Fish Kills in the Shenandoah River Basin: Preliminary Report of the Shenandoah Basin Science Team

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1. Background

For the past three springs (2004-2006), extensive fish kills have occurred in the Shenandoah River drainage. In 2004, the fish kills affected nearly the entire length of the North Fork of the Shenandoah River. In 2005, over 100 miles of the South Fork Shenandoah River were impacted. In 2006, the fish kills returned to the North Fork, a portion of the South River, and the mainstem Shenandoah River. These fish kill events were preceded by a similar fish kill in 2002 in West Virginia's South Branch of the Potomac River. The fish kills in the North and South Forks of the Shenandoah River resulted in an estimated loss of 80% of the adult smallmouth bass—an economically important (but nonindigenous) sport fish. A task force of regional biologists, water quality scientists, volunteer monitors, and local stakeholders has been investigating these fish kills under the leadership of Donald Kain (VDEQ) and Steve Reeser (VDGIF). Task force members represent the Virginia Department of Environmental Quality (DEQ), the Virginia Department of Game and Inland Fisheries, James Madison University, Virginia Tech, Virginia Commonwealth University, George Mason University, the U.S. Geological Survey, and a wide range of other agencies, institutions, and stakeholders.

The cause or causes of the Shenandoah river fish kills remain unknown and no less than 20 separate theories (Table 1) regarding causality have been advanced and several of these (e.g. ammonia toxicity, water temperature increases) have been pursued actively by task force members and promulgated by the popular press. None of these theories, however, are supported unequivocally by the (limited) relevant data. Much of the research activity to-date has focused on water quality monitoring and *post-hoc* responses to specific fish mortality events. No comprehensive research plan has emerged as a result of task force activities, although some significant data sources have been (or are being) developed by task force participants. At its core, the problem in the Shenandoah is a aquatic living resources problem, and a fish assemblage problem specifically. Water quality and land use are likely to be important factors, but fish biology and aquatic ecology may be the disciplines most likely to identify a cause or causes for the observed events.

The Shenandoah River has a long history of pollutant loading from a variety of sources. Long-term analysis of chloride, a conservative tracer of anthropogenic inputs to surface waters, indicates that the South Fork Shenandoah River has received increasing anthropogenic loadings of chlorides since 1952; levels have been relatively constant, however, since 1965 (Sherwood 2005). Based on this analysis, four major sources contribute 92.2 percent of the anthropogenic chlorides (1) deicing salts—4,149 tons/yr, (2) domestic sewage—3,015 tons/yr, (3) livestock and poultry wastes—2,458 tons/yr, and (4) commercial fertilizers—2,249 tons/yr. Among these four sources, the last three also contribute substantially to nutrient enrichment and to a wide range of potential contaminants to surface water and ground water within the basin.

2. Shenandoah Basin Science Team

In November, 2006, the Virginia Department of Environmental Quality established the Shenandoah Basin Science Team, a technical advisory group initially comprised of two academic fish biologists (Drs. G. Garman and D. Orth) that work closely with the co-chairs (D. Kain and S. Reeser) of the Shenandoah Fish Kill Task Force. The Science Team was charged with developing and implementing an evolving, hypothesis-driven study plan to focus in on the most plausible causes of the recent fish kills. The plan was to employ a combination of data mining, integration, and analysis, and the targeted development of new databases, to develop a 'short list' of the most plausible putative causes of the observed events, whether biological, chemical, or both.

Specifically, the Science Team's current objectives are: 1.) identify, collate, and synthesize existing and relevant data for the region; 2.) develop key working hypotheses and identify significant data gaps related to these hypotheses; 3.) develop, publish, and update a dynamic data portal to provide researchers access to new and archival data and data sources; 3.) develop and assist with the implementation of an evolving, comprehensive hypothesis-driven research and monitoring plan to identify the specific cause(s) of fish mortality and morbidity, and 4.) identify additional technical disciplines and new potential members for Science Team expansion.

3. Current Observations and Working Hypotheses

The fish mortality and morbidity events in question have occurred primarily within a single Chesapeake Bay watershed (Shenandoah) and within one major basin (Potomac), suggesting that there may be something unique about that one geographic location. In addition, fish kills have been documented only for the largest tributaries and mainstem of the Shenandoah River, suggesting that smaller tributaries may be unaffected. Furthermore, effects on only a small number of taxa—all fishes—have been documented. It is problematic that direct effects on the vast majority of resident aquatic fauna have not been observed or may not be observable with limited, *ad-hoc* observations. The available information does not, in fact, definitively and consistently implicate biological (epizootic) agents *versus* physico-chemical stressors (e.g. chemical contaminants), let alone validate the above observations or assumptions or identify a specific cause or causes. However, if the above observations and assumptions can be formalized as hypotheses and evaluated, that knowledge would provide much-needed focus to the on-going efforts to understand and address the fundamental cause(s) of the Shenandoah fish kills and develop appropriate remedial actions.

The Science Team has, therefore, identified several *working hypotheses* as a framework on which to base recommendations for future monitoring and research efforts, as well as appropriate corrective actions within the Shenandoah basin. These hypotheses include: 1.) the observed mortalities involved only a few of the 40+ fish species in the Shenandoah watershed and did not involve other aquatic taxa (e.g. macroinvertebrates); 2.) the observed mortalities involved only adult (cp. larval and juvenile) life history

stages of affected fishes; 3.) the observed mortalities were restricted primarily to the North Fork, South Fork, and mainstem Shenandoah River (cp. smaller tributaries within the basin; and 4.) the events documented for the Shenandoah River are unique to that watershed and the upper Potomac basin. A summary of pertinent observations, along with selected working hypotheses (in **bold**) and other potentially relevant information regarding the Shenandoah river fish kills is provided below:

Observations *re*: fish kills in Shenandoah Basin
Initial pathology reports and subsequent observations on fish mortalities are consistent with diagnosis of chronic stress in smallmouth bass.
Relatively low numbers of fish are dead or moribund at any one time. The vast majority of observed mortalities represent adult life history stages of only a few fish species (5% of the nonmigratory ichthyofauna of the basin), including smallmouth bass (*Micropterus dolomieu*), redbreast sunfish (*Lepomis auritus*), green sunfish (*L. cyanellus*), and northern hogsucker (*Hypentelium nigricans*), and include native, introduced, pollution-tolerant and pollution-intolerant taxa. There are, however, almost no community-level data to test the **working hypothesis that other aquatic species and/or fish representing non-adult life history stages have not been affected**.
Gill lesions and skin lesions are common on dead and dying fish. This is not a new observation as lesions were observed as early as 1994.
Fish kills may occur in any time of the year; however, kills most typically start in March and continue through June.
Patterns of rainfall and surface runoff were inconsistent during the three years (2004-2006) of fish kills, suggesting that the fish kills are not associated directly with runoff events.
Fish kills have occurred in North Fork, South Fork, and mainstem Shenandoah River; however, there are no available data to test the **working hypothesis that fish or macroinvertebrate populations in tributaries have not been affected.**
The surface waters in the Shenandoah River basin have elevated levels of nutrients (primarily nitrogen and phosphorus) that have been increasing during recent decades.
Elevated nutrients and clear, shallow water appear to be linked to excessive production of periphytic algae (diatoms); some contaminants (e.g. trace metals) may be sequestered, and subsequently released, by periphyton during senescence.
Domestic sewage, livestock and poultry waste, and commercial fertilizers contribute to elevated nutrient loads. However, potential geospatial and temporal patterns involving point- and non-point pollution sources and fish kill events have not been evaluated adequately. We believe that the necessary spatial and nonspatial data exist to test the **working hypothesis that fish kills and pollution sources are not correlated in time and space**.
The pH in the river shows daily swings from circumneutral to strongly basic on days when water is clear; pH daily highs may regularly exceed 9.0.
April is the time of peak atmospheric concentrations of ammonia (NH_3) in the region based on air sampling stations at Big Meadows and the James Madison University farm. The specific contribution of atmospheric ammonia deposition to surface waters is unknown.

Findings from literature and data review
Physiological stress in largemouth bass (a congener of smallmouth bass) occurs at ammonia concentrations substantially below NH_3 regulatory standards.
High ambient pH (>9.0) affects the oxygen affinity of fish hemoglobin, and may result in sublethal oxygen stress.
High ambient pH (>9.0) may increase the solubility and toxicity of some contaminants, including trace metals.
A wide variety of potential contaminants (arsenic, selenium, pesticides, endocrine disrupting compounds and other xenobiotics) have been identified in poultry litter and livestock waste within the Shenandoah basin and elsewhere.

Uniqueness of the region
Augusta and Rockingham counties at the upper regions of the watershed have the highest reported poultry waste transfer levels in Virginia.
Surface waters contain perennial groundwater inflows and flow pathways are regionally conflicted by carbonate-epikarst and non-carbonate siltstones, sandstones, and shales in the valley floor. The unique karst geology of the basin suggests that s**ubstantial groundwater inputs to the Shenandoah River and its tributaries may represent an additional and undocumented source or sources of excess nutrients or contaminants.**
Poultry and livestock waste contains estrogens (beta estradiol in livestock feed) and have direct and indirect access to groundwater and surface waters.
A significant lack of riparian vegetative buffers may exacerbate nutrient and contaminant transport into surface waters of the Shenandoah basin, compared to other river systems.

4. Current Theories

Given the significant gaps in the information available and the complexity of large ecological systems, the Science Team is not able at this time to identify a specific causal agent or agents that would fully explain the current observations regarding fish kills in the Shenandoah River basin. A preliminary review of the literature supports the general view that multiple chronic stressors are contributing factors involved in the Shenandoah River fish kills. Possible symptoms of chronic stress in smallmouth bass from the Shenandoah River include bacterial lesions, parasite infestations, thickening of gill filaments, and macrophage aggregates in livers (Blazer et al. 2006). The fact that observed dead and dying smallmouth bass tend to be the larger and older specimens within the population suggests that sublethal stressors are acting in a temporally (or trophically) cumulative fashion—possibly as a result of bioconcentration (Southworth et al. 2004, Chen and Folt 2000), synergism among stressors, or long-term exposure—leading to immunosuppression. In contrast, studies elsewhere typically show that early life history stages (e.g. larvae and juveniles) are more directly sensitive (cp. adults) to

pollutants. Finally, there is no reasonable explanation at present for the observation (one of the Science Team's working hypotheses) that the 2004-2006 fish kills have involved only a few aquatic species (e.g. smallmouth bass) and that only one of the affected taxa (northern hogsucker) is considered pollution-intolerant. Smallmouth bass are not native to Atlantic Slope rivers and introduced populations may exhibit limited genetic diversity, which could, in turn, affect disease resistance (Snyder, et al. 1996).

At least 20 distinct theories (listed below) regarding the putative cause or causes of the recent (2004-2006) fish kills have been promulgated by members of the task force and others. These theories implicate the full range of biological pathogens, ecological effects, chemical contaminants, and physicochemical stressors. However, because of significant data gaps, there exists no unequivocal evidence at present that should promote one or more specific theories, or even implicate biological and ecological *versus* physical or chemical causal factors. Some reasonable theories involve both biological and chemical agents. The Science Team has prioritized these theories based on the 'fit' of each to the available information. Recommendations for further action, provided later in this report, are focused on evaluating Tier I (high priority) and Tier II (moderate priority) theories using an hypothesis-driven framework (the 'working hypotheses' presented earlier).

Most of the theories presented below are neither independent nor mutually exclusive. In fact, the current level of uncertainty in decision making and the complexity of coupled human-natural ecosystems makes it likely that multiple and interactive causal factors will eventually be implicated. The priority rating of individual theories will likely change substantially as data are generated, knowledge gaps are filled, and working hypotheses evolve into a single, comprehensive theory, which will eventually help determine the most effective remediation actions. Classification of Tier I and Tier II theories is based on a preliminary review of available literature, data, and observations, on factors that may be unique to the Shenandoah basin, and on the best professional judgment of Science Team members.

Tier I Theories (high priority)

- Cumulative (temporal and/or trophic) effects of primarily agricultural chemicals and additives, including biocides, trace metals, hormones, and drugs
- Immunosuppression in affected fishes, leading to infection by secondary opportunistic biological pathogen(s)
- Direct effects by unknown biological pathogen(s), including bacteria, protist or mesomycetozoan parasites, viruses, or fungi

A number of trace metals (e.g. arsenic, selenium) are added to poultry and livestock feeds as biocides, growth promoters or immuno-stimulants (Brown et al. 2005, Lemley 2004, Simmons and Wallschlager 2005). Levels of these and other contaminants in poultry litter and related sources have not been documented adequately, although arsenic levels in smallmouth bass tissues from the Shenandoah River are higher than in other Virginia river basins (A. Barron,

VDEQ, pers. comm.). Many organic wastewater contaminants are detectable in U.S. streams (Kolpin 2002) and some are known or suspected to disrupt normal endocrine function (Orlando et al. 2004). Nitrates at high concentrations may also disrupt normal endocrine function (Guillette and Edwards 2005). In the Shenandoah River, the high occurrence of intersex male smallmouth bass may correspond with concentrations of poultry feeding operations and land application of poultry litter, which contains estrogens, testosterone, and progesterone (Finley-Moore et al. 2000; Hakk 2005; Yonkos 2005). The possible immunosuppressant effect of intersex condition (i.e., endrocrine disruption) in fishes has not been ruled out or confirmed by scientific studies.

The association of potentially virulent pathogens with Shenandoah River fish kill events (V. Blazer, USGS, pers. comm.) is consistent with the multiple stressor hypothesis. Infectious disease outbreaks in fishes may coincide with a variety of stresses, such as eutrophication, sewage and metabolic waste (e.g., ammonia), industrial pollution, and pesticides (Snieszko 1974). The unique character of the Shenandoah basin is supported by data showing higher levels of nitrogen and phosphorous, greater densities of animal feeding operations, and high arsenic concentrations in fish tissues, compared to watersheds elsewhere in the region (Smedley and Kinninburg 2002). These unique characteristics may contribute to sublethal stresses that combine to predispose some fishes to disease outbreaks.

Lesions on smallmouth bass from the Shenandoah basin generated isolates of opportunistic bacteria, including: *Aeromonas salmonicida, Enterobacter* sp., and *Flavobacteria columnare* (Blazer et al. 2006). These bacteria are common in centrarchid fishes and other game fishes in freshwaters and outbreaks involving *A. hydrophila* are stress-mediated events that may cause death at temperatures > 9.4° C. The presence of high nitrite concentrations (>6 mg/L) increased the susceptibility of channel catfish to bacterial infection in a study by Hanson and Grizzle (1985). Fish lesions may also be caused by amphizoic amoebae (Webb et al. 2002), protozoan parasites (Webb et al. 2005), and mesomycetozoan parasites (e.g. *Ichthyophonous*; Jones and Dawe 2002). None of these potential biological pathogens have been evaluated adequately for the Shenandoah events. A limited application of novel genomic techniques by Dr. Pat Gillevet at George Mason University (2006) found no differences in putative pathogen communities between lesion and non-lesion fish (on surface mucosa) from the Shenandoah system.

Tier II Theories (moderate priority)

- excess anthropogenic nutrients and eutrophication of surface and ground waters
- chronic and/or episodic pH values above 9.0; diurnal shifts in pH > 1 unit
- groundwater contamination of surface waters
- episodic ammonia spikes following precipitation events
- bioconcentration of toxic compounds affecting older and predatory fish

Nutrient enrichment can occur from sewage treatment plants, agricultural storage and disposal of liquid and solid waste, and atmospheric deposition. Nutrient enrichment can lead to stressful conditions which may be contributing factors in the occurrence of fish kills. These conditions include hypoxia, extreme or dynamic pH levels, and harmful algal blooms. Harmful algal blooms have been recognized globally as causes of fish kills in eutrophic estuaries (Codd 1995; Glasgow and Burkholder 2000; Anderson et al. 2001) but diatoms, which are the dominant components of periphyton in the Shenandoah system, are not known to produce toxins.

Primary production is stimulated by favorable light and elevated nutrient conditions in the Shenandoah River and daily fluxes in pH and dissolved oxygen concentrations have been documented for the system. Although we do not have evidence to link such physicochemical fluxes and fish kill events, the evidence from the literature suggests that extreme pH conditions may release sequestered contaminants from sinks, shift chemical equilibria to favor more toxic chemical forms (e.g. ammonia *vs*. ammonium), impair physiological homeostasis of aquatic organisms, and injure gill tissues.

Alabaster and Lloyd (1982) concluded that pH levels between 9.0 and 9.5 are likely to be harmful to fish if present for considerable lengths of time. Extreme pH levels can eliminate certain fish species from highly eutrophic systems (Kann and Smith 1999). Mortality related to high pH appears due to the corrosive actions of the alkaline medium to the integument; gill tissues appear to be particularly sensitive. At pH levels > 9.0, fish gill tissues may exhibit separation between the epithelial cells and the pilaster cells and hypertrophy of mucous cells on gill filaments, exposing capillaries to the ambient medium and contaminants (Masscheleyn et al. 1991).

Un-ionized ammonia (NH_3) is the more toxic form (cp. ammonium NH_4^+) of this metabolite and is more permeable to gill membranes. In salmonid fishes the reported lethal concentrations of ammonia are 0.2 mg/L and Alabaster and Lloyd (1982) recommend ammonia concentrations of 0.025 mg/L as a water quality standard. The proportion of un-ionized ammonia (*vs*. ammonium,) is positively correlated with water temperature and pH and Bergerhouse (1992) documented the interactive, additive toxicity of high pH and ammonia in fish larvae and juveniles. Some fishes appear to be able to acclimate quickly to high ammonia concentrations (Tomasso et al. 1981), while elevated but sublethal ammonia levels cause degenerative gill and kidney damage, reduce oxygen carrying capacity of hemoglobin, and increase plasma stress hormones in other fishes (Smart 1976; Sousa and Meade 1977; Thurston et al. 1978; Tomasso et al. 1981; Suski, pers. comm.).

Current Theories *re*: Causes of Fish Mortality in the Shenandoah River

BIOLOGICAL and ECOLOGICAL

Periphyton (algae) excess production, causing:
- -toxins from HABs
- -hypoxia
- -Release of sequestered contaminants during senescence

Change in the trophic basis of production of the system from autochthonous to allochthonous

Trophic cascades from introduced fish predators (e.g. smallmouth bass, muskellunge)

High bacteria concentrations from Nonpoint and WWTP sources

Decline in crayfish populations (a primary prey of smallmouth bass)

As yet unknown pathogen(s), including parasites, viruses, bacteria, and fungi

Bioconcentration of toxic compounds affecting older fish and species at higher trophic levels

Immunosuppression, leading to infection by biological agent(s)

Intersex condition in fish

Spawning stress in smallmouth bass

Population collapse in smallmouth bass (carrying capacity 'correction')

PHYSICOCHEMICAL

Ammonia (lethal or sub-lethal concentrations) in surface and/or groundwater; atmospheric deposition

Agricultural chemicals in water, groundwater, or sediments, including:
- Additives to poultry litter or livestock feed
- Drugs
- Pesticides

Excess nutrients from agriculture (e.g. spring manure application) and WWTPs

Increasing water temperatures as a consequence of development within the basin

Chronic and episodic high pH (>9.0)

Groundwater contamination of surface waters

Endocrine disruptors and pharmaceuticals

Hypoxia

Biosolids applications

Dumping of illicit drugs

Based on *in-situ* monitoring conducted in 2006, ambient ammonia concentrations under baseflow conditions in the North Fork and South Fork Shenandoah River, were relatively low and not likely to affect centrarchid fishes. No measurements of ammonia concentrations during or immediately following significant storm events are available for the system. Atmospheric deposition may be an additional source of ammonia within the Shenandoah basin.

5. Preliminary Recommendations

The following recommendations have been developed by the Science Team based on an initial review of the relevant, available information. These recommendations are prioritized to focus limited resources on evaluating key working hypotheses and high priority theories. Recommendations and their priorities will likely change as additional information becomes available. The Science Team did not consider cost as a factor in its rankings.

Tier I Recommendations (high priority)

a.) Expand the scope of fish pathology and toxicological studies to formally evaluate the role of novel or taxon-specific pathogens, including bacteria, parasites, viruses, fungi, etc. *and* to look for evidence of lethal or sublethal toxic effects by chemical agents, and thereby refine the Profile. A significant number of fish samples from the 2004-2006 fish kill events were collected and provided to appropriate scientists but only a few limited reports *re*: analysis of those samples have been available. Both conventional (e.g. histopathology) and novel (genomic/molecular) approaches to the question of biological agents should be employed.

b.) Explore the composition of novel or unique additives in agricultural products (e.g. poultry and livestock feeds, bedding, etc.) as potential sources of important contaminants within the basin. Several recent and ongoing studies within the basin suggest that agricultural additives, including trace metals and other compounds, could be contributors to environmental stress. Because many of these products are proprietary, the Task Force or Science Team may need to conduct laboratory analyses of products purchased from local suppliers and identify cooperating local farm operators through the task force.

c.) Implement new field studies, database development, and data mining efforts to test key working hypotheses (see above) concerning the fish kill events and thereby revise or confirm prioritized theories. Specific working hypotheses (worded here as *null* models) include: impacts are not limited to a small number of resident aquatic taxa (*vs*. community-wide effects), impacts are not limited to adult life history stages of those fishes, impacts are not limited to the mainstem and major tributaries (*vs*. catchment and watershed scales), and that events in the

Shenandoah are not unique to that basin, compared to other watersheds within the region.

d.) Develop a *Contaminant Profile* of selected characteristics of putative chemical compound(s) or agent(s) that would have actions or effects consistent with the observed fish mortality and morbidity events in the Shenandoah basin during the period 2004-2006. This profile should be dynamic—evolving as more information becomes available and members are added to the Science Team—and should serve to refine progressively the field of potential causal or contributing chemical agents. A preliminary inventory of surface water and ground water contaminants within the fish kill geographic area (F. Borsuk, USEPA, pers. comm.) is approaching 100 different contaminants. An initial Contaminant Profile, based on a review of the archived data and relevant literature, is provided below:

- Demonstrated direct toxic effects or associated with immunosuppression effects in freshwater fishes and especially adult (*vs.* early) life-history stages.
- Demonstrated or suspected bioconcentration or biomagnification action within natural food chains, suggesting disproportionate impacts on predator guilds, adult life-history stages, and/or long-lived species.
- Documented or suspected (ideally unique) source(s) within the Shenandoah basin.
- Toxic effect enhanced by high (>9.0) ambient pH in surface waters. In contrast, most chemical contaminants have greater toxicity or availability at acidic pH levels.
- Documented in both groundwater and surface waters and river sediments within the basin

e.) Expand the Science Team by two or possibly three additional members to represent additional areas of needed expertise. These areas include: the chemistry of profile contaminants in natural waters and fish pathology/toxicology.

Tier II (moderate priority)

a.) Evaluate potential groundwater sources of contaminants that fit the Profile and begin to document the interaction between groundwater and surface water base flows in the mainstem and tributaries. Specifically, identify existing groundwater wells in the basin, especially those within the floodplain, and work with cooperating agencies to develop a contaminant monitoring program for selected groundwater sources.

b.) Document the temporal and spatial patterns of high (>9.0) pH events within the mainstem and tributaries. Specifically, expand the current network of *in-situ* pH monitors. Attempt to test the hypothesis that high pH events are directly related to periphyton production stimulated by excess nutrient availability.

c.) Use GIS technologies to explore broad geospatial patterns among fish kill events, land use, and likely sources of Profile contaminants. Develop and maintain an online and interactive geospatial data portal and related databases to support data and GIS analyses by members of the Task Force and Science Team. A draft version of this application is currently under development by VCU (see #6 below).

d.) Leverage other ecological research and environmental monitoring efforts and programs in the region, including DEQ's ProbMon and fish tissue programs, South Fork and South river mercury testing, CADDIS, INSTAR (VCU and DCR), and DuPont South River Ecological Study, to enhance data development and advance Task Force and Science Team goals. Coordinate all 2007 field research activities within the basin.

e.) Continue intensive weekly water quality monitoring by DEQ within the basin and possibly expand this program to include smaller tributaries within the basin. Monitor ammonia concentrations at several river and stream locations only during significant precipitation events to evaluate possible storm-driven pulses in ammonia concentrations.

f.) Explore the feasibility and design of mesocosm studies at the VDGIF Front Royal Hatchery. Studies would be planned for 2008.

6. Geospatial Data Portal

A draft version of a database designed to support an online and interactive geospatial data portal is currently being developed. The ACCESS database consists currently of information from 61 sources—primarily peer-reviewed journals, agency reports, Task Force members, and CADDIS—that are relevant to one or more of the prevailing theories regarding fish kills in the Shenandoah River. An additional 50 potential resources are currently under review for inclusion. The database is dynamic and will be updated regularly and refined as more information becomes available. Each entry in the database includes specific information on the source of the data, a citation, location information, contacts for more information (if available), a project description, results to date, and key words. Each entry is also categorized according by which of the prevailing theories on causes of the fish mortality in the Shenandoah it is relates to (e.g. contaminants, nutrients, physiological stress). The geospatial portal is expected to be completed in early summer of 2007.

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