event highly suspect but, in light of the recent unexpected findings that there are blood-borne primordial germ cells in certain mammals (*2*), we became intrigued that such a happenstance might occur in frogs, notwithstanding the seemingly restricted opportunities for the germ cells to enter the bloodstream. It was a source of keen satisfaction for us to realize a method of testing the idea, although we lay no claim to having provided a definitive answer. Although the hypothesis may seem improbable to Wallace, we would remind him that many advances in science have issued from investigators' pursuing far-flung or unorthodox ideas. The results of Wallace's study (*3*) can not be fully appraised without additional experimentation, but it is our earnest hope that his work and ours will arouse interest and stimulate further inquiry.

E. PETER VOLPE
SHERILL CURTIS
Department of Biology,
Tulane University,
New Orleans, Louisiana

References

1. E. P. Volpe and S. Curtis, *Science* **160**, 328 (1968).
2. S. Ohno, J. M. Trujillo, C. Stenius, L. C. Christian, R. L. Teplitz, *Cytogenetics* **1**, 258 (1962); K. Benirschke and L. E. Brownhill, *ibid.* **2**, 331 (1963).
3. H. Wallace, *J. Embryol. Exp. Morphol.* **10**, 212 (1962).

13 June 1968

DDT Residues and Bermuda Petrels

Wurster and Wingate (*1*) state that Stickel (*2*) found DDT residues of 10.6 ppm in bald eagle eggs, that others have reported DDT residues of 120 to 227 ppm in herring gull eggs, and that both species are declining. From this they reason that the suggestion is "that susceptibility varies widely between species," and they conclude that residues of 6.44 ppm in the petrel eggs may account for the fluctuating decline in the reproductive success of the small, protected, and increasing population of the once-considered-extinct Bermuda petrel.

With regard to Stickel's information (*2*), he states that the residues in the eagle eggs "provide little basis for suspecting that DDT in the eggs prevented hatching." Stickel further notes that, while an "occasional" eagle may obtain enough pesticide to place it "in hazard," "most eagles that die in the United States today die of causes other than pesticide poisoning."

Herring gulls and ring-billed gulls have undergone a population explosion to such an extent that the U.S. Fish and Wildlife Service (*3*) has considered measures to reduce their numbers. Dr. W. H. Drury (*4*), director of research, Massachusetts Audubon Society, has urged the immediate burying of garbage at dumps to stop the population explosion of the gulls.

Even if the fluctuations in the ability to hatch of the eggs of the few pairs of Bermuda petrels observed by Wurster and Wingate are not within normal limits for that little-known species, Stickel's comments and gull populations provide little basis for suggesting that DDT residues of 6.4 ppm, which is less than the amount permitted in foods, are of significance.

LOUIS A. MCLEAN
525 Happ Road,
Northfield, Illinois 60093

References and Notes

1. C. F. Wurster, Jr., and D. B. Wingate, *Science* **159**, 979 (1968).
2. L. F. Stickel *et al.*, in *Trans. North American Wildlife Natural Resources Conf. 31st* (1966), pp. 190–200; cited in (*1*).
3. The ring-billed gull has been undergoing a population explosion in the upper Great Lakes and is currently the subject of an intensive and continuing study by J. P. Ludwig of the University of Michigan. J. J. Hickey, J. A. Keith, F. B. Coon, "An Exploration of Pesticides in a Lake Michigan Echo System," *Final Original Report on Contract 14-16-0008-659 to Board of Sport Fisheries and Wildlife* (Univ. of Wisconsin Agric. Exp. Sta., Madison, 6 March 1965); also Hartford (Connecticut) *Courant*, 13 May 1964.
4. W. H. Drury, quoted in *Newsday* (Garden City, Long Island, N.Y.), 27 May 1963.

18 April 1968

We did not say that herring gulls were declining, but that they had "very low reproductive success" on Lake Michigan. The two factors must be kept separate. Gull populations are exploding because increased pollution with human wastes expands their niche, a factor that more than offsets the effects of insecticides. An explosion in scavenging, garbage-eating gulls, accompanied by declines in predatory birds, is a good example of ecosystem degradation whereby a diverse avifauna is being replaced by large numbers of fewer species.

Stickel's paper was written before the importance of hepatic enzyme induction by chlorinated hydrocarbon insecticides was appreciated. These enzymes are relatively nonspecific and cause hydroxylation of steroids, including estrogen (*1*). Estrogen mediates calcium metabolism in birds, and hormonal balance affects reproductive success and population size (*2*). The sudden appearance of calcium-deficient eggshells (*3*), associated with declining reproduction and population size among carnivorous birds on two continents, can be explained by the coincidental introduction of DDT into the world environment during the late 1940's (*4*).

It was recently reported that DDT has estrogenic activity in rats, and it may suppress gonadotropic hormone secretion (*5*)—a distressing thought now that much of the world's biota are contaminated with DDT residues.

Hepatic enzyme induction occurred in rats fed as little as 1 ppm of DDT in the diet, and was associated with the storage of 10 ppm of DDE or DDT in fat (*6*). Since humans average 12 ppm in their fat (*7*), present tolerances in foods may be obsolete. We wish that environmental scientists could share McLean's complacence that current residues in petrels, people, and other organisms are without significance.

CHARLES F. WURSTER, JR.
Department of Biological Sciences,
State University of New York,
Stony Brook 11790
DAVID B. WINGATE
Department of Agriculture and
Fisheries, Hamilton, Bermuda

References

1. D. B. Peakall, *Nature* **216**, 505 (1967); A. H. Conney, *Pharmacol. Rev.* **19**, 317 (1967); D. Kupfer, *Residue Rev.* **19**, 11 (1967).
2. K. Simkiss, *Biol. Rev.* **36**, 321 (1961); J. J. Christian and D. E. Davis, *Science* **146**, 1550 (1964); E. O. Höhn, *Ibis* **109**, 445 (1967).
3. D. A. Ratcliffe, *Nature* **215**, 208 (1967); J. J. Hickey and D. W. Anderson, personal communication.
4. C. F. Wurster, First Rochester Conference on Toxicity, University of Rochester, 4–6 June 1968.
5. R. M. Welch, W. Levin, A. H. Conney, *ibid.*; D. J. Jefferies, *Ibis* **109**, 266 (1967).
6. G. Gerboth and U. Schwabe, *Arch. Exp. Pathol. Pharmakol.* **246**, 469 (1964); U. Schwabe, *Arzneimittel-Forsch.* **14**, 1265 (1964); F. K. Kinoshita, J. P. Frawley, K. P. DuBois, *Toxicol. Appl. Pharmacol.* **9**, 505 (1966).
7. G. E. Quinby *et al.*, *J. Amer. Med. Ass.* **191**, 175 (1965).

13 June 1968