

In an earlier experiment with pyridoxine-depleted rats, GPT values ranging from 500 to 700 percent above normal were observed after 4 weeks of treatment with 5 mg of hydrocortisone per rat per day. In the studies summarized in Table 1, the GPT levels were doubled after 2 days of treatment, and maximum GPT activity in liver occurred after the seventh day of treatment with 2.5 or 5 mg of hydrocortisone per day. Although some variation in the control values for GPT was noted on different days of the experiment, the standard deviation of each group remained small. On the 28th day, 14 days after the administration of hydrocortisone was discontinued, the GPT activity was still considerably higher than normal. Whether this observation can be attributed to the normal turnover rate of this enzyme in liver, or to the prolonged retention of hydrocortisone or an active metabolite, cannot be answered at this time.

The administration of large amounts of pyridoxine to animals receiving hydrocortisone did not alter the magnitude of the GPT response. Furthermore, deple-

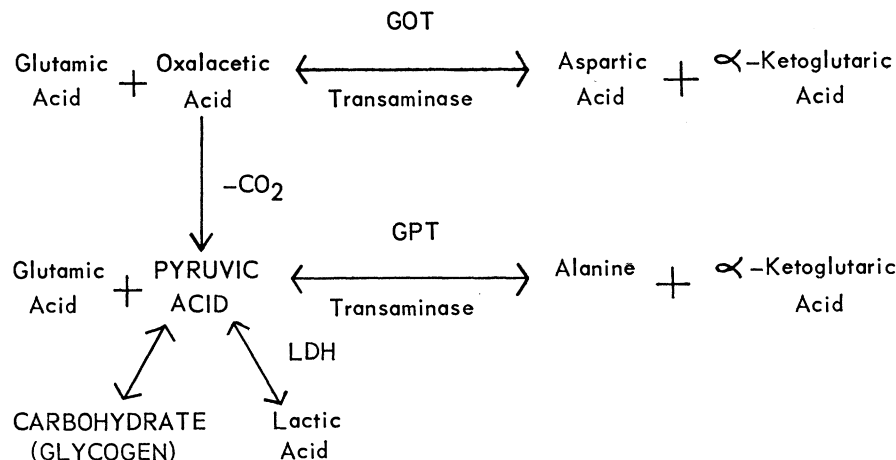


Fig. 1. Transaminase reactions and pathway to carbohydrate synthesis.

tion of rats of pyridoxine for 8 weeks did not impair the increase in GPT following daily injections of hydrocortisone (5 mg) for 1 week. Thus, neither depletion of pyridoxine nor administration of this vitamin appears to affect the changes in hepatic GPT levels produced by hydrocortisone.

It was of interest to determine whether other corticosteroids increased hepatic GPT activity to the extent observed with hydrocortisone. The daily subcutaneous injection of rats with Prednisone (2.5 mg) or cortisone acetate (5 mg) for 1 week resulted in more than a five-fold increase in liver GPT activity in each case. In a comparable experiment, deoxycorticosterone was administered (3 mg daily); the hepatic GPT values were not increased, and they appeared to be somewhat lower than those of the untreated control animals. The relationship between the stimulation of hepatic GPT activity and the gluconeogenic potency of related steroids is under study to evaluate the hepatic GPT response to corticosteroids as a method of assay.

Negative nitrogen balance is also an important aspect of treatment with glucocorticosteroids. These studies do not indicate whether the effect of hydrocortisone is limited to alterations in GPT activity or whether transamination processes other than GOT are also affected. It is apparent, however, that increased transaminase activity can be related directly to amino acid imbalances which initiate protein catabolism and negative nitrogen balance.

Of the many amino acids which have been studied for gluconeogenic activity, alanine, aspartic acid, and glutamic acid are unique with regard to their high gluconeogenic potency (7). The relationship between the substrates in the transaminase reactions studied herein and the pathway to carbohydrate synthesis is shown in Fig. 1. Pyruvic acid appears to be the common intermediate in the conversion of these amino acids to glycogen.

Both pyruvic acid and lactic acid are readily converted to carbohydrate. Moreover, both of these metabolites occur in elevated concentrations in the blood of patients with Cushing's syndrome and in subjects receiving glucocorticosteroids (8). These facts, added to the observation that a substantial rise in hepatic GPT occurs in rats treated with hydrocortisone, in contrast to treatment with deoxycorticosterone, strongly suggests that the control of hepatic levels of GPT by glucocorticosteroids is importantly related to the mechanism whereby these compounds exert their gluconeogenic activity (9).

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References and Notes

1. F. Gavosto, A. Pileri, A. Brusca, *Biochem. et Biophys. Acta* 24, 250 (1957).
2. O. H. Lowry, N. R. Roberts, M.-L. W. Chang, *J. Biol. Chem.* 222, 97 (1956).
3. O. H. Lowry, N. R. Roberts, J. I. Kappahn, *ibid.* 224, 1047 (1957).
4. O. H. Lowry *et al.*, *ibid.* 193, 265 (1951).
5. O. H. Lowry *et al.*, unpublished.
6. F. Wroblewski and J. S. LaDue, *Proc. Soc. Exptl. Biol. Med.* 91, 569 (1956).
7. S. Soskin and R. Levine, *Carbohydrate Metabolism* (Univ. of Chicago Press, Chicago, Ill., ed. 2, 1952), pp. 146, 147.
8. T. F. Frawley, *Ann. N. Y. Acad. Sci.* 61, 464 (1955); D. H. Henneman and J. P. Bunker, *Am. J. Med.* 23, 34 (1957).
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Table 1. Effect of two levels of hydrocortisone on glutamic-pyruvic transaminase (GPT), glutamic-oxalacetic transaminase (GOT) and lactic acid dehydrogenase (LDH) activities of rat liver. There were 20 animals in each group; five animals sacrificed on days 2, 7, 14, and 28. The animals were maintained on Purina mouse diet. The weight of rats in the untreated group was controlled during days 1 to 14 by restricting diet; thereafter, the rats were allowed to eat *ad libitum*. Hydrocortisone was injected subcutaneously (2.5 and 5.0 mg per rat per day). The administration of hydrocortisone was discontinued on day 15. The animals were killed by severance of the carotid artery and were exsanguinated before the tissues were removed.

Hydrocortisone treatment (mg)	Enzyme activity (mmole of substrate utilized/g of protein, per hour at 38°C)		
	GPT	GOT	LDH
<i>Second day</i>			
0	12.1 ± 3.2*	107 ± 10	281 ± 12
2.5	18.9 ± 2.0	112 ± 9.2	271 ± 12
5.0	25.4 ± 3.4	103 ± 5.2	259 ± 9.0
<i>Seventh day</i>			
0	8.7 ± 2.9	128 ± 3.6	290 ± 19
2.5	44.7 ± 4.1	140 ± 8.9	277 ± 33
5.0	53.0 ± 5.1	157 ± 3.9	268 ± 19
<i>Fourteenth day</i>			
0	16.1 ± 1.8	122 ± 13	266 ± 6.0
2.5	66.4 ± 4.4	152 ± 5.8	247 ± 25
5.0	71.0 ± 2.3	142 ± 14	242 ± 24
<i>Twenty-eighth day</i>			
0	17.5 ± 2.0	143 ± 6.8	299 ± 16
2.5	30.3 ± 4.3	157 ± 2.8	246 ± 16
5.0	52.5 ± 2.0	172 ± 7.7	226 ± 14

* Standard deviation = $(\sum d^2/n)^{1/2}$.

Corrosion of Pure Aluminum and Tin in Salt Spray

Despite the widespread and continued use of the salt spray test as a specification for plated metal parts (1) and as an accelerated corrosion test (2), very little is known about the actual mechanism of corrosion under these conditions. In

recent work on tin-plated copper (3) it was found that the rate of corrosion in salt spray could be expressed by an equation of the form

$$C = Kt^2 + b$$

where t is the time of exposure. It was not known, however, whether the observed rate was that of pure tin or whether the rate was influenced by the copper substrate. The latter situation would imply the existence of a galvanic couple in which the tin was anodic.

The technique described in this report was developed in order to study the corrosion of thin metallic films on a relatively inert substrate (4). It is believed that it can be used as a simple, inexpensive technique for measuring corrosion rates in a wide variety of environments.

Aluminum and tin films were evaporated onto cleaned glass slides, 2 by 2 in., by means of conventional high-vacuum techniques (5). The light transmission was determined for the central square inch by mounting the glass in aluminum slide-holders and by using an ordinary 35-mm slide projector. The projector had been modified so that the light path was enclosed by an opaque tube, and the lens position had been changed so that the image of the slide was focused onto a ground-glass screen just in front of a 931A photomultiplier tube. The output of the tube was read by a microammeter, and the intensity of the light was adjusted to give 100 percent transmission for holes of known diameter drilled into brass plates.

After measurement of the light transmission had been made, the specimens were removed from the aluminum holders and placed in a Lucite rack designed to hold them at the recommended angle of 15 degrees from the vertical. They were then placed in a standard salt spray test cabinet operating at 95°F (1); a 4 percent NaCl solution was used. When 4 hours had elapsed, the samples were removed, washed with cold water and alcohol, then dried, and the light transmission was determined. The procedure was repeated for as long as practical.

The results for tin and aluminum are shown in Fig. 1. Each graph represents the weighted average (with allowance made for the variation in individual

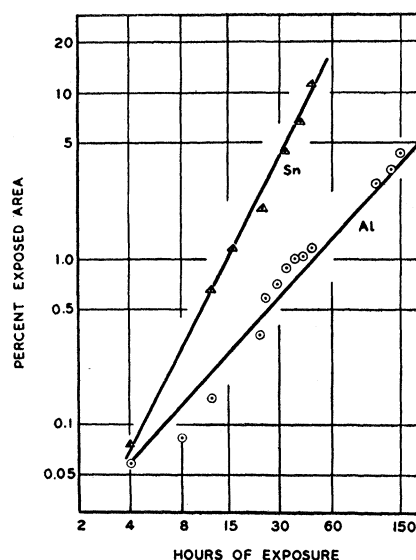


Fig. 1. Log-log plot of the corrosion of thin aluminum and tin films on glass: hours of exposure to 4 percent sodium chloride spray versus percentage of metal areas exposed.

thicknesses) of ten samples. The data are plotted as the logarithm of percentage transmission (compared to the total area) versus the logarithm of hours of exposure to salt spray. As can be seen, the corrosion rate for aluminum appears to have a slope of 1, and that for tin, a slope of 2. This means that the data may be represented by

$$C_{Sn} = Kt^2 + b$$

$$C_{Al} = K't + b'$$

where C_{Al} and C_{Sn} are the exposed areas resulting from corrosion and t is time of exposure.

Gross examination of the tin specimens indicates that corrosion occurs by pitting. If the attack of a salt solution is linear with time, then the exposed area would increase at the square of the rate of increase of the radius of the pit. Since the corrosion as measured by light transmission is total exposed area, the actual corrosion in one dimension is probably linear with time. The agreement in rate for thin tin films on copper and glass suggests that the same mechanism is operative in each case and rules out

mechanisms involving the substrate material.

Since the corrosion of tin proceeds as the square of the time of exposure, great caution must be exercised in expressing corrosion rate data in standard terms, such as inches per year or milligrams per decimeter per day (6).

For aluminum, the results indicate that no protective film is formed under salt spray conditions. It is not known whether this is due to the attack by the primary corrosion products, as suggested by Schikorr (7) for sodium chloride solutions, or due to the ability of the chloride ion to penetrate the oxide coating. The results are in agreement with those of Champion (8), who measured hydrogen evolution and oxygen absorption for aluminum suspended in potassium chloride solution. He found a curve of the form

$$dC/dt = \alpha C + \beta$$

although the values of the constants changed several times over a period of 40 days.

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References and Notes

1. "Specifications and Tests for Electrodeposited Metallic Coatings," *Am. Soc. Testing Materials ASTM Standards* (Sept. 1955); or *Federal Specification QQ-M-151a* (amendment 4).
2. For many examples, see L. R. Voight, *Corrosion* 4, No. 10, 1 (1948).
3. M. S. Frant, N. Morelock, A. Imswiler, in an address presented before the 131st meeting of the American Chemical Society, Miami, Fla., April 1957. A manuscript is in preparation.
4. Other related techniques reported include the use of a visual colorimeter on thin films, by W. R. van Wijk [*Ind. Eng. Chem. Anal. Ed.* 7, 48 (1935)] and visual observation by D. Roller, [*W.A.D.C. Tech. Rept.* 56-237 (U.S. Dept. Com., Washington, D.C.)].
5. L. Holland, *Vacuum Deposition of Thin Films* (Wiley, New York, 1956).
6. See G. R. Hiers and E. J. Minarcik, "Use of tin and lead outdoors" in "Symposium on Atmospheric Corrosion of Non-ferrous Metals," *Am. Soc. Testing Materials Spec. Tech. Publ.* No. 175 (1955).
7. G. Schikorr, quoted in U. R. Evans, *Metallic Corrosion, Passivity and Protection* (Arnold, London, 1946), p. 362.
8. F. A. Champion, "Assessment of the susceptibility of aluminum alloys" in "Symposium on Stress-Corrosion Cracking of Metals," *Am. Soc. Testing Materials, Proc.* (1945).
9. The help of Harold Woolley in the design and construction of the equipment, and the technical assistance of Claire Maier and Anna Imswiler, are gratefully acknowledged.

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