have been lose to deep sediments, transported out of the area by ocean currents, concentrated in other trophic layers, or metabolized. Brown pelican productivity is still too low for population stability, and continued monitoring and indefinite protection of these colonies will be necessary. Nonetheless, these data are encouraging; we believe they illustrate a significant response by a wild population to distant and largely unrelated antipollution measures.

DANIEL W. ANDERSON
U.S. Fish and Wildlife Service,
Post Office Box C, Davis, California 95616
JOSEPH R. JEHL, JR.
Natural History Museum,
Post Office Box 1390,
San Diego, California 92112
ROBERT W. RISEBROUGH
Bodega Marine Laboratory, University of California, Bodega Bay 94923
LEON A. WOODS, JR.
California Department of Fish and Game,
987 Jedsmith Drive, Sacramento 95819
LAWRENCE R. DEWEESE
WILLIAM G. EDGECOMB
U.S. Fish and Wildlife Service,
Denver Wildlife Research Center,
Building 16, Federal Center,
Denver, Colorado 80225

References and Notes

1. R. W. Schreiber and R. L. DeLong, *Audubon Field Notes* **23**, 57 (1969); J. R. Jehl, Jr., *Condor* **75**, 69 (1973).
2. D. W. Anderson and I. T. Anderson, in preparation. A population decline of brown pelicans off California began in the late 1950's.
3. Extensive surveys have shown that the only major brown pelican breeding colonies north of Isla San Martín, 440 km southeast of Los Angeles, are those at Anacapa Island (occasionally including nearby Santa Cruz Island), about 110 km west of Los Angeles, and at Isla Coronado Norte, about 200 km southeast of Los Angeles. Public access to Los Coronados is restricted by the Mexican government, and the U.S. National Park Service has restricted access to the Anacapa colony since 1970.
4. K. F. Mais, *Calif. Dep. Fish Game Fish. Bull. 162* (1975).
5. J. D. Spratt, *Calif. Dep. Fish Game Mar. Res. Tech. Rep. 1* (1972).
6. During banding at Anacapa from 1972 to 1974, we examined stomach contents regurgitated by young pelicans; the material consisted almost exclusively of anchovies. Our observations of feeding adults before and during the breeding season also indicated a heavy reliance on anchovies.
7. Population studies in the Gulf of California, where eggshell thinning is slight (3 percent; $N = 157$ eggs), indicate that increased production in years of good food supplies results primarily from more adults nesting. In 1972, when anchovy populations were moderate, we estimated that the pelicans on the California coast represented at least 950 pairs old enough to nest, yet only 261 nests were attempted on Anacapa.
8. C. J. Henny, *An Analysis of the Population Dynamics of Selected Avian Species: With Special Reference to Changes during the Modern Pesticide Era* (Government Printing Office, Washington, D.C., 1972), pp. 41–46.
9. D. W. Anderson and J. J. Hickey, *Wilson Bull.* **82**, 14 (1970); *Proc. Int. Ornithol. Congr.* **15**, 514 (1972).
10. L. J. Blus, R. G. Heath, C. D. Gish, A. A. Belisle, R. M. Prouty, *BioScience* **21**, 1213 (1971); L. J. Blus, C. D. Gish, A. A. Belisle, R. M. Prouty, *Nature (Lond.)* **235**, 376 (1972); L. J. Blus, A. A. Belisle, R. M. Prouty, *Pest. Monit. J.* **7**, 181 (1974).
11. R. W. Schreiber and R. W. Risebrough, *Wilson Bull.* **84**, 119 (1972).
12. Names of chemical compounds referred to in the text: DDT (*p,p'*-DDT), 1,1,1-trichloro-2,2-bis(*p*-chlorophenyl)ethane; TDE (*p,p'*-TDE), 1,1-dichloro-2,2-bis(*p*-chlorophenyl)ethane; and DDE (*p,p'*-DDE), 1,1,1-trichloro-2,2-bis(*p*-chlorophenyl)ethylene.
13. B. J. LeBoef and M. L. Bonnell, *Nature (Lond.)* **234**, 108 (1971); R. L. DeLong, W. G. Gilmartin, J. G. Simpson, *Science* **181**, 1168 (1973); F. Gress, R. W. Risebrough, D. W. Anderson, L. F. Kiff, J. R. Jehl, Jr., *Wilson Bull.* **85**, 197 (1973).
14. R. W. Risebrough, *Proc. Berkeley Symp. Math. Statist. Prob.* **6**, 443 (1972).
15. J. S. MacGregor, *NOAA Fish. Bull.* **72**, 275 (1974).
16. R. Burnett, *Science* **174**, 606 (1971); J. L. Cox, *ibid.* **170**, 71 (1970).
17. W. Hom, R. W. Risebrough, A. Soutar, D. R. Young, *ibid.* **184**, 1197 (1974).
18. T. T. Schmidt, R. W. Risebrough, F. Gress, *Bull. Environ. Contam. Toxicol.* **6**, 235 (1971).
19. F. D. Dryden, *Calif. Coop. Oceanic Fish. Invest. Rep.* **16**, 44 (1972); C. W. Carry and J. A. Redner, Progress Report, County Sanitation Districts of Los Angeles County (1970); J. A. Redner and K. Payne, *ibid.* (1971).
20. Review by A. S. Cooke, *Environ. Pollut.* **4**, 85 (1973); D. B. Peakall, J. L. Lincer, R. W. Risebrough, J. B. Pritchard, W. B. Kinter, *Comp. Gen. Pharmacol.* **4**, 305 (1973).
21. R. W. Risebrough, D. W. Anderson, R. W. Schreiber, in preparation.
22. J. G. Edwards, statement to U.S. Congress Committee on Agriculture (18 March 1971); W. Hazeltine, *Nature (Lond.)* **239**, 410 (1972).
23. L. J. Blus, C. D. Gish, A. A. Belisle, R. M. Prouty, *Nature (Lond.)* **240**, 164 (1972); I. C. T. Nisbet, *ibid.* **242**, 341 (1973).
24. Analyses for organochlorine residues were conducted by the Pesticides Investigations Laboratory, California Department of Fish and Game, for fish samples; by the Bodega Marine Laboratory for 1969 and 1972 egg samples; and by the Denver Wildlife Research Center for 1973–74 egg samples. Methods used by these three laboratories are described, respectively, by W. T. Castole and L. A. Woods, Jr. [*Calif. Fish Game* **58**, 198 (1972)]; R. W. Risebrough, G. L. Florant, D. D. Berger, *Can. Field-Nat.* **84**, 247 (1970); and M. A. Haegele, R. K. Tucker, R. H. Hudson, *Bull. Environ. Contam. Toxicol.* **11**, 5 (1974).
25. Wilcoxon two-sample test; R. R. Sokal and F. J. Rohlf, *Biometry: The Principles and Practice of Statistics in Biological Research* (Freeman, San Francisco, 1969), pp. 391–395.
26. M. A. Haegele and R. H. Hudson, *Arch. Environ. Contam. Toxicol.* **2**, 356 (1974).
27. J. H. Enderson and D. D. Berger, *BioScience* **20**, 355 (1970).
28. F. Gress, *Calif. Dep. Fish Game Wildl. Manage. Br. Rep. 70-6* (1970); and personal communication.
29. T. G. LaMont, G. E. Bagley, W. L. Reichel, *Bull. Environ. Contam. Toxicol.* **5**, 231 (1970); J. O. Keith (personal communication) provided additional data.
30. We are grateful to W. H. Ehorn, H. H. Hoover, H. W. Hunt, F. Jacot, H. R. Leach, and F. S. Todd for their help. The National Park Service and Sea-World (San Diego) provided additional transportation. R. W. R.'s part in this study was supported by NSF grants GB-11649 and GB-36593.

18 April 1975

Reduced Incidence of Spontaneous Tumors: Another Statistical Analysis

Lacour *et al.* (*1*) report a reduced incidence of spontaneous mammary tumors in C3H/He mice treated with polyadenylate-polyuridylate. They gave only a very crude statistical analysis of their data, and a more careful appraisal may be more enlightening.

The mice were observed for 380 days, with all survivors being killed at this time. Table 1 shows their data immediately before day 380 when the survivors were killed. The appropriate statistical analysis for ascertaining a difference in the incidence of tumors in the two groups up to this point is to calculate the mammary tumor rate, correcting for the intercurrent deaths; that is, deaths from causes other than mammary tumor. This cannot be done properly with the data as they are without making certain assumptions: (i) that on the average the animals dying of other causes were observed for half the observation period, and (ii) that the mammary tumors occurred at a constant rate during the observation period.

On these assumptions, the corrected mammary tumor rates may be calculated by dividing the total tumors by (the total animals observed minus one-half the number of intercurrent deaths). In this case, the rate for controls is $55/[127 - 0.5(29)] = 49$ percent; for treated mice it is $30/[83 - 0.5(31)] = 44$ percent, an obviously not significant difference.

The proper statistical methodology to use in analyzing such an experiment without making these assumptions requires knowledge of the actual times of intercurrent deaths and times of diagnosis of mammary tumors and is fully described by Peto (*2*). The major difference between the treated and control group was, as the authors note, in those mice still alive on day 380 (Table 2: 58 percent versus 23 percent; $P < .025$).

One may thus tentatively draw the conclusion that treatment appears to prevent tumors in a proportion of animals but not delay the appearance of the tumor in those mice it fails to protect completely. An

Table 1. Mammary tumors and total deaths observed up to day 380.

Group	Mammary tumors	Other deaths	Survivors	Totals
Control	55	29	43	127
Treated	30	31	22	83

Table 2. Mammary tumors discovered at autopsy of survivors.

Group	With mammary tumors	Without tumor	Total
Control	25	18	43
Treated	5	17	22

analysis of their complete detailed data by rigorous methods (*2*) may be able to show whether the data truly support this.

J. CASAGRANDE
M. C. PIKE
Departments of Community Medicine and
Pathology, School of Medicine,
University of Southern California,
Los Angeles 90033

References and Notes

1. F. Lacour, G. Delage, C. Chianale, *Science* **187**, 256 (1975).
2. R. Peto, *Br. J. Cancer* **29**, 101 (1974).
3. Supported by NIH contract PH43-NCI-68-1030 within the Virus Cancer Program and NIH grant PO ICA 17054-01.

3 February 1975

Immunity in Cancer

Lacour *et al.* (*1*) demonstrated "a significant reduction in the occurrence of mammary tumors" in C3H/He female mice given injections of poly(A)·poly(U). Practically identical results were obtained in the same strain of mice by Riley (*2*) with living LDH-virus. Seibert *et al.* (*3*) also showed similar significant immune response against spontaneous C3H tumors with a heat-killed vaccine from a bacterium (C3H ♀ 27 Brtu) isolated from a C3H spontaneous mammary tumor and shown to be filterable through 0.1-μm Seitz filters. At the peak period of tumor reduction in all three sets of experiments there was an increment of survival of about 20 percent treated mice over control untreated mice.

The necessity, even though tedious, of continuing such experiments until death of mice, not done by Lacour *et al.* (*1*), was demonstrated by both Riley (*2*) and Seibert *et al.* (*3*), who found 90 to 100 percent tumor incidence in both control and treated mice after the 380-day period, probably due to loss of cellular immunity with age. However, since delay of spontaneous tumor development as affected by all of these agents is most desirable in middle age, such agents may be of immediate practical value.

The advantages of a sterile autogenous or homologous vaccine, as used by Seibert *et al.* (*3*), are evident from the problems with viral vaccines recently discussed by Kolata (*4*). Caution is indeed timely. Agents, whether chemicals, including that used by Lacour *et al.* (*1*), or tumor extracts, viral vaccines, and the like must, throughout their entire preparation, be made so as to avoid contamination with bacteria, alive or dead, even in their filterable forms, since bacterial products are potent immunogens (*5*). Tumors frequently used for extracts do harbor viable bacteria (*5*) and their products, and whether filterable forms or phages are also always present is not yet known.

FLORENCE B. SEIBERT
Research Laboratory,
Veterans Administration Center,
Bay Pines, Florida 33504

References

1. F. Lacour, G. Delage, C. Chianale, *Science* **187**, 256 (1975).
2. V. Riley, *ibid.* **153**, 1657 (1966).
3. F. B. Seibert, J. A. Baker, J. Dierking, R. Abadel, R. L. Davis, *J. Intern. Res. Commun.* **1**, 53 (1973).
4. G. B. Kolata, *Science* **187**, 522 (1975).
5. F. B. Seibert, F. Yeomans, J. Baker, R. L. Davis, I. R. Diller, *Ann. N.Y. Acad. Sci* **34**, 504 (1972).

28 February 1975

Dates for the Middle Stone Age of East Africa: A Discussion

Two new K-Ar dates from Ethiopia (*1*) augment a small, but growing, body of data implying that the beginnings of the African "middle Paleolithic" and of the associated "late Pleistocene" fauna are at least as early as 200,000 years ago. This recent acquisition is also an important one. It invites reexamination of relationships between the middle Paleolithic and late Pleistocene of Africa and presumably comparable successions in other parts of the world. We wish to comment on the authors' treatment of this aspect of their data.

In South Africa, as in Europe (*2*), late Pleistocene is commonly equated to Last Glaciation (Weichsel) plus Last Interglacial (Eem). Through this equation, the authors suggest that the new dates may also provide new minimum ages for the beginning of the Last Interglacial (Eem) in northwest Europe, as well as in South Africa. This extension, however, introduces "conflict with a series of K-Ar dates for Laacher See Volcanics (West Germany), which are incorporated in Rhine terraces . . . [four of which] place the period between late and early Saale (Riss) at about 145,000 years ago . . . ." Further, "the most serious conflict . . . [is with] dates obtained by analyses of ^{230}Th and ^{234}U in shells from Atlantic and Mediterranean beaches and raised coral reefs regarded as of Eem age . . . [which] suggested a time range between 80,000 and 140,000 years ago."

The conflicts envisioned by Wendorf *et al.* (*1*) are not introduced by radiometric ages and their relationships to local stratigraphies (Fig. 1). Rather, they arise from an unwarranted use of the equation late Pleistocene = Last Glacial + Last Interglacial = Middle Stone Age in widely scattered areas. They require explication.

The Eem beds, in the type area, record a marine transgression directly overlying Saale till. In northwestern Europe, these and correlative deposits do represent the *last* interglacial. They have also been taken, arbitrarily, as the base of the late Pleistocene in that area. Their absolute age is not directly determinable. Extrapolation from ^{14}C dates of younger sediments (*3*) yields a minimum age of 75,000 years. On the other hand, the K-Ar age of 145,000 years for Laacher See Volcanics dates materials recognizable as detritus in the younger Middle Terrace of the Rhine (*4*) which downstream "at the Saale drift border, includes glacial-lake rhythmites and is overlain by Saale till" (*5*). Thus, both Saale (including Warthe) glaciation and Eem interglacial may be younger than 145,000 but older than 75,000 years.

As pointed out by Richmond (*6*), the implications of the Laacher See dates are consistent with a series of K-Ar dates from Yellowstone Park, where Bull Lake glaciation postdates a flow 150,000 years old and antedates a flow 70,000 years old. Additionally, an intra-Bull Lake interval of deglaciation may be 105,000 years old.

The ^{230}Th/^{234}U ages of molluscan shell from Mediterranean and Moroccan beaches do not suggest a time range for the Last Interglacial of 80,000 to 140,000 years. Rather, two intervals of (interglacial?) high sea level are indicated (*7*). The younger, Neotyrrhenian, yielded ages between 75,000 and 95,000 years. It is separated by strong regression (glacial?) from the older, Eutyrrhenian, which yielded ages between 115,000 and 220,000 years. The ^{230}Th/^{234}U ages of molluscan shell are ininherently unreliable (*8*), but ^{230}Th/^{234}U ages of coral from the Salentine peninsula confirm at least the age range of the Eutyrrhenian (120,000 to 220,000 years) (*9*).

Dated Eutyrrhenian deposits on Mallorca can be interpreted as belonging to two separate episodes of high sea level, separated by regression about 160,000 years ago (*10*). The same inference could be drawn from stratigraphic relations of Ravagnese and Cafari, Calabria [dates included in (*8*)] and near Cerveteri, Roma (*11*). A break in the series of dated reef-terraces in New Guinea (*12*) may record the same interval.

The classic littoral deposits of the Grotte du Prince (*13*) have traditionally been regarded as last interglacial (Riss-Würm) and late Pleistocene. They are older than