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IDEAS AND PERSPECTIVES

Parasites in the food web: linking amphibian malformations and aquatic eutrophication

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Abstract

Emerging diseases are an ever-growing affliction of both humans and wildlife. By exploring recent increases in amphibian malformations (e.g. extra or missing limbs), we illustrate the importance of food web theory and community ecology for understanding and controlling emerging infections. Evidence points to a native parasite, *Ribeiroia ondatrae*, as the primary culprit of these malformations, but reasons for the increase have remained conjectural. We suggest that the increase is a consequence of complex changes to aquatic food webs resulting from anthropogenic disturbance. Our results implicate cultural eutrophication as a driver of elevated parasitic infection: (1) eutrophication causes a predator-mediated shift in snail species composition toward *Planorbella* spp., (2) *Planorbella* are the exclusive first intermediate hosts of *R. ondatrae* and (3) *Ribeiroia* infection is a strong predictor of amphibian malformation levels. Our study illustrates how the effects of anthropogenic disturbance on epidemic disease can be mediated through direct and indirect changes in food web structure.

Keywords

Amphibian malformations, deformities, emerging disease, eutrophication, food webs, parasites, *Ribeiroia*.

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INTRODUCTION

A central tenet of community ecology is that species dynamics are inextricably linked within a nexus of interactions among species and their abiotic environment (Elton 1927). As environmental factors change through space or time, particularly owing to anthropogenic influence, interactions among species also change. Recent studies have shown how a variety of anthropogenic factors, including increases in temperature, carbon dioxide, acidity and nutrient deposition, can significantly alter the interactions, diversity and very structure of communities (Petchey *et al.* 1999; Reich *et al.* 2001; Zavaleta *et al.* 2003).

Beyond its more conspicuous effects on competitive or predatory relationships, environmental change also affects cryptic interactions among species such as those between hosts and parasites. Although infrequently considered in food web dynamics, parasites and pathogens play an important role in trophic interactions (Marcogliese & Cone 1997). Virtually all species serve as hosts for one or more parasites, and many parasites have dramatic impacts on host abundance, behaviour, development, fecundity and population cycling (Scott 1988; Hudson *et al.* 1998; Moore 2002). Through their influence on biological interactions such as host competitive ability (e.g. Kiesecker & Blaustein 1999) and predator avoidance behaviour (e.g. Lafferty & Morris 1996), parasites may also be important determinants of community structure and composition (Minchella & Scott 1991). Taken together, such studies suggest that omission of parasites and pathogens from food web studies may exclude a key endogenous driver, leading to potentially serious misunderstandings of species interactions.

Changing the ecological context within which parasites and their hosts interact can have dramatic, oftentimes unanticipated consequences (e.g. Lafferty & Kuris 1999; Ostfeld & Holt 2004). For example, over-fishing of molluscivorous fish in Lake Malawi led to increases in the habitat range and abundance of snail hosts for blood flukes (*Schistosoma haematobium*), which in turn caused increases in infection prevalence and haematoria (bloody urine) among school children (Stauffer *et al.* 1997). Likewise, declines in vertebrate biodiversity can elevate the prevalence of Lyme disease spirocheates in white-footed mice populations through loss of the 'dilution effect' (Ostfeld & Keesing 2000), thereby increasing disease risk in humans.

In this paper, we focus on an emerging phenomenon that has created considerable public and scientific interest – that of malformed amphibians in North America. We present data from wetland systems across western and central USA to put forth the hypothesis that the recent increase in amphibian malformations results from a complex set of interactions among a parasite that causes malformations, the parasite's intermediate host (aquatic snails) and anthropogenic changes to the food web within which the snails are embedded (cultural eutrophication).

PARASITE INFECTION AND AMPHIBIAN MALFORMATIONS

Although malformed amphibians have been observed sporadically for nearly 300 years (e.g. Ouellet 2000), reports of deformed amphibians increased dramatically in many parts of North America over the past decade (Fig. 1; Souder 2000) (although this may indicate an increase in surveillance rather than in actual malformations, see Ward & Lafferty 2004). Contemporary observations were unusual in their (1) severity, often involving frogs with missing, extra, or misshapen limbs, (2) frequency, sometimes affecting >50% of the amphibians in a wetland and (3) wide distribution, involving more than 60 species from 46 states in the USA and parts of Canada (reviewed by Blaustein & Johnson 2003). Although heightened surveillance undoubtedly accounts for some observations, historical studies indicate that, at least in some areas, the increase is real (Hoppe 2000; Johnson *et al.* 2003).

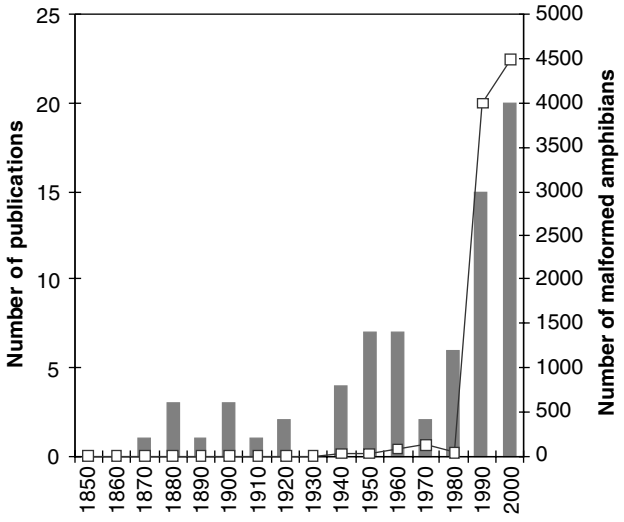

Figure 1 Numbers of published articles (bars) and malformed amphibians (line) observed in wild amphibian populations within the USA and Canada (1850–2003). Literature prior to 2000 follows Ouellet (2000).

Recent evidence implicates infection by a parasitic flatworm (*Ribeiroia ondatrae*) as a widespread cause of limb malformations in amphibians (particularly supernumerary limbs) (e.g. Sessions & Ruth 1990; Johnson *et al.* 1999). *Ribeiroia* is a digenetic trematode with a complex life cycle involving snails in the family Planorbidae (Pulmonata), amphibians and water birds. The parasite reproduces asexually within snail hosts, producing free-swimming cercariae that actively encyst around developing limbs of larval amphibians, sometimes leading to severe errors in limb development. For *Ribeiroia* to complete its life cycle, an infected amphibian must be consumed by a suitable definitive host such as a water bird, allowing for sexual reproduction and egg production (Johnson *et al.* 2004). In laboratory experiments, exposure of larval amphibians to realistic numbers of *Ribeiroia* cercariae caused high levels of mortality and limb malformations. These malformations were similar to those observed at field sites, including missing, extra and deformed limbs (Johnson *et al.* 1999; Stopper *et al.* 2002; Schotthoefer *et al.* 2003). Similarly, field studies have revealed links between *Ribeiroia* infection and the presence and frequency of amphibian malformations in wetlands within the western USA (Johnson *et al.* 2002), the Midwest (Lannoo *et al.* 2003; Johnson, unpublished data) and the Northeast (Kiesecker 2002; Stopper *et al.* 2002).

LINKING MALFORMATIONS AND EUTROPHICATION

Eutrophication is a pervasive and growing problem in marine and freshwater ecosystems worldwide (Smith 1998; World Water Council 2003). It is caused by elevated levels of nutrients (particularly phosphorus), often owing to runoff sources associated with agriculture, cattle or urbanization (National Research Council 1992; Carpenter *et al.* 1998). Attempts to reverse or ameliorate the effects of eutrophication are hindered by internal cycling of phosphorus within lakes, the buildup of phosphorus in upland soils and nonpoint sources of nutrient runoff (Carpenter *et al.* 1998; Bennett *et al.* 2001). This is particularly true in the pond systems in which *Ribeiroia* and malformations frequently occur, many of which are constructed to support cattle or irrigation (Johnson *et al.* 2003; Lannoo *et al.* 2003).

We suggest that the rise in amphibian malformations may have resulted through complex changes within wetland food webs. Specifically, as anthropogenic factors (e.g. agricultural fertilizers, cattle grazing) lead to increased nutrient loading in freshwater ecosystems (particularly ponds), the resulting acceleration of eutrophication shifts the community composition of aquatic snails from small species to larger planorbid species that serve as intermediate hosts for *Ribeiroia* through a complex interaction of top-down and bottom-up food web processes. Eutrophication is generally

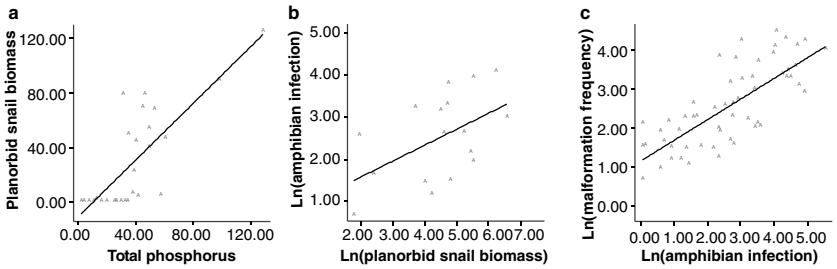

Figure 2 (a) Relationship between total phosphorus (μg L^{-1}) and planorbid snail biomass (g m^{-2}) among 27 Michigan ponds ($R^2 = 0.616$, $P < 0.0005$). (b) Relationship between planorbid snail biomass and *Ribeiroia* infection (mean abundance) in amphibians ($R^2 = 0.262$; $P < 0.05$). (c) Relationship between *Ribeiroia* infection (ln[mean abundance]) in 11 species of amphibians and malformation frequency (ln[%malformed]) among 56 populations in western and central USA (CA, OR, WA, MT, MN, WI) ($R^2 = 0.581$; $P < 0.0005$). Amphibian species include *Hyla regilla*, *Bufo americanus*, *Rana luteiventris*, *R. pretiosa*, *R. catesbeiana*, *R. cascadae*, *R. aurora*, *R. pipiens*, *R. clamitans*, *R. septentrionalis* and *R. sylvatica*.

expected to decrease the impact of infection on hosts (e.g. Lafferty & Holt 2003). In the case of trematodes and snails, this will decrease the mortality of infected snails thereby increasing the period over which snails release parasites and overall infection prevalence. Collectively, such changes will lead to an increase in amphibian infection intensity and malformations. Our hypothesis is based on observations and experiments from wetland ecosystems across western and central North America:

1 Among 27 ponds in Michigan, we found that increased total phosphorus levels were associated with increases in the biomass and proportional representation of *Planorbella* snails (Fig. 2a; methods are described in Chase 2003a). This result was not due to an overall increase among all snail species; rather, the proportion of the total herbivore community composed of *Planorbella* snails increased at higher phosphorus levels (R^2=0.396; $P < 0.0005$). Recent evidence points to an interaction between nutrients and the snails' predators in causing this shift (Chase 2003a,b). At low nutrient levels small species of snails (e.g. *Physella*, *Gyralus*, *Lymnaea*) are favoured due to their lower resource requirements. With increasing nutrient levels that result from cultural eutrophication, however, these smaller herbivores become disadvantaged as predator densities increase, allowing larger species of snails (e.g. *Planorbella*) to increase in density (Chase 2003a,b). Increases in periphyton production also tend to increase planorbid snail growth, survivorship and the number of generations produced per season (Chase 2003a).

2 *Planorbella* snails are the exclusive first intermediate hosts for the trematode *R. ondatrae* and are expected to limit the parasite's distribution and local abundance (Johnson *et al.* 2002). *Planorbella* snails frequently occur in smaller ponds, many of which are used to support cattle or irrigation. In surveys of 16 malformation hotspots in western and central USA, we found that higher densities of *Planorbella* snails were associated with increased levels of *Ribeiroia* infection in amphibians (Fig. 2b). Among these systems, the density of planorbid snails was positively and significantly related to total phosphorus concentrations.

3 Among 56 amphibian populations from California, Oregon, Washington, Minnesota and Wisconsin, we found that *Ribeiroia* infection levels were a strong predictor of malformation frequency for 11 species of frogs and toads (Fig. 2c, methods described in Johnson *et al.* 2002). Our results are further supported by previous field data and experiments with *Ribeiroia* and several species of frogs and toads (Johnson *et al.* 1999; Kiesecker 2002; Stopper *et al.* 2002; Schotthoefer *et al.* 2003). Factors that increase the density of planorbid snails (e.g. nutrient inputs) are therefore likely to increase the intensity of parasite infection and the frequency of malformations in amphibians (Johnson *et al.* 1999, 2002).

MULTIPLE TIME SCALES: CLIMATE AND LAND USE PATTERNS

Eutrophication is often considered a slow process, requiring decades of nutrient loading, whereas malformations seemed to 'appear' quite suddenly in the mid-1990s (Souder 2000). However, eutrophication can be accelerated when large pulses of nutrients enter a wetland, as frequently occurs during heavy storms. Flooding events are an important force in transporting excess nutrients (e.g. from agricultural fields) from terrestrial to aquatic habitats. In fact, up to 90% of the phosphorus exported to wetlands each year occurs during large storms (Pionke *et al.* 2000). In Minnesota, where the issue of amphibian malformations was catalysed in the general public, the 1990s were marked by several of the most severe floods in the last 100 years following a prolonged drought in the late 1980s (e.g. the floods of 1993 and 1997).

Considering that croplands and livestock are a billion dollar industry in southern Minnesota, with over 170 million kilograms of phosphate fertilizers applied annually, these floods undoubtedly delivered large quantities of phosphorus and nitrogen to surrounding wetlands. Our hypothesis would predict increases in planorbid snail biomass, parasite production and malformation frequency as a consequence of heightened productivity.

CAVEATS

Despite our supposition that the apparent rise in amphibian malformations is the result of a series of direct and indirect food web interactions instigated by anthropogenic changes in the number of eutrophied wetland ecosystems, some caveats exist.

1 We assume that increased nutrient loading to wetlands will increase the amount of resources (i.e. periphyton) available to snails. In some circumstances, nutrient loading can increase the biomass of phytoplankton, thereby decreasing the biomass of the periphyton through shading (e.g. Vadeboncoeur *et al.* 2002). This effect may be particularly important in deep lake ecosystems, whereas in shallower wetland ecosystems that are primary breeding grounds for amphibians, nutrient loading seems to have a positive effect on periphytic resources (Chase 2003a).
2 Although the relationships that we have shown in Fig. 2 are linear, the actual responses to eutrophication may be nonlinear. There may be critical thresholds above which these positive relationships are dampened or reversed. In Fig. 2a, for example, there is evidence suggestive of a threshold in the response of planorbids to phosphorus levels. Moreover, considering that *Ribeiroia* infection is known to castrate infected snails and elevate their mortality, extreme levels of infection are likely to cause a reduction or crash in snail populations (Pointier 1989; Johnson *et al.* 2004). More empirical work is needed to identify these thresholds and evaluate their significance. We have also ignored the effects of eutrophication on *Ribeiroia* definitive hosts, and there is some evidence to suggest fish-eating bird activity will increase around eutrophied systems (Wisniewski 1958; Esch 1971).
3 We have not considered the role of secondary stressors that could interact with parasite infection to influence malformations levels (e.g. pesticides and ultraviolet radiation). Although evidence is mounting that *Ribeiroia* is often the primary culprit in causing amphibian deformities (reviewed in Blaustein & Johnson 2003), other factors may independently or interactively influence amphibian deformities. A more general exploration into the role of anthropogenic changes on amphibian deformities will need to examine not only the role of increased levels of parasite infection owing to shifts in food web interactions, but also the effects of chemical and UV pollution and the interactions among these stressors.

IMPLICATIONS AND CONCLUSIONS

An unprecedented number of diseases have emerged or re-emerged in recent years, frequently owing to changes in the ecological interactions among a pathogen, its hosts and the environment in which they co-occur (Daszak *et al.* 2000). In well-studied examples, it is clear that changes in ecosystems often drive disease emergence, whether directly or indirectly (Dobson & Foufopoulos 2001). In most cases, however, little is known about the drivers of pathogen emergence or the consequences of parasitism for host populations and ecosystem processes. In their synthesis of the Grand Challenges in Environmental Sciences, the National Research Council (2001) listed infectious disease as one of the eight most pressing environmental issues, advocating a 'systems-level' approach to understanding disease emergence. Correspondingly, in aquatic ecosystems, eutrophication is considered one of the greatest threats to global freshwater resources (Smith 1998; World Water Council 2003). Considering that human and wildlife populations depend on freshwater for survival and that many diseases are transmitted via waterborne stages, interactions between pathogens and aquatic eutrophication are an important frontier for understanding current – and forecasting future – disease epidemics.

We examined the interface between these issues, exploring the consequences of eutrophication on a host–parasite system and the food web within which it occurs. Although malformed amphibians rarely survive to sexual maturity, the long-term effects of parasite-induced malformations on amphibian population dynamics are unclear. Increased levels of *Ribeiroia* infection are expected to exacerbate mortality in the larval and metamorphic stages of many amphibian species. We suspect, however, that the effect of parasite-induced malformations and mortality on amphibian populations will depend critically on the demographic contribution of recent metamorphs to population growth rates (e.g. Vonesh & De la Cruz 2002). For example, if adults are long-lived relative to juveniles, then a population is unlikely to be limited by mortality in the juvenile stages (Caswell 2001). Alternatively, if adults are short-lived, juvenile mortality is likely to have a larger effect on population growth rates (Caswell 2001). Thus, amphibians with short-lived adults, such as treefrogs in the family Hylidae, might be more susceptible to population-level effects of parasite-induced malformations than amphibians with long-lived adults, such as frog species from the family Ranidae. If our hypothesis linking eutrophication and

increased amphibian malformations is correct, the continued inputs of excess nutrients into aquatic systems could cause shifts in the abundance and composition of adult amphibian communities. These results emphasize the importance of incorporating food web theory into efforts aimed at understanding and mediating emerging diseases.

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