TABLE I. Detection of Mumps Virus Antigen and Isolation of Mumps Virus.

Tissue	Detection of antigen	Isolation of virus
Left parotid gland	+	+
Right " "	+	+
Kidney	—	—
Liver	—	—
Spleen	—	—
Adrenal	—	—
Lung	—	—
Heart	—	—
Skeletal muscle	—	—
Pancreas	—	—
Lymph node	—	—
Fat	—	—
Stomach	—	—
Small bowel	—	—
Large bowel	—	—
Testis	—	—
Meninges	—	+
Cortex	?	+
Cerebellum	?	+
Medulla	+	—
Cervical cord	+	+
Lumbar cord	+	+

tunately the blood was not examined for the presence of virus. Since, however, the virus could not be detected in the other organs examined (with the exception of the parotid glands) it seems unlikely that it was present in the blood in high concentration at the time the animal was sacrificed. Had the experiment been permitted to run longer than 96 hours the distribution of virus might have been different. The results are compatible with the clinical observation that the CNS of man may be invaded by the virus during the course of natural infections.

The data presented in the present paper furnish additional evidence as to the value of fluorescein labelled antibody as a histochemical tool. Fair agreement was produced by the two methods—virus isolation and demonstration of viral antigen—employed to investigate the distribution of mumps virus in the experimentally infected monkey. In 14 of the 22 tissues examined the presence of virus could not be demonstrated by either method. In 4 tissues both methods yielded positive results. Virus was isolated from 2 tissues (cortex and cerebellum) in which the application of labelled antibody yielded questionable results due to technical difficulties. One tissue (medulla) gave a positive reaction with labelled antibody, but no virus was isolated from it. Conversely, no mumps antigen could be detected in the meninges, although the virus was itself isolated from both samples of this tissue that were tested. Thus, of 22 trials the methods were in agreement 18 times. In two instances they yielded divergent results, while in the case of 2 tissues, the results could not be compared because of technical difficulties.

Summary. The distribution of mumps virus in one experimentally infected monkey 96 hours after inoculation has been investigated by simultaneous labelled antibody and virus isolation studies. The results furnished by the two methods were in fair agreement. Virus was demonstrated in CNS (brain and spinal cord) as well as in both parotid glands, although the animal had shown no neurological signs. No virus was demonstrated in the other organs examined.

Received February 15, 1951. P.S.E.B.M., 1951, v76.

Lack of Effect of Folic Acid on Scurvy in Guinea Pigs. (18562)

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The frequent association of scurvy with megaloblastic anemia in infants was pointed out by Zuelzer(1) in his initial description of this hematologic disorder. Scurvy was then considered only as a manifestation of the poor nutritional status of the patient and other-

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1. Zuelzer, W. W., and Odgen, F. N., *Am. J. Dis. Child.*, 1946, v71, 211.

wise unrelated to megaloblastic anemia. The possibility of a closer relationship between vitamin C and folic acid has been suggested by reports of folic acid deficiency in rats which responded to the administration of ascorbic acid(2,3). More recently May(4) has stated his belief that chronic deficiency of ascorbic acid leads to a deficiency of folic acid or related compounds which results in a megaloblastic pattern in the marrow.

In an attempt to isolate the effect of folic acid on megaloblastic anemia and scurvy, a 6-month-old male infant in this hospital, afflicted with both diseases, was placed on a low vit C diet and given therapeutic amounts of folic acid. The hematologic response was excellent and corresponded to that described by Zuelzer. Serial roentgenograms of the long bones suggested that the scorbutic process was also ameliorated during the administration of folic acid in the presence of a low and presumably inadequate vit. C intake. These observations led us to undertake studies to determine the effect, if any, of folic acid on vit. C deprivation in the guinea pig.

Male guinea pigs weighing approximately 150 g were fed, *ad libitum,* a scorbutogenic diet(5). Normal growing guinea pigs on this diet develop signs of scurvy within 18 to 25 days and die shortly after. Ten animals were given 1.5 mg of folic acid† twice daily by subcutaneous injection. Nine animals were given an equivalent volume of physiological saline solution subcutaneously twice daily. Two animals receiving no treatment were given ascorbic acid beginning on the twenty-first day of the diet. The animals were weighed daily and at death were autopsied. As this was initially considered a pilot experiment, only death rates were compared in the 2 groups; phosphatase determinations as described by Gould and Shwachman(5) were not made. The cumulative death rates were practically identical, all the animals in both groups having died by the thirty-second day. The experiment was therefore repeated using 250 g male guinea pigs and 3 mg of folic acid† twice daily; the control animals were given an equivalent volume of physiologic saline solution. Cumulative death rates were the same as in the initial experiment. Six untreated animals on the scorbutogenic diet died between the nineteenth and twenty-ninth days. The combined cumulative mortality rates for the 2 experiments are shown in Fig. 1. Of the 2 animals given ascorbic acid beginning on the twenty-first day of the diet in the first experiment, one died after 6 days. The other recovered, and was sacrificed on the thirty-seventh day of the diet. Histologic study of sections from the region of the knees in representative animals in each group showed characteristic changes of active scurvy in all except the animals treated with ascorbic acid; these showed healing or healed scurvy.

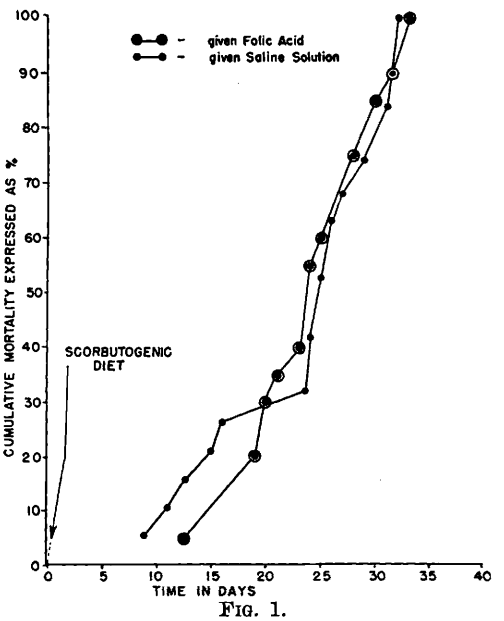

FIG. 1.

Cumulative mortality rates of guinea pigs on scorbutogenic diet.

Discussion. The evolution of scurvy and the death rates are those to be expected in

2. Johnson. B. C., and Dana, A. S., *Science,* 1948, v108, 210.

3. Woodruff. C. W., and Darby, W. J., *J. Biol. Chem.,* 1948, v172, 851.

4. May, C. D., Nelson, E. N., Lowe, C. U, and Salmon, R. J., *Am. J. Dis. Child.,* 1950, v80, 191.

† Folic acid was administered as Folvite supplied through the courtesy of Lederle Laboratories.

5. Gould, B. S., and Shwachman, H., *J. Biol. Chem.,* 1943, v151, 439.

guinea pigs on the scorbutogenic diet if antiscorbutic substances are withheld. Folic acid apparently did not affect the animals favorably or adversely. An adverse effect might have been expected if larger doses of folic acid were given, since the guinea pig is known to be more susceptible to toxic effects of folic acid than other animals(6). On a weight basis, the dosage given was greater than that used therapeutically in humans. The development of scurvy in monkeys receiving folic acid has been observed by May (4). The roentgen observations of amelioration were therefore puzzling. Later, a careful review of records revealed that a single dose of 200 mg of ascorbic acid, administered intravenously as part of a tolerance test performed to establish the diagnosis of scurvy, could have been responsible for the regression of the skeletal lesions noted roentgenographically.

Conclusions. Under conditions of vit. C deprivation, folic acid has no apparent antiscorbutic effect in guinea pigs.

6. Harned, B. K., Cunningham, R. W., Smith, H. D., and Clark, M. C., *Ann. New York Acad. Sc.*, 1946, v8, 289.

Received February 16, 1951. P.S.E.B.M., 1951, v76.

Thioglycollate Inactivation of Posterior Pituitary Antidiuretic Principle as Determined in the Rat.* (18563)

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Recently, Ralli and her collaborators(1) suggested that "the antidiuretic activity of commercial Pitressin can be fractionated into at least two substances, one of which is active in the rat but not in the dog." This suggestion was based upon the observation that the antidiuretic action of Pitressin could be abolished by treatment with 0.01 M sodium thioglycollate(2) as determined by intravenous assay in dogs but not by similar treatment of Pitressin when assayed by intraperitoneal injection in rats. On the other hand, the chloruretic action of large doses of Pitressin treated with thioglycollate was reduced 45% in comparison with untreated Pitressin. (Whether the chloruretic action was abolished cannot be determined from the published experiments which lacked control groups receiving no Pitressin.) Ralli and her colleagues also concluded that in the dose range of 10-100 milli-units the chloruretic effect of Pitressin in rats was related to its antidiuretic action.

In their study of the antidiuretic action of thioglycollate-treated Pitressin, Ralli *et al.*(1) reported control experiments only with Pitressin containing a similar concentration of acetate. Sodium thioglycollate (1 ml of 0.01 or 0.05 M solution) has no antidiuretic effect after intravenous injection into the dog(2). However, its action on water diuresis in the rat after either intraperitoneal or intravenous injection has never been reported. In the experiments about to be described it was found that solutions of sodium thioglycollate have an "antidiuretic" action after intraperitoneal or subcutaneous injection into rats but not after intravenous injection.

Material and methods. Adult rats of either sex of the Long-Evans strain were used. In all the experiments a special lot of Pitressin†

* This investigation was in part supported by grants from the Playtex Park Research Institute and E. R. Squibb and Sons.

1. Ralli, E. P., Raisz, L. G., Leslie, L. H., Dumm, M. E., and Laken, B., *Am. J. Physiol.*, 1950, v163, 141.

2. Ames, R. G., Moore, D. H., and van Dyke, H. B., *Endocrinology*, 1950, v46, 215.

† The Pitressin was kindly furnished by Dr. A. C. Bratton of Parke, Davis and Co.