Comment on "Zebra Mussel Destruction by a Lake Michigan Sponge: Populations, in Vivo ^{31}P Nuclear Magnetic Resonance, and Phospholipid Profiling"

SIR: We take issue with the paper by Early and Glonek (*1*) dealing with a remarkable biological interaction between native freshwater sponges and invading zebra mussels in the Great Lakes. The authors describe the effects of luxuriant overgrowth by the sponge *Eunapius fragilis* on zebra mussels and interpret this interaction as a form of predation on mussel tissues. They imply that their study is the first to identify this native species "exerting a significant impact [on zebra mussels] on a local level".

However, our study published in 1995 (*2*) showed that (i) sponge overgrowth (particularly by the species *E. fragilis*) on zebra mussels occurs frequently in Lake Erie and the St. Lawrence River, (ii) this overgrowth causes mussels to become emaciated and significantly increases population mortality, and (iii) sponge-induced mortality has little effect on zebra mussel densities beyond a local scale because of high mussel recruitment and environmental constraints on sponge growth. Early and Glonek have cited our study erroneously in reference to the effects of sponges *on native unionid mussel species*.

Regarding Increased Sponge Populations in Lake Michigan. In the same paper (*2*), we proposed mechanisms by which sponge growth may be enhanced by zebra mussel activity including mussel filtration, which (i) reduces suspended silt that fouls sponge pores; (ii) removes large phytoplankton, thereby shifting plankton populations toward smaller cell sizes that are processed more efficiently by suspension-feeding sponges; and (iii) increases transparency of the water column, thus allowing light to stimulate metabolic activity of the sponge's algal symbionts (*3*). Another beneficial mechanism may be sponge exploitation of bacteria associated with decomposing mussel feces. Thus, we hypothesized that sponge abundance will increase in the Great Lakes concomitant with zebra mussel colonization, creating a feedback cycle that could promote competition locally. Early and Glonek claim that the sponge population in Lake Michigan has indeed increased, based on their interpretation of numerical density. Unfortunately, this is a very poor indicator of abundance. A large sponge tends to shrink and fragment at the end of a growing season, resulting in higher numbers of colonies but lower biomass. Conversely, sponge colonies of the same genetic strain may fuse and grow confluently (*3*, *4*). Therefore, the only meaningful measures of sponge growth are biomass and percent aerial coverage. Sponges may very well be increasing in Lake Michigan; alternatively, divers may be noticing more of them as a result of the unprecedented transparency of the water caused by zebra mussel filtration.

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Regarding "Sponge Predation" on Zebra Mussels. Early and Glonek contend that sponges are feeding either directly on mussel tissues (through chemical digestion) or indirectly on bacteria associated with sponge-enhanced decomposition of mussel tissues. While the authors maintain a realistic uncertainty about this interaction, they draw a dubious comparison with the predatory activity of certain deep-sea sponges that use adhesive filaments to capture crustaceans (*5*). The precise mechanism of nutrient transport to these deep-sea sponges remains undocumented and may involve exploitation of bacteria associated with decomposing crustacean tissue; however, the comparison certainly ends there as the sponge employs a specialized mode of prey capture. In the freshwater sponge/zebra mussel interaction, no adaptations to prey capture exist—nor would we expect any given that these two organisms evolved independently of each other. The mussels are killed by either anoxia or starvation, and nutrient transfer probably occurs by a free bacteria loop. It is very unlikely that this process involves lysosomal enzymes secreted by the freshwater sponge. Sponges are not known to produce extracellular digestive exoenzymes, although some marine species produce histolytic secretions and secondary metabolites that play a role in substrate defense (*6*, *7*). Similarly, sponges have been known to kill corals (presumably by suffocation) and benefit from their degradation (*8*). Again, an exoenzyme use has not been demonstrated in this example. If the freshwater sponge/zebra mussel interaction indeed involves a free bacteria loop as nutrient transfer, this might be more analogous to a situation in which one animal inadvertently kills another and feeds on the flora grown on its corpse. In such a case, the "prey" tissues are not directly utilized, so it could hardly be considered predation. If, however, the degradation products of mussel tissue are directly caught and ingested by the sponge, then this might be more accurately described as a form of saprophagous feeding behavior.

As we indicated previously (*2*), we consider the sponge/zebra mussel interaction to be an unequivocal example of interference competition (*sensu* Russ (*9*)). Similar examples are rare in freshwater systems but quite common in marine intertidal communities dominated by primary-space occupiers such as mussels, barnacles, bryozoans, and sponges (*7*−*11*). Marine sponges commonly overgrow bivalves and other sedentary animals but do not necessarily kill them (*10*, *11*). Overgrowth appears to be an evolved response by sponges to outcompete their smaller, slower growing neighbors for space (*10*). In freshwater sponges, this response was previously inconspicuous because of the absence of dominant competitors but has manifested itself in the presence of invading zebra mussel populations in the Great Lakes.

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