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Commentary

Science Ethics and its Role in Early Suppression of the Pfiesteria *Issue*

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A recent paper by David Griffith (1999) presented the *Pfiesteria* issue as an example of exaggerated environmental risk. The anthropologist falsely alleged that there is no evidence for serious human health impacts from the issue. He seriously erred on the science of *Pfiesteria* and miscast the issue as a politically safe issue for affluent white people (p.125). In falsely based personal attacks, the anthropologist depicted Burkholder and her colleagues as having acted mainly for vested interests in monetary gain (pp. 120, 121, 123, 125). Griffith described the organism from the wrong habitat (p. 119), used nonexistent dinoflagellate "groups" such as "*Gynovidinium breve*" (p. 125), and fabricated Burkholder's having had to abandon use of *Pfiesteria* from the Pocomoke (p.125). Here we present the facts that will stand in a court of law — about *Pfiesteria* science, suppression of the *Pfiesteria* issue by certain state officials, participation by natural and social scientists in such efforts, and serious human health impacts that have been linked to this toxic dinoflagellate by medical specialists.

The First False Allegation—No Serious Health Threat from *Pfiesteria*

The most serious misinformation presented in Griffith (1999) is reflected in his first statement (p. 119), "mounting evidence [indicates] that *Pfiesteria picicida* [misspelled], a marine organism that releases a neurotoxin, poses no serious threat to human health.... [Exposure to toxic *Pfiesteria*] is believed to have caused temporary memory loss in laboratory technicians and mild cognitive impairment in individuals exposed to fish kills (p. 120).... The few individuals who have suffered from contact with the organism have recovered fully, usually within a few hours to days (p. 120).... The handful of people who claimed to have suffered from exposure to *Pfiesteria* suffered no long-term health problems that could be attributed to exposure.... One lab technician's problems ranged from mild irritability and disorientation to bursts of rage and memory loss (p. 124).... Anecdotal evidence supporting the health threat hypothesis was accumulating outside laboratory settings (p. 124).... These cases...remained anecdotal and had neither clinical nor epidemiological support" (p. 124).

The facts are as follows. Medical researchers report *increasing* evidence that toxic *Pfiesteria* can seriously affect human health. The impacts are being studied in affected people in Maryland (G. Morris et al., University of Maryland) and North Carolina (D. Schmechel et al., Duke Medical Center). Impacts also are being examined in research with small mammals as surrogates for humans (Levin et al. 1999). Severe cognitive exposure lasting three months or more was sustained by several laboratory workers (Glasgow et al. 1995). Grattan et al. (1998) described severe cognitive dysfunction that lasted for several months, as well, in some people who were first clinically evaluated shortly after environmental exposure to small toxic *Pfiesteria* outbreaks (Burkholder and Glasgow 1997; DNR 1998). Some individuals tested in the *bottom* 2 percent of the U.S. population in their ability to learn or remember, once normalized for age and education. In two cases normal cognitive functioning was not regained until approximately six months after exposure.

Thus, the people Griffith describes as having "claimed" to have suffered "mild" impacts (pp. 120, 124, 125) were clinically evaluated and some of them suffered *profound* cognitive dysfunction that lasted for months, rather than "a few hours or days." There is also evidence that certain serious impacts on the nervous system and the immune system from exposure to toxic *Pfiesteria* last years. Note that we use "serious" to indicate impacts such as chronic infections and neurological dysfunction that have caused lifestyle changes (equated to Music's [1997a] term "moderate"; Music reserved "serious" only for impacts that cause death, which is not how nonmedical people use the term). Griffith repeatedly mis-

stated (pp. 120, 124, 125) that our concerns about *Pfiesteria* as a potentially serious hazard to public and environmental health were based on "anecdotal evidence from a handful of self-selected, self-diagnosed individuals who claimed to have been afflicted by the dinoflagellate" (p. 124). The reality is that the people upon whom our health concerns were based were clinically evaluated by medical specialists (at Duke Medical Center and elsewhere). Our concerns about *Pfiesteria*'s impacts on estuarine ecosystems were based on peer-reviewed, published data (Burkholder et al. 1992, 1997; Burkholder, Glasgow, and Hobbs 1995; Burkholder and Glasgow 1997).

Three remaining points were given by Griffith in support of this first false allegation. The first (p. 120) was that "*Pfiesteria* has been implicated in sickness of fewer than 100 people." North Carolina health officials were informed of the laboratory workers' cases in early 1994 and conducted the DEHNR's (North Carolina Department of Environment, Health and Natural Resources) first epidemiology surveys in late 1995 following public outcry over lack of response by these officials to major toxic *Pfiesteria* outbreaks (Neuse River Foundation 1995). In conducting early surveys, the health officials did not prevent people from going into areas affected by toxic *Pfiesteria* outbreaks, but asked how they felt afterward (Morris 1996). People with respiratory distress and sore throats were discounted if they had had a cold within the previous eight weeks. More than 40 people described these and/or other symptoms such as disorientation, severe headaches, memory problems, nausea, burning eyes, burning skin, etc. during one of many documented toxic outbreaks (Burkholder, Glasgow, and Hobbs 1995; Burkholder and Glasgow 1997). Some people avoided the state health officials because they distrusted their motivation (e.g., Brodeur 1995; Barker 1997). Thus, there is no way to know the number of people who have been affected.

Laboratory exposure evidence indicated there was recovery from the symptom that offered the clearest available diagnostic—severe cognitive impairment—within 1-12 weeks after exposure (Glasgow et al. 1995). However, the state health officials waited 6-9 months before clinically evaluating three people from the 1995 Neuse toxic *Pfiesteria* outbreak. The lack of conclusive information was widely publicized by the health officials as evidence against serious health problems from *Pfiesteria*. A representative from the Centers for Disease Control and Prevention (CDC) reexamined the NC DEHNR survey data and found indication of neurocognitive impairment and visual, respiratory, and gastrointestinal problems in people who had been exposed to a verified toxic *Pfiesteria* outbreak (McGeehen 1997). The citizenry were not informed of the CDC communication.

The second point offered by Griffith (p. 121) was that "researchers began reporting that long-term health problems from Pfiesteria were doubtful" and cited Smith and Music (1998) and Swinker (1998). The study clinically evaluated people from North Carolina who, at some time during the previous five years, recalled being in "fish kill waters"—that is, in areas where fish kills had at some point occurred. Many fish kills are caused by factors other than *Pfiesteria* (North Carolina Department of Environment and Natural Resources fish kill database, Raleigh; Burkholder, Mallin, and Glasgow 1999). *There was no way to discern whether these subjects had actually been in a fish kill that involved toxic Pfiesteria*, while fish were diseased/dying, which is when most toxin exposure would be expected to occur (Burkholder and Glasgow 1997; Grattan et al. 1998). As mentioned, the clearest diagnostic to indicate environmental exposure to toxic *Pfiesteria* is severe cognitive impairment (Grattan et al. 1998). That symptom disappeared within three months in most known cases as the subjects regained normal cognitive function. Subjects in Savitz (1998) were evaluated at least four months after the most recently documented toxic *Pfiesteria* outbreak (Burkholder and Glasgow 1997). Thus, no one in the Savitz (1998) study was known to have been exposed for certain to a toxic *Pfiesteria* outbreak and, even if they had been, the study was conducted too late after the most recent outbreak to enable detection of the reliable field diagnostic. Other medical specialists have stated that there is no way to relate this study with certainty to *Pfiesteria* exposure (e.g., W. Roper, School of Public Health, University of North Carolina-Chapel Hill, pers. comm.).

The third point offered by Griffith (p. 124) was that there were no increased reports of sickness in wildlife and domestic animals during toxic *Pfiesteria* outbreaks (from Smith and Music 1998). Thus far, human exposure has occurred via contact with water or breathing the overlying air in the immediate areas where fish are diseased or dying and toxic *Pfiesteria* is present (Burkholder and Glasgow 1997; Grattan et al. 1998). Dogs, cats, and cows do not swim in or move over toxic outbreak areas and would not be expected to have been hurt. Humans who were hurt from laboratory or environmental exposure to toxic *Pfiesteria appeared* normal (Glasgow et al. 1995; Grattan et al. 1998). Thus, wildlife *appearance* would likely provide little information about impacts from *Pfiesteria*.

The Second False Allegation—No Acts to Suppress the Issue

> *There are two kinds of science. One is purchased and manipulated, and dispersed at industries'* [or agencies'] *whims. The other cannot be bought.*
>
> David Brower
> (in Wilkinson, 1998)

Barker's Book

An investigative writer wrote a nonfiction book (Barker 1997) that described misconduct of state health officials in their repeated attempts to falsely discredit the *Pfiesteria* research. Griffith repeatedly cast the book as fabricated extremism (pp. 123, 124). Perhaps he is unfamiliar with the old adage that "a book should not be judged by its cover." Book

jacket, advertising hype, and dramatized writing style aside, the contents of the book provide an accurate account of state officials' response to the *Pfiesteria* issue. Barker stated that this information was corroborated by many witnesses in taped interviews and written documentation. Barker accurately related the story of a state in denial: of certain health officials who strategized about how to discredit Burkholder when she provided evidence (e.g., science publications) of environmental and human health impacts from *Pfiesteria*; of health officials' harassment of doctors concerned about such issues; of health officials' plans to redirect funding that had been designated by the governor for use in *Pfiesteria* research; and of scientists with questionable ethics who, for reasons of self-gain, provided assistance to certain state officials in their attempts to discredit the *Pfiesteria* research and negate the issue. The book's release prompted blanket denial from the health officials and their affiliates, and more intensive attacks of Burkholder's personal/professional character (e.g. Burkholder 1997a; Lancaster 1997; Barker 1998).

Harassment of Concerned Medical Specialists

Toxic *Pfiesteria* outbreaks had been documented in North Carolina's estuaries nearly every year since 1991, with well over 1 billion fish affected (Burkholder et al. 1992; Burkholder and Glasgow 1997). Well before the Chesapeake's toxic *Pfiesteria* outbreaks involving approximately 30,000 fish in 1997 (DNR 1998), North Carolina medical specialists had expressed concern for patients who suffered illness after contact with Neuse Estuary water. Their concerns, including but not restricted to the *Pfiesteria* issue, had been shared by about 135 other coastal physicians whose patients had experienced similar illness. The physicians were harassed by coworkers of state health agency official Dr. G. Smith (of Smith and Music 1998), apparently in an effort to silence them. (Note that Smith is acknowledged in Griffith [p.119] by his adjunct position, but he was/is an employee of the state health agency.) A coastal physician formally demanded a prominent state health official to stop the ongoing harassment (Delaney 1997). Smith and Music were later removed by the state's present health director from further role in the *Pfiesteria* issue apparently because of inappropriate conduct (Associated Press 1998).

The physicians eventually petitioned Vice President Gore to assist in improving Neuse water quality (Morgan 1997). They were publicly described by certain state health officials as having taken irresponsible action that could negatively impact tourism and other aspects of the coastal economy. When a reporter asked why the state health officials were so certain that *Pfiesteria* could not cause serious human health impacts, the official's videotaped response was, "Look, nobody's falling out of the boats dead yet, ok?" (Music 1997b). The officials' behavior did not inspire confidence among the citizenry that the environmental/health agency meant to consider their health proactively in the *Pfiesteria* issue (e.g., Brodeur 1995). During 1997 the state health officials finally presented informational sessions for the coastal physicians, ostensibly about *Pfiesteria*. Physicians complained that, rather than describing symptoms of *Pfiesteria*-related illness, the health officials appeared intent to discredit *Pfiesteria* as a nonissue (e.g., Music 1997c,1997d). Griffith quoted health official Music (n.d., actually a 1997c e-mailed letter to a reporter), but failed to mention that the writing falsely cast Burkholder as having stated that people were dying from *Pfiesteria*, which—had Burkholder not acted in immediate rebut—could have promoted the panic that Griffith falsely portrayed her research team of having designed (pp. 120, 123).

State *Pfiesteria* Research Funding (1994-1996)

Griffith (p. 121) stated that the "common scientific review process was portrayed [by Burkholder] as a way the state could stall investigation into the potential threat [of *Pfiesteria*]." Was it a "common" scientific review process, and were the concerns about delays legitimate? We address the latter question first.

In May 1994, the governor authorized the state health director to direct approximately $600,000 to the *Pfiesteria* issue (DEHNR file on *Pfiesteria* funding, Raleigh, N.C., 1994). The governor's action was prompted by a coastal senator in response to concerns of her constituency. She had requested that funding be directed to Burkholder's research team, known as foremost experts on *Pfiesteria* (Jones 1996a). The state health director agreed. But as soon as he had secured the funds he informed Burkholder that there were serious reservations about her competence and that her research team would not receive funding (Burkholder 1994a). After eight months, a reporter's inquiry (Franklin 1995) prompted the health official to transfer the funds to a local granting agency for, ostensibly, a competitive grant process. After nearly two years, funding became available for designated laboratories (N.C. Sea Grant records on the 1995-1996 *Pfiesteria* grant process and director's memorandum, September 5, 1996).

The vice chancellor for research at North Carolina State University (NCSU) later verified that concerns raised by Burkholder (1994b) about the grant process had been well founded (Moreland 1996). One of four grants funded in the competitive review process was not submitted by the submission deadline, nor formally reviewed by the panel (N.C. Sea Grant records, Raleigh, N.C., October 13 1995, September 5, 1996; Jones 1996a, 1996b). Funded researchers were told by the granting agency director, without Burkholder's knowledge or consent, that her laboratory would provide expensive materials that they needed (Burkholder 1996a). A proposal by Griffith and Borré (1995) was funded despite having been evaluated in external review as poor in quality (N.C. Sea Grant records, Raleigh, N.C.). Among various serious errors, the proposal stated that crucial information maps of toxic *Pfiesteria* outbreaks would be provided by an inland biologist who had no maps (P. Rublee, UNC-Greensboro, pers. comm. 1996, 1999). A member of Griffith's

team informed Burkholder that state health officials had instructed them to avoid our laboratory as a credible source — providing an alternate explanation (other than lack of knowledge about publications) as to why Griffith failed to mention our laboratory as the only source with that information.

In respected granting agencies such as the National Science Foundation, panelists involved in funding decisions on grants must abstain from decisions about grants submitted by colleagues from the same university, certainly from the same department; and they are supposed to abstain from other known personal or professional conflicts of interest. This grant process, by contrast, involved clear conflicts of interest among panelists (N.C. Sea Grant records, Raleigh, September 5, 1996). A state health official from the source agency for the funding heavily influenced at least one member to direct funding away from Burkholder's laboratory (Barker 1997); another panelist was a scientist from the same center in the same university as Griffith; another was a state water quality official whose immediate supervisor repeatedly had been hostile to Burkholder in public and other forums (Barker 1997). A fourth panelist was a member of the North Carolina State University higher administration who had little familiarity with *Pfiesteria* science or with Burkholder and colleagues.

A fifth panelist was a scientist from the same department in the same university as a scientist who was funded by the grant process. The funded scientist had publicly negated any role of *Pfiesteria* in fish kills (e.g., Burkholder et al. 1999). He had not previously worked with *Pfiesteria*, yet was judged by his panelist colleague as more competent than Burkholder (who, by that time, had worked with *Pfiesteria* for five years) to conduct the research (Jones 1996c). After receiving the funding, in a taped interview the lead scientist disparaged the *Pfiesteria* issue and stated his intent to disprove linkages between *Pfiesteria* and nutrient pollution, which would have been favorable to agriculture and other interests (Barker 1997). But the scientists were unable to detect or identify *P. piscicida* and eventually reported "pfiesteria-like cells" (Paerl and Pinckney 1998; see below).

The North Carolina Sea Grant director was asked to step down from his at-large position shortly after the irregularities in the grant review process came to public attention (Jones 1996d). He previously had been ordered by the NCSU administration to apologize to Burkholder in writing for having publicly maligned her (N.C. Sea Grant records, Raleigh, NC, August 28, 1996). An audit was conducted on the grant process, but the narrow directive allowed only assessment of whether paperwork was on file describing the request for proposals and whether a review process had, in some form, been conducted. The state auditor expressed to Burkholder his regret that the audit had not been more widely framed and his belief in irregularities in the process that had extended beyond the "paper trail." Books have been written about the fragility of the review process in science, which can be easily tainted, "wired" for a predetermined end, and otherwise misused (Guston and Keniston 1994; Marshall 1995; Moran 1998). There was evidence for such misuse in this case.

Motivations

Griffith indicated that "vested interests," namely, millions of dollars in funding (pp. 120, 121), motivated the actions of the early *Pfiesteria* researchers (citing Burkholder as chief among them; p. 121) in their attempts to advance public understanding of the *Pfiesteria* issue. The facts show otherwise. Burkholder's actions were unpopular with the environmental/ health agency, and her laboratory was overlooked for major funding prospects by early invitation or contractural agreement. She was informed by agricultural factions that, because she had conducted research which demonstrated significant aquatic impacts from the swine industry (Burkholder et al. 1997), including stimulation of toxic *Pfiesteria*, her name would be a liability on grants submitted to agricultural agencies/interests, or to the environmental/ health agency, for research to further examine such impacts. Her actions were conducive neither to favor nor funding from important local sources of grant support.

Our research team received outstanding evaluations from all external reviewers in the above grant process, but funding in the grant award was substantially cut (N.C. Sea Grant records, Raleigh, November 1995, summarized in the director's memorandum of September 5, 1996). Most funding directed to our laboratory was for production of *Pfiesteria* culture and toxin material for use in other colleagues' research. When the grant process irregularities were exposed, Burkholder rejected the funding as tainted (Betts 1996), but committed to provide the material (through other support) needed by her colleagues who had accepted N.C. Sea Grant funding in their segments of her multi-investigator grant (Burkholder 1996b). She later accepted the funding after an unforeseen mechanism made it possible for another official to administer the grant (Moreland 1996). Most support for *Pfiesteria* research reached our laboratory after Maryland experienced small toxic *Pfiesteria* outbreaks, completed the first clinical evaluations of people shortly after they were known to have been exposed to toxic *Pfiesteria* outbreaks, and reported compelling evidence in support of *Pfiesteria* as a human health issue. These events could not remotely have been expected. We also note a detailed inquiry into Burkholder's character by the American Association for the Advancement of Science (AAAS). As a result, and contrary to Griffith's false allegations, Burkholder received the AAAS Scientific Freedom and Responsibility Award (1998) *for* her efforts in the *Pfiesteria* issue.

The Congressional Hearing

Griffith (p. 120) misstated that a September 1997 congressional hearing was conducted over "concern about the organism." Burkholder (1997a), but not Griffith, attended because she was invited to testify. The hearing, conducted

by the Committee on Government Reform and Oversight, focused in large measure on governments' previous failures to address the *Pfiesteria* issue. This was the same committee (chaired by C. Shays, R-Conn.) that confronted issues such as the Internal Revenue Service's conduct in mistreating citizens and military administrators' having attempted to deny that Gulf War veterans returned from battle with serious neurological illness. Mr. Shays informed some who testified that he wished to know why certain sectors of North Carolina government had ignored this legitimate human health issue for years, while using various unethical techniques in attempts to discredit our laboratory. Thus a congressional hearing addressed suppression of the *Pfiesteria* issue by certain North Carolina state officials. Earlier, the secretary of North Carolina DEHNR had formally apologized for inappropriate remarks and acts that had been directed against Burkholder by certain agency personnel (Howes 1996; Lancaster 1997). As a matter of public record, the agency did not deny such conduct and, in fact, formally acknowledged and apologized for it. Yet Griffith (pp. 120, 121) claimed that such conduct and actions by certain state health officials to suppress the *Pfiesteria* issue had not occurred.

The Third False Allegation—High Caliber of an Occupational Survey

Griffith (pp. 122, 123) stated that his occupational survey of crab fishermen was not accepted by scientists who had "vested [lucrative] interests" in the *Pfiesteria* issue. We present an alternate view, namely, that Griffith' work remains poorly received because it cannot be related to *Pfiesteria.*

The project team (Griffith et al. 1998) was described (p. 121) as having "paid particular attention to those areas where toxic dinoflagellates were associated with fish kills. These . . . data allowed closer analysis of the incidence of illness among crabbers in areas known to have hosted fish kills which are highly correlated with *Pfiesteria*-like dinoflagellates." However, letters from Griffith (1997) indicate that he and his coworkers did not have such information during the design (late 1995—early 1996 or earlier) or the duration (grant study period, 1.5 years) of their survey. His first letter (February) stated that he would consider our data and maps if we wished to send them. His second letter (July) requested all of our maps. But from April 1997 on, Griffith had informed the press that he did not need maps from our laboratory and that North Carolina had no human health impacts of consequence from *Pfiesteria* (Leavenworth 1997; N.C. Sea Grant 1997).

Griffith (1997) indicated that DEHNR had supplied the fish kill records for his study. From1991 through 1997, however, the agency had refused to include nearly all available data on *Pfiesteria*-related fish kills. Thus, his source would have contained very little about fish kills related to *Pfiesteria.* He had failed to indicate knowledge of an early (and, by 1996, incomplete) map of known toxic outbreaks (Burkholder, Glasgow, and Hobbs 1995). We provided (only) that public information for Epstein et al. at that time (1997; workshop attended by Burkholder but not by Griffith). In reply to Griffith's second letter, Burkholder (1997b) summarized his remarks about *Pfiesteria* as a nonissue. She explained that she and her students/staff had spent many years of research to obtain the maps and requested a science-based explanation as to why he wanted them so late in his project. He failed to respond.

Griffith stated (p. 121) that his study was not taken seriously until 1998 (e.g., Smith and Music 1998). In reality, his survey remains poorly regarded by medical specialists (e.g., L. Fleming in Wright 1998; L. Grattan and D. Oldach, pers. comm., University of Maryland). It is as Griffith described it in March 1996 when he admitted that he "did not know how, or whether, [his] survey of crab fishermen could be related to *Pfiesteria*" (N.C. Sea Grant 1996). It cannot be, based on the science of *Pfiesteria. Pfiesteria* is usually benign but is triggered to produce toxin when it detects sufficient fresh fish excreta/secreta (Burkholder and Glasgow 1997). Diseased or dying fish are used as "sentinels" for the possible presence of actively toxic populations, followed by tests of water samples with fish to confirm whether toxic *Pfiesteria* is present. Yet, Griffith (p. 123) focused on fishermen "specifically in the absence of fish kills"–when toxic *Pfiesteria* would have been minor or absent (Burkholder and Glasgow 1997).

Human health risks from *Pfiesteria* occur during toxic *Pfiesteria*-related fish-kill/disease events (Grattan et al. 1998). *Pfiesteria* has been linked to such impacts as nausea, severe headaches, burning eyes and skin, respiratory problems, and impaired memory (Glasgow et al. 1995; Grattan et al. 1998). Although there are subtle and sometimes serious lingering effects, the best diagnostic at this time is most clearly detected during or shortly after exposure to those specific events, which Griffith (p. 123) did not track. Many of the other symptoms, as indicated by the laboratory toxic *Pfiesteria* exposures, are nonspecific—nausea, easy infections, headaches, and flu-like symptoms have been indicated during/following known *Pfiesteria* environmental exposure (e.g., McGeehen 1997). Griffith's study design, with focus away from fish kills, would have ensured that it was impossible to relate such impacts to *Pfiesteria.*

Griffith's survey, from its inception, was embraced by certain state environmental/health officials who had repeatedly attempted to negate the validity of *Pfiesteria* as a human health issue (e.g., Smith and Music 1998; Associated Press 1998). However, many medical specialists have evaluated it as too poorly designed to contribute useful information (e.g., Wright 1998:12): "To date, the epidemiological data [on *Pfiesteria* in N.C.] consist of two clinical series and one-cross-sectional study [Griffith et al. 1998].... In all of these reports there is no consistent case definition, no consistent objective measure of human health effects, and no consistent objective measure of exposure.... There is a need for general epidemiological surveillance to detect any illness due to *Pfiesteria*-related toxins." Thus, after considering Griffith's survey, the panel identified a need for quality epidemiology studies to evaluate *Pfiesteria* health impacts in North Carolina.

The Fourth False Allegation—Our Scientific Incompetence and Unprofessionalism

Identification of *Pfiesteria*

Griffith fabricated (p. 125) that *Pfiesteria* was misidentified in the Maryland outbreak (actually three outbreaks; see below) and that the organism was identified as something entirely different by a scientist in Florida who had "more precision equipment." The instrument required is a scanning electron microscope (used throughout the past near-decade of our *Pfiesteria* research; e.g., see Burkholder et al. 1992; Burkholder and Glasgow 1995, 1997; Steidinger et al. 1996 also contains several of our scanning electron micrographs). The further reality is that we are regarded by the Florida scientist, Dr. K. Steidinger (Florida Dept. of Environmental Protection–Florida Marine Research Institute, St. Petersburg), as highly competent in *Pfiesteria* identification. We provide formal training in such analyses to other specialists, as well.

Appropriately conducted fish bioassays are required to test for the presence of toxic *Pfiesteria* (Burkholder, Glasgow, and Hobbs 1995; Burkholder, Mallin, and Glasgow 1999; EPA 1999). These tests are done by adding live fish to a natural sample within an expensive, specialized biohazard III containment system to avoid the potential for human exposure to aerosolized *Pfiesteria* toxins. If dinoflagellates that resemble *Pfiesteria* in light microscopy (rapid technique, but inadequate for species confirmations) are common during fish death (EPA 1999), then we continue to add live fish until the cell densities are sufficiently high to enable species identification (of suture-swollen cells) with scanning electron microscopy. Florida colleagues, lacking biohazard III facilities, attempted to test for the presence of *Pfiesteria* by adding algal prey for them to eat, instead of fish. Algal prey are not known to stimulate toxin production by *Pfiesteria*, but—unknown to the Florida scientists—the test also often fails to detect even benign or nontoxic populations of *Pfiesteria* (EPA 1999; cross-confirmed in multiple tests by our laboratory and by H. Marshall, Old Dominion University). Algal assays thus should *not* be used to test for *Pfiesteria* in water samples taken during fish kill/disease events (EPA 1999).

The Florida scientists thus correctly reported that they had not detected *Pfiesteria* in algal assays from the same Pocomoke samples in which we had detected *Pfiesteria* using fish prey, but they had used an inappropriate assay. We sent photographs documenting our identifications on Pocomoke *Pfiesteria* to Dr. Steidinger, who cross-confirmed our species identification of *P. piscicida* as the toxic organism that was present in the Pocomoke fish kills (based on fish bioassays, our scanning electron micrographs, and supporting information). The identification was further corroborated by the independent laboratories of P. Rublee, UNC-Greensboro, and D. Oldach, University of Maryland, using molecular techniques. We similarly confirmed *P. piscicida* and/or the second known toxic *Pfiesteria* species in later samples from the Pocomoke outbreak as well as samples from the other two toxic outbreaks in Maryland. In a sample from one outbreak, the Florida colleagues did detect *P. piscicida* with algal assays; however, that population was later tested by two independent laboratories and cross-confirmed as a benign strain (incapable of killing fish; see "never-toxic" in EPA 1999). When we added fish to the same sample, our technique selected for a toxic *P. piscicida* strain that increased in abundance, in association with fish death.

Smith and Music (1998), state health officials who repeatedly attempted to discredit the *Pfiesteria* issue (see Barker 1997; Associated Press 1998), were supported in their writing by a signed reviewer who took natural river-water sample from our laboratory during early 1996 without our knowledge or consent (records for toxic culture transfer to individuals outside our laboratory; Department of Environmental Health Safety, NCSU). The sample culture was about to be discarded, as the federal scientist was informed, because the dinoflagellates contained in it were weakly toxic and unsuitable for use in developing research-quality clones. The scientist, who had/has not published peer-reviewed research on *Pfiesteria,* conducted algal assays on the mixed-species sample and, not surprisingly, missed the presence of *P. piscicida.* Florida specialists were used to identify the benign dinoflagellates that were detected. The scientist then announced to the press that our laboratory "did not have" *Pfiesteria piscicida.* The report falsely cast our laboratory as "highly suspect" in our past and present research. The timing of the announcement (after the Chesapeake outbreaks, and approximately 1.5 years after the sample was taken) was used in an unsuccessful attempt to obtain a biohazard III facility for toxic *Pfiesteria* research. To various witnesses the scientist tried to justify the request based, in part, on our alleged incompetence that the scientist falsely claimed to have demonstrated.

This scientist then provided what was claimed to be *Pfiesteria piscicida* to a molecular biologist of good reputation to determine its 18S ribosomal DNA sequence. The species was later shown to have been misidentified. The sequence had to be withdrawn from the GenBank (AF-080098) and relisted as "pfiesteria-like species." Our research team's genetic sequence for *P. piscicida* remains the first correct GenBank entry (AF-077055; Rublee et al. with our laboratory; species identification cross-confirmed by three independent laboratories). Ironically, this individual and certain other supporters/press—Griffith's apparent sources—who had miscast our laboratory as not "having" *P. piscicida,* and "having failed to corroborate [our] species analyses," had described not our scientific method but, rather, their own.

Griffith could not have obtained another stated fabrication (p.125, Note 1) from any scientist with cursory knowledge of this field, or from any basic reference because 1) there is no such toxic dinoflagellate "group" as "*Gynovidinium breve*"; and 2) the organism *Gymnodinium breve,* which he may have meant, is a marine toxic dinoflagellate that looks/acts nothing like *Pfiesteria* spp. (see Steidinger et al. 1996; Tomas 1997). No dinoflagellate specialists would be "led to lump" *Pfiesteria* into a "group" (actually, one species) known

as *Gymnodinium breve*, or into the imaginary "group" (nonexistent species) that Griffith mentioned.

In referring to our use of "*Pfiesteria*-like" under certain conditions, Griffith failed to recognize the careful science reflected by our actions. Until we complete scanning electron microscopy of suture-swollen cells (a tedious process), we do not publish a species identification. For resource managers, the most important question to resolve quickly during a fish kill/disease event is whether our fish bioassays have yielded an actively toxic dinoflagellate population. If so, we report a "positive fish bioassay for the presence of a toxic *Pfiesteria*-like species." Thus, during the Pocomoke outbreak, our laboratory reported positive fish bioassays as soon as we had verified active, fish-killing populations that closely resembled *Pfiesteria* under light microscopy. Maryland officials then acted immediately to prevent people from entering affected areas. When there was time after the toxic outbreaks, we completed the species identifications, which we did publicly report as *Pfiesteria piscicida*, alone or with the second toxic *Pfiesteria* species (DNR 1998). It is because of our demonstrated expertise and strict quality control/assurance that we are relied upon as the reference laboratory to diagnose the presence of toxic strains of *Pfiesteria* spp. for officials in Maryland, Delaware, Virginia, North Carolina, and Florida (NOAA and EPA 1998-1999).

Use of Language

Griffith also miscast our writing as unprofessional. He stated (p. 123) that in describing *Pfiesteria* as an "ambush predator," we used words "more appropriate to military history than biology.... Surely a view of Pfiesteria as the 'ultimate biological threat' became possible through the ways in which scientists [previously identified as Burkholder and associates] and then reporters wrote about...the organism [these scientists] endowed the organism with anthropocentric qualities, claiming that certain human behaviors trigger it."

Here Griffith's writing demonstrated lack of familiarity with *accepted terminology in the field of aquatic biology* for describing predatory behavior in protozoans (including dinoflagellates) and other creatures. For example, the freshwater aquatic insect larva, *Chaoborus* (known as the phantom midge) is routinely described as an ambush predator (e.g., Riessen et al. 1984; Tjossem 1990). Words such as "gorged" and "engorged" are commonly used as well. We use "ambush predator" to signal readers that the two known toxic *Pfiesteria* spp. are animal-like toxic dinoflagellates that behave very differently than nearly all other toxic dinoflagellates, which are plant-like creatures (Steidinger 1993). We describe *Pfiesteria*'s effects on fish and human health as insidious (Glasgow et al. 1995) in accord with Webster's dictionary, stated to mean "working harmfully in a subtle manner" (e.g., see Peterson 1995). We repeatedly discourage use of such terms as "cell from hell" or "ultimate biological threat," as Betts (1996, 1997), Barker (1997), Guynup (1999), and many other journalists would verify.

The Fifth False Allegation—*Pfiesteria* Is Just an Issue of Affluent White People

Griffith falsely alleged (p. 125) that politicians have supported the *Pfiesteria* issue because it is a safe, affluent white people's issue. We pose two questions. First, where was Griffith when Governor Glendening took the courageous step of announcing the findings of the Maryland medical team *as soon* as he received them? The governor received the news the day before the 1997 Labor Day weekend when, if the information was announced and the citizenry failed to understand it, tourism and fishing industries stood to lose millions of dollars. A massive education effort had been launched, but the citizenry had continued to erroneously believe that the entire Chesapeake Bay was affected. Mr. Glendening was informed that the medical team had found compelling evidence that some people exposed to toxic *Pfiesteria* outbreaks had sustained profound cognitive impairment. This political leader was not willing to take unnecessary risks with citizens' health. For those actions, Mr. Glendening withstood a firestorm of protest from the tourism industry, the fishing industry, and, later, agricultural industries for passing the toughest legislation in the nation to strengthen control of nonpoint nutrient pollution that had been linked to stimulation of *Pfiesteria* (e.g., Dresser and Dewar 1998; DNR 1998). Thus, Griffith also erred (p. 125) in his claim that "the nutrient loading issue is one that [for politicians] may eventually involve taking on organized agricultural interests."

People such as this governor have shown they are willing to take the often-great personal and political risk of erring on the side of protecting the health of one person or one hundred, numbers too small to matter according to Griffith's writing (p. 120). Griffith does great disservice to all of these people, including North Carolina's governor and present state health director, who also have shown commitment to a proactive stance in protecting public health in this and many other more important issues. We note that North Carolina's present state health director is of African American as well as Caucasian heritage. His actions have shown the sincerity of his intent to strengthen health protection in issues ranging from *Pfiesteria* to swine industry impacts to air pollution *because*, as he has stated it, such issues can affect *all* people, surely including the economically depressed (Henderson 1999).

The second question is: Where was Griffith in 1991-1997 when concerned citizens, including members of our laboratory, repeatedly attempted to explain *Pfiesteria* to economically depressed people *because* of concern for their health? These people were not difficult to find—they were fishing or wading in the waters of toxic *Pfiesteria* outbreaks and collecting dead fish that had bleeding sores. They informed us of their intent to cut out the sores before cooking and eating the fish. They commonly complained of burning hands, or difficulty in breathing, or burning eyes when engaged in such activities. State health officials of that period were notably absent. When asked why there had been no effort to even visit a fish kill prior to late 1995–when massive kills and

health complaints had been ongoing for years–G. Smith (DEHNR) stated that the health officials had had insufficient travel funds to support the two-hour drive from Raleigh to New Bern (Neuse River Foundation 1995).

In many public forums on our own time, we/our colleagues explained that *Pfiesteria,* like many other common microbial pathogens, is cause for concern but not for alarm (records given to the Governor's Eastern Office, New Bern, N.C., April-July 1997). We designed an early warning system to proactively monitor for toxic *Pfiesteria* activity. We explained that fish kills, whether *Pfiesteria*-related or not, are innately unhealthy places because of rotting fish and associated bacteria. We advised that fish should not be eaten if dead or diseased when caught, and we pressed for cautionary signs to be posted about fish kills (Dill 1995). We were requested to write open letters in education outreach for fishermen and other coastal inhabitants/visitors (letters submitted to the Governor's Eastern Office, New Bern; DNR, Annapolis). Our staff and students frequently assisted children's recreational camps (Neuse—Camp Seagull, Camp Seafarer). We helped coastal aquaculturists eliminate *Pfiesteria* contamination (e.g., Bear Creek Shellfish near Swansboro, N.C.). We do not know where Griffith was—he avoided fish kills, even in his survey—but he is grossly in error to project such conduct onto others in casting *Pfiesteria* as a safe issue for affluent white people.

Numerous Other Errors in Griffith (1999)

Scholarship in peer-reviewed literature is supposed to be based, in part, on careful fact-checking with literature citations that support written statements. There are many other points of misinformation in Griffith's writing. As a few examples, Griffith (p. 120) miscited Paerl and Pinckney (1998) as a *Vibrio* reference. He falsely alleged (p. 120) that Maryland has downplayed *Pfiesteria* health issues for a nutrient focus (Hughes 1997, Grattan et al. 1998). No such agency as the North Carolina Department of Natural Resources has existed throughout the *Pfiesteria* issue (p. 125); Sprinter (Springer) is misspelled (pp. 123, 127); Burkholder et al. (1992) and Smith and Music (1998) citations are incomplete (pp. 126, 127). Griffith (p. 124) falsely ascribed to Burkholder et al. (1992) and Burkholder (1998) accounts of a researcher's experiences when affected by toxic *Pfiesteria.*

In more serious misinformation, Griffith falsely alleged (pp. 120, 125) that our laboratory worked with high concentrations of *Pfiesteria* which "have never been documented in the wild." The reality: 1 of 12 people who were affected worked with a culture that was at 90,000 cells/mL; the rest worked with culture at 300-2,000 cells/mL. Glasgow et al. (1995) (misquoted by Griffith) stated that cell densities up to 250,000/mL have occurred in *Pfiesteria*-related fish kills (reconfirmed using our species-specific molecular probe, developed by P. Rublee with our laboratory). Densities in the 10^4 range also have been reported (Burkholder, Glasgow, and Hobbs 1995; reconfirmed with our species-specific molecular probe). Note that in September 1997, state health official S. Music (1997a)–a frequent source in Griffith's writing–corrected similar misinformation that had been widely conveyed by his agency.

Griffith also seriously misstated (p. 120) that the available evidence indicates that *Pfiesteria* is "on par with other well-known marine irritants such as ciguatera, a coral-reef neurotoxin, or the better-known red tide." Ciguateratoxin causes serious neurological illness (called ciguatera, a term misused by Griffith) that can recur for years after exposure (see Burkholder 1998). It is not merely an irritant, nor is *Pfiesteria* merely an irritant (Glasgow et al. 1995; Fairey et al. 1999). Some "red tide" dinoflagellates produce among the most potent biotoxins known; for example, saxitoxins and derivatives, which have caused human deaths in many parts of the world (Falconer 1993). About 40 species of toxic dinoflagellates have been newly recognized in the past 25 years (Burkholder 1998). There is strong precedent for toxic dinoflagellates to cause serious impacts on human health (Falconer 1993). These facts, considered with the sometimes-serious health impacts that have been linked to toxic *Pfiesteria,* make Griffith's false portrayal of *Pfiesteria* as a nonissue and other toxic dinoflagellates as only irritants, especially troubling. Improved diagnostics for *Pfiesteria*-related illness will be developed once the toxins have been identified (Fairey et al. 1999); but that does not make *Pfiesteria* unusual either, since the toxins from various other harmful algae/dinoflagellates remain poorly characterized (Falconer 1993).

Nutrient linkages with *Pfiesteria,* as first reported by Burkholder et al. (1992), were politically unpopular data to agricultural interests, developers, and certain regulatory officials. Griffith miscast (pp. 124-125) this important aspect of the *Pfiesteria* story in his writing: "evidence of this [nutrient stimulation of *Pfiesteria*] remains a matter of debate in the biological literature." Two respected scientific panels critically evaluated all available evidence about *Pfiesteria* and nutrients (Boesch 1997; Wright 1998). They reached unanimous accord that there is strong evidence for stimulation of some stages of *Pfiesteria* by nutrient pollution. What *remains* to be determined (i.e., "in debate") is the relative importance of various sources of nutrients, from sewage to cropland fertilizer runoff to swine wastes, in stimulating *Pfiesteria* (Burkholder and Glasgow 1997; Wright 1998).

The reference used by Griffith to support his misstatement about nutrients and *Pfiesteria* was available (in draft form) to Wright's (1998) panel. Like Griffith et al.'s (1998) survey, it was not well received. The Paerl and Pinckney (1998) study indicated no increase in "pfiesteria-like cells" under nutrient enrichment–but actually, such cells generally were sparse in unenriched controls as well. The experimental system was not conducive to *Pfiesteria* growth in part because an inappropriate salinity had been used (ca. 2 psu, near freshwater; range 0.1-5.2 psu). Strongly brackish conditions are required for rapid growth of *Pfiesteria* spp. (e.g., Burkholder, Glasgow, and Hobbs 1995); they can grow at 2-5 psu salinity, but slowly over several weeks. These tests

lasted only 6 days. As the authors could not find/identify *P. piscicida*, results were given as "*Pfiesteria*-like cells." Griffith thus cited a technical report that, despite its title, is not about *Pfiesteria,* but failed to cite multiple peer-reviewed international publications that actually were about the organism or consensus reports from two respected science panels.

The Role of Science Ethics in Environmental/ Health Issues

A final aspect of this issue remains to be addressed. Griffith's writing casts environmental/health officials, from tobacco issues to AIDS, as consistently having acted responsibly. A wealth of literature on the discovery of new environmental issues that affect human health unfortunately refutes his view. The 19th century play, "An Enemy of the People," (Ibsen 1882; Greenberg 1998) captured the reality of what is frequently encountered, especially involving new pathogens that are not easily tracked without obvious human sickness or (as Music 1997b had stated) people "falling out of the boats dead." News is unwelcome if it threatens economic development; further study for years, with funding often directed to scientists without expertise but with stated bias against the issue, is a common fallback position, while little is done to err on the side of protecting natural resources and human health (Gelbspan 1997; Wilkinson 1998). The health arena is prepared to deal with new, obviously lethal pathogens, but does not deal as well with new pathogens linked to more subtle, chronic health impacts (e.g., Liegner 1994; Kaiser 1995).

The AIDS virus is such a chronic pathogen. Shilts's (1987) writing on the discovery of this virus and its link to human illness and death was cast by Griffith (p. 119) merely as a "dramatized account with a doomsday theme." What early researchers observed about that virus and its impacts was sufficiently compelling to cause them to risk their careers, to combat scientists with compromised ethics and various government officials who were reluctant to act, and to sustain personal and professional attacks. Considering the rapid trajectory of the AIDS epidemic within the past two decades, and the evidence for delayed government response (e.g., Aldhous 1991), it would seem that the concerned actions of these researchers merit more respect than Griffith afforded them.

Industries, government agencies, and politicians are most often blamed for lack of progress on environmental issues and for the associated serious human health and natural resource impacts that can result. The damage contributed by ethically compromised scientists–not only as personnel in certain industries, but as free agents in academia–is also great, and seldom brought to light. Certainly, it is not usually revealed by the press who are taught to cover both sides of a given issue, while evaluation of scientific expertise is omitted from their training. Intimidated by science, well-intended journalists often forget what they do best—namely, to investigate (Gelbspan 1997). They often fail to assess (e.g., by requesting the scientist's peer-reviewed international science publications) whether a scientist who declares him/herself an expert in an area is telling the truth. They also fail to determine whether the scientist has been a paid consultant for a vested interest that the individual staunchly supports. In so doing, the media often miss the most important story of the "controversy," namely, the motive and damage of scientists who, as falsely proclaimed experts, denigrate research about which they know little or nothing (e.g., O'Neill 1994).

Scientific misconduct is usually defined within narrow constraints. Many professional societies consider it to include only data fabrication/falsification and plagiarism, taken from the National Science Foundation's definition (1996:254): "Misconduct means fabrication, falsification, plagiarism, or other serious deviation from accepted practices in proposing, carrying out, or reporting results... or, retaliation of any kind against a person who reported or provided information about [such] suspected or alleged misconduct and who has not acted in bad faith." Such narrow scope does not adequately address compromised ethics within the scientific "community," where there is evidence of serious dysfunction (Marshall 1995; Ryan 1996). For example, Swazey, Anderson, and Lewis (1993:552) concluded, "Scientific misconduct, as narrowly defined to include plagiarism and data falsification, takes place less frequently than other types of ethically wrong or questionable behavior by [science] faculty."

In the environmental/health arena are scientists who commonly misrepresent their expertise to obtain grants (e.g., O'Neill 1994; Gelbspan 1997). In some cases, scientists—although ethically required to abstain from reviewing grant proposals or manuscripts by other scientists with whom they have personal or professional conflicts of interest—ignore that requirement (e.g., O'Neill 1994). In other cases, scientists evaluate another scientist's grant proposal as poor in quality because they covet the research and then obtain the grant funding to do it by using the ideas/writing of the researcher whom they falsely condemned (e.g., Marshall 1995; Moran 1998). In still others, scientists inform a potential funding source that they will find what is in the best interest of the funding source to find, if given the support (e.g., O'Neill 1994). Or, scientists lacking expertise on a subject fabricate damaging misinformation about experienced scientists, with motivation in greed, jealousy, or personal malice (e.g., Wilkinson 1998). None of these breaches in ethics easily fits the generally accepted, narrow definition of scientific misconduct (NSF 1996, part 1)–but all have played roles in the *Pfiesteria* issue (Barker 1997) and many other environmental/health issues (e.g., Shilts 1987; Gelbspan 1997), sometimes with dangerous potential or realized outcomes (e.g., O'Neill 1994; Wilkinson 1998).

Science as a general discipline has little in the way of effective mechanisms to combat such serious ethical breaches, except for the "test of time" —years of publications for a historical verdict that may occur long after the scientists who told the truth, based on sound data rather than false claims, are deceased (e.g., Miller 2000). Ironically, a scientist who engages in the above unethical actions is usually not punished but, rather, rewarded (e.g., Gelbspan 1997), often with

grants and, thus, enhanced recognition and power, with the scientist's increased control over the issue through design of the research and interpretation of the data. It is easy for such scientists—deliberately, or through lack of expertise—to design studies that "fail" or find nothing, so that the work is used by those who sanctioned it to falsely negate the issue. The net outcome can be years required to correct the misinformation, with cost of careers and sometimes lives as well as destruction of public-trust natural resources for the lucrative benefit of few (O'Neill 1994; Wilkinson 1998).

What the *Pfiesteria* issue illustrates is not, as Griffith misstated (p. 124), an example of "anecdotal information, presented without additional checks required in scientific reporting, [that has led] to flawed conclusions." It provides, instead, an example of an environmental/health issue that was nearly and falsely negated because certain agency officials' misconduct received strong support/attempted legitimization by ethically compromised scientists. The *Pfiesteria* issue has been invoked by the eminent scientific society, AAAS, as an illustration of the need for mechanisms to infuse higher ethics in science. As mentioned, following investigation into Burkholder's conduct and ethics, AAAS vindicated her from the false allegations made by Griffith and the like. As a second outcome, AAAS is exploring mechanisms through which scientists can obtain assistance when confronted by powerful forces that attempt to discredit politically unpalatable data by falsely defaming the personal/professional character of researchers who present the information.

Are such mechanisms needed? Ryan (1996:163) wrote, "The scientific community has been reluctant to discourage misconduct and sloppy research.... Even the National Academy of Sciences has indicated that such standards are strictly voluntary. The current research environment seems to foster cynicism about simple virtues such as honesty and fairness, and it clearly fosters hostility toward anyone who makes claims about misconduct." In formally correcting Griffith's personal attacks and other false allegations, Burkholder stands among relatively few in the scientific community who have been critically investigated by societies such as AAAS, and then honored for the integrity of their character and actions as well as the quality of their research. We view inquiry into the conduct of environmental/health scientists (here, including social socientists) as action that should be taken on a broader scale to strengthen science ethics. Such practice could lift the issue beyond the reach of local controlling forces. If carefully and constructively conducted, it could also provide a means to more objectively evaluate the scientists and the data involved. It could offer, additionally, strengthened means to correct a failed peer review process, exemplified by this journal's acceptance of the many false allegations and errors contained in Griffith's writing. Such evaluations, if more frequently practiced, could help to address the critical need identified by Ryan (1996:163) for additional mechanisms to "stimulate...wide-ranging discussion of integrity and misconduct that has been lacking in the scientific community for so long."

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

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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 dinoflagellate (Burkholder et al. 1992; Noga et al. 1993). 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 of 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 1998), 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 hyperbole (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, but 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, but *leaving open* the possibility that humans might be 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), but when extrapolating to estuarine exposure, Burkholder and coauthors have consistently characterized the problem as *potential*. For example, Burkholder, Glasgow, and Hobbs (1995:58) stated that "Apart from anecdotal information provided by local fishermen, carefully designed epidemiological studies are lacking to determine whether humans who frequent estuaries with toxic outbreaks might be adversely affected." Also, Burkholder and Glasgow (1997:1073) stated that "Anecdotal information...points to the potential for this dinoflagellate 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.122), 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 Borré 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 claims that [*Pfiesteria*] posed a serious threat to public and *environmental health*," emphasis added). However, the *Pfiesteria* issue cannot be divorced from the impacts on fish. All finfish 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 (Noga 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 are 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 *Gymnodinium 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. 1992; Burkholder and Glasgow 1995; Steidinger et al. 1996; versus Tomas 1997. 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 *Saggita,* 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, but 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; for 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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Regarding Pfiesteria

David Oldach

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As an investigator participating in Maryland's CDC-sponsored *Pfiesteria* and human health studies, I find it interesting that Dr. Griffith (1999:119) can conclude that *Pfiesteria piscicida* "poses no serious threat to public health," while so many questions about the organism, its putative toxins, and their neurotoxicology remain unanswered. The fact of the matter is that we have not measured the impact of this organism on public health. Dr. Griffith states that there is "mounting evidence" (p. 119) that the organism has no impact on human health; reviewing the available data, the opposite conclusion can easily be justified (Oldach, Grattan, and Morris 1999). Cohort studies designed to address the question of *Pfiesteria* and *Pfiesteria*-like dinoflagellates' impact on human health have been initiated in Maryland, North Carolina, and Virginia. In each of these studies systematic, uniform, and prospective protocols are being used to assess health outcomes. Study participants' performance on sensitive tests of cognitive ability is being monitored over time. These data will be analyzed in linkage with extensive environmental monitoring data collected to monitor for the presence of *Pfiesteria piscicida* and its possible effects on fish health. As the results of these investigations become available, we will be in a better position to objectively assess the risk of illness linked to estuarine water exposure in the presence (or absence) of *Pfiesteria piscicida* and related dinoflagellates.

Dr. Griffith's study of North Carolina watermen's health is interesting, although incomplete in addressing these questions. The author himself clearly stated some of the methodological flaws that accompany retrospective survey data. Other study design concerns could be debated, but they are outside the intended scope and length of this letter. Dr. Griffith does point out that these watermen were not exposed to fish-kill events: what then does "exposure" mean? Does studying individuals without exposure to such events, (i.e., when the organism, if present, is presumptively "nontoxic") support the global conclusion that the organism has no impact on public health? Be that as it may, the essential observation from this study is that over 300 North Carolina watermen with exposure to estuarine waters were interviewed, and they did not report symptoms of cognitive disturbance in excess of that reported among the control population. This is an important observation and possibly informs us regarding the risk of *Pfiesteria*-related illness in this setting (in the absence of fish-kill exposure). The study may also inform us, however, of the stoicism of North Carolina watermen, or of their perception of the study's potential impact on their livelihood. Thus, despite his protests over funding for further *Pfiesteria* research, the article actually underscores the need for studies that incorporate prospective evaluation of signs and symptoms among "at-risk" persons and the use of objective measures of cognitive function and *Pfiesteria* exposure.

Dr. Griffith raises alarm regarding overutilization and overinterpretation of anecdotal clinical data, and particularly, its dissemination in the popular media. This point is well made; there is no question that *Pfiesteria* has been dramatically (and often completely inaccurately) presented in the media. For this reason, most investigators involved in ongoing epidemiological studies of possible *Pfiesteria*-related human health effects have adopted a decidedly low-key approach to the media. Regarding anecdotal data, however, I must take exception to Dr. Griffith's contention that the cases (in North Carolina), while alarming, "had neither clinical nor epidemiological support" (p. 124). In fact, "anecdotal" illnesses among the laboratory workers were evaluated by a highly respected neurologist at Duke University School of Medicine (Donald Schmechel); were found by him to be clinically compelling; and were reported in a peer-reviewed publication (Glasgow et al. 1995). We found Dr. Schmechel's insights to be invaluable as we prepared for our investigations of reported illness among Maryland watermen. That investigation found both clinical and epidemiological support for the occurrence of a novel syndrome of memory disturbance among individuals with exposure to *Pfiesteria*-related fish kills and was sufficiently robust, scientifically, to pass peer review and publication in the journal *Lancet* (Grattan et al 1998). Dr. Griffith also states that individuals in our investigation "recovered fully, usually within a few hours or days" (p. 120). In fact, our article states that individual's test performance had returned to within normal ranges within 3-6 *months*. Furthermore, in that study we could not measure individuals return to their own normal level of function, as preexposure cognitive skills had not been measured. Some study participants continue to complain at this time of persistent illness.

Although I found Dr. Griffith's musings on shark bites, sting rays, and sea nettles amusing, the comparisons are irrelevant, for we have a pretty good idea of what happens when humans encounter these organisms, and when it has occurred. In contrast, we have not defined the spectrum of human illness related to exposure to *Pfiesteria*-associated toxins, nor have we defined clinically significant exposures, and we do not as yet have a laboratory assay to detect the toxin(s) in human tissues or secretions (if detectable). How then can one compare them? At one time, one was also more likely to suffer a shark bite than to be diagnosed with Legionnaire's Disease, Lyme Disease, domoic acid intoxication (amnesic shellfish poisoning) or any of a number of recently recognized illnesses. Each doubtless existed long before its recognition, and came to light scientifically as a result of investigations of disease clusters, or (as in the case of Lyme Disease) in response to the activism of family members of individuals with "anecdotal" illness. In the case of *Pfiesteria,* clusters of otherwise unexplained illness occurred first among laboratory workers in North Carolina and later among watermen on the Chesapeake Bay. These events led to initial investigations that implicated possible exposure to *Pfiesteria* toxins as a cause of illness. The results of those investigations were sufficiently compelling to lead to the design and implementation of the more definitive epidemiological and basic science investigations presently underway. Until such evaluations are complete, conclusions regarding the impact of this organism on human health are, in my view, premature.

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Placing Risk in Context

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The Seamen call it looming. *Philosophy is as yet in the rear of the seamen, for so far from having accounted for it, she has not given it a name. Its principal effect is to make distant objects appear larger, in opposition to the general law of vision, by which they are diminished. I knew an instance at Yorktown, from whence the water prospect eastwardly is without termination, wherein a canoe with three men, at a great distance, was taken for a ship with its three masts.*

Thomas Jefferson
Notes On the State Of Virginia, 1787

Thomas Jefferson's observations recognize that appearances can be deceiving and that expertise sometimes develops beyond the halls of academics. In the incident he describes, the passage of time and a shift in perspective revealed that what was believed to be a great ship was, in fact, nothing more than a passing canoe. Much of cultural anthropology has been directed toward pointing out that humans are often misled by superficial explanations and that individuals with the best of intentions can benefit from and fuel misperceptions from platforms of recognized authority. Emily Martin's (1987) critique of the medical profession's gender bias, Bonnie McCay and James Acheson's (1987) accumulation of evidence questioning assumptions of the trag-

edy of the commons, and E. Paul Durrenberger's (1995) empirical challenge to central assumptions of economic science are three such examples. The work of anthropologists along these lines involves contextualization: simply, gaining a new perspective by placing behaviors, ideologies, bodies of knowledge, and other social and cultural phenomena into the broader human contexts that produced them. My most recent work on *Pfiesteria* (Griffith 1999a), which the authors of the rebuttals presented with this response evidently failed to understand, was one such endeavor.

Because so many of the comments in the rebuttals were irrelevant to my argument, I confine my response to those areas where the criticisms attempt to reveal its weakness: namely, *Pfiesteria* as an exaggerated risk and the design of the study that led me to argue this in the first place. The two issues are closely related. In attempting to counter the argument that *Pfiesteria* represents an exaggerated risk, the authors point to the organism's history of causing illness in humans, something which I acknowledge in the article several times. The fact that *Pfiesteria* can cause people to get sick, however, is a separate issue from the extent to which people in general, and people with water-based occupations in particular, are at risk from its toxin. Many plants and animals in the natural environment—including, as one rebuttal notes, ticks that carry Lyme disease—can cause illness and death, yet many that are far more dangerous than *Pfiesteria* have not received the media and political attention that *Pfiesteria* has. I mentioned *Vibrio* in the article, but I could have easily used much more common examples from households across the world. Annually, some 300,000 individuals in the United States experience sickness from eating chicken eggs, and over 200 of these individuals die (GAO 1999). Despite such alarming statistics, most people do not fear eggs. Studies have shown that people tend to be poor judges of the risks they face, placing the risks of such incidents as nuclear power plant meltdowns above the risks of driving automobiles, consuming alcoholic beverages, or smoking cigarettes (Fischoff, Lichtenstein, and Slovic 1989; Rodericks 1992). Despite that the Grattan et al. (1998) study was based on fewer than 15 individuals, let us suppose that in fact up to 40 individuals have been affected by *Pfiesteria*, as one of the rebuttals claims. There are over 6,500 watermen in North Carolina alone. Leaving aside the hundreds of thousands of tourists, fishery biologists, coastal residents, and others who come in contact with the waters of North Carolina and the Chesapeake, 40 afflicted individuals represent less than 1 percent of just the population of watermen—the people most likely to come in contact with *Pfiesteria*.

Based on the best epidemiological evidence to date (Griffith et al. 1998; Morris et al. 1991), and on the small numbers of people to have claimed to have been afflicted by *Pfiesteria* over the years since its discovery, *Pfiesteria* poses low risks to the general swimming, fishing, and boating public of eastern North Carolina. With each passing season of few individuals reporting *Pfiesteria* poisoning, the evidence for low risk mounts. In any case, it is certainly not, as Roderick Barker (1997) claims in his book's subtitle, "the ultimate biological threat." (I would encourage readers to examine Barker's book themselves, as well as others he has written, to judge whether or not his work represents sensationalist reporting.)

People from the popular media and others were able to exaggerate *Pfiesteria's* threat, however, based in part on raw materials provided by the scientists working with the dinoflagellate. It may be common practice to use terms like "phantom" and "ambush" in the biological literature, but in the context I discuss in my article, these characterizations provided grist for the mills of media coverage, environmentalism, and politics. Again, it is the overall context that I address in my argument, not its specific components in isolation from one another, and trends in sensationalist reporting, conspiracy theories, and doomsday predictions are part of that context. Clearly, the reasons that *Pfiesteria* has been exaggerated are as multiple as the causes of fish kills.

Regarding the design of the study that led me to write about *Pfiesteria* as an exaggerated health threat, the authors of the rebuttals were mistaken about the purpose of our study. Had we been purposefully looking for people who had become ill due to exposure to the water of eastern North Carolina, the authors of the rebuttals would have been correct in their assertions that our study was poorly designed. This was not, however, our purpose. We were interested in determining whether, under normal ecological conditions, a public health threat existed from *Pfiesteria* and, if such a health threat did exist, what were its characteristics regarding the distribution of symptoms associated with *Pfiesteria*, the individuals affected, and other factors that could enable health workers to marshal an effective response. Repeatedly, during and after our research, individuals from the press, along with others with an interest in exaggerating the threat of *Pfiesteria*, criticized us for not interviewing the "right" people in our study (that is, people sickened by *Pfiesteria*). We did not intentionally include or exclude these individuals. As Marcia Angell (1995:99-103) and other medical researchers know well, one does not estimate the incidence of a disease in a population by seeking out only those individuals experiencing the disease. This would be like attempting to determine the incidence of bee stings in a population by designing the study to include only beekeepers.

By the same token, we did not specifically include or exclude individuals who had been exposed to fish kills. Fish kills are exceptional environmental events that most people avoid because they smell awful, look dreadful, and produce other unpleasant signals that something is terribly wrong with the water. In both our report and in the article, we reiterated the common-sense warning that people should exercise caution around such events. Yet to focus exclusively on human interaction with events of this nature would have, again, biased our study, and biased it in a way that exaggerated the threat of *Pfiesteria* to the general public.

Instead, as the article makes explicit, we used a random-sampling design among a population that would have been

particularly at risk from *Pfiesteria,* had the organism constituted a true health threat. For several reasons, crabbers were the ideal candidates for the study. They had been coming in daily contact with the waters of eastern North Carolina for years, they move their traps and bait nets through various territories during the crabbing season (including areas that had experienced fish kills and algae blooms), nearly two thirds (64% - 65%) had experienced fish kills, and they were a large enough population to make comparisons in terms of important variables such as reported symptoms, fish-kill areas, and crabbing territories. Our research assistants and I had had extensive experience and rapport with this population from years of previous research (Griffith 1996, 1999b). Finally, as with Jefferson's seamen, many watermen possess generations of knowledge and lifetimes of personal experience with water quality, characteristics that helped us consider the *Pfiesteria* issue in light of its broader context.

It is true we did not acquire maps of fish-kill areas until after we had collected most of the data. This was deliberate. We did not want to bias our observations by knowing ahead of time exactly where fish kills had taken place. By the time we did acquire maps, we had done enough preliminary data analysis to know that very low levels of symptoms associated with *Pfiesteria* were being reported, regardless of crabbing territory, and that few differences existed between the control and the target groups in the study. In short, little health threat existed. Still interested in the relationships between fish-kill areas and sickness, however, we did compare the territorial data we collected from the crabbers to maps showing fish-kill data that others, in the spirit of scientific cooperation, were responsible and kind enough to share with us. Again, based on these comparisons, little health threat existed. Our study, despite weaknesses I acknowledged in my article, along with the low number of people afflicted by *Pfiesteria*, remain the best evidence that the public health threat from *Pfiesteria* has been grossly exaggerated. I am confident that time, a slight shift in perspective, and additional well-designed epidemiological studies will show that *Pfiesteria* is not as looming a threat as a ship with three masts, but more like a few people passing by in a canoe.

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