Waldmeier (*2*) has pointed out that in past minima the polar prominence zone has remained stationary at about 50° latitude prior to the sunspot minimum. During the minimum its heliographic latitude sinks to about 45° before resuming a poleward drift. Observers who have access to detailed prominence data can watch this additional criterion for solar minimum.

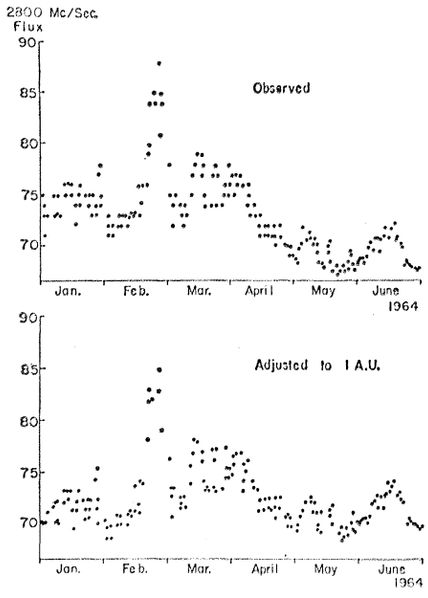

Fig. 5. Daily values of 2800 Mcy/sec solar flux (Ottawa) January to June 1964. New cycle spot visible on solar disk for the surface of the earth in watt/m^2 per cycle of band width per second ($\times 10^{-22}$). The lower chart shows the flux adjusted to the standard distance of 1 A.U.

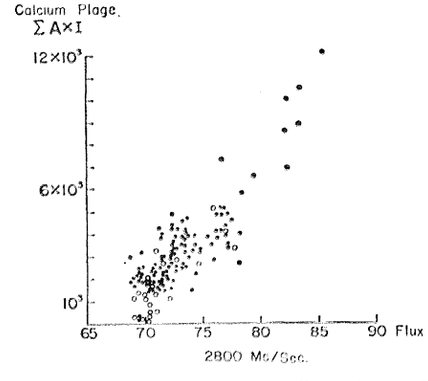

Fig. 6. Relation between daily values of solar radiation at 2800 Mcy/sec adjusted to 1 A.U. (Ottawa) and calcium plages (McMath-Hulbert Observatory). The flux is measured in watts per square meter per cycle of band width per second ($\times 10^{-22}$). The daily calcium plage measurement is the sum of the product for each plage of excess intensity and area in millionths of hemisphere. The open circles identify the days with no visible spots.

In January we suggested (*1*) that the present solar minimum was developing in a manner that resembled circumstances in 1944 more closely than those in 1923, 1933, or 1954. This continues to be true. Unless there is a sudden drop in the development of new cycle activity, the 1964 minimum will occur without long intervals of true solar calm. The exact date of minimum, based on smoothed sunspot means, will be a matter of future statistics. Persons planning IQSY programs designed to study the truly quiet sun should try to take advantage of the brief intervals of solar quiet which are recognized and reported—but all too seldom forecast in advance—by solar astronomers.

HELEN W. DODSON
E. RUTH HEDEMAN
FREDERIC L. STEWART
McMath-Hulbert Observatory, University of Michigan, Pontiac 48055

References and Notes

1. H. W. Dodson and E. R. Hedeman, *Science* **143**, 237 (1964). See also bibliography, *ibid.*, p. 240.
2. M. Waldmeier, *Z. Astrophys.* **59**, 205 (1964).

3. Supported in part by contract with the Office of Naval Research.

17 July 1964

Conversion of p,p′ DDT to p,p′ DDD in the Liver of the Rat

Abstract. *Feeding experiments with rats demonstrate that* p,p′ *DDT [1,1,1-trichloro-2,2-bis(*p*-chlorophenyl)ethane] is converted to* p,p′ *DDD [1,1-dichloro-2,2-bis(*p*-chlorophenyl)ethane] in the liver.*

Relatively high concentrations of *p,p′* DDD in fish liver oils have recently been reported (*1*). Although some *p,p′* DDT and *p,p′* DDE [1,1-dichloro-2,2-bis(*p*-chlorophenyl)ethylene] were found in these oils, the amounts were significantly smaller than the amount of *p,p′* DDD and, judged by the usual pesticide practices, there appeared to be no immediate explanation for the high *p,p′* DDD concentrations. In more recent work, Kallman and Andrews (*2*) showed by the use of C^{14}-labeled *p,p′* DDT and paper chromatography that yeast can effect a reductive dechlorination of *p,p′* DDT to *p,p′* DDD. The study with yeast was repeated in this laboratory with pure *p,p′* DDT and a gas chromatographic technique. The result supported the evidence for formation of

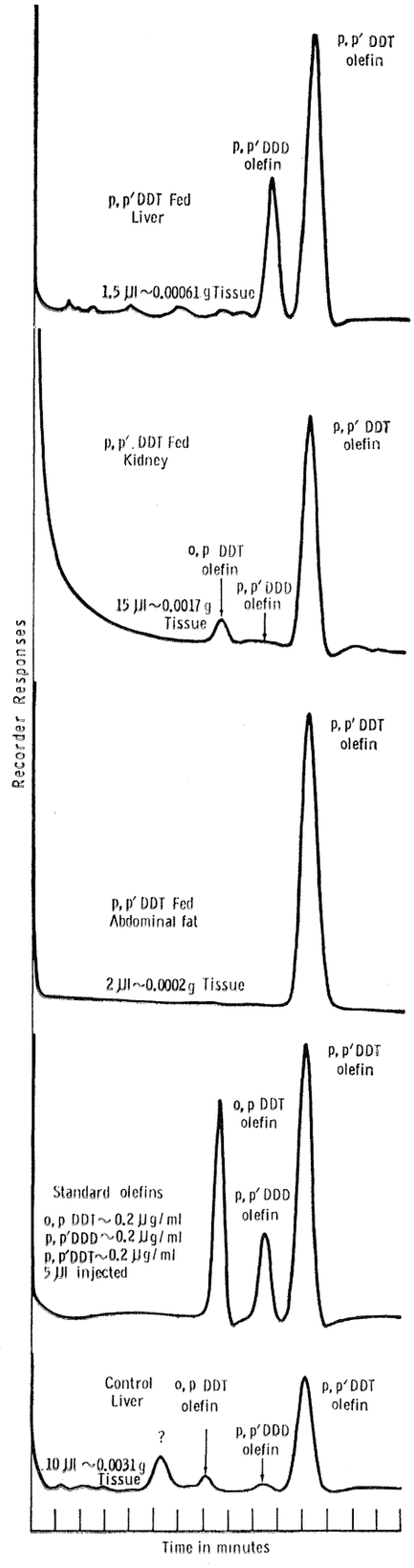

Fig. 1. Representative gas chromatograms of the olefins derived from *p,p′* DDT and *p,p′* DDD in the defatted tissue extracts of rats fed *p,p′* DDT and a control rat.

p,p′ DDD from *p,p′* DDT. These results suggested a possible reason for the high concentration of *p,p′* DDD in fish liver oils. The ability to dechlorinate *p,p′* DDT may not be limited to yeast but may also reside in higher animal forms. Although the conversion of *p,p′* DDT to such metabolites as DDE (*3*) and DDA (*4*) has been reported for species from the fly to man, there has been no evidence thus far that *p,p′* DDT can be dechlorinated to *p,p′* DDD in forms higher on the evolutionary scale than yeast. In this report we present gas chromatographic data that such a conversion occurs in at least one mammalian species, the rat.

Eighteen mature male and female Sprague-Dawley rats, each weighing about 210 to 320 g, were divided into three groups in which the members of each were matched in weight and sex. One group served as controls, and the animals were maintained on a standard diet of ground Purina laboratory chow. The two experimental groups were maintained on the same diet, but *p,p′* DDT or *p,p′* DDE (50 parts per million each) was added. Each experimental rat consumed approximately 0.75 mg of *p,p′* DDT or *p,p′* DDE daily.

After 12 weeks on these diets the animals were killed, and the livers, kidneys, and abdominal fat were removed for analysis and weighed. The tissues were extracted with ethyl ether and each extract was made up to 50 ml. A portion was evaporated, and then the residue was taken up in petroleum ether distilled from an all-glass system and treated with acetonitrile (*5*) to remove fat. Although gas chromatography of the acetonitrile-treated petroleum ether extracts clearly distinguished between *p,p′* DDT and *p,p′* DDE, the peak for *p,p′* DDD, if present, exactly coincided with that for *p,p′* DDT. In order to distinguish between *p,p′* DDT and *p,p′* DDD the method of Klein and Watts (*6*) was employed. This consisted in preparing the corresponding olefins by hydrolysis of the defatted tissue extracts with alcoholic sodium hydroxide.

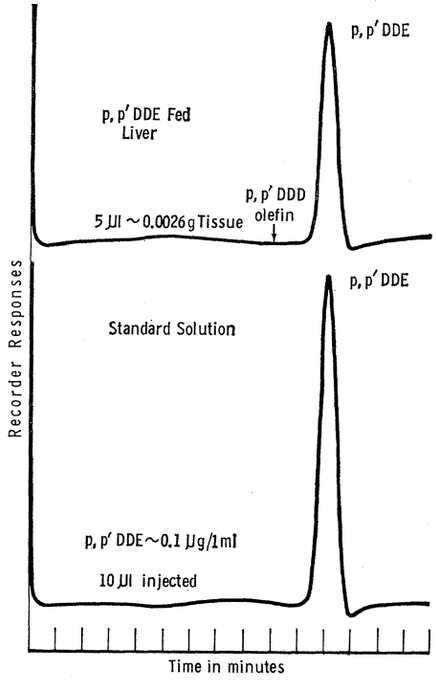

Fig. 2. Gas chromatogram of a defatted liver extract from a rat fed *p,p′* DDE.

The gas chromatographic analyses were carried out in a Jarrell-Ash model 700 gas chromatograph with a 90-cm U-shaped glass column which was mildly polar. The column consisted of a 2.5 percent coating of a mixture of silicone fluid 96 (General Electric) and 2,2-diethyl-1,3-propanediol-isophthalate polyester (1:1 by weight) on Celite. The column temperature was set at 175°C, the electron capture detector at 200°C, the splitter and injector units at 190°C, and an 18-volt potential was applied to the cell. The system was able to detect readily 5×10^{-4} µg of the olefin of *p,p′* DDT, 2.5×10^{-4} µg of the olefin of *p,p′* DDD, and 5×10^{-4} µg of the olefin of *o,p* DDT.

If the results for tissues of rats fed *p,p′* DDT (Fig. 1) are compared with the result for liver tissue from a control rat, it is evident that considerably more *p,p′* DDD is present in the liver of rats fed *p,p′* DDT. It should be noted, however, that under the conditions of these experiments no *p,p′* DDD was observed in either kidney or fat. Traces of *p,p′* DDD were also observed in the liver of the control animal as well as *p,p′* DDT and *o,p′* DDT. This is attributed to the presence of mixed DDT residues in the commercial rat chow. *p,p′* DDD was found in the livers of all experimental and control rats, and there was no demonstrable difference between the amounts found in males and females. Figure 2 shows that the liver of a rat fed *p,p′* DDE does not contain *p,p′* DDD. No *p,p′* DDD was found in the kidneys or fat of rats fed *p,p′* DDE.

It seemed of interest to compare the relative amounts of *p,p′* DDD olefin and total *p,p′* DDT olefins present in livers of rats fed *p,p′* DDT. In six rats, where a graphic analysis was made, the ratio of *p,p′* DDD to total *p,p′* DDT olefins is approximately 1:1. However, when account is taken of the *p,p′* DDE [*p,p′* DDT olefin] which was already in the liver as a result of metabolic processes, then a ratio of the *p,p′* DDD to unconverted *p,p′* DDT stored in the liver is approximately 2:1.

Our results thus show that *p,p′* DDT is converted to *p,p′* DDD in the rat liver and that this conversion does not require the intermediate formation of *p,p′* DDE.

P. R. Datta
E. P. Laug
A. K. Klein

Food and Drug Administration, U.S. Department of Health, Education, and Welfare, Washington 25, D.C.

References and Notes

1. Food and Drug Administration, unpublished reports.
2. B. J. Kallman and A. K. Andrews, *Science* **141**, 1050 (1963).
3. A. W. Lindquist, A. R. Roth, W. W. Yates, R. A. Hoffman, J. S. Butts, *J. Econ. Entomol.* **44**, 167 (1951); A. M. Mattson, J. T. Spillane, C. Baker, G. W. Pearce, *Anal. Chem.* **25**, 1065 (1953).
4. J. D. Judah, *Brit. J. Pharmacol.* **4**, 120 (1949); W. C. White and T. R. Sweeney, *Public Health Rept. U.S.* **60**, 66 (1945).
5. P. A. Mills, J. H. Onley, R. A. Gaither, *J. Assoc. Offic. Agr. Chemists* **46**, 186 (1963).
6. A. K. Klein and J. O. Watts, *ibid.* **47**, 331 (1964).

7 July 1964

Redifferentiation of Connective Tissue Cells in Serial Culture

Abstract. *When cell cultures are initiated from aortic connective tissue of rats and maintained as monolayers on glass, the differentiated property of collagen production disappears. However, these same cells, when placed in diffusion chambers in the peritoneal cavities of other rats, produce collagen, as indicated both morphologically and by the accumulation of hydroxyproline within the chambers.*

Some of the early workers in tissue culture, notably Champy (*1*), regarded the loss of identifiable morphologic and biochemical characteristics of differentiation, which occurs in tissue culture, as a process of dedifferentiation —from which state cells could again return. With the more recent common use of cell culture techniques this concept of dedifferentiation and redifferentiation has been questioned (*2*). Although specialized functions such as synthesis of collagen (*3*) or acid mucopolysaccharides (*4*) will, in rare instances, persist when cells are grown