

To: Ron Larson, USFWS

From: Tamara Wood, USGS

Re: Water Quality sections of the 2001 Biological Opinion for the Klamath Project

As you requested, I have looked at those sections of the 2001 Biological Opinion (BO) that deal specifically with water quality issues, and I have the following review comments to offer for your consideration.

As you know, those sections of the BO that deal specifically with water quality rely heavily on a report from R2 Resource Consultants (Welch and Burke 2001, hereafter referred to as the R2 report). It quickly became apparent that I needed to go back to the source to properly assess many of the arguments presented. It was unavoidable, therefore, that much of the following pertains directly to the R2 report rather than to the BO.

After struggling for some time with how best to present my comments in a form most useful to you as you prepare the new BO, I settled on the following. I believe that there are seven basic arguments that make their way into the 2001 BO regarding the dependence of water quality on lake elevation. I have organized my comments around those seven arguments. Please note that, in the interests of focusing attention on the “big picture”, I have declined to make editorial comments on the BO itself. In an earlier review of the draft 2001 BO a USGS colleague, Chauncey Anderson, made extensive comments on the text of the document itself, and I believe that many of those still warrant attention.

It is my sincere hope, in preparing these comments, that they are useful and that they inject some fresh perspective into the controversy surrounding the BO, rather than simply muddling the issues further.

### **Argument 1. Higher lake elevation leads to lower water-column light availability, which leads to light limitation of algal growth and reductions in chlorophyll-a.**

*Summary.* This argument has a sound theoretical basis, and has been confirmed by observations elsewhere in the world, but the effect in UKL is quantitatively overstated in the R2 report. My recalculation of the growth curves presented in the R2 report (see explanation below) yielded a maximum calculated reduction in the 30-day biomass of 75

ug/L chlorophyll-a over the range of observed June lake elevation (1.8 to 2.7 m average depth) from 1990 to 2000. For comparison, the measured range of the median lake wide June chlorophyll-a concentration, as calculated from the 1990-2000 data from UKL, is 230 ug/L. The predicted underlying trend in the data due to light limitation with increasing depth, therefore, is about a third of the overall observed variability in the data. A trend of this magnitude should be evident in the 11 years of UKL monitoring data; because it is not, it is likely that the magnitude of the effect is smaller than indicated in the BO or the R2 report. This may be because *A. flos aquae* is able to regulate its location in the water column, or there may be other confounding factors as well. Thus, while it would be reasonable to assume that some increase in light limitation, and consequent decrease in biomass and pH, occurs with increased lake elevation in UKL, this dependence on lake elevation is small compared to the larger inherent variability in these quantities due to climatic and other factors.

*Supporting details:*

1. (Recalculation of R2 analytical results.) I attempted to duplicate the information in Equation 3-4 and in Table 3-2 of R2 using the constants that are provided in the text. I was unable to do so; it appears that a programming error involving the length of the photoperiod may have resulted in the unrealistically large biomass predictions provided, but I cannot confirm this.

Furthermore, it appears from the explanation in the text on page 3-7 that the equations have been manipulated such that the production of biomass is independent of the amount of incident light,  $I_o$ . This is done by making both the half-saturation constant used in the Michaelis-Menton formulation and the saturating light intensity used in the Steele formulation proportional to incident light. While it is true that these quantities can change, depending on the light regime to which the algae are acclimated, the effect of this strict proportionality is that the biomass produced would be the same if  $I_o$  were 6 or 600 W/m<sup>2</sup>, which seems unrealistic. I was unable to find the justification for this proportionality in either of the references cited, although the Pechan (1992) reference is quite long and perhaps I missed something. I chose instead

to use an incident light based on the data collected during the biweekly monitoring program, and came up with a representative spring value of 500 W/m<sup>2</sup>. The EPA reference (Bowie, et al., 1985) does indicate that optimal conditions for photosynthesis have been observed to occur at the point in the water column where light is about 30% of incident, so I used that relation for saturation intensity. A representative value for light half-saturation (used in the M-M model, but not found in the Pechan, 1992 reference that was cited) was taken from a table in Bowie, et al. (1985). I am providing you with the spreadsheet so that you can check my assumptions or try out some of your own.

The end result of this exercise is that the Michaelis-Menton and Steele chlorophyll-a concentrations I was able to obtain are probably more realistic than those in the R2 report. I was able to determine the predicted reduction in chlorophyll-a concentration as water column depth increases. A 60-day time frame is used in the R2 report, but I used a 30-day time frame for the comparison provided in the summary because at 60 days growth has already started to level off. As indicated above, this value is equivalent to about a third of the overall range in June chlorophyll-a concentration in the 11 years of monitoring data.

2. The considerable uncertainty in this analysis was not acknowledged in the BO. This type of exercise can be quite useful for checking assumptions, as long as the results are not taken too literally. In this case, it seems that the effect of light limitation on the blue-green bloom in Upper Klamath Lake is less than this simple theory would suggest.

The literature on blue-greens describes versatile organisms that demonstrate a remarkable ability to adapt to light conditions—both low and high. It is unclear from the text in R2 which of the references cited involve lakes where AFA or another buoyant blue-green dominates the algal assemblage. This information is of particular interest for Lake Vörtsjärv, as direct analogies are made between this lake and UKL. The references I was able to obtain for Lake Vörtsjärv do not entirely clarify the composition of the algal assemblage. Nöges, et al. (1997) refer to an “abundance” of the blue-green *Planktolyngbya subtilis*, an *Oscillatoria* type of filamentous blue-green

that contains gas vacuoles but has no nitrogen-fixing capability. A decreasing trend in the biomass of this blue-green, which might well be expected to respond to light conditions similarly to AFA, as well as several “minor” phytoplankton groups like chlorophytes and chrysophytes, was observed to correspond with the increasing trend in lake elevation. It is also noted, however, both in Nöges, et al. (1997) and Nöges and Jarvet (1995), that *Limnothrix redekei*, another buoyant, filamentous blue-green, successfully competed with *P. subtilis* and increased in biomass dramatically when the lake elevation became higher. *L. redekei* appears to make up a large fraction of the smaller bloom that occurred at the recent, higher lake elevations. Thus the effect on some blue-greens could be the opposite of what is predicted by the light limitation argument.

AFA is without doubt periodically mixed throughout the water column in UKL. It is also clear, however, that the AFA much of the time is concentrated in mats that may be at or under the surface of the lake, suggesting that the cells do regulate themselves to optimize their position in the water column, and that they can do so quickly. USGS divers who spent several weeks in the lake during an SOD study confirmed that AFA was not well-mixed through the water column during that time. The amount of time that the cells spend in mat formation vs. mixed through the water column is an unknown, but if a large fraction of the time the algae are optimized with respect to their location in the water column, that would certainly diminish the effectiveness of the light limitation effect with increased water column depth.

3. The reader is reminded at several points in the R2 report that one should not expect a simple, univariate relation between chlorophyll-a and lake elevation because interactions with weather obscure such relations. Nonetheless, the figures shown for Lake Vörtsjärv apparently do show such a univariate relation, and so one must ask how apt the comparison is between these two lakes. Quantitative comparisons of biomass with UKL are not possible, because no guidance is given as to how to convert the BFP (total phytoplankton biomass in  $\text{g m}^{-3}$ ) to chlorophyll concentration. The R2 report indicates that Lake Vörtsjärv operates in a different trophic regime than UKL, as evidenced by the pH values, which are lower than those observed in UKL.

The R2 report notes that the lakes are similar in terms of overall average surface area and average depth. Nõges and Jarvet (1995) indicate that the annual fluctuation in water level in Lake Võrtsjärv has averaged about 1.4 m over the years of record, comparable to the annual fluctuation in UKL. It appears, therefore, that in terms of water column depth, both the mean value and the annual fluctuations, the comparison between UKL and Lake Võrtsjärv is appropriate.

One could ask if the response in Lake Võrtsjärv appears more dramatic because the change in depth was more extreme than what has occurred over the length of record in UKL. The increase in depth that occurred in Lake Võrtsjärv between the years prior to and after 1978 was about 1.1 m. The total range in lake elevation and average depth (assuming the relation between the two is approximately one-to-one) in UKL for the summer months during the decade between 1990 and 2000 has been about a meter (June/July/August mean elevation 4139.14 to 4142.44 ft). Thus the water quality record in each lake covers a comparable range in depth. Yet, there is little indication in UKL of a dramatic dependence of chlorophyll-a on lake elevation, such as seen in Lake Võrtsjärv. Direct analogies between these two lakes should therefore be made with caution, but perhaps a further examination of the *differences* between these two lakes could provide insight into important processes.

## **Argument 2. Higher lake elevation leads to a lag in spring temperature response to an increase in air temperature, thus delaying, and limiting the size of, the bloom.**

*Summary.* This argument has a sound theoretical basis but the effect is smaller than the BO implies. Fluctuations in observed daily-averaged water temperatures track fluctuations in daily-averaged air temperatures with a lag of only 1 to 3 days. One question that might deserve further investigation is the dependence of the magnitude of the diel fluctuations in water temperature on lake volume.

### *Supporting details:*

1. The best way to examine this question is by looking directly at temperature data. To demonstrate, I took the temperature data collected at the USGS RUSS buoy station last summer (located north of Ball Pt.) and compared it to air temperature

collected at the airport, obtained from the Western Region Climate Center. Only August through September data were available at the RUSS buoy, but the lake should respond similarly to a warming and cooling trend in air temperature. The daily-averaged temperature data from the uppermost spot in the profile (usually around 0.5 m; closer to 1 m in the first half of August) is plotted with daily-averaged air temperature in figure 1. The depth at the location of the RUSS buoy varied from about 3 m at the beginning of August to closer to 2.5 m at the end of October. The fluctuations in lake temperature follow those in air temperature closely with only a short lag between distinct peaks and valleys in the respective datasets; indeed, the correlation coefficient between these two datasets is a maximum of 0.84 at a 3-day lag (correlation coefficients computed with the SAS statistical package). Undoubtedly there are other datasets that could be used to examine this question more carefully than has been done here, but it seems unlikely that the lag time between a change in the daily-averaged air temperature and the response in water temperature at any lake elevation reaches the number days (7-30) reported in Table 3-3 of the R2 report.

2. (Limitations of R2 presentation.) The temperature results are presented in Section 3.3 with little discussion of the model used to generate the numbers or the meteorological data that must have been used to properly model heat transfer at the surface of the water column. The quote of a “range” in equilibrium temperature, with no reference to a “range” in the air temperature used to generate the warming scenario, is confusing. With so little to go on, it is impossible to carefully evaluate what was done to arrive at the numbers and the conclusion in the R2 report.

It also is unclear what the initial conditions were for the scenarios presented, which would make a big difference. In reality, the lake does not respond to a step function increase in daily-averaged air temperature, but rather to a relatively gradual increase through the early spring. Thus, lake temperature adjusts incrementally as the season progresses. In visualizing this process, it helps to realize that the diel fluctuations in air temperature are much greater than the day-to-day or even week-to-week fluctuations, and that diel fluctuations in air temperature bracket entirely the smaller diel fluctuations in water temperature. Thus, the water temperature actually crosses the equilibrium temperature twice a day. For this reason, perhaps a more

interesting question to ask would be whether the lake volume has a significant affect on the magnitude of diel swings in temperature, which could theoretically have some effect on the daily maximums (and minimums) in water temperature.

### **Argument 3. Higher lake elevation leads to a lower probability of exceeding pH of a given value.**

*Summary.* To the extent that higher lake elevation limits the size of the bloom, it clearly follows that the overall distribution in pH values should be lower and fewer pH exceedences should occur. However, the establishment of a quantitative link between higher lake elevation and smaller bloom size remains elusive. This link cannot be made empirically with the monitoring data collected since 1990; it must be made, therefore, based on theoretical arguments. Those theoretical arguments are accompanied by the additional constraints that a) one would not expect to observe this link in the monitoring data collected since 1990 because the effect is small compared to the larger variability imposed on the system by climate, and b) even though the effect is small, it is significant when attempting to improve the odds for endangered species. Whether one accepts the theoretical arguments linking bloom size and lake elevation, and the associated constraints, remains, unfortunately, a matter of belief and opinion, about which knowledgeable experts can legitimately disagree.

In developing the BO it is important to acknowledge where observation leaves off and theory takes over, and that line is often not made clear in the 2001 BO. A USGS colleague who provided comments on the draft version of the BO noted this same problem—"I suggest you make it very clear where the report is referring to hypotheses, so that readers can understand where the decision is based on solid evidence vs. assumptions or theories," (C. Anderson, 2001, unpub.) The lack of a clear distinction between hypothesis and observational evidence is particularly evident in the discussions in the BO concerning the relation between lake elevation and bloom size, leading to a relation with pH exceedences. The argument is taken beyond that which can be directly supported by data (i.e. empirical relations among total phosphorus, chlorophyll, and pH), and into the realm of hypothesis (i.e. relating phosphorus to lake level). As a result, the quantitative relations developed between hypothetical minimum lake elevation and the

corresponding reduction in the pHs that would have occurred over the last decade should be viewed with skepticism.

*Supporting details:*

1. (Evaluation of the theoretical arguments presented in R2.) The R2 presentation is actually in 3 distinct parts. The empirical relations developed between chlorophyll-a, pH, and exceedence frequencies are robust, and, with the Kann and Smith (1999) reference, well documented. This constitutes a useful way of analyzing the data and converting changes in chlorophyll-a to changes in the frequency distribution of pH. It would be very useful for setting “target” reductions in chlorophyll-a to achieve improvements in pH conditions. The second part, the conversion of total phosphorus concentration to chlorophyll-a concentration, is based on relations that are less robust, but still acceptable.

The weakest part of this presentation is the calculation of a revised total phosphorus concentration covering the 11 years of data, based on the assumption that internal loading would remain the same and the effect of lake elevation would be to dilute phosphorus concentrations. Documentation of the procedures is minimal, but I assume that the baseline phosphorus model is fundamentally a mass balance calculation based on the biweekly monitoring data. The mechanisms that control internal loading of phosphorus in UKL are still poorly understood. Eleven years of monitoring data have shed little light on this particular problem, but these data do not provide evidence that dilution plays much of a role in determining year-to-year differences in phosphorus concentration. Therefore, the calculation of a revised historical total phosphorus concentration based on a) keeping internal loading the same, and b) incorporating volume dilution when lake elevation is assumed to have been higher than actually occurred, is simplistic and not likely to be very accurate.

The argument is further weakened because the presentation of the model does not conform to standard modeling practice. The model results are presented with little discussion of uncertainty, other than to declare that all assumptions were conservative and that the effects could only be greater than predicted, not less. It is indicated that the model has been calibrated, but there is no indication as to how. Most importantly,



there is no presentation of how well the “calibrated” model performs in reproducing the observed values of chlorophyll-a, and pH on the calibration and validation datasets. Without this kind of information, it is impossible to evaluate how well this model performed even before the imposition of the assumptions made in order to calculate a revised historical phosphorus concentration.

#### **Argument 4. Higher lake elevation has only a weak effect on water column stability, and therefore is not important in bringing on the conditions that lead to a fish kill.**

*Summary.* The dependence of water column stability (as measured by RTRM) on lake elevation was not fully explored in the R2 report, largely because the relevant independent variable is not lake elevation, but rather water column depth, which varies over the lake at any given elevation. Further study and data collection is required to establish water column stability as both a spatial and temporal phenomenon. The transient nature of the water column stability cannot be fully appreciated with biweekly monitoring data, and it is likely that in many cases stability that appears persistent in the biweekly data is, in fact, routinely dissipated by diel temperature fluctuations and re-established the following day.

The role of water column stability in setting up the conditions for a fish kill also needs to be further developed. On close inspection, the connections between water column stability and the low dissolved oxygen and high ammonia concentrations that lead to the fish kills are not as obvious as they first appear. Given the choice, therefore, between managing the lake during the mid to late summer in order to avoid stability or to increase the volume dilution of ammonia and SOD, the better choice for protecting endangered species may well be to manage for volume dilution, which is discussed below with Arguments 6 and 7.

##### *Supporting details:*

1. (*Discussion of RTRM in the R2 Report.*) The R2 report does not adequately make the case that water column stability is only weakly affected by lake elevation, for the following reasons:

First, the significance of lake elevation in determining water column stability RTRM is missed, largely because the relevant independent variable is not actually lake elevation, but rather water column depth, which varies spatially over the lake for any given lake elevation. Also, because RTRM is a purely physical quantity calculated from densities, and because stability can set up and dissipate quickly, there's no compelling reason to work with averages, either spatial or temporal, of this parameter when investigating dependencies. If the data are separated into 3 groups- the shallow sites Wocas Bay and Shoalwater Bay, the mid-lake sites Midlake, Midnorth, North Buck Island, Pelican Marina, and the deep-lake site Eagle Ridge by itself- then the water column depth appears to define an upper limit for the RTRM. The actual value of the RTRM is determined in part by wind, but it appears that air temperature also is significant. For example, the value for 1996, which appears as an outlier on several plots (e.g. 5-2 and 5-3), is probably due to exceptionally high air temperature. Whether lake elevation is important to stability or not depends on determining a "critical" RTRM, loosely defined as the threshold for persistent stability (i.e. stability that is not broken down on a nearly daily basis), and determining what total area of the lake would allow a critical RTRM at a given elevation.

Second, the modeling effort suffers from a lack of supporting documentation. The reference cited for the model used to calculate RTRM is focused on artificial aeration in ice-covered lakes, and is not particularly useful for understanding how the model transfers heat and momentum, so it is difficult to evaluate how appropriate the model is. Furthermore, the same criticism that was made of the pH model applies here: there is no attempt to show a calibration/verification process for the model, and thus no way to evaluate its performance. RTRM values are presented in fig. 5-5 with no indication of where the observations during the same time period would fall on the graph. Furthermore, the logic behind choosing a water column much deeper than most of the lake for calculating RTRM values is confusing. It seems that a deeper water column would exhibit less dependence on lake elevation than a shallower one, because the relative depth change of a few feet in elevation is smaller. If the point is

to demonstrate how RTRM values would depend on lake elevation over most of the lake, however, it seems that a shallower test case would be a better choice.

2. The role that water column stability plays in bringing on the fish kills still needs to be fleshed out. Perkins, et al. (2000) showed that thermal stability was measured on several sampling dates prior to the 1995, but particularly prior to the 1996 and 1997 fish kills. The authors also showed that the stability had collapsed prior to the actual fish kills. It was suggested, however, that the persistent stability that preceded the kills for several weeks was responsible for generating the low dissolved oxygen conditions that were identified as the cause of death, as well as high ammonia concentrations that stressed the fish for weeks prior to that point. Comments with regard to both of these aspects of water quality follow.

*(Water column stability and low dissolved oxygen concentration.)* If the fish kills had coincided directly with water column stability, it would be relatively straightforward to associate low near-bottom dissolved oxygen conditions with the cause of fish death. Once the water column mixes, however, the overall concentration is a volume-weighted mixture of the two layers. If the upper layer is supersaturated due to photosynthetic activity, then the overall water column concentration may well be much higher than the previously depleted bottom layer. It appears from the data that the low dissolved oxygen concentrations at the time of the fish kills probably had at least as much to do with the cessation of photosynthesis, in combination with ongoing oxygen-demanding processes including SOD, BOD, and nitrification, as with the mixing of low-DO near-bottom water into the water column.

The interpretation of the role of stability in bringing on fish kills is made even more difficult by the biweekly nature of the data, since stability that appears persistent may not be at all. Recent data from the USGS RUSS Buoy indicates that water column stability sets up and dissipates on a daily basis (figure 2.), but if sampling was done only once a day in the afternoon it would appear to be persistent. The question of the role that stability plays in depleting the water column of oxygen and leading to fish kills is intriguing and needs some further investigation,

particularly with datasets designed to investigate stability as a temporal and spatial phenomenon.

*(Water column stability and ammonia concentration.)* The data on ammonia is truly extraordinary. A large increase in ammonia concentration, starting in 1996 and persisting through 2000 (the latest data that I have), is attributed to an increase in water column stability caused by low-wind summers. Unless I missed something, it was never made explicit in either the BO or the R2 report what exactly is the proposed biological or chemical mechanism behind this connection, but it apparently does rely on the development of a near-bottom layer of water, characterized by low or near-zero dissolved oxygen conditions, in a persistently stable water column.

A couple of logical possibilities for this mechanism come to mind. The first is that dissolved oxygen concentrations get so low as to turn off nitrification, thus interrupting the normal cycling of nitrogen through the compartments in the water column and allowing ammonia to build up. Another possibility is that reducing conditions develop near the sediment-water interface, thus converting nitrogen in the sediments to ammonia, which is then released to the water column. (Note that in the discussion of possible sources of ammonia, it is appropriate to refer to total [ionized plus un-ionized] ammonia. The toxic, un-ionized form of ammonia is simply a fraction of the total that varies primarily with pH. Using un-ionized ammonia to investigate sources obscures the problem.)

There are a few features of the data that don't fit either of these scenarios. The first is that the higher ammonia concentrations observed from 1996 to 2000 were already observed in June, at values of the same order of magnitude as later in the summer, before the setup of persistent water-column stability and before dissolved oxygen concentrations were low enough to either inhibit nitrification or reduce nitrate in the sediments. Furthermore, in the first scenario one might expect the high ammonia concentrations to be offset by a corresponding decrease in nitrate concentrations, thus preserving about the same amount of total nitrogen. The nitrate data, however, while variable, show no obvious change in character like the ammonia data starting in 1996. Finally, in the summer of 2000 July/August winds were back to

the same levels seen earlier in the decade, even though ammonia concentrations remained high. Data from the summer of 2001 will be very interesting, as it appears that 2001 winds were actually higher than the average earlier in the decade.

Because of the difficulties with explaining higher 1996-2000 ammonia concentrations with water column stability alone, it may be worthwhile to consider alternative explanations. Ammonia concentrations suddenly increased in 1996, after 2 years of high flow in the Klamath Basin. The ammonia that appears in June could be seen as simply the excess, above and beyond what the algae can use, resulting from the decay of a big pulse of nutrient-rich sediment that made its way into the basin as a result of these high flows. 1997 was an even bigger runoff year in the Klamath basin than was 1996; it is possible that 3 years in a row of increasingly larger flows washed a great deal of sediment into the lake and that the effect of that pulse was still apparent in 2000.

In the table below, we provide the recurrence interval of flows in the basin associated with known fish-kill years. Most of these years also were years characterized by spring runoff high enough to be considered “extreme”. It also is noteworthy that the loading of sediment to the lake associated with extreme flows might have become much greater during the last century, as wetland loss accompanied development.

Fish Kill Year	Spring Runoff Recurrence Interval (estimated)
1932	Extreme low flow
1971	7 yr
1986	15 yr
1995	7 yr
1996	15 yr
1997	20 yr

There are, of course, pieces of this story that don’t fit the available data either. The extreme low flows of 1932 don’t fit the pattern. Also, spring runoff in 1993 had a 10 year recurrence interval and there was no fish kill in that year. Nonetheless, this is an idea that probably deserves further thought and scrutiny. It is also worth noting an

even more fundamental question: How could it be possible to build up the kind of ammonia concentrations (several hundred  $\mu\text{g/L}$ , approaching 1  $\text{mg/L}$ ) that were observed in the spring and summer of 1996-2000? In the presence of oxygen, why doesn't nitrification kick in and convert that ammonia to nitrate? Does high pH inhibit nitrification? We have not been able to find the answer to the latter question in the literature. This is a question that should be investigated if it has not been previously.

### **Argument 5. Higher lake elevation leads to weaker blooms, lower pH values, and therefore lower internal loading**

*Summary.* The connection between lake elevation and bloom timing and magnitude is discussed above. Both the R2 report and the BO contain numerous references to the “feedback loop” between high pH and internal loading. This connection is stated as if it were fact when in reality it is still a hypothesis. Caution is urged when invoking this argument.

#### *Supporting details:*

Work done by the USGS last summer and fall with Klamath Lake sediments in the laboratory indicates that high pH alone can account for only a small fraction of the internal loading required to support the algal blooms. Because the results are still being analyzed, and because the final report of the work has been delayed by more pressing requests, specific details are not available to provide here. For the time being, it would be prudent to present this argument as hypothetical.

### **Argument 6. Higher lake elevation provides more volume to dilute the oxygen depletion caused by sediment oxygen demand (SOD).**

*Summary.* The argument is conceptually straightforward and theoretically sound and was probably not given as much emphasis as it should have been in the BO. The datasets required to quantify this effect or to attempt some kind of probability risk assessment is not available. It is important to recognize that dissolved oxygen is inherently a spatially and temporally-varying variable and that the worst conditions are

likely to occur in places and at times that are not routinely monitored. In order to properly conduct a risk assessment one would also need to know how the likely spatial occurrence of low dissolved oxygen intersects with areas occupied by fish.

*Supporting details:*

In order to properly evaluate this argument, two pieces of information are needed: First, the biweekly monitoring data are inadequate to map the extent of poor water quality, both spatially and temporally. The temporal component could be particularly important here because photosynthetic production of oxygen turns off every night. The effect of lake elevation dilution on oxygen depletion would be most important in the shallower areas of the lake where a relatively small change in depth (a fraction of a meter) might theoretically mean the difference between repeatedly depleting oxygen overnight or not.

Miranda, et al. (2001) developed a probabilistic risk assessment for a shallow lake in Mississippi. In that study, the probability of DO dropping below 1.5 mg/L in any given dawn increased rapidly in those areas of the lake where depth was less than about 2 m. The total amount of area of the lake where depth was less than 2 m changed with lake elevation, resulting in a strong dependence on lake elevation in the probability of low dissolved oxygen conditions occurring in the lake overnight.

Given better temporal and spatial information, a similar type of assessment could be attempted for UKL, but the second piece of required information is what areas of the lake fish prefer to occupy. In developing a probability risk assessment, areas of the lake should be weighted by importance according to the frequency with which fish will occupy those areas.

**Argument 7. Higher lake elevation provides more volume to dilute high ammonia concentrations that can result in high concentrations of the toxic, un-ionized form.**

*Summary.* The ammonia data collected over the 11 years from 1990 to 2000 presents an intriguing question. The increase in ammonia concentration in the last 5 years of that dataset over the first 6 years is dramatic. It also seems likely that these high concentrations contributed in some way to the occurrence of the largest fish kills of the

last 10 years in 1996 and 1997. The reason for the sudden shift in ammonia concentrations is still unclear, but it is unlikely to be caused entirely by increased water column stability, for the reasons discussed above. If the recent, high ammonia concentrations were caused by a pulse of sediments brought into the lake during high flows in 1995-1997, then in the absence of future extreme events, those concentrations should begin to taper off at some point. In the meantime, if ammonia in excess of what the bloom incorporates continues to be liberated by decay processes in the sediments that initiate with warmer temperatures in the spring, it would make sense that more lake volume should provide some dilution.

*Supporting details:* None.

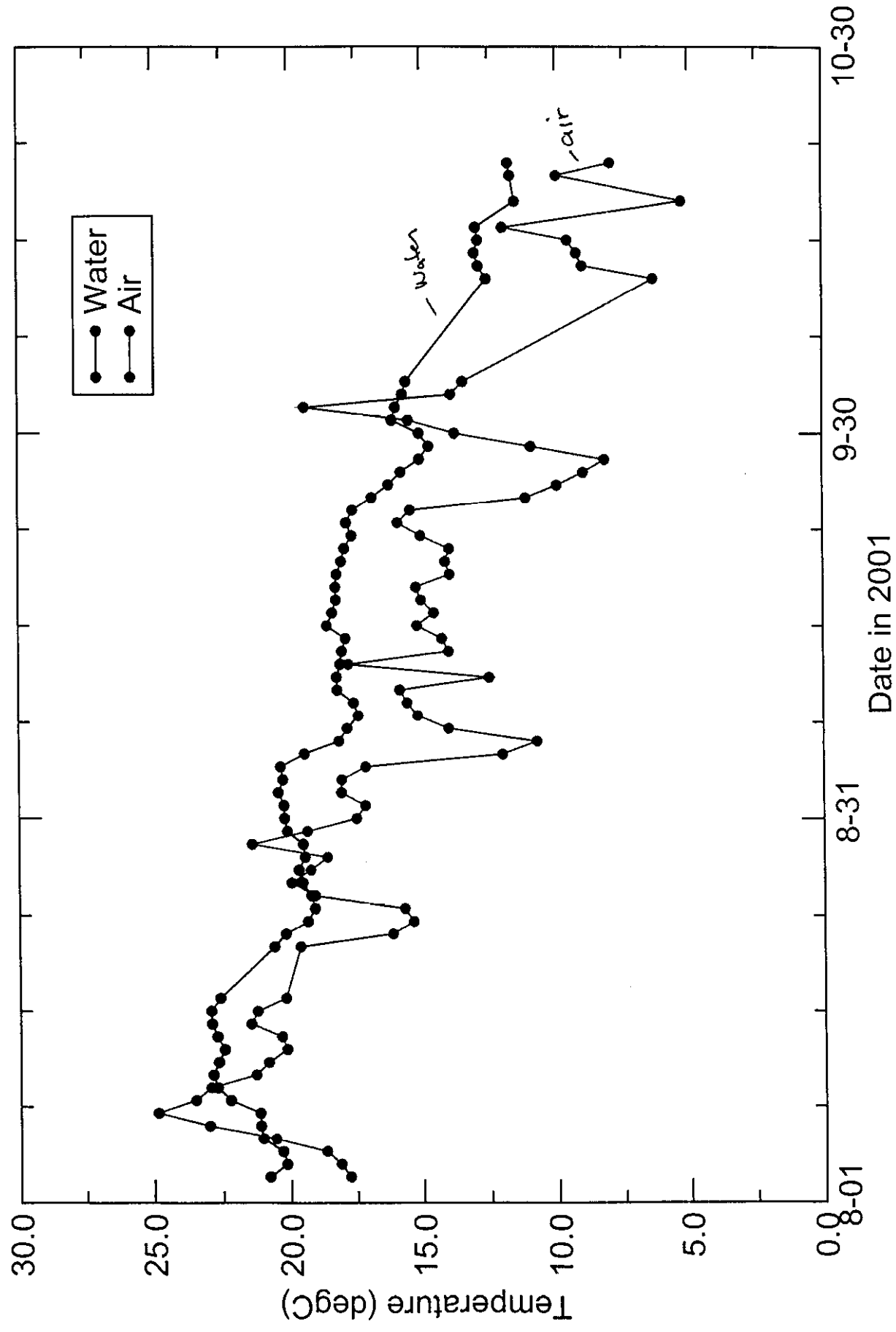
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Temperature at RUSS Buoy, Top of Water Column



RTRM at Russ Buoy in 2001

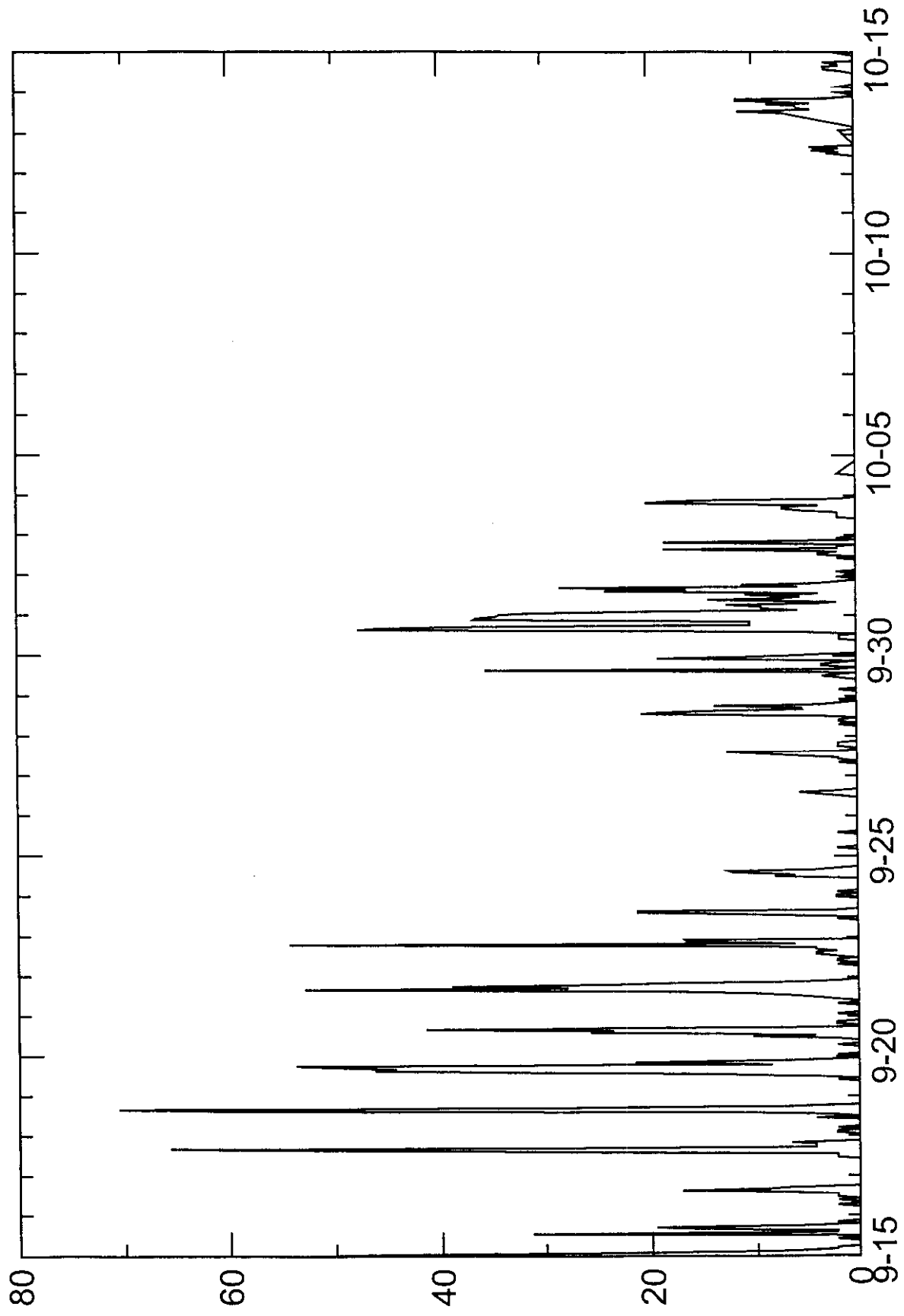


Table 3-2. Predicted maximum algal biomass (chl-a in ug/L) after 60 days  
Water Column Depth (m)

Modeling Method	Mean May-June Temperature	1.5	2	2.7
Michaelis-Menten	14 deg C	252	149	66
Steele	14 deg C	87	34	8
Michaelis-Menten	17.8 deg C	350	223	118
Steele	17.8 deg C	162	74	18

Modified Table 3-2. Predicted reduction in biomass as a percentage of the  
biomass at 1.5 m after 60 days:

Modeling Method	Mean May-June Temperature	1.5	2	2.7
Michaelis-Menten	14 deg C	-	41%	74%
Steele	14 deg C	-	61%	91%
Michaelis-Menten	17.8 deg C	-	36%	66%
Steele	17.8 deg C	-	54%	89%

Modified Table 3-2. Change in 30-d chl-a concentration per meter depth,  
based on slope between 1.8 and 2.7 m depth (representing June depths)  
Slope of regression (ug per L chl-a  
per m depth)

Modeling Method	Mean May-June Temperature	
Michaelis-Menten	14 deg C	-26
Steele	14 deg C	-11
Michaelis-Menten	17.8 deg C	-75
Steele	17.8 deg C	-26

Modified Table 3-2. Change in 60-d chl-a concentration per meter depth,  
based on slope between 1.8 and 2.7 m depth (representing June depths)  
Slope of regression (ug per L chl-a  
per m depth)

Modeling Method	Mean May-June Temperature	
Michaelis-Menten	14 deg C	-130
Steele	14 deg C	-45
Michaelis-Menten	17.8 deg C	-163
Steele	17.8 deg C	-94