Water link to frog deformities strengthened

In 1995, middle school students on a visit to a Minnesota wetland were startled to find frogs missing limbs or sprouting three and four hind legs. Significant numbers of deformed frogs soon turned up at other sites in the Midwest, as well as in the northeastern, southern, and western United States and in Canada.

Since then, numerous explanations for the deformities have sprouted, including parasites, pesticides, and ultraviolet radiation from ozone loss. Federal and Minnesota state researchers last week announced results from continuing studies that point back to the students' initial suspicion: Something in the water can induce deformities.

"We believe that we have shown there is something operating or active in the water," says Mark Gernes of the Minnesota Pollution Control Agency in St. Paul. The next step is "to try in essence to pull that water apart."

Results of chemical analyses of the water samples are expected within 2 months. Researchers are looking for specific pesticides, metals, and chlorinated contaminants, while also considering all the chemicals present.

Public interest and health concerns prompted the announcement of the early results, which have not yet been published or released in full. In what officials called a precautionary measure, the agency began providing bottled water to Minnesota residents who live near the sites.

Water from several wetland sites had a pronounced effect in laboratory tests, says Gernes. Water samples were sent to toxicologists at the National Institute of Environmental Health Sciences (NIEHS) in Research Triangle Park, N.C., who oversaw laboratory tests using African clawed frog (*Xenopus*) embryos. Researchers grew the embryos in different concentrations of the water samples for 96 hours, then recorded the numbers that died or developed abnormally.

Undiluted pond water from two of the Minnesota sites caused abnormalities in 100 percent of the embryos, James Burkhart of NIEHS told SCIENCE NEWS. Pond sediments, groundwater, and tap water from nearby private wells also affected the embryos' development.

Water from one site without deformed frogs did not harm the embryos. When water from another of the wetlands considered to be unaffected resulted in some abnormal embryos, researchers returned to the site and found deformities in about 7 percent of the frogs collected there.

"There's complete concordance with an increased incidence of abnormal frogs and the [frog embryo] assays," says Burkhart.

It's common to find a small number of malformed frogs at any site—about 1 percent—the researchers say. The worrisome wetland sites have had much larger numbers, 10 percent or more. Such sites have been reported in 54 of Minnesota's 87 counties. In some hot spots, among species such as the mink frog, which spends 2 years developing in water, 75 percent of the frogs are deformed, says Gernes.

The widespread emergence of high numbers of deformed frogs (SN: 7/12/97, p. 31) is probably contributing to the general decline of some frog populations, says David M. Gardiner of the University of California, Irvine. He held a workshop last week to brief developmental biologists about the phenomenon.

Because frogs live both in water and on land, they are generally considered to be accurate indicators of environmental health. The embryos' direct exposure to water during development makes them particularly vulnerable to defect-causing agents, says Gardiner. Although human embryos are protected from many environmental influences as they develop, the hormonal pathways controlling limb development in frogs and people can be affected by the same agents, he says.

*This leopard frog (*Rana pipiens*) with an extra leg was collected from a Minnesota wetland.*

"If it does it to frogs, it'll do it to people. There's no question about that."

Although water is now the main focus of research, there are still many questions about other environmental influences on frogs in the wild. "To say that any one thing is the cause of all we've seen isn't viable," says Burkhart.—*C. Mlot*

Plant estrogens may ward off breast cancer

Imagine a dinner that starts with hummus, a chickpea spread, slathered on bite-size slices of rye bread. Then move on to a tomato-soybean casserole laced with hot sauce. The side dish consists of shredded carrots dressed with lemon juice, cumin, and mint. Cap the meal with blueberry pie.

Sound good? Not to aspiring breast cancers.

Each course contains a significant helping of phytoestrogens, a family of plant-based compounds that weakly mimic the animal kingdom's primary female sex hormone. Women who eat foods rich in these compounds appear to have a significantly lower risk of breast cancer, a new Australian study finds.

David Ingram and his colleagues at the Queen Elizabeth II Medical Centre in Perth recruited 144 volunteers—age 30 to 84—newly diagnosed with breast cancer and an equal number of demographically matched women without the cancer. They sampled urine excreted over a 72-hour period for lignans and isoflavonoids, two types of plant estrogens.

Whole-grain foods, berries, and certain root crops are rich sources of lignans. Legumes, especially soy beans, contain isoflavonoids. The researchers relied on the concentrations of these substances in urine to gauge how much phytoestrogen-rich food a woman was typically eating. At least five earlier studies by other researchers had found some evidence that such compounds might reduce breast cancer risk.

In the Oct. 4 LANCET, Ingram's team reports that "for all phytoestrogens, [cancerfree] women had higher median excretion rates." Indeed, the healthy women typically excreted 50 percent more of the lignan enterolactone than did the cancer patients.

As a group, the 25 percent of women who excreted the least enterolactone proved three times as likely to have cancer as the 25 percent excreting the most. For the isoflavonoid equol, women excreting the least were four times as likely to have cancer as women shedding the most.

"In 1982, we found exactly the same thing"—far lower urinary excretion of enterolactone and equol in breast cancer patients, notes Herman Adlercreutz of the University of Helsinki in Finland. The difference, he says, is that analyses back then could not quantify the low concentrations of these compounds seen in some women today. Moreover, his study followed only 10 women, though it tracked them for a year, not just 72 hours.

While many studies have indicated that diet plays a role in cancer prevention, not one "has shown a degree of risk reduction similar to that found for some phytoestrogens in this study," Ingram's team argues.

Certainly, this "intriguing" new study "is consistent with the idea that diets high in phytoestrogens decrease breast cancer risk," observes Stephen Barnes of the University of Alabama at Birmingham. However, it's not clear which, if any, of the phytoestrogens inhibited cancer, he and two colleagues note in an accompanying commentary.

Explains Barnes, "they might just be markers for something in the diet that wasn't measured." —*J. Raloff*