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Conclusions Drawn from the Malformity and Disease Session, Midwest Declining Amphibians Conference, 1998

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While the general problem of amphibian declines is well known, no issue recently has engendered as much concern by the public as amphibian malformities (Ouellet et al. 1996, Tietge 1996, Gray 1998, Helgen et al. 1998a and 1998b, D. Johnson 1998, T. Johnson 1998, Meteyer and Converse 1998). As a result, Gary Casper, Chris Phillips and I decided to assemble this symposium. The session was open to anyone who wished to participate. Nineteen papers were presented (there was one cancellation) and a panel discussion followed. I will organize this summary of, and conclusions drawn from, our symposium into two sections: 1) the ecological importance and 2) proximal causes of the amphibian malformities that now command our attention.

How important are malformities?

I will begin tangentially. Among organisms, awareness proceeds along a spectrum from sensation to perception to conception, and one can divide neurobiologists into two camps: those that believe that animals other than humans can form concepts and those who do not. An old joke in neuroscience is that you can tell these people apart by proxy, by simply noting whether or not they own dogs. Likewise, in herpetology, you can divide researchers into two camps: those who believe that malformities are an important component of amphibian declines, and those who do not. You can discern these people by simply asking them whether or not they have ever experienced a wetland with a large number of malformed amphibians. In fact, the observation that many field herpetologists have never encountered more than a few malformed amphibians over their years of fieldwork offers an important clue in resolving the cause of malformities.

There are several other reasons why the malformity problem may not seem important to many field herpetologists. For one, wetlands characterized by amphibian malformities are frequently isolated and surrounded by wetlands that do not produce malformities. Malformities thus become a local (i.e., unimportant) phenomenon. Another reason is that wetlands with large numbers of amphibian malformities tend to be restricted geographically to the Upper Midwest, Quebec, and New England. Again, regional issues are deemed to be less important than global issues. Yet another reason is that affected animals tend to be from common species that are geographically widespread. There have been, as yet, no species extinctions that can be ascribed to malformities.

While we differ on our opinions of the importance of malformities, there can be no doubt that being malformed affects the fitness of an individual amphibian. In populations where malformed animals are observed, malformities are always more common among newly-metamorphosed animals, and almost never observed in older breeding

animals. This observation suggests that malformed animals do not survive their first winter and almost never breed.

Having a large number of malformed animals also affects the health of a population (see Green 1997 for a discussion of using populations to quantify amphibian declines). Both Hoppe (1998) in Minnesota and Ouellet et al. (1996) in Quebec have observed reduced numbers of breeding animals over time in populations with malformities. Clearly (assuming no immigration), a population that does not reproduce cannot hope to persist beyond the lifespans of its youngest individuals.

The two cases of amphibian declines most widely cited are the presumed extinctions of the golden toad in the Monte Verde rainforest of Central America and the Australian gastric brooding frog (e.g., Phillips 1994). Both of these species were restricted in geographic range and may have each consisted of only one population. If, rather than using species extinction as the criterion for importance of decline, we use the unconventional approach of counting the numbers of populations affected (or simply use a body count) malformities have resulted in a greater loss of amphibians than losses reported in the most famous examples of declining amphibians.

What are the causes of malformities?

I know of no more contentious issue in the field of amphibian declines than determining the cause(s) of amphibian malformities. I will preface these remarks by noting that we are first members of a culture, and we are secondly scientists, and that it is therefore easy to slip from the logic of the scientific method to the less formal debating styles used by society in general. For example, when challenged on their data on causes of malformities, one colleague reverted to citing the university where they were trained and the person who trained them. Another colleague responded by citing the number of animals and microscopic sections examined. Unfortunately, most reporters covering the malformity issue (e.g., Kaiser 1997) have been trained as journalists and therefore do not understand the power or the mechanics of the scientific method. These folks can be persuaded by such responses, which then reach the public. The utterings of scientists, by themselves, do not constitute science. A related problem is that information disseminated through meetings, press conferences, and through conversations with the press is not peer reviewed.

For the record, the scientific method typically consists of observations, hypothesis formation, and hypothesis testing. Hypotheses are never proven to be correct, they can only be shown to be incorrect by data that do not bear out the predictions of the hypothesis (remember T. H. Huxley's classic quip "A beautiful hypothesis destroyed by one ugly little fact."). In science, the basic incontrovert-

ible unit is the fact; facts trump hypotheses. Hypotheses that are proven incorrect do not discredit their inventor, for they narrow the field and help point the way to the truth. In contrast, American society as a whole emphasizes winners and losers (perhaps because of our infatuation with sports) and believes that winners can be made through sheer force of will, independent of tangibles.

Three other important points must be made about the mechanism of scientific inquiry. First, peer review is a major component of modern science (see above). Second, the most parsimonious answer is provisionally considered to be the correct answer. Third, we must make a distinction between what is generally true and what is specifically true.

In the debate over amphibian malformities the emphasis has been not on facts, but rather on hypotheses. Some researchers have tended to defend their favorite hypothesis against facts, and aspects of this debate have more closely resembled a theological discussion (belief system versus belief system) than a scientific investigation. It is time to emphasize facts and to move the debate on causes of malformities back into the realm of science.

Within the past five years malformed amphibians have been noted at a large number of sites in the northern United States and eastern Canada. According to data collected by Hoppe (1998) the rates and the types of malformities currently observed differ from historical observations.

Three causes have been proposed for the outbreak of amphibian malformities: trematode parasite infestations (Sessions and Ruth 1990, Sessions 1996, Christiansen and Feltman 1998); xenobiotic chemicals (Ankley et al. 1998, Blumberg et al. 1998, Gardiner and Hoppe 1998, Helgen et al., 1998a and 1998b, Sparling 1998); and UV-B exposure (Ankley et al., 1998). Each of these causes has been determined to produce amphibian malformities in laboratory studies. The answer to the general question do trematode parasite infestations, xenobiotic chemicals, and UV-B radiation each cause amphibian malformities is, as far as we know, yes. The next question is more specific: do these causes produce the types and ratios of malformities that have been recently observed in natural populations across the northern United States? Addressing this question will occupy the remainder of this contribution.

Using the scientific method we can treat each of the three potential causes for amphibian malformities as hypotheses (i.e., the hypothesis is that trematode parasite infestations are causing the recent outbreaks in amphibian malformities, etc.). We can then make predictions about the type of malformities found in nature based on the findings of each hypothesis (predicted malformities). Finally, we can use the animals collected in nature (observed malformities) to test each hypothesis.

Predicted Malformities

The parasite hypothesis predicts three features: 1) limb and pelvic malformities only; 2) a characteristic type of limb malformity predominates (multiple limbs or portions of limbs arranged in a mirror image fashion); and 3) a correlation, at least in older tadpoles and newly-metamorphosed animals, between parasite infestation and the presence of a malformity (Sessions and Ruth, 1990, Sessions 1996, Christiansen and Feltman 1998).

The UV-B radiation hypothesis predicts two features: 1) a characteristic type of limb malformity consisting of bilateral (symmetrical) limb taperings (limbs appear to be missing but in fact have all or most of their components; distal elements attenuate as they form at progressively smaller sizes); and 2) high mortality suggests that systemic effects involving other organ systems may also be occurring (Ankley et al. 1998).

The xenobiotic chemical hypothesis predicts a wide suite of mal-

formities involving limbs, a variety of organ systems, and/or a number of biochemical/physiological processes (Muneoka 1996, Gardiner and Hoppe 1998, Blumberg et al. 1998, Sparling 1998).

Observed Malformities

In 1997, I received through the Minnesota Pollution Control Agency 64 malformed and 38 normal ranids (*Rana pipiens* and *Rana clamitans*) from 17 Minnesota sites. Size and unresorbed opercular flap absorption scar indicated that most animals were young of the year (i.e., recently metamorphosed from tadpoles). Of the 64 malformed frogs, 42 had missing hindlimbs or parts of hindlimbs (all but one asymmetrical), 8 had orbital or eye malformities, 5 had missing forelimbs or parts of forelimbs, 3 had hind limb duplications, 3 had cranial other than orbital malformities, 2 had pigment malformities only, and 1 had an abdominal hydrocele. The types of malformities as well as their ratios are similar to reported malformities from Minnesota in 1998 (Helgen 1998a and 1998b), Wisconsin (DuBois 1996; Gilbertson et al. 1998) and Quebec (Quellet et al. 1996), New England (Converse et al. 1998), and animals in my possession from Indiana.

The observed cranial, visceral, pigment, and behavioral abnormalities, and the low number of limb duplications appear to exclude the parasite hypothesis alone as the causal agent for these malformities. The asymmetry associated with the limb malformities (only one symmetrical malformity) appears to exclude UV-B radiation alone as the causal agent. Only xenobiotic chemicals have been reported to produce the full range of malformities that we now observe in natural populations of anuran amphibians (Gardiner and Hoppe 1998). In fact, one class of malformity, so-called bony triangles, have been only reported to be induced by xenobiotics, specifically, retinoid-like compounds (Blumberg et al. 1998, Gardiner and Hoppe 1998, Sparling 1998).

Does this conclusion that xenobiotic chemicals explain the range of malformities observed in nature mean that trematode parasites and UV-B do not cause amphibian malformities? No. This conclusion simply means that with the data collected up to this point in time (1999), parasites and UV-B radiation cannot be said to cause the majority of the malformities that are being observed at any particular site. Therefore, these hypotheses are not parsimonious. Does this conclusion mean that with more data, parasites and UV-B radiation will not be shown to cause the malformities now observed in nature? No. New data will result in a reconsideration of causes. Science is self correcting, and conclusions, therefore, are provisional. Does this conclusion mean that parasites and UV-B radiation will not be shown to cause malformities at sites other than the ones that are being studied? No. In fact, because the currently-studied sites consistently produce malformities year after year, it would be instructive to revisit and observe Sessions and Ruth's (1990) Santa Cruz County, California wetland.

In arguing for the parasite hypothesis, several researchers have concluded that this hypothesis in combination with amputations due to failed predation attempts can explain the observed range of malformities. This view has been widely perpetuated both among herpetologists and the public at large. Yet from my perspective there are several problems with this story that must be addressed (one role as U.S. Coordinator of the DAPTF is to ensure that we are proceeding along reasonable and rational lines of inquiry in pursuing causes of amphibian declines). If we view the above scenario (parasites cause extra limbs, failed predation attempts cause missing limbs) as a hypothesis, the following thirteen observations would appear to negate it.

1. The parasite/predation hypothesis is not the most parsimonious hypothesis about causes of malformities to be proposed. It in-

- okes two causes (parasites plus amputations), when one cause (xenobiotic chemicals) is sufficient.
2. Data to date do not support a relationship between trematode infestations and malformities (Meteyer 1998; but see Christiansen and Feltman 1998 for a new Iowa site).
 3. Neither parasitic infestations nor failed predation attempts explain some of the cranial, or any of the visceral and pigment malformities.
 4. Surveys conducted in Minnesota in 1997 (Helgen et al. 1998a and 1998b) show that sites with missing limb malformities are also sites with multiple limb malformities. Why should high levels of predation be associated with high levels of parasitism? One would reasonably expect that predation pressures would be independent of snail numbers/parasite infestations. Furthermore, one would expect that if failed predation caused missing limbs, that more field herpetologists would be observing large numbers of animals with missing limbs. There are no predators on amphibians restricted solely to the areas of North America where amphibian malformities are highest.
 5. Missing limbs occur in the absence of predators. Malformed *Rana clamitans* were observed in 1997 at a newly constructed wetland east of Indianapolis. Most malformities were missing limbs, yet for the first year there were no aquatic predators (fishes, turtles, or invertebrates) at what was essentially a sterile basin.
 6. Developing amphibian limbs cannot be easily amputated by being pulled from the body (as would occur if limbs were grabbed by animals with mouthparts designed for swallowing prey whole, as in most non-avian vertebrate amphibian predators) but instead must be severed by a shearing masticatory apparatus. What kind of predators have this type of shearing masticatory apparatus (and attack from below, an approach necessary to gain access to developing hind limbs)? Aquatic turtles are candidates, but what size turtle population would it take to produce malformity rates of upwards of 70% in a frog population? Would all those turtles not be noticed?
 7. Why would predators selectively choose hind limbs? Failed predation attempts on tadpoles by aquatic vertebrates typically result in lost tails. Failed predation by birds on tadpoles in drying wetlands frequently produces 'v' shaped notches, corresponding to beak morphology, in the tadpole's dorsal fin.
 8. If predation is causal, why are there rarely signs of wound repair in newly metamorphosed animals? Among developing limbs, the older the limb, the longer the limb, and the likelihood of a limb being grabbed by a predator must be proportional to its length. It therefore seems that the closer a tadpole gets to metamorphosis, the more likely it is to lose a limb, and so the more likely it would be to show obvious signs of wounding.
 9. Radiographs of missing limbs nearly always show abnormal spongiform-like bone malformities proximal to the sight of the absence. This morphology is inconsistent with known inflammatory responses, for example following violent amputations.
 10. Pigment abnormalities, especially dorsally, are often present on the proximal portions of missing limbs. Why should amputation convert, for example, a barred pigment pattern to a mottled pigment pattern some distance away from the allegedly traumatized area in the absence of scarring?
 11. The argument for multiple limbs actually negates the argument for amputations. If trematode parasites produce multiple limbs compromising the locomotory ability of the host and therefore making it more susceptible to predation (Sessions and Ruth 1990, Sessions 1996), why would predators not then focus on the multiple limbed animals rather than focusing on normal animals (to produce the missing limbs)? If sites with multiple limb malformities are associated with the presence of predators, and if predators take multi-limbed animals disproportionately (Sessions and Ruth 1990, Sessions 1996), would not the number of multi-limbed animals be reduced, or the number of limbs they possess be reduced? And if this occurred, would not the perception of the magnitude of the malformed frog problem be diminished?
 12. Some animals missing whole limbs are also missing portions of the associated pelvic or pectoral girdles. It is unlikely in my view that this degree of trauma is consistent with life (i.e., what is the probability of an animal surviving the trauma associated with violently losing a hind leg and a hemipelvis)? And under such circumstances would you not expect scarring?
 13. Some newly metamorphosed animals exhibit complete loss of single forelimbs. In tadpoles, forelimbs develop under opercular flaps. Why would predators specifically focus predation attempts on a structure they cannot see, and how could they take it without including other structures or producing scarring?
- Finally, I'll conclude this discussion on predation by noting that missing limbs in older animals (the first thing a predator can grab on an escaping adult frog is a hind limb) and missing limbs associated with wounding or scarring are undoubtedly due to failed predation attempts. Yet animals that fit these descriptions rarely occur at malformity sites.
- An aside. The comments above are by no means to be taken as a criticism of Sessions and Ruth (1990). This paper is a wonderful example of how laboratory studies can confirm causal phenomena underlying field observations. Many of us are incapable of doing science at this level. But the quality of Sessions and Ruth's (1990) work does not necessarily translate into universal application. Indeed, will anyone truly be surprised if the cause of an isolated occurrence of malformities in California does not apply to a more generalized problem across the Upper Midwest, Quebec, and New England? And if parasites are not the primary cause of amphibian malformities in the Midwest and east, Sessions and Ruth (1990) cannot be faulted.
- Given that xenobiotic chemicals offer the best explanation for amphibian malformities and that xenobiotic chemicals have also been implicated in amphibian declines (Fellers 1996, Britson and Threlkeld 1998, Hirsch and Temple 1998, Huang et al. 1998, Jofre and Karasov 1998, Reeder et al. 1998, Rosenshield and Jofre 1998, Zaga and Little 1998), the distinction herpetologists make between malformities and declines may be artificial; either way, the fitness of affected animals is near zero. The distinction between causes of malformities and declines may simply be one of temporal scale and proximal mechanism. Whereas direct effects of exposure to xenobiotics tend to produce death by a more-or-less immediate failure of biochemical/metabolic systems, malformities tend to produce death through longer term ecological mechanisms (desiccation, freezing, predation).
- In summary, parasitic infections, UV-B radiation, and xenobiotic chemicals have all been determined to induce amphibian malformities in the laboratory. Comparing the type of malformities from animals captured in nature to malformities induced by exposure to the causes listed above, only xenobiotic chemicals offer a parsimonious explanation for the field malformities. Stated another way, certain types of malformities from animals collected in nature must cause scientists to reject the parasite and the UV-B radiation hypotheses as stand-alone possibilities. The parasite hypothesis combined with the scenario of amputation due to failed predation attempts requires an improbable suite of behavioral/ecological scenarios and coincidences. At this juncture the xenobiotic hypothesis must be considered as the most probable cause of Midwestern amphibian malformities. This conclusion may change if further experimentation on par-

asites and UV-B radiation produces a wider variety of malformities, and if field studies establish clear cause and effect relationships.

Addendum: Since this symposium was held, there have been several advances in our understanding of natural and unnatural causes of amphibian malformations. For an update, as well as some behind the scenes insight, I refer the reader to William Souder's recently published book *A Plague of Frogs: The Horrifying True Story* (Hyperion Press, 2000).

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