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9 July 1976

Volume 193, No. 4248

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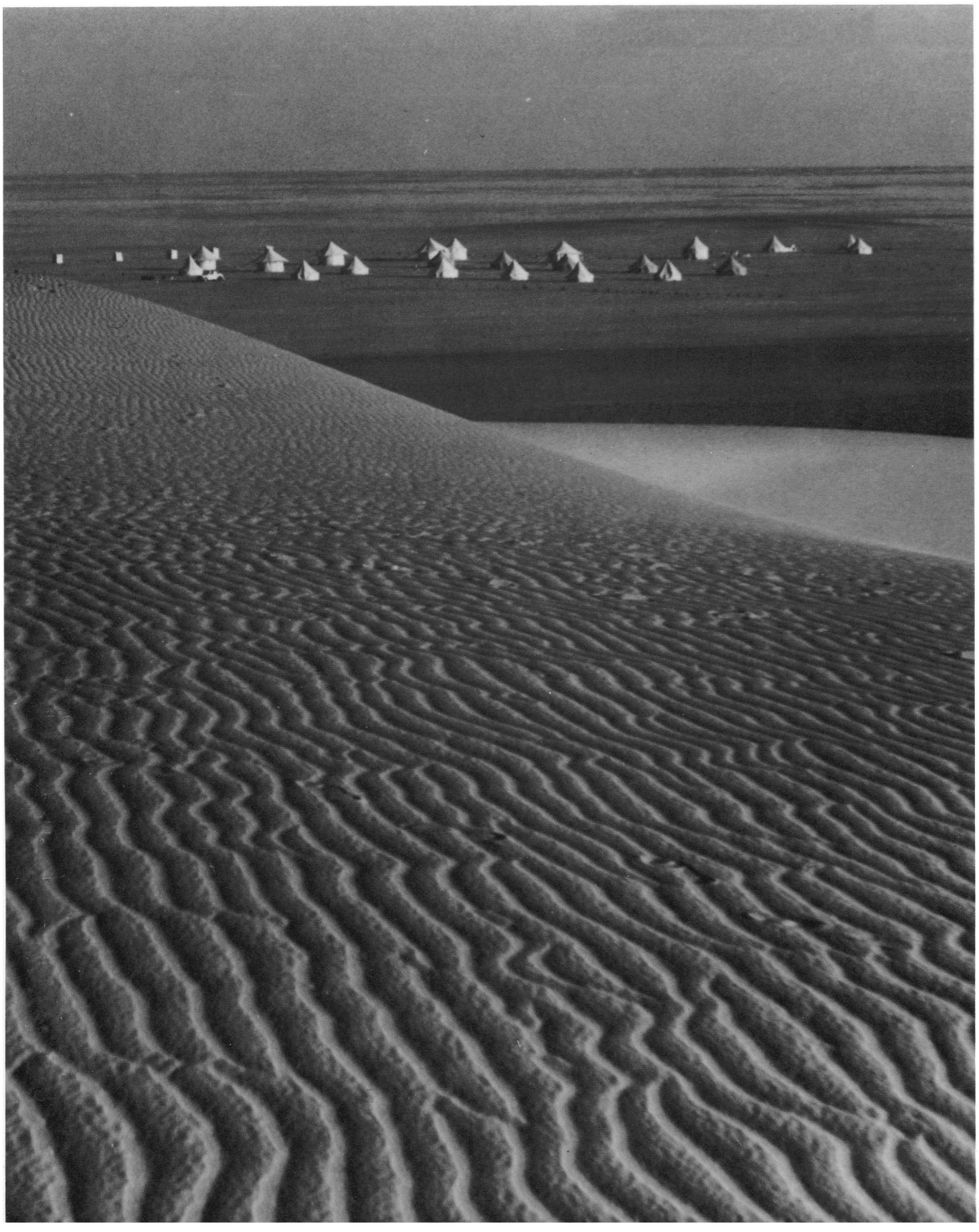

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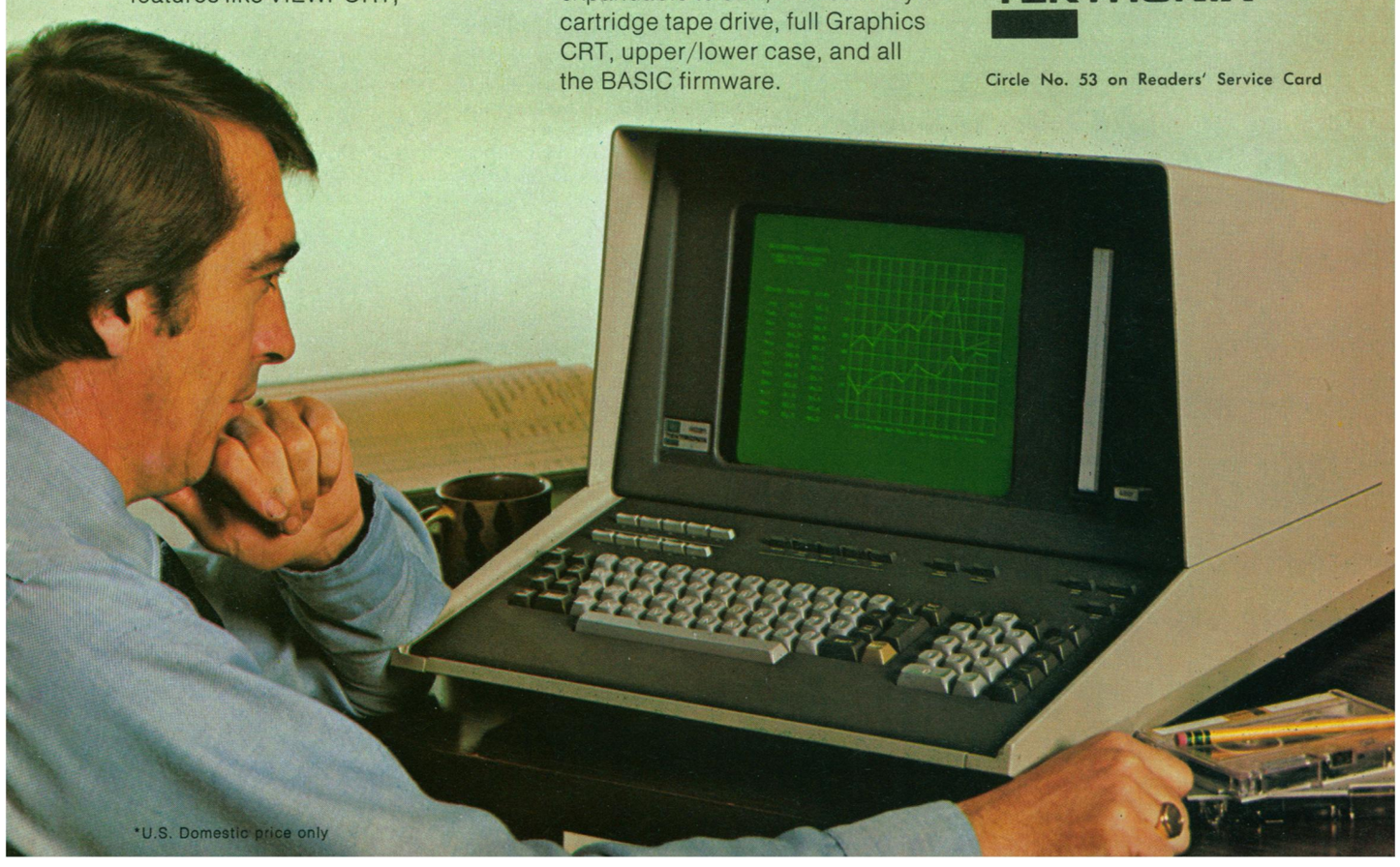

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COVER

Camp at archeological site Nabta Playa, Western Desert of Egypt. See page 103. [Fred Wendorf *et al.*, Southern Methodist University, Dallas, Texas]

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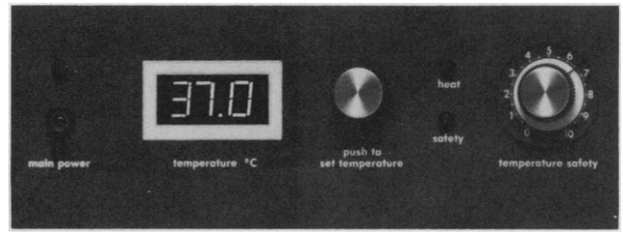

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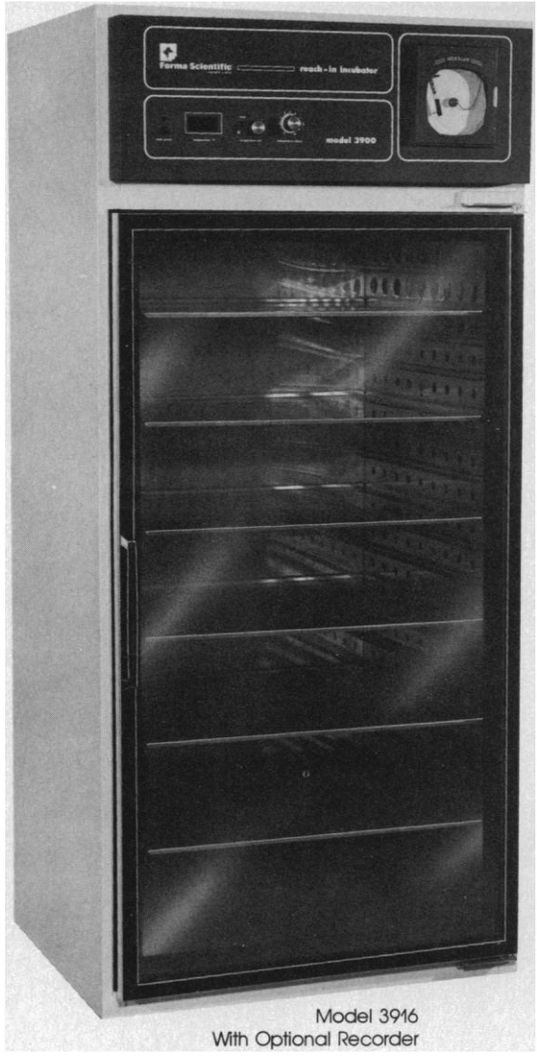

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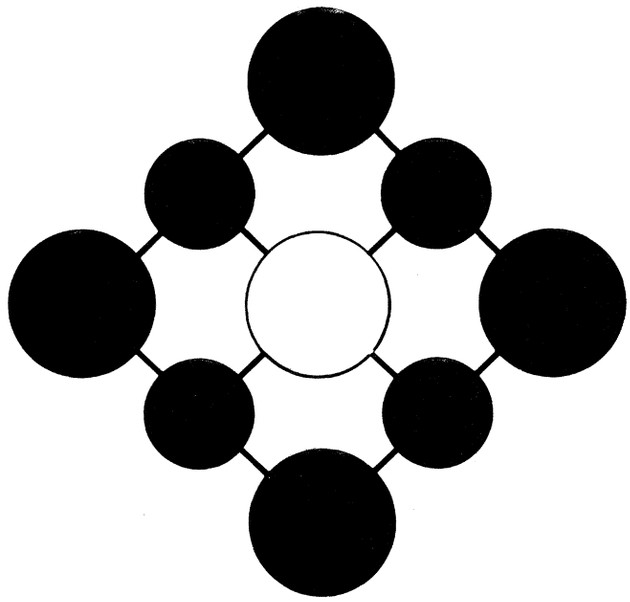

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Edited by
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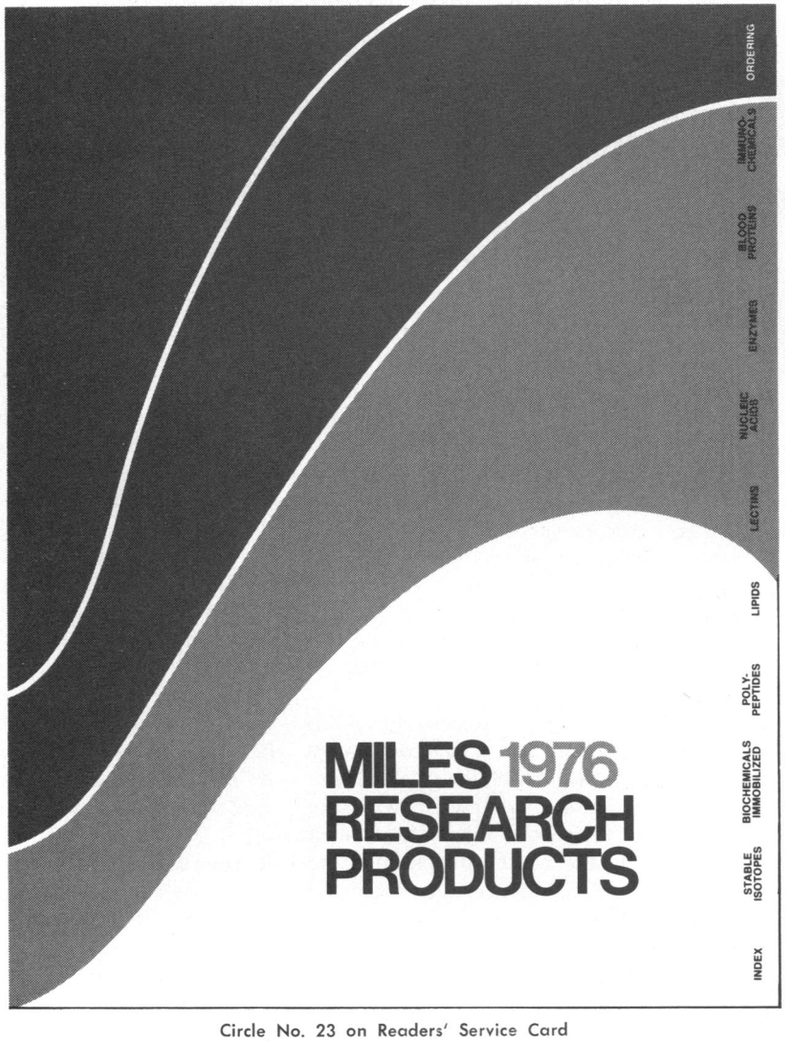

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LETTERS

Kepone Chronology

In a recent issue of *Science* (News and Comment, 7 May, p. 529), Nicholas Wade reports that Umberto Saffiotti resigned as director of the National Cancer Institute's (NCI's) chemical carcinogenesis bioassay program. According to Wade's article, part of the reason for Saffiotti's resignation from this position relates to the lack of staff assigned to the program and the attendant inability to promptly assess and disseminate experimental results. Without assigning responsibility for the bottleneck, the following chronology may serve to illustrate the consequences of such a failure to make information available.

The chemical Kepone (chlordecone) was tested in the NCI bioassay program, and the results were released on 8 April 1976 (*1*). (Preliminary results were available in January 1976.) The studies, conducted under contract to the NCI, were begun in November 1971 and May 1972 (of mice and rats, respectively). According to the NCI, the mice were killed in August 1973 (after 90 weeks), and the rats were killed in July 1974 (after 112 weeks). Both groups were dosed for 80 weeks. All pathology was held, and it is not yet clear when the slides were actually evaluated.

Life Science Products Company of Hopewell, Virginia, the firm that made Kepone, was formed in the fall of 1973 but did not begin manufacturing operations until March 1974. At that time, the NCI mouse bioassay had been completed but had not been evaluated. The rat study was completed 4 months later. If both studies had been evaluated within 6 months of the termination of the longest bioassay (that of rats), the results would have been available in January 1975. The Life Science plant was ordered closed by the state of Virginia on 25 July 1975.

Information on the toxicology of Kepone was minimal in the period between 1949 and 1958. In 1958, the Allied Chemical Corporation contracted with the Medical College of Virginia to conduct studies on the acute, subchronic, and chronic toxicity of Kepone in several species. These reports were used in connection with petitions to register Kepone as a pesticide; the data in the petitions were kept confidential. A memorandum dated 1958 was referred to in *Clinical Toxicology of Commercial Products* (*2*). This information, supplied by Allied Chemical, showed the rabbit to be the most sensitive species; the single lethal dose that killed 50 percent of the animals (LD_{50}) was 65 mg/kg; the rat was less sensitive (the single LD_{50} was 95 mg/kg). The minimum single lethal dose in the dog was 250 mg/kg. No information on the chronic toxicity of Kepone was released by Allied Chemical.

In the 1960's, two investigations of the subchronic toxicity of Kepone in mice were reported (*3*, *4*). They showed that Kepone produced tremors and ataxia. The report by Good *et al.* (*3*) suggested that effects on reproduction are detectable when the diet contains Kepone concentrations of 5 ppm. Both reports alluded to the cumulative nature of the toxin.

As a result of the disease that Kepone produced in exposed workers and the attendant court actions, the studies sponsored by Allied Chemical have become available (*5*). In these reports, the single LD_{50} in rats of Kepone dissolved in corn oil was 132 mg/kg. The estimated LD_{50} from a 3-month feeding study was 3.2 mg/kg per day. The 6-month LD_{50} was lower, 1.5 mg/kg per day. This indicates that Kepone is a cumulative poison and that at least 6 months are required for toxic concentrations to be reached with low doses. A chronicity factor, calculated from these data, is the ratio of the single LD_{50} value in repeatedly dosed animals. The values are 41 and 88 for 3- and 6-month periods, respectively. Only Mirex, an analog of Kepone with a 3-month chronicity factor of 60.8, appears to be a more cumulative toxin (*6*). Mirex has a comparable 3-month LD_{50} value, 6 mg/kg, but a larger single LD_{50} value, 365 mg/kg.

From the observations on the acute and chronic toxicity of Kepone, it can be said that a reduction in the exposure time would have benefited the employees at the Life Science Products plant. From the chronology, the NCI could have been instrumental in this reduction. Using the airborne Kepone concentration in the plant of 3 mg/cm^3 measured by state of Virginia officials in July 1975, it can be estimated that the workers could have received daily doses of Kepone that were within a factor of 12.5 of the dose calculated as a 6-month LD_{50} for the rat. The NCI carcinogenesis bioassay data and the data found in the toxicity studies sponsored by Allied Chemical indicate that Kepone concentrations of 10 ppm in the diet cause cancer in mice and rats. This dose is within a factor of 5 of the daily amount that estimates suggest could have been absorbed by the exposed workers. It remains to be seen whether the 15-month exposure period in humans will be enough to produce cancer.

RUDOLPH J. JAEGER
Department of Physiology, School of Public Health, Harvard University, Boston, Massachusetts 02115

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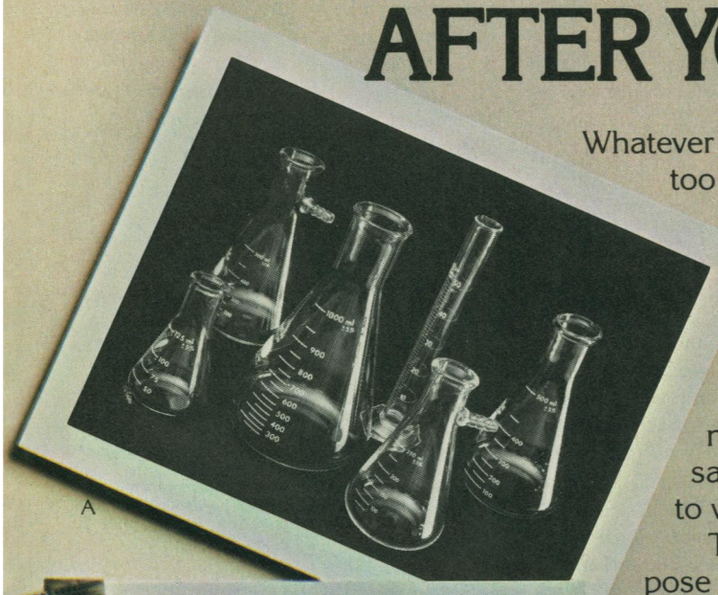

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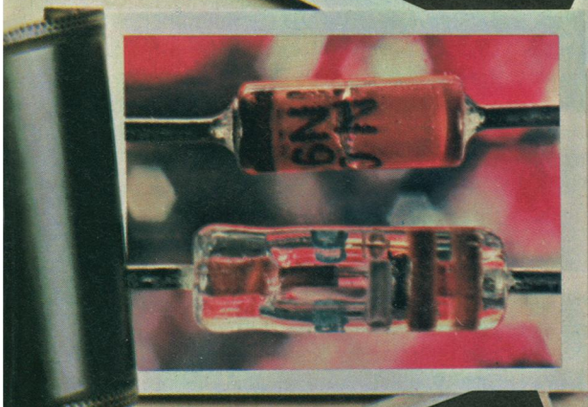

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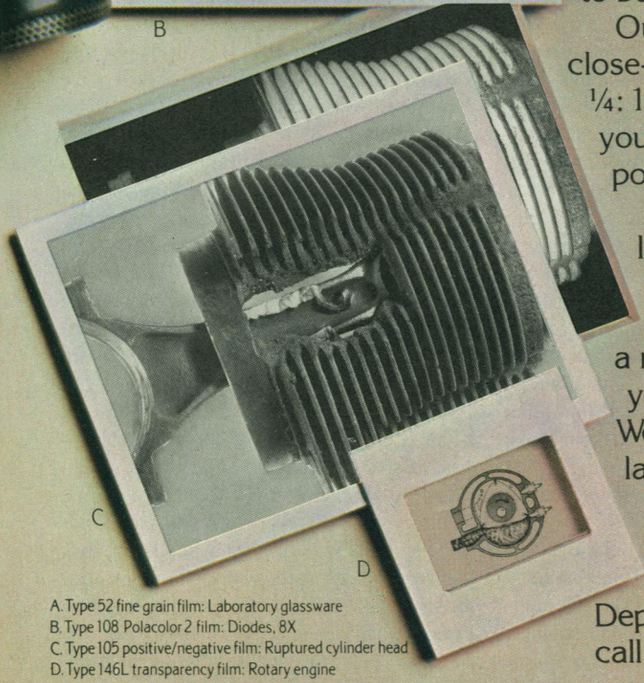

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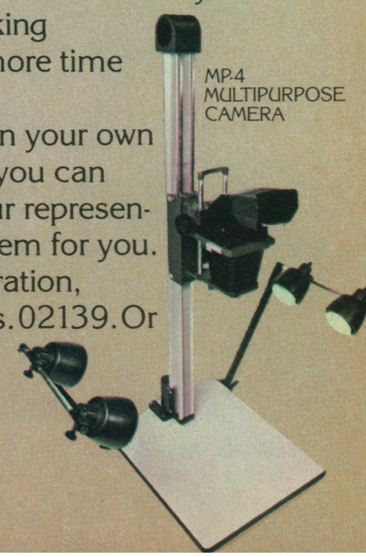

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Brown Pelican Reproduction

D. W. Anderson *et al.* (Reports, 21 Nov. 1975, p. 806) attribute the improvement since 1970 of brown pelican reproduction in Southern California to lower DDT residues in "the discharge at a sewage outfall associated with a Los Angeles plant that manufactured technical DDT."

The following observations are pertinent. Before 1970, the effluent from the factory mentioned by Anderson *et al.* (the Montrose plant) had drained into Southern California coastal waters by way of the Los Angeles County sewage treatment plant for more than 20 years without any reported effect on brown pelicans. During this period, the DDT in the effluent from the plant's settling pond averaged 10 to 15 pounds per day.

One month after the 1969 Santa Barbara oil spill, Risebrough reported that the pelicans were not reproducing because of high DDT residues (*1*). At the Wisconsin DDT hearings, which were characterized even by an Environmental Protection Agency (EPA) attorney as "a circus" (*2*), he had testified (*3*) that the brown pelican on Anacapa Island, off the Santa Barbara coast, was "extinct," "gone." Under cross-examination at the 1971–72 Washington DDT hearings, however, he retracted this statement (*4*). The pelican was not extinct after all.

In 1974, L. R. Axelrod of the EPA testified (*5*) before the House Appropriations Committee's subcommittee on agriculture that mercury was the principal suspect in eggshell thinning. The concentration of mercury in crude petroleum has been reported to be as high as 18 parts per million (*6*).

Noise, fright, and intrusion also cause birds to produce eggs with thin shells. Frequent visits to the brown pelican colonies by investigators, sometimes in helicopters, were stopped by the Department of the Interior as a result of protests. The pelicans have since made a quick recovery.

If DDT persists for decades, how could the pelicans have recovered so quickly? Either DDT is not as persistent as its detractors maintain, or it was not the cause of the pelicans' decline.

I believe the final judgment of the scientific community will be that DDT is not responsible for the depletion or extinction of living organisms except for insect pests.

MAX SOBELMAN
Montrose Chemical Corporation of California, Post Office Box 147, Torrance 90507

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The excessive DDT contamination of the Southern California Bight in the late 1960's and early 1970's was associated with a number of wildlife problems that have been examined in a series of other reports (*1–3*).

Of the points raised by Sobelman, only three refer to the data and conclusions of our *Science* report. Observations before 1969 include a report of thin-shelled eggs from Anacapa Island in 1962 (*4*), a decline of Southern California brown pelican populations beginning in the mid- to late 1950's (*2*), and suggestions of reproductive problems on Los Coronados as early as 1963 (*5*). Investigations specifically directed toward this problem were begun in 1969 (*5, 6*).

We believe it important to stress our conclusion that this contamination resulted principally from an industrial discharge rather than agricultural or urban runoff. Sobelman maintains that the high DDT residues in the sewer pipe below the Montrose plant (*7*) and the more than 200 metric tons of DDT in the sediment offshore from the sewer outfall (*8*) did not originate at the Montrose plant. DDT residues entering the waste treatment plant of the Los Angeles County sanitation districts dropped sharply in 1970, after the changeover by Montrose from a settling pond disposal to a sanitary landfill disposal (*9*). Sobelman has so far not published any data supporting his conclusion that the effluent from the settling pond contributed only 10 to 15 pounds of DDT per day to the sewage system. Nor has he published any description of the analytical methodologies employed. The wide interest generated by this prob-

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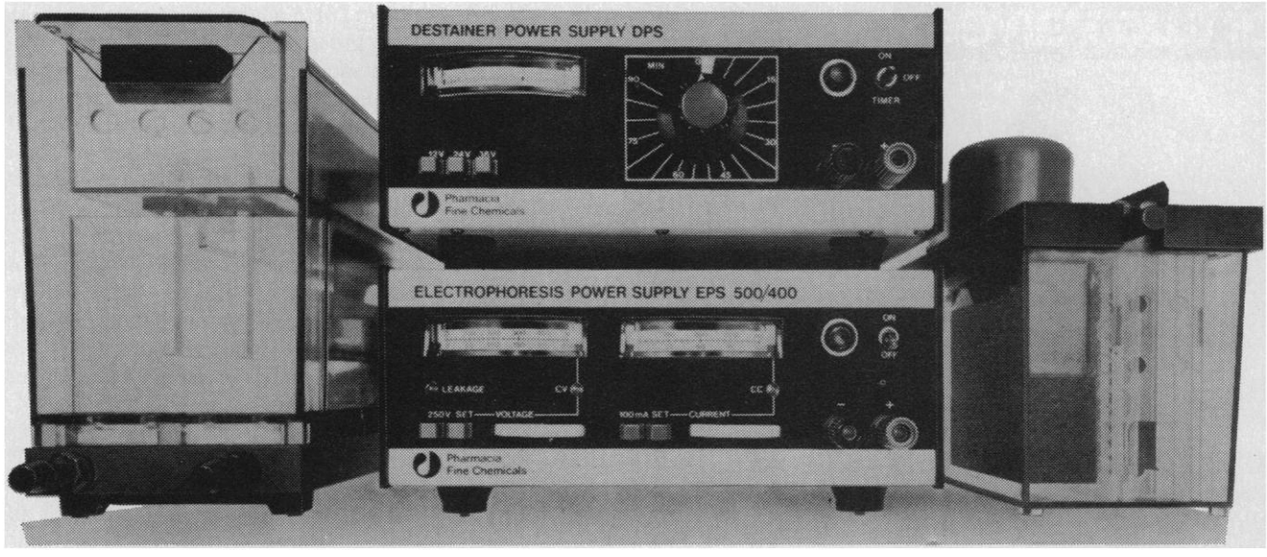

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lem and its implications to global marine pollution suggest that he do so without delay.

As documented in our report and in a more recent article (*3*), the brown pelicans are still laying thin-shelled eggs on Anacapa Island, and reproduction is not yet normal. Nor have the DDT residues declined to acceptable levels. Aerial fallout of DDT, originating largely from point sources near the Montrose factory and its sanitary landfill site, is now a major source of DDT input to the Southern California Bight (*10*).

As yet, there is insufficient support for the hypothesis that a visit by a scientific investigator to a seabird colony can cause eggshell thinning in eggs laid on previous days.

DANIEL W. ANDERSON
Division of Wildlife and Fisheries Biology, University of California, Davis 95616

ROBERT W. RISEBROUGH
Bodega Marine Laboratory, University of California, Bodega Bay 94923

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Errors in Mathematical Proofs

Gina Bari Kolata's article "Mathematical proofs: The genesis of reasonable doubt" (Research News, 4 June, p. 989) contains one passage which is both out-of-date and somewhat misleading. She alludes to an unresolved debate over a result in homotopy theory, in which two investigators are described as possessing long, complicated, and mutually contradictory proofs which could not be reconciled. Kolata is referring to a paper by myself and Emery Thomas of Berkeley (*1*), which for a time was contradicted by work of H. Toda and S. Oka in Japan. In fact, the issue remained open for somewhat more than a year but was settled in July 1974—2 years ago—when Toda and Oka found an error in their proof (*2*). The conflict drew attention precisely because such controversies are almost unheard-of in mathematics, as opposed even to physics and chemistry.

To say that the proofs were so long and complicated as to be "nearly impossible to check" is also a red herring. Our proof, for example, takes 13 pages (not 400) and has been used and generalized by a number of other workers. Actually, the conflict persisted as long as it did only because just one outside person, J. F. Adams, took the trouble to verify the details of our proof independently. This is the real problem: many published mathematical articles undoubtedly contain serious undiscovered errors, not because the mistakes are too difficult to find, but because contemporary pure mathematics has become so abstract and fragmented that few people bother to look carefully for errors.

RAPHAEL ZAHLER
Department of Mathematics, Rutgers University, New Brunswick, New Jersey 08903

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Linguistic Deterrent?

Gina Bari Kolata, in her article, "Strategies for the control of gonorrhea" (Research News, 16 April, p. 245), notes that the incidence of gonorrhea has declined remarkably in Sweden, whereas this has not happened in Denmark. I suggest that a major factor here may be the fact that in Sweden protective devices are referred to as *kondoms*. The Danish word for contraceptive is *svangerskabsforebyggende middel*. The sheer effort of uttering all ten syllables must surely be a deterrent to their purchase and use.

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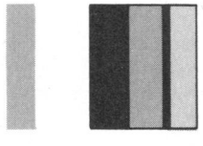

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9 July 1976, Volume 193, Number 4248 **SCIENCE**

R&D in the New Budget System

Innovations in federal public management are usually triggered by trouble of one sort or other. It is a classic and immutable sequence and worth thinking about amidst the congratulatory ceremonies of a bicentennial year.

Reform of government's budgeting methods, 55 long years after the executive budget was first introduced, is coming about not because of the tedious parade of annual deficits, but because of bitter quarrels between Presidents and the Congress over fiscal responsibility. Congress came to realize that it had yielded too much budgetary power to the executive, and is bent on putting matters to rights. What is emerging is a complicated budget control system. Strict limits on spending are to be self-imposed each year by the Congress in order to enforce fiscal discipline based on appraisals of the national economy and the relationships between ends and means. When the means are insufficient to the ends, the Congress will have to cut the cloth or face the necessity for higher taxes rather than the easy road of deficit budgeting.

All this is of more than academic interest to the scientific and technical communities. Disciplined budgeting has its price, and part of the price is a more competitive and risky context for the claims of research and development. The good old days of incremental bargaining and friends at court are on their way out. Henceforth it will be futile to try to shoehorn a little more of this or that into the annual budget as it makes its tormented journey through the two houses of Congress. The scientific community, along with other unwary clienteles, will have to awaken to the news that the government's budgets are being made up with different rules. Hereafter the dimensions and the mix will be locked into place well in advance of the budget year. We are now in the transitional quarter between fiscal '76 and'77, but the budget for 1978 is already on the drawing boards, and if the 1979 budget amounts are to be affected on behalf of R & D priorities, the time is already at hand.

It is now clear that there is no such thing as "the R & D budget." Except for the National Aeronautics and Space Administration, the Energy Research and Development Administration, and the National Science Foundation, government agencies treat research and development as subordinate elements in their program budgets. This means that the final amounts announced for research and development are an after-the-fact compilation derived painfully from a multitude of program and functional decisions. This is scarcely calculated to raise much consciousness on behalf of research and development in the Congress as the Battle of the Budget wears on.

These and other facts of life came out on 15 and 16 June at the first AAAS Colloquium on Research and Development in the Federal Budget. Some 200 persons assembled in Washington on short notice to examine the public policy problems in budgeting for research and development. They came from the White House, the Congress, government agencies, universities, industry, the media, and state and local government. A landmark AAAS report by Willis Shapley for the Committee on Science and Public Policy (to be published in August 1976) served both to demolish lingering myths and pose issues for the future. For once, all of the concerned parties were brought together in a lively exchange. If there were more questions than answers, most participants went home having learned something worth knowing.

Henceforth the merits of R & D as claimants on an embattled budget will have to be articulated more convincingly than in the past. Choices at the margins will be made on pragmatic rather than altruistic grounds, whether we like it or not. Congress and the Office of Management and Budget need help in making a strong case for the advancement of science. The AAAS Colloquium broke new ground, but it was only a beginning. To imagine that the future of scientific research can be left to faith, hope, and charity is to miscalculate the public policy climate.—WILLIAM D. CAREY

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during aggregation of platelet-rich plasma with PGH_2 (Fig. 4A) (*3*), as well as the inability of the potent vasoactive TA_3 to induce aggregation (Fig. 4B), indicate that the two properties of thromboxanes—that is, the powerful rabbit aorta contraction and platelet aggregation—are dissociated. Some pertinent observations have been reported. Addition of PGG_2 to platelet-rich plasma caused aggregation but little if any thromboxane production (*3*). In contrast, incubation of arachidonate or PGG_2 with washed platelets resulted in irreversible aggregation, generation of a potent rabbit aorta contractile factor, and in recovery of TB_2 (*7*).

The biosynthesis of thromboxanes is apparently not essential for platelet function. For example, primary aggregation, the release reaction, and irreversible aggregation induced by physiological concentrations of thrombin are not abolished by aspirin (*15*). In addition, a genetic deficiency of platelet cyclooxygenase resulted (in one patient) in a mild hemostatic defect associated only with a modest prolongation of bleeding time (*3*). The implication from our data and the earlier reports is that TA_2 formation is not an essential process in platelet aggregation. The primary physiological function of TA_2 is presumably as a potent localized vasoconstrictor that enhances hemostasis primarily by sharply reducing the blood vessel lumen, and perhaps secondarily by augmenting aggregation.

PHILIP NEEDLEMAN
MARK MINKES
AMIRAM RAZ

Departments of Pharmacology and Surgery, Washington University Medical School, St. Louis, Missouri 63110

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12. Radioactive C_{20} : 5 is not available. ^{14}C-Labeled dihomo-γ-linolenic acid was used as the tracer in the synthesis (SSV), isolation, and quantitation of PGG_3 and PGH_3. The ^{14}C-labeled PGH_1 did not interfere with the vasoconstrictor experiments since no TA_1 was formed. In addition, PGH_1 is reported not to inhibit PGH_2 platelet aggregation (*13*). No difference was observed when the PGH_3 was prepared without the ^{14}C-labeled C_{20} : 3.
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19. We thank A. Wyche, S. L. Key, and S. E. Denny for technical assistance and the Upjohn Company for the endoperoxide 9-epoxy-CEE. Supported by SCOR HL-17646, RCDA HL-19586, HE 14397, HEW surgical training grant 55552, and American Heart Association grant-in-aid.

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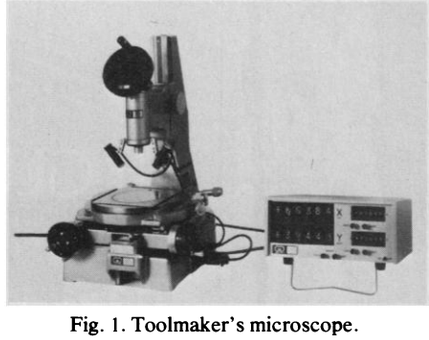

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