a basis for correlation of two previous observations that had no apparent common relationship, namely protein tends to prevent and fat[5] tends to aggravate the lesions. In fact, Hoelzel and Da Costa[4] have maintained that the changes are produced by a protein deficiency. Since methionine and perhaps other amino acids have a choline-like action, protein would be expected to help prevent the lesions. Because choline is necessary for normal fat metabolism or transport a choline deficiency would tend to follow ingestion of a high fat diet.

Summary. Choline is necessary to help maintain normal squamous epithelium in the forestomach of rats fed white flour. This observation explains why protein tends to prevent and fat tends to increase hyperplasia of the forestomach epithelium.

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Salt after Adrenalectomy. III. Carbohydrate Stores in Adrenalectomized Rats Given Various Levels of Sodium Chloride.*

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(Introduced by Herbert M. Evans.)

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In a preceding communication[1] it was shown that giving 1% sodium chloride solution to adrenalectomized rats enabled them to grow and maintain a satisfactory state of health, and on the other hand, a larger amount of sodium chloride proved to be deleterious. The capacity of the adrenalectomized rats to store fed glucose has been studied under these 2 types of treatment. A group of 35 adrenalectomized rats and 21 "sham adrenalectomized" control rats were allowed to drink 1% sodium chloride solution in place of tap water. The average NaCl intake was 940 mg daily. Another group of 5 adrenalectomized rats and 4 controls were given tap water to drink. The NaCl intake was 339 mg daily for the former and 601 mg for the latter. The rats were kept in individual cages in a constant temper-

[4] Hoelzel, Frederick, and Da Costa, Esther, *Am. J. Digest. Dis. and Nutrition*, 1937, **4**, 325.

[5] Fujimaki, Yoshitomo, *Trans. Jap. Path. Soc.*, 1931, **21**, 708.

* We wish to acknowledge the assistance of the Federal Works Progress Administration, Project No. OP 65-1-08-62, Unit A-5, and the Christine Breon Fund.

[1] Anderson, E., Joseph, M., and Herring, V., Proc. Soc. Exp. Biol. and Med., 1940, **44**, 477.

ature chamber at 28.5° C. The food and fluid intake and the gain in body weight were carefully measured. These data are given in Chart II of the previous communication.[1] The methods used and the standardization of the conditions necessary for this experiment have been described previously.[2]

Another group of 8 adrenalectomized rats and 8 controls on 1% NaCl were followed for 176 days. These animals were tested for completeness of adrenalectomy by withdrawing salt and allowing them to go into a state of adrenal insufficiency. This appeared within 24 hours. A striking decrease in food intake occurred and the loss in weight averaged 10 g daily. It was not safe to permit the animals to continue without salt more than 48 hours. At autopsy a block of tissue was removed from each adrenal site and serial sections made. No evidence of adrenal tissue was found. This group of animals continued to grow during the entire period of the experiment. Fig. 1 shows the gain in body weight of these animals compared with the gain in weight of the control rats. The gain in weight is practically the same for both groups.

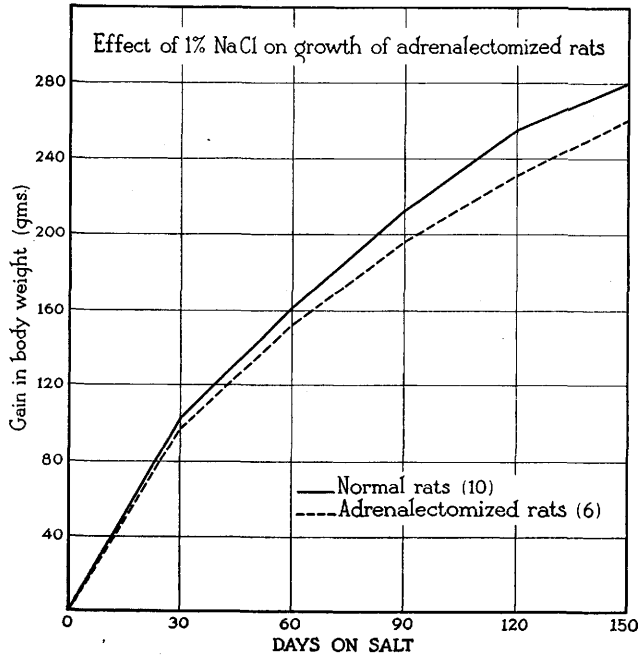

Fig. 1.
Showing the Gain in Body Weight of Adrenalectomized Rats Given 1% NaCl.

[2] Anderson, E., Joseph, M., and Herring, V., Proc. Soc. Exp. Biol. and Med., 1939, **42**, 785.

TABLE I.
Effect of 1% NaCl on Carbohydrate Stores of Adrenalectomized Rats.

	No. animals	Glucose fed, g	Intestinal absorption %	Blood sugar mg %	Muscle Glycogen mg %	Liver glycogen mg %
Group I 15 days post-oper.						
No NaCl soln. adr.*	5	1.2	73.3 ± 6.8‡	133 ± 15.6	535 ± 44.3	432 ± 224
No NaCl soln. contr.†	4	1.2	91.3 ± 5.1	138 ± 6.2	558 ± 54.8	1,977 ± 264
Group II, 10 days post-oper.						
1% NaCl adr.	10	1.2	92.7 ± 2.0	133 ± 6.2	606 ± 53.4	1,494 ± 54.1
1% NaCl control	6	1.2	95.5 ± 1.1	138 ± 5.8	712 ± 19.3	1,760 ± 266.6
Group III 15 days post-oper.						
1% NaCl adr.	7	1.2	88.1 ± 3.6	137 ± 12.7	543 ± 30.5	1,074 ± 178.4
1% NaCl control	4	1.2	97.2 ± 1.0	128 ± 17.9	760 ± 73.6	2,150 ± 337.2
Group IV 20 days post-oper.						
1% NaCl adr.	7	1.2	93.8 ± 3.5	143 ± 6.7	589 ± 35	1,056 ± 189.1
1% NaCl control	4	1.2	98.5 ± 1.3	155 ± 5.0	800 ± 128	1,711 ± 427
Group V 50 days post-oper.						
1% NaCl adr.	4	1.2	93.2 ± 4.5	147 ± 20.9	—	1,412 ± 160
1% NaCl control	3	1.2	98.0 ± 1.0	195 ± 28.8	—	1,933 ± 202
Group VI 176 days post-oper.						
adr.	8	2.5	89.6 ± 2.2	123 ± 11.4	—	1,233 ± 175
control	8	2.5	73.3 ± 3.2	177 ± 27.6	—	1,886 ± 145

*Adrenalectomized.
†Sham adrenalectomized.
‡Standard errors used throughout this table.

TABLE II.
Effect of High NaCl Intake in Adrenalectomized Rats upon Carbohydrate Stores.

	No. animals	NaCl daily g	Intestinal absorption %	Blood sugar mg %	Muscle Glycogen mg %	Liver glycogen mg %
Adrenalectomized rats 15 days post-oper.						
5% NaCl { adr.	7	0.8†	86.7 ± 1.4	155 ± 16.5	124 ± 28.7	630 ± 159.1
5% NaCl { control*	4	0.8†	95.7 ± 1.5	141 ± 4.9	435 ± 70.8	1,202 ± 292
1% NaCl { adr.	7	0.32‡	88.1 ± 3.6	137 ± 12.7	543 ± 30.5	1,074 ± 178.4
1% NaCl { control	4	0.41‡	97.2 ± 1.0	128 ± 17.9	760 ± 73.6	2,150 ± 337.2
Adrenalectomized rats 20 days post-oper.						
5% NaCl { adr.	6	0.8†	89.3 ± 3.0	126 ± 7.9	415 ± 60.7	676 ± 87.9
5% NaCl { control	3	0.8†	94.0 ± 4.4	137 ± 15.9	423 ± 52.8	1,433 ± 74.5
1% NaCl { adr.	7	0.34‡	93.8 ± 3.5	143 ± 6.7	589 ± 35	1,056 ± 189.1
1% NaCl { control	4	0.49‡	98.5 ± 1.3	155 ± 0.5	800 ± 128	1,711 ± 427

*Control rats subjected to a sham adrenalectomy.
†Amount of NaCl given by stomach tube.
‡Amount of NaCl taken voluntarily in 1% solution.
Standard errors have been used throughout this table.

The rats were sacrificed on the 10th, 15th, 20th, 50th and 176th days post-adrenalectomy. The animals were fasted for 20 hours, and then fed a standard glucose meal (5 cc of 25% solution=1.2 g glucose to all the animals except those of Group VI which received 10 cc of 25%=2.5 g glucose) and 4 hours later anaesthetized and the blood and tissues taken for the following determinations: intestinal absorption of glucose, blood sugar, liver glycogen and muscle glycogen. The results are given in Table I. It will be seen from this table that 1% NaCl enables the adrenalectomized rat to absorb fed glucose and to store liver glycogen almost as well as its corresponding control animal. The value for liver glycogen remains high in all the groups through 176 days. The levels of blood sugar were not significantly different in the experimental and control groups over the entire period of 176 days. The muscle glycogen values were unchanged over the period in which they were determined.

In another experiment the effect of high NaCl intake on carbohydrate storage was studied and compared with the effect of 1% NaCl. Thirteen adrenalectomized rats and 7 control rats subjected to a "sham adrenalectomy" were given 4 cc of a 5% NaCl solution 4 times a day. The food, fluid and salt intake on this set of animals has been reported in the preceding communication.[1] The total NaCl intake was 1234 mg daily for the adrenalectomized rats and 1352 mg daily for the controls. The animals were sacrificed on the 15th and 20th days post operative. Similar determinations were made as for the groups described above. The data are given in Table II. On inspection of these data, it is apparent that with the higher NaCl intake there is an impairment in the storage of liver glycogen from fed carbohydrate by the 15th post operative day and this is further substantiated by the findings on the 20th day after adrenalectomy. Furthermore, the high NaCl intake seemed to prevent the high glycogen storage seen in intact animals on 1% NaCl.

Long, Katzin and Fry[3] state that so long as adrenalectomized rats are fed, normal levels of liver and muscle glycogen are found. This appears to be true after adrenalectomy provided a certain optimal amount of NaCl is given. For instance, the daily intake of 650 to 940 mg NaCl enables the adrenalectomized rat to store fed glucose almost as well as the normal animal for 176 days post operative but adrenalectomized rats kept on NaCl levels below or above this optimal range are not able to store fed glucose as normal animals. For instance, the adrenalectomized rats of Group I, Table I given tap water to drink had a marked reduction in liver glycogen by the 15th day post operative.

[3] Long, C. N. H., Katzin, B., and Fry, E., *Endocrinology*, 1940, **26**, 309.

Summary. The adrenalectomized rat given 650-940 mg NaCl daily stores fed glucose as liver glycogen almost as well as does an intact animal. A NaCl intake of 1200 mg seems to interfere with glycogen storage in both adrenalectomized and intact animals.

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Electrical Conveyance of the Melanophore Hormone.

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The treatment of vitiligo[1] with the melanophore hormone as developed in this Institute has all the disadvantages peculiar to intradermal injections. Electrical conveyance has now been explored as a means of facilitating therapeutic application. Experiments have been carried out along 2 lines, *i. e.*, conveyance of the hormone *in vitro* and conveyance through the skin.

1. *Conveyance in vitro.* The apparatus employed (Fig. 1) consists

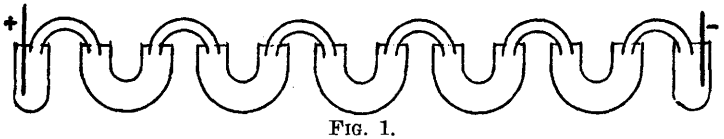

FIG. 1.

of a series of 7 vessels joined together by means of siphons filled with saline. The solution containing the melanophore hormone, which has been purified by a modification of Stehle's method,[2] is placed into the central vessel. The distribution of the hormone in the system was ascertained by biological test after 1 mA current had passed through the apparatus for 24 hours.

Dietel's method[3] of testing, adapted to the native species *Leptodactylus ocellatus,* was employed. In all the anodic vessels the test was negative, whereas decreasing amounts of melanophore hormone were found in the 2 vessels nearest to the central one. Saline solution was used in the experiments described here, which were preliminary, because we lacked the necessary potential for experiments with dis-

1 Mussio Fournier, J. C., Cervino, J. M., and Conti, O., *Bull. de l'Acad. de Med.,* 1938, **120**, 770; Mussio Fournier, J. C., Cervino, J. M., and Conti, O., in press.

2 Stehle, R. L., *J. Pharm. and Exp. Therap.,* 1936, **57**, 1.

3 Dietel, F. G., *Klin. Wochenschr.,* 1932, **11**, 2075.