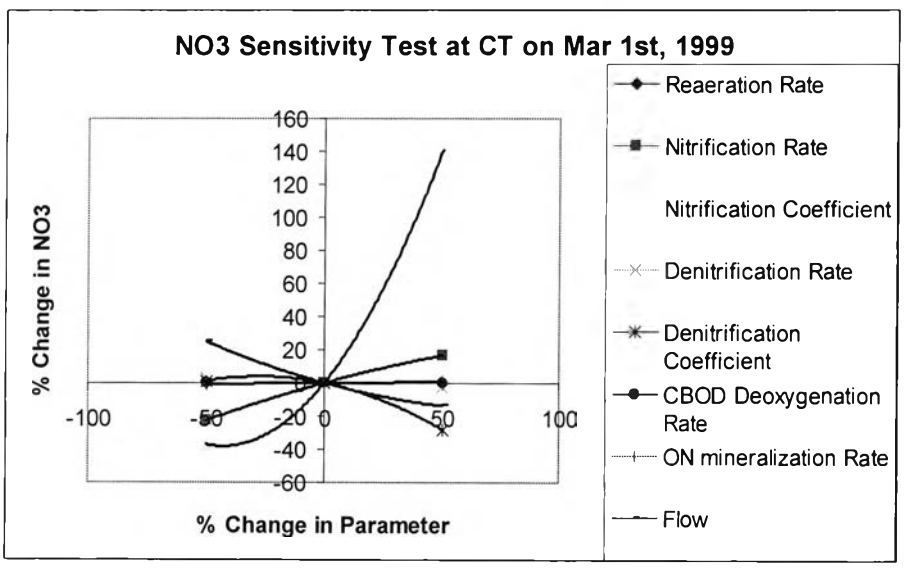



Figure 4.126. $NO_3$–N Sensitivity Analysis in segment CT on March 1$^{st}$, 1999

The above sensitivity analysis demonstrated that, under spatial and temporal variations, the changes of the conventional nutrients were similarly affected by the same constants, coefficients and variables over reasonable ranges. To reduce the size of the algal bloom, the Dam flow must be manipulated properly, and will be discussed next.

4.3.3 Modeling-Based Management Solutions for the Fish-Kill Prevention

After the sensitivity analysis on chlorophyll *a* and conventional nutrients was performed, and SIs of constants, coefficients and variables such as Dam flow were compared, the management solutions could be suggested. Since the cause of the fish kills was assumed to be the algal bloom, the Dam flows which significantly affected *Chl a* would be manipulated in order to reduce algae to the safe level for aquaculture.

According to the ambient water quality criteria in the state of North Carolina (US EPA, 2003), the chlorophyll *a* limit was established at 15 □g/L for trout waters. This study should have also established the chlorophyll *a* limit in the vicinity of this figure, assuming that trout and Nile tilapia responded similarly to algae. Chlorophyll *a*, however, was not monitored in the 1999 and 2000 studies, and thus the model simulation of *Chl a* in this study could not be calibrated and used to predict chlorophyll *a*, quantitatively. With this limitation, this model could still provide management solutions to achieve two goals: 1) the percent reduction of chlorophyll *a* in the river; and 2) the reduction of chlorophyll *a* to an unharmful level for Nile tilapia - by setting chlorophyll *a* before the bloom as the target.

1. Percent Reduction of *Chl a* at the CT aquaculture

For an environmental management of the river to prevent fish kills, it would be very helpful to have a tool that could guide EGAT and RID staff on how to reduce algae by a certain percentage through manipulating the Dam flow. This strategy only worked under an assumption that there were no or less algae in the reservoir than in the river, and that the water from the reservoir was used to flush out algae in the river. Aquaculturalists could be assigned the responsibility of monitoring chlorophyll *a* at their sites by using a submersible, portable

fluorometer. The value of chlorophyll *a* could be read off the meter immediately. If chlorophyll *a* was found too high, EGAT and RID staff could make use of the environmental tool and decide on how much water should be released.

In this study, a diagram shown in Figure 4.127 was constructed as an environmental tool for reducing algae to a certain percentage, under assumptions that there was no loading of algae from the reservoir and runoff. The period from April 21$^{st}$-June 30$^{th}$, 1999 was selected for the adjustment of the flows. Figure 4.128 is another diagram created by changing the flows during the period from April 27$^{th}$-June 30$^{th}$, 1999. The first diagram shows a higher reduction percentage of *Chl a* in the beginning, as expected, because the flows were adjusted earlier. However, both diagrams leveled off at about the same percent flow increase. It was clear that the earlier the flows were adjusted, the faster the reduction of algae. According to the diagrams in Figures 4.127 and 4.128, whatever the amount of chlorophyll *a* might be in the river, it could be reduced by 50-60% if the flow was increased by 100%.

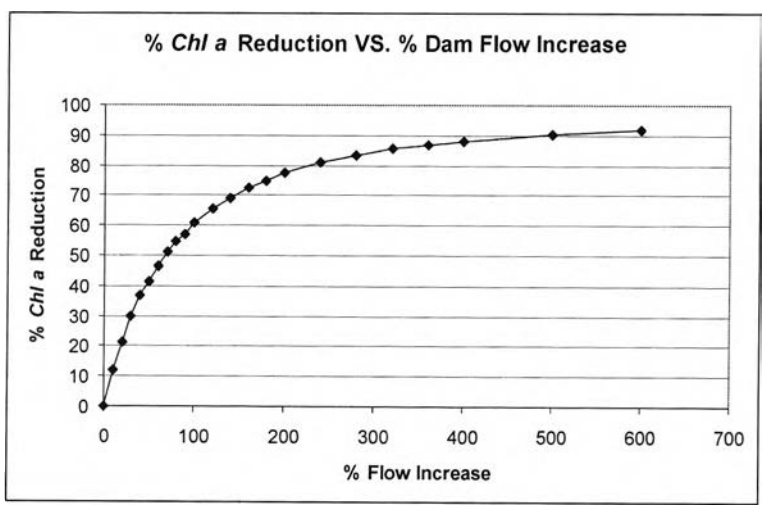



Figure 4.127. Percent reduction of *Chl a* at the CT aquaculture versus percent flow increase from April 21$^{st}$, 1999.

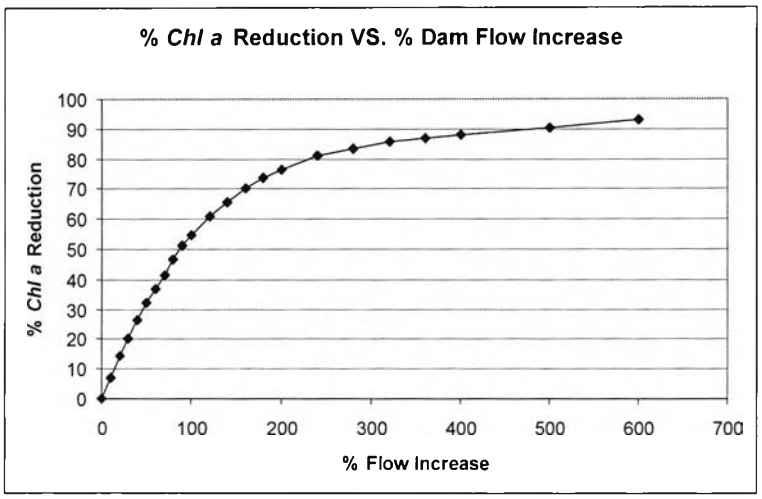



Figure 4.128. Percent reduction of *Chl a* at the CT aquaculture versus percent flow increase from April 27th, 1999.

If there were algae in the reservoir, the above diagrams could not be used. Figure 4.129 was determined under similar conditions as Figures 4.127 and 4.128, except there was 5 μg/L algae in the reservoir water. The baseline scenario in this case was run with 5 μg/L algae in the reservoir water. According to Figure 4.129, when the flow was increased in the beginning, algae at CT actually increased as seen by a negative value of percent reduction. It was because the flow was not high enough, and the algae from the reservoir proliferated from the available nutrients in the river. If there were 5 μg/L algae in the reservoir water, the Dam flows had to be increased by 800% in order to achieve an algal reduction of approximately 45%. Compared to Figures 4.127 and 4.128, it seemed more practical and possibly economical to keep the algae in the reservoir water under control for flushing the bloom in the river downstream.

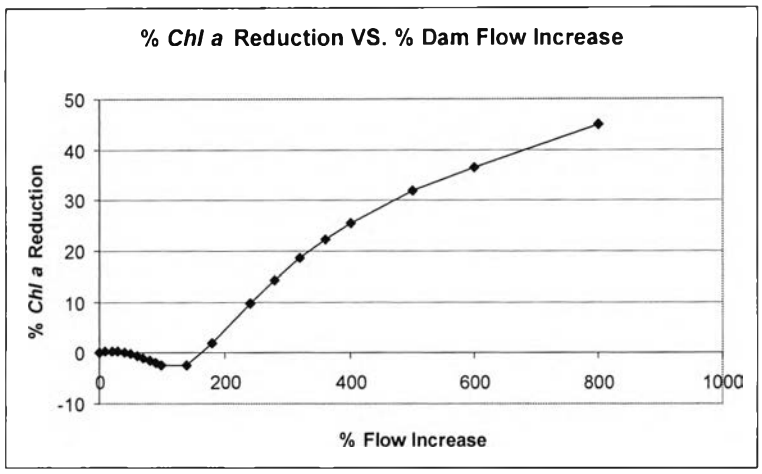



Figure 4.129. Percent reduction of *Chl a* at the CT aquaculture versus percent flow increase from April 21st, 1999, with 5 µg/L *Chl a* in the reservoir water.

Figure 4.130 was constructed under assumptions that there were existing algae in the river water, and no algae in the reservoir. On April 29th, there were 2.24 µg/L *Chl a* at CT. With increased flows of a hundred fold, *Chl a* could be reduced by 42%. Figure 4.131 was constructed under similar conditions as Figure 4.130, except that the amount of algae in the river was adjusted higher. On April 29th, there were 4.06 µg/L *Chl a* at CT. With increased flows of a hundred fold, *Chl a* could be reduced by 47%. Both Figures 4.130-4.131 demonstrats that similar percent reductions of algae at CT could be achieved for the same amount of percent flow increase; the difference of existing algae in the segment produced a very small effect on these two figures.

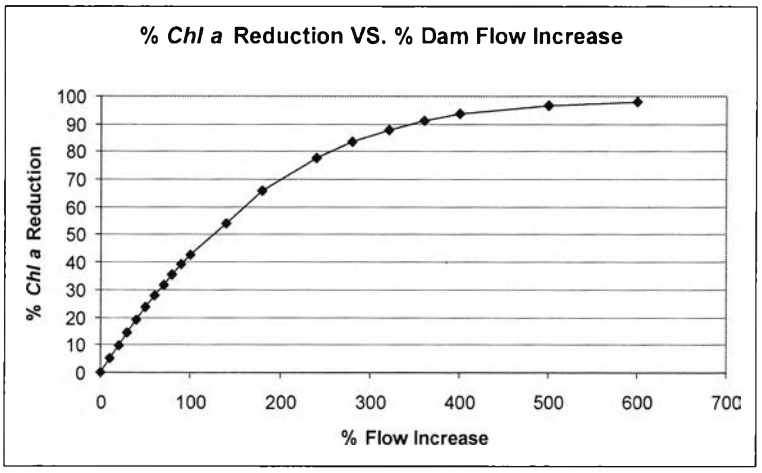



Figure 4.130. Percent reduction of *Chl a* at the CT aquaculture versus percent flow increase from April 27th, 1999 with no *Chl a* in the reservoir water. On April 29th, *Chl a* was 2.24 µg/L.

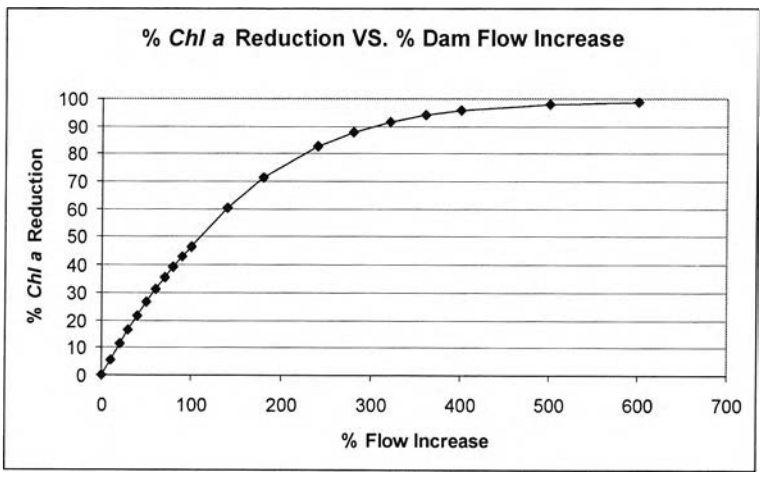



Figure 4.131. Percent reduction of *Chl a* at the CT aquaculture versus percent flow increase from April 27th, 1999 with no *Chl a* in the reservoir water. On April 29th, *Chl a* was 4.06 µg/L.

2. Reduction of algae to an unharmful level for Nile tilapia

By assuming that the *Chl a* level in the baseline scenario before the bloom was acceptable for N. tilapia aquaculture, this level was set as a target for simulation to determine the flows. Figure 4.132 illustrates that *Chl a* reduced to approximately the same level as before the bloom when the flow was increased by 500%.

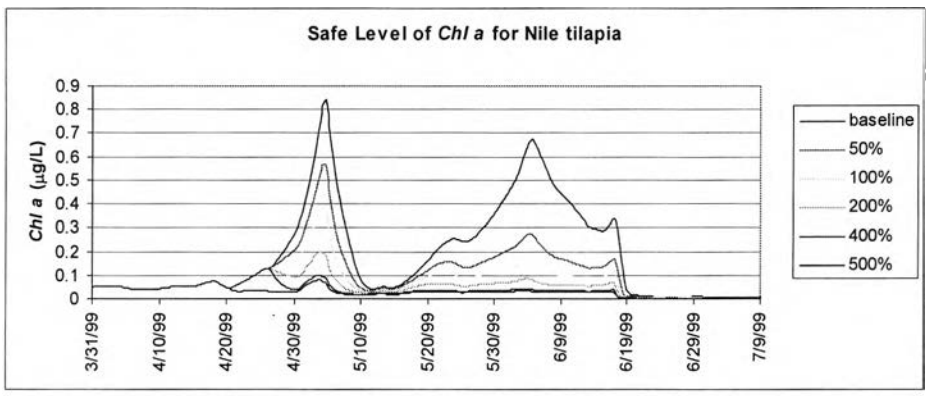



Figure 4.132. Percent flow increase to reduce *Chl a* to the safe level for tilapia.

If increasing the Dam flows by 500% from the average of 0.49 MCM/day between 4/27/1999 – 6/17/1999, was uneconomical during this period, other alternative methods shown in Table 4.11 were derived to meet both economic and safety objectives. Figure 4.133 illustrates the comparison of *Chl a* between the baseline scenario with scenarios with flows of 500% every day, 500% every other day (designated as 500% 1L-1H), 500% every two days (designated as 500% 2L-1H), every two days with minimum of 1 MCM/day, continuous 1 MCM/day, and continuous 1.2 MCM/day. The reduction of algae was achieved at 100, 81, 62, 84, 75 and 82%, respectively. The best algal reduction was achieved when the Dam water was released at 500% of the past flows. The amount of water saved, compared with the release of 500% from April 27$^{th}$ - June 17$^{th}$, 1999 was 41, 57, 45, 66, and 59% for releasing 500% every other day, 500% every two days, 500% every two days with minimum of 1 MCM/day, continuous 1 MCM/day, and continuous 1.2 MCM/day, respectively.

If the reduction efficiency was defined as the amount of algae reduction per water release, the efficiencies were 5.7, 7.8, 8.1, 8.7, 12.5, and 11.4% for scenarios of releasing the flows with 500% every day, 500% every other day, 500% every two days, every two days with

a minimum of 1 MCM, continuous 1 MCM and continuous 1.2 MCM, respectively. Releasing a constant flow of 1 MCM yielded the best efficiency, but not the most percent reduction. The most percent algal reduction was achieved by releasing water at 500% of the past flows.

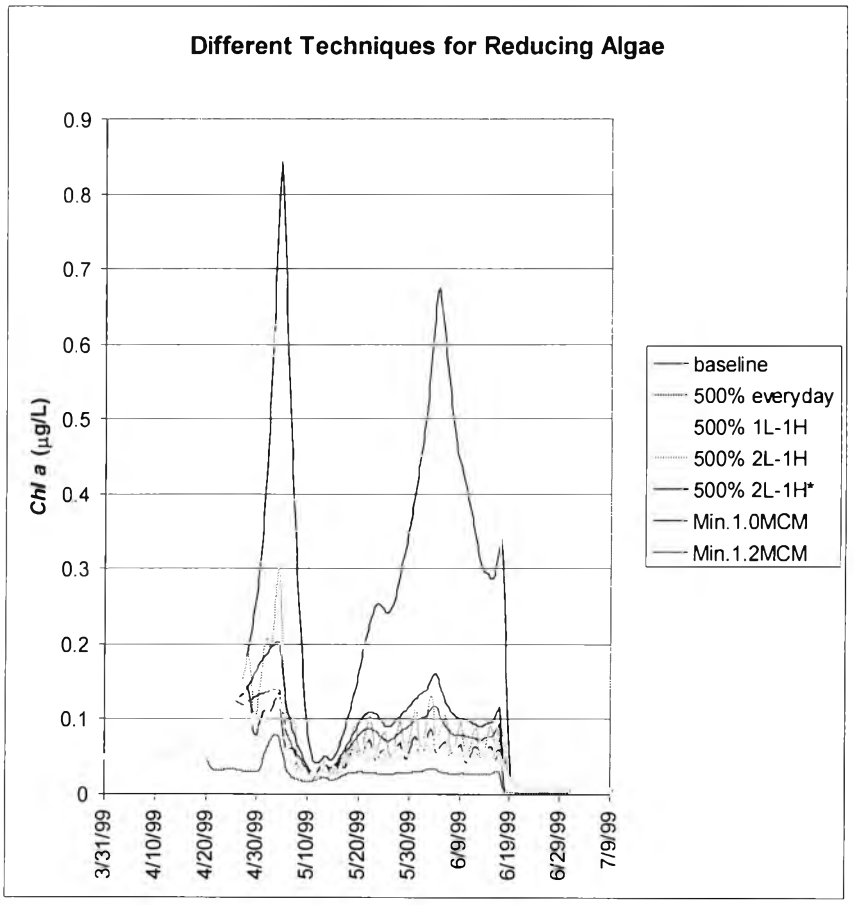



Figure 4.133. Comparison between releasing 500% of water every day, every other day (1L-1H), every two days (2L-1H), every two days with min. of 1 MCM, continuous 1 MCM (2L-1H*), and continuous 1.2 MCM.

Table 4.11 Different methods for reducing algae and their associated benefits and efficiencies from April 27th-June 17th, 1999.

| Method | Method Descriptions | % Algal Reduction | % Water Saved | % Efficiency in |
|---|---|---|---|---|

| No. | | from Actual Case | Compared to Method 2 | Algal Reduction/ water released |
|---|---|---|---|---|
| 1 | Actual flows | 0 | - | - |
| 2 | 500% of actual flows every day | 0 | 0 | 5.7 |
| 3 | 500% of actual flows every other day | 81 | 40.71 | 7.8 |
| 4 | 500% of actual flows every 2 days | 62 | 56.57 | 8.1 |
| 5 | 500% of actual flows every 2 days with min. 1 MCM | 84 | 44.81 | 8.7 |
| 6 | Continuous 1 MCM (EGAT's Recommendation) | 75 | 65.73 | 12.5 |
| 7 | Continuous 1.2 MCM | 82 | 58.86 | 11.4 |

From the above illustrations, it was recommended that algae should be kept under control in the reservoir, in order to lend its water's effectiveness in flushing the algae out of the river. Since *Chl a* was not calibrated in this model, it would be very difficult to be exact about how much water should be released from the Dam to rid of the algal bloom. Assuming that there was no algae in the reservoir and run-off, this model could predict that the highest efficiency of algal reduction per water release was achieve by releasing 1 MCM per day to flush out algae efficiency, but for the best percent algal reduction, the flow should be increased to 500% of the actual flows or approximately 3 MCM/day. All above methods would become ineffective if the Dam had algae because the nutrients in the river only worsened the problem.

Similar to the reservoir, algae in the Chot lagoon and Sua Ten should also be kept under control because with high initial algae, they could multiply very quickly and bloom in the Lake. This might explain why fish at the ST aquaculture died quite often. Longer residence time in the river also allowed time for algae to grow more, resulting in more fish deaths every year at the last aquaculture site, KP/BN, in the segment with the longest residence time.

The case of the 1999 fish kills with low DO was considered more severe than recent years. The fish death in the river was only 1-2% everyday from the end of June to July in 2002. A drop in DO did not stand out. Possibly, the size of the bloom in recent years was less due to numerous reasons. First of all, the mill stopped releasing its effluent into the Chot lagoon; even though the effluent was treated, it still contained about 15.76 mg/L of OP, 5.2 mg/L of TP and 2.3 mg/L of TKN (CMS, 1995). These nutrients at a loading rate of 15,750 $m^3$/day would sustain the high algal population in the Chot lagoon even when there was no agricultural runoff. The flood at the end of 2002 might also dilute most of the existing algal population in the river. The bottom of the outer part of the Chot lagoon which was in 2002 covered with an unmistakable thick mat of green algae on submerged aquatic plants, became less visible; most of the aquatic plants and algae disappeared.

Secondly, water hyacinth had been regularly removed from the fish pond in recent years. This activity, in essence, removed OP, soluble reactive phosphate (SRP), TP, $NH_3$-N, $NO_2$-N and $NO_3$-N in the water column as well as TN and TP in the sediment, and induced heterogeneity in the planktonic community required for fish growth (Saha and Jana, 2003). Although there was no release of the effluent in the fish pond in recent years, the remnants of the past effluent's spill might have caused the re-suspension of N and P from the sediment – even though it was once dredged.

Thirdly, Dam flows in recent years have not been as continuously low as 0.2-0.5 MCM per day for a long time (52 days in 1999). From 2001-2004, the period that the Dam released water below 1 MCM lasted only 12 days in 2002.

Finally, the conditions required for a maximum bloom must have occurred in the correct sequence described. To begin with, heavy rain around the reservoir would bring about N and P from leaching and runoff. These nutrients would produce a large algal population in the reservoir to be released into the river. If the Dam released water very slowly, the residence time in the river would allow algae from the reservoir to multiply with more nutrients from the runoff. And the last condition of the low light extinction coefficient must follow after the large bloom was produced, to cause the bloom to die. Its cellular degradation would produce low DO as detected. If any of the mentioned conditions did not occur in the correct sequence, only small blooms would result and the microcystin toxin might produce a small amount of fish kills and physical properties such as popped-out eyes and rashes.

## 4.4 RESEARCH LIMITATION

1) The water quality inside Lake Sua Ten was not monitored; therefore, only the typical nutrient values of waterbody were used. This waterbody was different from others because there were fish kills around the lake and their nutrient data should not have been similar to the typical ones.

2) $PO_4$ was not monitored in 1999 and 2000; therefore it could not be calibrated in the model. $PO_4$ from the reservoir was assumed to be zero in the model. Even if $PO_4$ was monitored in the river in 1999 and 2000, it would have been very difficult to detect it because $PO_4$ was a limiting nutrient for algal uptake in freshwater body, and it would have been readily

taken up by the algae in this eutrophic river. For this reason, TP was often measured instead of $PO_4$, but TP could not be applied with WASP6.1. Therefore, a direct measurement of algae would be the best way for modeling eutrophication in the river.

3) The algal growth from $NO_3$ as the secondary choice of nutrient was not considered. The algorithm only allowed $NH_3$-N as the nutrient for algae. In 1999, there was a nitrate leaching into the reservoir, this could be an alternative supply of nutrient for algae which was not accounted for. Thus, in 1999, there could have been more algal population than predicted from $NH_3$-N only.

4) Benthic nitrogen and phosphorus fluxes which could represent internal nitrogen and phosphorus loadings of 78% and 50%, respectively, particular during high temperature and low DO (Tufford and McKellar, 1999), were not considered. These sources affected the algal bloom.