In sound tests, the group exhibited the normal low percentage of seizures up to 9 weeks of treatment. Following that, the percentage rose sharply to 50% just 2 weeks before all the mice died.

The mice in the groups breathing air saturated with Chlordane reacted quite differently. They immediately ceased feeding and drinking and huddled together, seeming loath to move at all. They apparently became blind and within relatively few hours lost all power of coordination. All the mice in the group exposed continuously died within 4 days, four of the 20 dying during the first day. The mice in the groups exposed for 15 hr/day and 9 hr/day were exposed for only 4 days. Half of the 15hr group were dead at that time, and the others died within a few days. Two of the mice in the 9-hr group were dead at the end of 4 days (36 hr of exposure), and all but four died within the next 10 days. This confirms the statement of Radeleff (10) that animals "poisoned by Chlordane do not seem to recover, once they have manifest toxic symptoms." There were no observable anatomical differences between these mice and normal mice, except for extreme emaciation in mice which lived for some days. No sound tests could be given because the mice survived too short a time and seemed incapable of moving about with rapidity after 6 hr-8 hr of exposure to the vapor.

In general, results of this study indicate that Chlordane is very similar to DDT in its toxic action in mammals. The first system affected is the nervous system, and nervous symptoms predominate in acute toxicity. In chronic intoxication, however, the liver seems to be most affected. We were unable to determine to our satisfaction whether Chlordane accumulated in the fat of the mice or not. Possibly the cumulative toxicity is owing merely to destruction of the liver cells, along with effects on other organs of the body such as the kidneys.

Because of the widespread use of Chlordane in structural pest control, the rather striking toxicity of the vapor is significant. In this particular, Chlordane differs from DDT, just as it does in the apparent relative ease with which it is absorbed through the unbroken skin. A surprising feature is the relatively high toxicity of wettable powder preparations, since it is usually assumed that this type of preparation is less readily absorbed than kerosene solutions. All these results indicate that repeated exposure to Chlordane preparations on the skin or continued inhalation of the vapor may be deleterious.

Another striking point is that the treated animals gained weight normally, and except for greater irritability than the controls, seemed in good health. If transposition of results on mice to man is permissible, this indicates that the general level of health, appetite, weight maintenance, and the like would be a poor index of early chronic intoxication by Chlordane. The established fact that the chlorinated hydrocarbon insecticides have a similar cumulative action makes it imperative that clinical tests for detection of early intoxication in man be developed.

In experimental animals, the method of "audiogenic" seizures seems promising as a test for early intoxication. From our results with Chlordane, it is seen that the incidence of "audiogenic" seizures in a population of treated mice rises rather sharply a few weeks before elinical symptoms and deaths appear. With proper standardization of the test situation and with selected mice, this test may provide a good indication of early intoxication.

Finally, the results suggest that Chlordane may have value as a rodenticide. Since it is toxic to animals when absorbed through the skin and lungs, as well as when ingested, it might be used under favorable conditions for control of mice and possibly rats.

## References

- BUSHLAND, R. C., WELLS, R. W., and RADELEFF, R. D. J. econ. Entomol., 1948, 41, 642.
- DRAIZE, J. H., NELSON, A. A., and CALVERY, H. O. J. Pharm. exp. Therap., 1944, 82, 159.
- 3. FINGER, F. W. Psychol. Bull., 1947, 44, 201.
- 4. FITZHUGH, O. G. and NELSON, A. A. J. Pharm. exp. Therap., 1947, 89, 18.
- 5. HALL, C. S. J. Heredity, 1947, 38, 1.
- 6. INGLE, L. J. econ. Entomol., 1947, 40, 264.
- 7. LAUG, E. P. and FITZHUGH, O. G. J. Pharm. exp. Therap., 1946, 87, 18.
- LEHMAN, A. J. Bull. Assoc. Food and Drug Offic., 1948, 12, 82.
- 9. Ibid., 1949, 13, 65.
- 10. RADELEFF, R. D. Vet. Med., 1948, 43, 342.
- 11. STAINBROOK, E. Psychosom. Med., 1947, 9, 256.

# Stability of the Adenosinetriphosphatase System in Animal Tissues<sup>1</sup>

## Mona Marquette and B. S. Schweigert

Division of Biochemistry and Nutrition, American Meat Institute Foundation, and Department of Biochemistry, University of Chicago, Chicago

In the course of experiments on the stability of respiratory enzyme systems in beef tissues obtained from carcasses of different grades and aged for varying periods, the stability of the adenosinetriphosphatase (ATP-ase) system in intact tissues was investigated. Data on the nature and occurrence, metabolic function, and specificity of this system have been reviewed  $(\mathcal{Z}, \mathcal{Z})$ ; however, little attention has been devoted to studies on the stability of the system in intact tissues stored for varying periods of time after the animals are killed. In the present study the ATP-ase activity of animal tissues stored at  $-2^{\circ}$  C and at  $+5^{\circ}$  C for periods up to 15 days was investigated.

Preliminary experiments were conducted with rat tissues and the study was then extended to beef muscle tissue. The procedure used for determining the ATP-ase activity of the tissues was essentially that described by Umbreit, Burris, and Stauffer (5). Young adult rats that were fed stock ration were killed by decapitation. Approximately 1 g of muscle tissue, and in certain experiments liver and kidney, were removed and prepared

<sup>&</sup>lt;sup>1</sup> This work was supported in part by a contract with the Production and Marketing Administration, U. S. Department of Agriculture, under the Research and Marketing Act.

for the ATP-ase assays. The tissues were homogenized in a Waring Blendor for 5 min and each tissue was assayed at levels of 1 and 2 mg of fresh tissue per assay. The equipment used was kept cold during preparation of the tissues and ice was added during the blending period.

After the tissues were obtained for determination of the ATP-ase activity, the remainder of the carcass was wrapped in moistened paper towels and stored at either  $-2^{\circ}$  C or at  $+5^{\circ}$  C. Samples of tissue were removed  $\hat{\Sigma}$ , 4, 8, or 15 days later and the ATP-ase activity was determined. In some experiments two different samples of muscle tissue were taken from the same carcass on the same day, and the values obtained for the two samples were in good agreement.

## TABLE 1

ADENOSINETRIPHOSPHATASE ACTIVITY OF RAT TISSUES AS INFLUENCED BY STORAGE CONDITIONS\*

Tissue or organ	Storage tempera- ture	Time of storage (days)					
		0	2	4	8	15	
Rat No. 1							
Muscle		14.8	15.0	13.0	15.3	16.2	
Kidney		9.9	12.4				
Liver	- 2° C	8.1	7.8	6.9	6.4	5.2	
Rat No. 3							
Muscle		13.4		12.3	14.4	11.2	
Liver		6.5		5.9	5.0	3.3	
Rat No. 2							
Muscle		14.1	12.2		15.5		
Kidney	+ 5° C	12.2	12.8				
Liver		7.5	8.0				
Rat No. 4							
Muscle		15.8		11.9	13.3		

\* Values expressed as µg phosphorus liberated per mg of fresh tissue in 15 min at 37° C (1).

#### TABLE 2

ADENOSINETRIPHOSPHATASE ACTIVITY OF BEEF MUSCLE TISSUE AS INFLUENCED BY STORAGE CONDITIONS\*

Sample†	Storage temperature	Time of storage (days)				
		0	5	9	15	
No. 1		9.3	9.0	8.8	9.0	
No. 2	– 2° C	7.9	8.1	6.9	7.2	
No. 1A	+ 5° C	7.3	8.5	8.5	8.7	
No. 2A		7.3	7.1	7.8	7.2	

\* Values expressed as µg phosphorus liberated per mg of fresh tissue in 15 min at 37° C.

† Samples 1 and 1A were obtained from the same carcass and were adjacent sections, 11 in. thick of the longissimus dorsi muscle. A similar pairing was made for samples 2 and 2A from another carcass.

The rib-eye muscle (longissimus dorsi) from the 11th and 12th rib cut was obtained from steer carcasses that weighed 631 and 637 lb. These samples were obtained 24 hr after the steers were killed and the rib-eye muscle was then dissected free of fat and connective tissue. Two samples were prepared from each carcass; one was stored at  $-2^{\circ}$  C, and the other at  $+5^{\circ}$  C. The samples were approximately 11 in. thick and were wrapped in paper and stored as indicated. The outer portions of the muscle were removed prior to sampling and 3-4-g samples were taken for ATP-ase assays at 0, 5, 9, and 15 days of storage.

The ATP-ase system was found to be essentially stable in both intact rat and beef muscle tissues during storage for periods up to 15 days with the test conditions used (Tables 1 and 2). The slight increases in activity after storage may have been due to a reduction in water content during storage of the tissues. Results obtained for tissues stored above freezing (approximately  $+5^{\circ}$  C) were essentially similar to those obtained for tissues stored at  $-2^{\circ}$  C in that after 8-15 days no appreciable changes in ATP-ase activity had occurred. Some reduction in the ATP-ase activity occurred in liver as evidenced by the results obtained with tissues stored at  $-2^{\circ}$  C.

Values obtained for rat muscle, kidney, and liver were somewhat lower than those reported by DuBois and Potter (1). No comparable data are available for beef tissues. The technique of homogenization used in the present work may not have been as effective in rupturing the cells as that used by these workers. The ATP-ase activity was not increased by increasing the homogenization time to 10 or 15 min. The ATP-ase activity of beef muscle was lower than that obtained for rat muscle.

It is significant, therefore, that the ATP-ase system in these intact tissues is essentially stable for a considerable period after the animal is killed. This is in contrast to the instability of certain other enzyme systems such as the oxalacetic acid oxidase system (4).

### References

- 1. DUBOIS, K. P. and POTTER, V. R J. biol. Chem., 1948, 150, 185.
- 2. ENGELHARDT, V. A. Adv. Enzymol., 1946, 6, 147.
- 3.
- POTTER, V. R. Adv. Enzymol., 1944, 4, 201. POTTER, V. R., LEPAGE, G. A., and KLUG, H. L. J. biol. 4. Chem., 1948, 175, 619.
- 5. UMBREIT, W. W., BURRIS, R. H., and STAUFFER, J. F. Manometric techniques and related methods for the study of tissue metabolism. Minneapolis: Burgess Publishing Co., 1947. P. 97.

## A Modern Periodic Chart of **Chemical Elements**

## John D. Clark

## 252 W. 78th Street, New York City

Since the discovery and elucidation of the actinide series of elements, a reexamination of the perennial problem of the position of the rare earths (and now the actinides) in the periodic chart of chemical elements appears to be desirable. The disposition of these series is far from obvious. Emeléus (3) and others have pointed out that the actinides are quite analogous to the lanthanides, but Anderson (1) has emphasized their resemblances to the other elements. The accompanying chart (Fig. 1), a development of one published by the author (2) in 1933 and recently republished (5) in a slightly modified form. but with a much more extensive treatment of the two