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## **Fish Kills and Bottom-Water Hypoxia in the Neuse River and Estuary: Reply to Burkholder et al.**

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## COMMENT

**Fish kills and bottom-water hypoxia in the Neuse River and Estuary:  
reply to Burkholder et al.****Hans W. Paerl<sup>1,\*</sup>, James L. Pinckney<sup>2</sup>, John M. Fear<sup>1</sup>, Benjamin L. Peierls<sup>1</sup>**<sup>1</sup>Institute of Marine Sciences, University of North Carolina at Chapel Hill, Morehead City, North Carolina 28557, USA<sup>2</sup>Department of Oceanography, Texas A&M University, College Station, Texas 77843-3146, USA

Burkholder et al. (1999) authored a comment in Marine Ecology Progress Series (MEPS) that selectively criticizes elements of our findings that appeared earlier in the same journal (Paerl et al. 1998). For the benefit of the readership of MEPS, it would have been useful to have had both their comment and our reply in the same volume. Unfortunately, we were not informed of their comment prior to its publication.

The lengthy comment by Burkholder et al. is focused on the relationship between hypoxia/anoxia and the occurrence of fish kills in the Neuse River Estuary. While the main criticisms by Burkholder et al. are addressed separately in this response, we must emphasize that our paper specifically examined the effects of organic matter loading on the oxygen dynamics of this estuary, and not the relationship between fish kills and hypoxia/anoxia as Burkholder et al. imply. The comment by Burkholder et al. revolves around one ancillary aspect of our paper, namely our conclusion that many of the 'reported fish kills appeared to reflect the magnitudes, areal coverage and duration of hypoxia and anoxia events' (Paerl et al. 1998).

Burkholder et al. present 5 specific criticisms and provide what they consider to be evidence to support their arguments. First, they contend that we 'lack depth profiles of dissolved oxygen data to support any of [our] conclusions about kills of surface schooling fish'. We do not lack these data; however we felt that the *vertical* profiles of dissolved oxygen (DO) concentrations for specific dates, times, and locations were not necessary to illustrate the overall spatiotemporal distribution of *bottom-water* hypoxia in the estuary, and therefore, these data were not included in the manuscript. Vertical profile data for DO and other physical parameters are collected by our laboratory and by colleagues, and have been pub-

lished biweekly since 1994 in a graphical format on our web site (<http://www.marine.unc.edu/neuse/modmon/>).

Second, Burkholder et al. assert that we have used 'unrecorded' or 'nonexistent' fish kill data and 'misconveying' data in the State of North Carolina's Department of Environment and Natural Resources (Division of Water Quality) data base. We believe this is a very serious allegation and one that is blatantly false. The data on fish kills in the Neuse River Estuary were obtained from official State records of documented events at the time our paper was submitted for publication (15 August 1997). In this regard, it is inappropriate and unfair for Burkholder et al. to present undocumented and unverified data that neither we nor State officials were aware of at the time of manuscript publication. To the best of our knowledge, the data that 'we failed to cite' have not been available to scientific colleagues and are not part of the official fish kill data archived by the State. A recent (June 1999) re-examination of the State's official fish kill data (North Carolina Department of Environment, Division of Water Quality; <http://esb.ehnr.state.nc.us/Fishkill/fishkill99.htm>) indicates that data on the numbers and locations of fish kills which were referred to in our study (Paerl et al. 1998) have not been altered or adjusted since the appearance of our paper. We accordingly stand by both our own and the State's data, which show spatial overlap between periods of bottom water hypoxia, anoxia and State-investigated fish kills.

Third, Burkholder et al. argue that our conclusion concerning the conditions conducive to fish kills is not supported because, in their opinion, we lack an understanding about the behavior of the resident fish populations. We strongly refute this point. Widespread mortality of resident fauna is one of many consequences of oxygen depletion in this system. Fish kills are inherently unpredictable because motile fauna usually vacate stressful conditions such as hypoxia and anoxia in stratified bottom waters before suffering mortality (Renaud 1986, Pihl et

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al. 1991, Breitburg 1992). On rare occasions, individuals or entire schools of fish may become 'trapped' when conditions change rapidly or escape routes are inaccessible.

The relationship between DO and the mortality of highly mobile finfish populations in the Neuse River Estuary is complex. In our paper we presented clear evidence of a spatiotemporal linkage between anoxic and hypoxic bottom water and fish kills in the Neuse during 1995 and 1996 (Fig. 1, Paerl et al. 1998). These data indicate that while the presence of anoxic or hypoxic bottom water did not guarantee a fish kill would occur, fish kills were always accompanied by low DO concentrations in the bottom water. This cooccurrence has further been confirmed in 1998 and 1999 (<http://www.marine.unc.edu/neuse/modmon/>; <http://esb.ehnr.state.nc.us/Fishkill/fishkill99.htm>). Burkholder et al. point out (1) that the fish kills in the mesohaline sections of the Neuse have been largely comprised of the surface-dwelling species Atlantic menhaden *Brevoortia tyrannus* and (2) that finfish will avoid areas where dissolved oxygen is less than  $2 \text{ mg l}^{-1}$  if more oxygen-replete habitat is available. They present weekly vertical DO profiles, each of which is the mean profile based on 6 separate locations in the mesohaline section of the estuary. Their data, which are consistent with the findings of our paper, showed that low oxygen water was typically confined to the bottom third of the water column during daylight hours. Based on this, they discount all possibility that the fish kills were oxygen-related for lack of a mechanism that would expose the surface-dwelling fish to the low oxygen bottom water.

Side-to-side sloshing (seiching) of the surface and bottom waters in the Neuse provides just such an exposure mechanism (Luettich et al. 1999a, b). In this scenario, wind blowing across the river pushes surface water toward the downwind shore where it downwells. A compensating flow of bottom water occurs in the opposite direction and causes upwelling of bottom water along the upwind shore. This lateral sloshing and concurrent upwelling and downwelling was observed on 2 cruises conducted during the summer of 1998 (Luettich et al. 1999a, b). During each cruise, lateral transects across the width of the mesohaline section of the Neuse were repeated every 6 to 12 h over the course of 36 h using a shipboard mounted acoustic doppler current profiler and a conductivity/temperature/depth/DO sensor. These data show that the cross-estuary response to variations in meteorological forcing is very rapid (time scales of a few hours) and that upwelled low-DO bottom water does reach the surface near the upwind shore (see Luettich et al. 1999a; <http://www.marine.unc.edu/neuse/modmon/publications/publications.html>).

There are several important implications of the sloshing/upwelling mechanism for fish kills. (1) The upwelled water is drawn from the near bottom regions of the estuary and only poses a threat to fish when the bottom water is oxygen depleted. Thus the presence of oxygen-

depleted bottom water as shown by Paerl et al. (1998) provides the potential for a fish kill. Clearly, a fish kill also requires the presence of fish and a sufficient physical forcing to cause upwelling to occur. (2) It seems plausible that pelagic fish may be able to react to and swim away from weak upwelling events that occur frequently within the Neuse. However, in a strong event, fish that happen to be in a near shore upwelling area will be rapidly engulfed by a large area of low DO water with no obvious escape route. (3) By the time a fish kill is reported and investigators arrive at the location of the dead fish (typically hours), the upwelling may have subsided and the water column returned to a state that does not reflect the conditions that preceded the fish kill. Therefore, DO levels measured in the presence of floating, dead fish may not be relevant to the cause of the fish kill. (4) Dead fish initially float to the surface and therefore may be transported horizontally in the direction of the surface current. This could cause fish that experienced upwelling near the upwind shore to be moved across the estuary and to accumulate near the downwind shore. A  $0.1 \text{ m s}^{-1}$  surface current travels a kilometer in approximately 3 h (Luettich et al. 1999a). In some cases, floating fish carcasses may be blown from one side of the estuary to the other before authorities document the kill. This also complicates the determination of the conditions that preceded fish mortality based on observations collected following a kill.

Burkholder et al. point to the dominance of menhaden in many of these fish kills as evidence of our misinterpretation of the link between low DO and fish kills. We agree with Burkholder et al. and others (Marotz et al. 1990, Hall et al. 1991) that, if an escape route exists, menhaden will leave areas when DO concentrations fall below some critical level. However, it is also entirely plausible that on occasion DO concentrations decrease very rapidly (as described above). Given their fairly low tolerance to hypoxic water (Hall et al. 1991), large schools of menhaden may expire before finding a suitable escape route. Our study is not the first to report highly mobile, pelagic species in fish kills caused by low DO (Renaud 1986, Burkholder et al. 1995). The numerical dominance of menhaden in fish kills may also simply reflect their relative abundance in highly productive areas that experience low DO. Large schools of menhaden aggregate in mesohaline, phytoplankton-rich (Friedland et al. 1989, 1996) areas, which our data show are areas most likely to experience low DO events. Consequently, we would predict that menhaden are the numerically dominant finfish species likely to be trapped in a rapid pulse of low DO water.

Fourth, Burkholder et al. declare that our paper omitted a large body of peer-reviewed, published information on fish kills in the Neuse River and misused literature citations. It appears from their supporting citations on this point that Burkholder et al. are referring to their own

research on the dinoflagellate *Pfiesteria piscicida*. However, because our paper was not focused on potential causes of fish kills in the Neuse River Estuary, we did not feel it necessary to cite anyone's work on *P. piscicida* and morphologically similar dinoflagellates. The absence of multiple citations for work on this organism by Burkholder and colleagues was not intended to exclude consideration of their work, but rather based on relevance to the central theme of the paper. We have cited the work of others who have addressed causes and effects of hypoxia on estuarine fauna appropriately and in context with the ideas presented in the preceding sentence, counter to the assertions of Burkholder et al.

Finally, Burkholder et al. criticize our original paper for the 'lack of any supporting statistical analyses to demonstrate relationships among field dissolved oxygen, nutrient, and fish kill data'. Nutrient enrichment and resultant eutrophication of estuarine ecosystems may promote the proliferation of opportunistic microbes and pathogens that compromise the immune systems of resident fauna, including fish and benthic invertebrates. Brief exposures to hypoxic/anoxic conditions, in combination with these additional stressors, may increase the susceptibility and lower the tolerance thresholds of fish species relative to healthy populations. Therefore, it is difficult to assign a single causal factor for widespread mortality in natural systems. Instead, these events are likely the result of a combination of interacting physical, chemical, biological, and behavioral factors that all contribute to the occurrence of fish kills. While Fig. 1 of Paerl et al. (1998) clearly shows spatiotemporal co-occurrence of bottom water hypoxia/anoxia and fish kills, statistical tests of *specific* causal hypotheses could lead to potentially false conclusions because behavioral responses of schooling fishes are unpredictable and influenced by *multiple* factors. Linking fish behavioral models with hydrodynamic and meteorological models to predict the frequency, magnitude, and location of fish kills was not the primary purpose of our paper. Furthermore, the absence of statistical validation of the linkage between fish kills and anoxia in our study has little bearing on the main theme of the paper, presentation and analysis of the data, or our conclusions. We reaffirm that internal and external loading of organic matter into the estuary results in bottom water oxygen depletion which produces conditions that are conducive to fish kills and mortality of benthic fauna.

In closing, we must reiterate that the central theme of our paper was that loading of organic matter from internal and external sources plays a major role in the oxygen dynamics of this system, a point Burkholder et al. do not argue and, in fact, similarly conclude in their comment. We are pleased that they share this important conclusion, for we believe it holds the key to understanding fish mortalities in this system. Although the topic was not the focus of our original paper, we stand by our interpreta-

tion of the available data and conclude that there is a reasonable and justifiable link between fish kills and the low DO events in the Neuse River Estuary.

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