histamine was produced from the corresponding histidine derivatives.

That the bacterial decarboxylase is not inhibited by acyl-histidines was shown by the following experiments:

Sample A: Bacterial suspension + 50 mg histidine.

Sample B: Bacterial suspension + 50 mg histidine + 50 mg acetylhistidine.

Sample C: Bacterial suspension + 50 mg acetylhistidine.

After 12 hours incubation only Sample C had no activity on the intestine, while Samples A and B contained equal amounts of histamine (35-40 mg), indicating that presence of acetylhistidine does not inhibit the decarboxylation of histidine. Analogous results were obtained with benzoylhistidine.

Summary. The reported experiments show that the ability of *E. coli* to decarboxylate histidine and its derivatives is highly specific, since the acylation of the amino group hinders this process. We cannot decide however whether or not the *E. coli* are able to convert peptides into "peptamines" by decarboxylation. If a free amino group should be necessary also in peptides, then such amino group could be provided not only by histidine but also by peptides containing histidine units with free carboxyl groups.

We are indebted to Dr. C. Niemann (California Institute of Technology) for valuable suggestions.

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Effect of Low Potassium Diet and Desoxycorticosterone on the Rat Heart.*

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In a previous publication[1] attention was directed to lesions produced in the heart by injections of desoxycorticosterone acetate. This was to be expected since heart lesions develop on diets low in potassium[2,3,4] and injections of desoxycorticosterone acetate produce a deficit of potassium and changes in muscle composition similar to those accompanying diets low in potassium.[1,5,6] The lesions in the heart are not necessarily accompanied by changes in the electrolyte composition of the heart, and in rats it has not been certain that loss of heart potassium is produced either by diets low in potassium or by injections of desoxycorticosterone acetate. Study of the effects of the combination of a diet low in potassium together with injection of desoxycorticosterone acetate was, therefore, undertaken to determine if heart potassium can be reduced in rats and if lesions are more quickly produced by this means.

The chemical methods and the low potassium diet were described in previous studies.[7] Owing to the size, the hearts of several animals were pooled and analyzed as a whole. Sufficient material was not available to include fat and chloride analyses. All values are expressed per 100 g of dried tissue.

Table I shows the results. While neither the diet low in potassium nor injections of desoxycorticosterone give more than a questionable lowering of heart potassium in 30 days, the combination of the diet low in potassium

* Aided in part by a grant from the Fluid Research Fund.

1 Darrow, D. C., and Miller, H. C., *J. Clin. Invest.*, 1942, **21**, 601.

2 Schrader, G. A., Pricket, C. O., and Salmon, W. D., *J. Nutrition*, 1937, **14**, 85.

3 Follis, R. H., Oerent-Keiles, E., and McCollum, E. V., *Am. J. Path.*, 1942, **18**, 29.

4 Thomas, R. M., Mylon, E., and Winternitz, M. C., *Yale J. Biol. and Med.*, 1940, **12**, 345.

5 Ferrebee, J. W., Parker, D., Carnes, W. H., Gerity, M. K., Atchley, D. W., and Loeb, R. F., *Am. J. Physiol.*, 1941, **135**, 230.

6 Heppel, L. A., *Am. J. Physiol.*, 1939, **127**, 385.

7 Miller, H. C., and Darrow, D. C., *Am. J. Physiol.*, 1940, **130**, 747.

TABLE I.
Heart: Content per 100 g of Dried Weight.

	No. of rats	Experimental, days	DOCA, mg/day	Water, g	Na mM	K mM	P mM
Control	6	0	0	334	17	37	31
DOCA	6	30	2	355	19	35	29
L.K.	6	30	0	359	15	35	31
L.K. + DOCA	6	4	2	355	21	32	31

DOCA rats received 2 mg of desoxycorticosterone acetate daily for 30 days.
L.K. rats received the diet low in potassium for 30 days.
L.K. + DOCA rats received both the low potassium diet and desoxycorticosterone acetate for 4 days.

and the injections of desoxycorticosterone acetate produce significant lowering of heart potassium in 4 days. It will be noted that heart sodium is raised significantly. The data do not demonstrate that the increase in sodium represents an intracellular replacement of the lost potassium by sodium since the small amount of tissue did not permit chloride analyses. However, such a replacement is almost certainly indicated since heart water is the same in this group as in the other experimental groups and total water would be increased in relation to tissue solids if extracellular sodium were appreciably increased.

In parallel experiments lasting a week, the hearts were saved for histological examination. Lesions consisting of necrosis of the myocardium and cellular infiltration were found in 4 of 6 rats. Since neither the diet low in potassium nor injection of desoxycorticosterone acetate produced lesions regularly before 20 days, the combination accelerates the production of lesions as well as brings out the loss of heart potassium.

These findings may have some clinical significance since the administration of large amounts of desoxycorticosterone acetate to a patient receiving a diet low in potassium or large infusions of glucose solutions may lead to loss of muscle potassium. Indeed, certain data[8] show that continuous infusions of salt solution alone lead to deficits of potassium.

Summary. A diet low in potassium when accompanied by injections of desoxycorticosterone acetate produces in 4 days loss of potassium and gain in sodium in the rat heart. Lesions are induced within a week by this treatment.

[8] Stewart, J. D., and Rourke, M. G., *J. Clin. Invest.*, 1942, **21**, 197.

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Role of Inositol in Alopecia of Rats Fed Sulfasuxidine.

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Nielsen and Elvehjem[1] reported that feeding of sulfasuxidine* to rats maintained on a synthetic diet resulted in a biotin and folic acid deficiency. This finding was confirmed by Welch and Wright,[2] Martin.[3] and Totter and Day.[4] The present report concerns the effectiveness of inositol in the pre-

[1] Nielsen, E., and Elvehjem, C. A., *J. Biol. Chem.*, 1942, **145**, 713.

* Sulfasuxidine (succinyl-sulfathiazole) was kindly supplied by Dr. W. A. Feirer of Sharp and Dohme, Glenolden, Penn.

[2] Welch, A. C., and Wright, L. D., *J. Nutrition*, 1943, **25**, 555.

[3] Martin, G. J., PROC. SOC. EXP. BIOL. AND MED., 1942, **51**, 353.

[4] Totter, J. R., and Day, P. L., *J. Biol. Chem.*, 1943, **147**, 257.