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LETTERS

edited by Jennifer Sills

The Spread of Grapevine Trunk Disease

THE GRAPEVINE DIEBACK DISEASES, ALSO CALLED GRAPEVINE TRUNK diseases, are the consequences of a complex of fungi that was described as early as the end of the 20th century. They attack the perennial organs of a vine and ultimately lead to the death of the plant. Over the past decade, the frequency of symptoms due to these fungi has considerably increased worldwide. For example, cumulated disease incidence values estimated for Italian vineyards may reach up to 50% (1). The lack of resources to fight the diseases and favorable environmental conditions worsen the situation. Sodium arsenic is the only treatment that has a potential effect against dieback diseases, but it has been prohibited in some countries. Some vineyards that have never been treated with sodium arsenic now present an exponential development of symptoms.

Vines at risk. Symptoms of grapevine trunk disease.



Why are these symptoms emerging today? Are they due to changes in the vine behavior, in the climate, or in the microbial equilibrium, or are they due to undiagnosed pathogens? Despite all the studies on the fungi associated with the disease, on the host-pathogen interactions, and on the symptoms, the actual causes for their development are still elusive.

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Eutrophication: More Nitrogen Data Needed

WE AGREE WITH D. J. CONLEY *ET AL.* ("Controlling eutrophication: Nitrogen and phosphorus," Policy Forum, 20 February, p. 1014) that there are many compelling reasons for controlling agricultural and industrial sources of nitrogen. In many areas, nitrate and ammonium are now the main pollutants causing damage by acidification and base cation depletion in forests and freshwaters (1). In some areas, nitrate concentrations in drinking water have increased enough to exceed health standards (2). However, at this time, we cannot agree that

reducing nitrogen is essential for controlling eutrophication, because there are insufficient whole ecosystem-scale data to show that removing nitrogen will reduce eutrophication.

Phosphorus control alone has succeeded in reducing eutrophication in many lakes [reviewed by (3)] and in at least one low-salinity estuary (4). In contrast, not a single ecosystem-scale study in any aquatic system has shown that reducing inputs of nitrogen decreases eutrophication.

The authors state that reducing phosphorus inputs has not reduced eutrophication in some lakes and many estuaries. In most cases, the reason is high "internal loading" of phosphorus from anoxic sediments. High concentrations of phosphorus and anoxia in surface sediments are the result of decades of high phosphorus loading causing increased settling and decomposition of organic matter. However, long-term studies of lakes in Europe (5, 6) have shown that internal loading decreases slowly after external sources of phosphorus are controlled, so that ecosystems recover over a period of years to decades.

Many of the arguments put forward by Conley *et al.* are based on physiological or short-term indices of nitrogen limitation,

which we have found to be spurious in our long-term, whole-lake manipulations. Simply put, over time algal and bacterial communities change to include species that fix nitrogen when fixed nitrogen limits the growth of other species. Small but long-term inputs of nitrogen through fixation and subsequent return from sediments eventually correct nitrogen deficits in ecosystems (7, 8). The importance of these long-term, adaptive processes cannot be evaluated by short-term incubations or dissolved nutrient concentration ratios (9).

Conley *et al.* state that the nitrogen-fixing cyanobacteria that are capable of correcting ecosystem-scale nitrogen deficiencies in lakes are absent in saline estuaries. However, recent measurements of N₂/Ar ratios indicate that there is considerable N fixation by bacteria and phytobenthos in shallow, saline estuaries (10, 11). Rates of fixation are similar to those that we have observed in lakes (8), and we anticipate similar results in overcoming N deficiencies. In fact, in whole systems, N removal accomplished at great expense in wastewater treatment may be offset by N fixation in natural open systems (8).

Globally, reducing inputs of nitrogen from sewage as well as phosphorus would require

Letters to the Editor

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spending many billions of dollars. The costs of removing both nutrients may even discourage any treatment in developing countries, particularly in the current economic depression. We believe that before the additional expense of nitrogen removal from sewage is to be imposed on society, it should first be demonstrated at ecosystem scales to effectively reduce eutrophication. **D. W. SCHINDLER¹* AND R. E. HECKY²**

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Eutrophication: Focus on Phosphorus

THE POLICY FORUM BY D. J. CONLEY *ET AL.* ("Controlling eutrophication: Nitrogen and phosphorus," 20 February, p. 1014) advocates expensive and unnecessary nitrogen (N) control in lakes.

Many demonstrations of successful phosphorus (P)-only control in lakes are found in the literature (1). In the 1970s, P control was implemented in the Laurentian Great Lakes, an important North American freshwater source (2). Total P (TP) in Lake Ontario decreased to half the maximum in response to P management and by half again after zebra mussel invasion, reducing phytoplankton standing crop and shoreline nuisance blooms of *Cladophora*. The lower phytoplankton N demand alleviated nitrate shortages. Thus, Lake Ontario was a real-time experiment to validate P control as a means to manage eutrophication.

I take exception to Conley *et al.*'s prediction that P-only reduction strategies will fail in Lake Apopka. Sediments deposited since 1947 provide the basis for estimating whole-lake historic TP sedimentation (3). The sediment inventory shows average annual deposition of 0.367 g TP m⁻² year⁻¹ (1947 to 1996). Some TP, however, was in a form that is not readily recycled (4, 5). External loading averaged 0.55 g TP m⁻² year⁻¹ in the 1990s when water-column TP was 0.320 g TP m⁻² (6). The large TP sediment sink and short TP residence time in the water column indicate that sediment recycling is low (6). Therefore, strategies that control external P loading will control eutrophication over time.

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Eutrophication: Model Before Acting

IN A RECENT POLICY FORUM ("CONTROLLING eutrophication: Nitrogen and phosphorus," 20 February, p. 1014), D. J. Conley *et al.* made a controversial case for a dual nutrient-reduction strategy to address eutrophication in lakes, estuaries, and coastal areas.

We believe that all asserted beneficial effects must be robustly predictable to ensure that society actually gets something in return for this effort, given the high cost of nutrient reductions. For instance, Swedish nitrogen (N) reductions in a very ambitious abatement plan for the Baltic Sea may not be possible to fulfill unless a large part of Swedish agriculture is permanently shut down, according to recent calculations by the Swedish Department of Agriculture (1).

Unfortunately, there are no general, validated mass-balance models for nitrogen that have been tested for independent coastal systems and been demonstrated to yield good predictive power. Any N model can be tuned, using different calibration constant sets for different

systems, to give perfect descriptive power. However, such tuning may obscure the true aspects of a natural system (2). In addition, the effects of N abatement on many coastal areas have been quite disappointing (3).

There is one general dynamic phosphorus (P) model (thus far) that has yielded good predictions of phosphorus and chlorophyll in all Baltic Sea basins without basin-specific tuning and without taking N concentrations into account. The abatement strategy for the Baltic Sea should therefore focus on cost-effective P reductions, such as urban sewage treatment (2). Strategies designed for other estuaries, coastal areas, and lakes should also be based on methods with documented cross-systems predictive power. **ANDREAS C. BRYHN* AND LARS HÅKANSON**

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Eutrophication: Time to Adjust Expectations

D. J. CONLEY *ET AL.* ("CONTROLLING EUTROPHICATION: Nitrogen and phosphorus," Policy Forum, 20 February, p. 1014) advocate a shift in strategies to control eutrophication of aquatic systems. We agree that the best hope for success rests with strategies couched in a systems perspective and founded on an understanding of interactions among biogeochemical cycles.

Current efforts to control eutrophication focus on repairing past damage, with systems expected to return to a desired state after obvious stressors are reduced. One approach is to restrict nutrient inputs to waterbodies that are declared impaired by limiting total maximum daily loads. Although costly, managing anthropogenic loads of macronutrients represents an essential, sensible, and feasible strategy for controlling eutrophication and reversing its effects. Increased loads of nutrients drive, support, or enable eutrophication.

Reducing loads of macronutrients, including simultaneous reductions for nitrogen and

phosphorus, may not yield desired responses. Beyond lags due to “legacy loads,” systems may not return to undamaged states along desired trajectories if they have entered alternative stable states or baseline conditions have shifted (1–3). For example, ocean acidification may prevent corals from regaining ascendancy on reefs, and impacts from overfishing may cascade through trophic webs to create stable but undesirable assemblages of consumers and producers. In such cases, reversing eutrophication may require restoration of habitats, repair of trophic webs, or relatively drastic projects that remove accumulated effects and thus shift systems toward previous states. In some cases, our best efforts may not produce systems that are structurally identical to a previous, desired state, so we will have to settle for restoring dynamic functions that consistently yield desired services (1, 2).

Sustainable control of eutrophication in aquatic systems requires all stakeholders to acknowledge our inability to predict the exact trajectory followed by any particular ecosystem in response to management interventions, including reduced loads of nitrogen and phosphorus. Stakeholders can foster

success by embracing an adaptive approach supported by monitoring that evaluates alternative actions and endpoints, promotes continual learning, and fosters progressive improvement (4).

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Response

WE ARE GRATIFIED THAT OUR POLICY FORUM has stimulated numerous responses on effective strategies for controlling phosphorus (P) and nitrogen (N) to reduce eutrophication in freshwater and coastal marine ecosystems.

Rigid application of P control—the only paradigm proffered by Schindler and Hecky—has been increasingly called into question

even for lakes (1, 2) and was rejected long ago for estuarine and coastal waters for the reasons discussed in our Policy Forum. The paradigm depends on sufficient N_2 fixation by cyanobacteria to meet the demands of algal growth that could be supported by the available P. While this is not always reached even in lakes (1, 2), quantitatively significant N_2 fixation simply does not occur in the water columns of coastal ecosystems except, as we pointed out, under low-salinity conditions found in the more freshwater portions of estuaries and the Baltic Sea. The evidence cited by Schindler and Hecky for estuaries is for N_2 fixation in bottom sediments, which is seldom important in the N economy of estuarine ecosystems.

Although demonstration at the whole-ecosystem scale advocated by Schindler and Hecky is powerful, it is not usually possible to intentionally make whole-ecosystem experiments in marine systems. Nonetheless, there is substantial evidence of N limitation in coastal marine ecosystems at the whole-ecosystem and large-mesocosm scale (3). In fact, the example by Schindler and Hecky of P control alone succeeding in reducing eutroph-

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ication in a low-salinity estuary, the Stockholm Archipelago, is only half of the story. Phytoplankton chlorophyll levels further declined after the waste treatment facilities substantially removed N (4).

Schelske's basic points are similar to those of Schindler and Hecky, namely, that N_2 fixation can alleviate N shortages for phytoplankton and that there have been many demonstrations of successful P-only control in lakes, such as in some of the Laurentian Great Lakes. Unfortunately, such P-only control has not been universally effective. In fact, the very reference Schelske provides to support the success of P-only control noted that it is important to consider not only P but also N loading (5). Shallow hyper-eutrophic lakes such as Lake Apopka frequently have blooms of cyanobacteria that do not fix N_2 and have not responded to P-load reduction, probably because of the large internal recycling of P from sediments. Schelske's calculations consider only external P loading and ignore P remobilization from sediments.

We agree with Bryhn and Håkanson that P reductions are required for improvements to be observed in the Baltic Sea; however, we

differ in the need for N reductions. Their model is only for P because they believe that it is not possible to construct adequate models for nitrogen mass-balances, despite the fact that N models are common. Their model parameterization for P cycling is unusual—the boundary conditions are very different from other models used in the Baltic Sea, and their conclusions differ substantially as well (6–8), bringing into question their validity.

Jacoby and Frazer agree with us that both P and N controls should be considered, but stress that this might not yield desired responses because the damaged ecosystems may have shifted to alternative stable states. This issue fell beyond the scope of our short Policy Forum, but we have addressed it elsewhere (9). We certainly agree that this makes the exact recovery trajectory somewhat unpredictable and requires an adaptive approach, the first step of which is aggressive control of nutrient loads based on a rational and context-specific two-nutrient strategy.

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