Human Health and Environmental Impacts from Pfiesteria: A Science-Based Rebuttal to Griffith (1999)

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Article:

David Griffith began his article, “Exaggerating Environmental Health Risk: The Case of the Toxic Dinoflagellate Pfiesteria” (Human Organization 58:119-127). with a quotation by Angell (1995) which notes that assuming a connection between an effect and a cause, and then searching for it, is an inefficient approach that can lead to bias. Griffith clearly implied this was the approach Burkholder and her colleagues took to link Pfiesteria to human health problems. Griffith was in error. The approach Drs. Burkholder. Noga, and others took began with an observation of fish dying in aquaria. followed by identification of the cause as an unknown dinollagellate (Burkholder et al. 1992; Noga et al. 19931. It was then hypothesized that this organism could potentially cause fish kills in the environment. This was followed by its identification in field samples at fish kills (Burkholder et al. 1992; Noga et al. 1996) and searches of historical records that suggested it also was a potential cause of some (but not all) fish kills during the years when phytoplankton count records were maintained. The association with human illness came after laboratory workers became mildly to seriously ill, and their symptoms were similar to those reported by watermen (Glasgow et al. 1995). Because of the potential for human health problems in nature. they called for studies. Initial questionnaires used to begin to address the potential for health problems from Pfiesteria in North Carolina yielded little definitive information (see Oldach, Grattan, and Morris 1999). Later, clinical studies by physicians from the University of Maryland and Johns Hopkins on individuals with confirmed exposure to toxic Pfiesteria outbreaks demonstrated cognitive impairment lasting up to six months (Grattan et al. 1998). It is also important to note that the long-term impact of exposure to these toxins is still unknown.

On page 120 and throughout. Griffith downplayed the health problems associated with Pfiesteria (e.g. “mild cognitive impairment,” “recovery within hours or days”) and suggested that concerns about human health hazards were declining. The actual results show quite the opposite. In contrast to Griffith's statements, cognitive impairment suffered by individuals was pronounced. with 10 or 13 individuals with high exposure to affected waterways scoring below the 2nd percentile on the Rey auditory. verbal, learning, and memory test compared to matched national norms (Grattan et al. 1998). This level of impairment "reflects a profound and potentially disabling deficit- (Oldach. Grattan. and Morris 1999:147). Recovery time was weeks to months in most cases (Glasgow et al. 1995; Grattan I998), but Oldach, Grattan, and Morris (1999:147) reported that some individuals complain that they still have not recovered completely. In fact, in referring to affected laboratory workers. Morris (1999:1191) stated that "Although most of the symptoms appear to have resolved, concerns remain … about persistent effects (including persistent neurocognitive deficits) 6-7 years after the acute incident."

Griffith noted that "popular writers embellish with hyperbole and speculation"(p.119) but the tone of Griffith's article is an example of the same embellishment and hyper-hole (e.g., raising the specter of AIDS and Ebola: his characterization of problems as "mild" vs. physicians' characterization as "severe" and "profound"). He continually charges that the scientists working with Pfiesteria characterized it as a "serious threat" to public health (pp. 120, 121. 122). The use of these descriptors (unsubstantiated with quotes or references' helped

elevate the level of concern for Pfiesteria as a human health potential problem to a “serious threat.” This distinction was important in helping Griffith create the atmosphere of exaggeration, hut it has no basis in fact. In contrast to Griffith's anecdotal account. Burkholder and colleagues have been conservative in assessing Pfiesteria's link to human health effects. recognizing that laboratory-related exposure is unnatural, hut leaving open the possibility that humans might he affected in natural settings. In peer-reviewed published accounts, it is the effect from laboratory exposure that has been considered 'serious" (e.g.. Burkholder and Glasgow 1997), hut when extrapolating to estuarine exposure, Burkholder and coauthors have consistently characterized the problem as potential. For example, Burkholder, Glasgow, and Hobbs (1995158) stated that "Apart from an-ecdotal information provided by local fishermen, carefully designed epidemiological studies arc lacking to determine whether humans who frequent estuaries with toxic outbreaks might he adversely affected.- Also. Burkholder and Glasgow (1997:1073) stated that "Anecdotal information...points to the potential for this dinollagellate to adversely affect human health in natural habitat." Such statements hardly qualify for exaggeration or the characterization that Griffith portrayed.

Why, as Griffith stated (p. I 22), was his epidemiological survey "dismissed or taken lightly" by outside panels of scientists (e.g., Wright 1998)? We recognize the inherent difficulty in drawing definitive conclusions from survey research and acknowledge that Griffith presented related weaknesses from such studies. However, criticism of-his study was mainly derived from serious problems, related to flaws in his study design. Griffith's survey was supposed to be designed to assess watermen's health in Pfiesteria kill areas and control areas. The only source of accurate Pfiesteria fish-kill maps was Burkholder's laboratory because North Carolina's environmental agency, did not allow most Pfiesteria data to be included in the state's official fish-kill database until after the Chesapeake outbreaks (North Carolina Department of Environment, Health, and Natural Resources fish-kill database records, Raleigh, 1991 - 1998). Griffith wrote his proposal (Griffith and Borre 1995) and conducted his survey without asking Burkholder for such maps until after his study was nearly completed (see the accompanying comment by Burkholder and Glasgow) and after he began to widely inform the press that his study had shown that Pfiesteria was a nonissue (e.g., North Carolina Sea Grant 1995; Leavenworth 1997).

In addition to not having accurate Pfiesteria fish-kill location maps, the other major reason why Griffith's survey cannot be related to Pfiesteria is that contact with fish kills was not assessed in the study. Griffith (p. 123) stated that the research goal was to assess "the effects of contact with the waters of Eastern North Carolina under normal ecological conditions (specifically in the absence of fish kills).- However, toxic Pfiesteria is only active during certain fish-kill/ disease events (Burkholder and Glasgow 1997; Burkholder et al. 1999); thus. if information on effects of pfiesteria on watermen is a research goal, it is imperative to assess the health of watermen contacting in-progress fish kills (that is, while fish are dying) or periods when active Pfiesteria-like lesions were present on fish. The Grattan et al. (1998) study was properly designed to do this and resulted in their findings of mild to severe cognitive impairment up to a six-month period after exposure Griffith et al.'s (1998) results. used as evidence against the link of Pfiesteria with public health problems, suffered from these critical design flaws. Evaluation of that survey as too poor in quality to provide solid information about health impacts from environmental exposure to toxic Pfiesteria was based on these critical design flaws-it was not the result of a conspiracy by scientists "benefiting from Pfiesteria research dollars" (p. 121) or "with vested interests" (pp. 122. 123).

Griffith attempted to base his article on human health risks, while at the same time disparaging Burkholder and her research associates for having expressed concerns about Pfiesteria's impacts on estuarine ecosystems (p. 120. Griffith's misstatement that these scientists “began making and embellishing Joints that [Pfiesteria] posed a serious threat to public and environmental health,” emphasis added). However, the Pfiesteria issue cannot he divorced from the impacts on fish. All fin fish and shellfish species exposed to toxic Pfiesteria cultures to date (predominantly commercially important species) have been shown to be susceptible, and kills linked with Pfiesteria are dominated by commercially important species (Burkholder and Glasgow; 1997). There is also strong scientific evidence that toxic Pfiesteria causes serious chronic/sublethal impacts on fish health (Nogg et al. 1996; Burkholder 1998).

These observations raised two critical concerns very early in the emerging picture of Pfiesteria: Can toxins accumulate in seafood and be transferred to humans? And, what are the impacts on coastal communities, especially impacts of Pfiesteria on the seafood and recreation industries? The first question is directly related to human health. Biomagnification of toxins through the food chain is a common mode of dinoflagellate toxin transfer (see Anderson and Garrison 1997), so the question is relevant to address in the case of Pfiesteria (Wright 1998). Fortunately, there is, as yet, no evidence that Pfiesteria toxins accumulate in affected finfish and shellfish, but note that this is a rare exception to general scientific understanding of dinoflagellate toxins.

The second question is relevant on several counts. First, fish kills can reduce the number of fish available for commercial harvest, although in estuarine kills the number of fish affected is believed to be small relative to the total number in the population. More importantly, chronic/sublethal impacts that impair fish reproduction, recruitment, and disease resistance–especially in estuarine fish nursery grounds as important as those where toxic Pfiesteria has been most active–would be expected to promote declines in fish populations over the long term (see Burkholder 1998). Either of these outcomes can lead to significant economic problems for the seafood industry. as can the perception of a problem. (We note also that this threat to the seafood industry–realized in a significant decline in sales in the Chesapeake Bay area during the summer of 1997 [Epstein 1998]–is a potentially strong incentive for participants in Griffith's study to have misreported health impacts). Furthermore, the estuarine tourism industry depends economically on both recreational fishing and water sports. If the quality of those activities arc either actually or even perceived to be risky, the economic ramifications may be significant. Thus. aside from the direct effect on human health problems. the indirect effect on fisheries remains an important motivation for understanding Pfiesteria.

The many misinterpretations and lack of background research demonstrated by Griffith's writing lead us to seriously doubt that reviewers of the paper had any knowledge of Pfiesteria research. Nonetheless, these falsely based attacks on the scientific caliber of studies by Burkholder and her associates were published. For example. it is commonly recognized throughout the scientific community in this field that Pfiesteria and Pfiesteria-like dinoflagellates do not include Gymnodiniam breve (if that is what Griffith meant–see note 1, p. 125). Note that Griffith's “Gynovidinium breve” does not exist; see Burkholder et al. I 992: Burkholder and Glasgow [995: Steidinger et al. 1996; versus Tomas I997. Also, which "better-known red tide" did Griffith mean on p. 120? There are many different red tides well known throughout the world. including along the eastern U.S. coastline (Anderson and Garrison 1997). Griffith described Pfiesteria, ciguatera, and "red-tide" dinoflagellates as 'irritants" with similar health effects. Exposure to toxic Pfiesteria has been linked to serious human illness (Glasgow et al. 1995: Grattan et al. 1998). Toxins from some "red tide" dinoflagellates have both caused serious illness and even killed people in many parts of the world (Anderson and Garrison 1997). Toxic dinoflagellate blooms are generally unpredictable. and because they can be so potent, resource managers and health specialists generally agree it is wise to err on the side of caution. For that reason, proactive monitoring programs have been developed for some of the better understood toxic dinoflagellates in the U.S. and elsewhere (Shumway 1990; Burkholder 1998).

Moreover, Griffith (p. 124) wrote (without citing any of the many available papers on the subject) that scientists working with Pfiesteria claimed that certain human behaviors, specifically nutrient loading, have been implicated as the primary cause of Pfiesteria's transformation from a dormant, plant-like state to an active killer of fish and other organisms. Actually. it is clearly stated in several peer-reviewed international science publications that substances secreted by schools of fish, principally Atlantic menhaden, cause the transformation of Pfiesteria from a nontoxic to a toxic stage (e.g., Burkholder et al. 1992; Burkholder, Glasgow, and Hobbs 1995; Burkholder and Glasgow 1997). It is also clearly stated that nontoxic stages of Pfiesteria have been shown to respond positively to increases in nutrient loading (Glasgow et al. 1995: Burkholder and Glasgow 1997).

As another example, Griffith took issue with terms such as "ambush predator" and "phantom," claiming that such terms were more appropriate to military history than biology. However, such descriptors are standard in aquatic biology, as any cursory survey of the literature would reveal. For instance, the term "ambush predator"

is used to classify a vast functional group of organisms in relation to their feeding behavior (Greene 1985). A good example is the chaetognath Sagglia, the "arrow worm." one of the most important predators in marine zooplankton communities (Fulton 1984). Its counterpart in freshwater systems is Chaoborus, the "phantom midge," one of the most well-studied aquatic invertebrates in the world (Riessen, O'Brien, and Loveless 1985).

In summary', there may be differences of opinion in scientific issues, hut these must be argued in an objective, factually based manner. We call for a dependency on hypothesis-driven. peer-reviewed science in international journals as the primary basis for understanding such issues; tor professional conduct so that respected scientists are not falsely disparaged: and for the peer-review process to include appropriate specialists to ensure that the quality of scientific information can be fairly evaluated, rather than cursorily condemned on false grounds. Griffith's accusations—that scientists involved with Pfiesteria research exaggerated the link to human health. and that there is no evidence for serious health impacts from this toxic dinoflagellate—have no basis in fact. This rebuttal refutes the critical points in his allegations on the basis of peer-reviewed international publications on the biological and medical science of Pfiesteria.

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