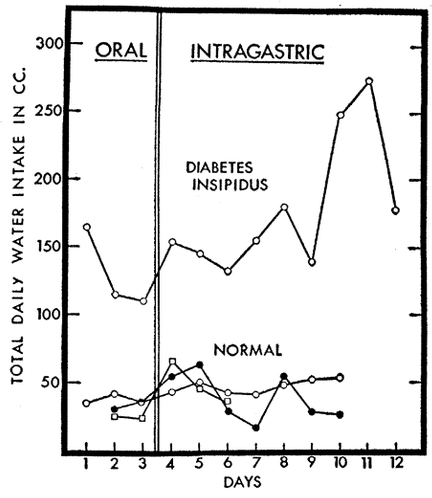

Fig. 1. Total daily intake of water obtained by three normal rats and a rat with diabetes insipidus during several typical days of bar pressing for oral intake and during a continuous period of bar pressing for direct intragastric injection.

of oral intake of water obtained by bar pressing and 3 to 9 nine days of direct intragastric water intake with each stomach load held within narrow limits for three normal rats and the diabetic rat. Note that total daily water intake remains within normal limits during the periods of direct intragastric self-injection. During these same periods the animals' weights remained essentially constant, and their average daily response totals were between 19.0 and 23.4 bar presses. In addition to demonstrating that the method described here can be used for long-term study of fluid intake, the relative constancy of these data show that preingestion factors are not necessary for the day-to-day regulation of water intake in the normal and the diabetic rat. This means not only that oropharyngeal sensations and feedback from consummatory responses are not essential here but also that this regulation can occur without the performance of the consummatory acts of licking and swallowing. Secondly, the data for the diabetic animal make it clear that the bar-pressing response used in the present method is sensitive to an increased need for water produced by a major alteration in the internal fluid balance.

The following additional facts support the view that bar pressing in this situation is related to the animal's need for water. First, the number of responses increases when the animal is required to press the bar several times for a single stomach load of constant size. A normal rat that had been pressing 20 to 25 times a day when every response was reinforced reached a peak of 74 responses when required to make five to six responses for a single stomach load. This adjustment required 2 or 3 days of experience with each of several successively higher ratios before normal regulation occurred. Second, the number of responses fell sharply in two well-trained animals (from 39 to 7, and from 46 to 9) when they were given *ad libitum* access to water by mouth with the bar in place but with the tube carrying water to the solenoid valve clamped shut. And in a third animal the solenoid valve was inadvertently disconnected overnight, depriving the animal of all water. In this case the number of responses rose from 55 during the previous day to 130 during the "deprivation" run. Thus the number of responses rises when water is more difficult or impossible to obtain and falls when it can be obtained freely by mouth.

The tendency toward excess intake that can be seen in Fig. 1 was dramatically revealed by progressively increasing the stomach load per injection between ranges of 0.75 and 42.0 ml for a normal rat and 0.9 and 63.3 ml for the diabetic rat over a period of several weeks. Despite a gradual fall in total daily responses, daily water intake rose rapidly from 30 ml/day to a plateau of 145 ml for the normal animal and from 200 ml/day to a plateau of 350 ml for the diabetic rat before falling off at the very high stomach loads. The normal as well as the diabetic animal produced large volumes of pale urine with the specific gravity of water during these periods of excessive intake. The loss of precise "metering" of water intake by feedback from licking and swallowing which has been suggested as an important role of preingestion factors may account for this phenomenon.

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References and Notes

1. R. T. Bellows, *Am. J. Physiol.* **125**, 87 (1939).
2. E. F. Adolph, *ibid.* **125**, 75 (1939).
3. H. D. Janowitz and M. I. Grossman, *ibid.* **159**, 143 (1949).
4. E. Stellar, R. Hyman, S. Samet, *J. Comp. and Physiol. Psychol.* **47**, 220 (1954).
5. P. Teitelbaum, *ibid.* **48**, 156 (1955).
6. N. E. Miller and M. L. Kessen [*ibid.* **45**, 550 (1952)], working with rats with chronic gastric fistulae, demonstrated that food injected into the stomach by the experimenter is a sufficient reinforcer for learning. This work suggested that the behavior required of the animal in the situation described here would be self-maintaining.
7. This work was done while I was a fellow of the National Foundation. I am grateful for the generous cooperation of Philip Teitelbaum in all phases of this research and to Eliot Stellar for his advice and criticism of the manuscript.

5 October 1959

Fatal Disease of Swine Due to Encephalomyocarditis Virus

Abstract. Encephalomyocarditis virus was isolated from the organs of swine dying during an outbreak of an acutely fatal disease occurring on a farm in Panama. The outstanding lesion was severe myocarditis. Pigs inoculated with the viral isolate developed a systemic infection with myocarditis.

During the past 20 years the encephalomyocarditis virus has been sporadically isolated from rodents and primates. It has been implicated as a cause of human disease. This report describes an outbreak of a fatal disease of swine due to the encephalomyocarditis virus. To our knowledge, the association of this virus with disease in domestic animals has not been previously recognized.

The outbreak occurred in July 1958 on a large commercial swine farm located 10 miles west of Panama City, Republic of Panama. Approximately 30 pigs died over a 20-day period in one overcrowded feed lot containing 300 3- to 5-month-old Duroc and Hampshire pigs. No deaths occurred in other pigs of the same age in an adjacent lot separated from the affected group by a narrow roadway.

The drove was frequently examined during the outbreak. The general appearance of the pigs was good. Some mild coughing and lameness were noted, which the owner did not consider unusual for his herd. All the pigs had been previously vaccinated for hog cholera. The feed ration consisted of corn with appropriate supplements.

Most deaths occurred at night; despite regular visits to the farm, the agonal stage was witnessed by only one of us, on a single occasion. The pig in question suddenly collapsed in severe dyspnea and died within a few minutes. The owner of the farm reported a similar observation.

Eight of the pigs that died during the outbreak were autopsied. Hydrothorax, hydropericardium, and ascites were frequently observed. The lungs were congested and edematous, with localized consolidation. The heart was soft and pale, with minute yellowish areas suggestive of necrosis. The meninges were slightly congested. Tissues for histopathological examination were selected from three animals which had died on the 6th, 10th, and 12th days, respectively, of the outbreak. The findings were severe myocarditis with round cell infiltration, vascular congestion, edema, and degeneration of the myocardial fibers; mild pneumonitis and pulmonary edema; mild meningitis and minimal congestion of the brain, with spotty areas of neuronal degeneration.

Bacteriological and virological studies were performed on two of these animals. Cultures of liver, spleen, brain, and blood were negative. Stained blood films were negative for blood protozoa.

An agent pathogenic for mice was repeatedly isolated from the lung and spleen of both of these pigs and from one of the brains. It was recovered from the brain, lung, and spleen of inoculated mice and passaged serially by the intracerebral, intraperitoneal, and intranasal routes. On initial passage in 2- and 21-day-old mice the average incubation period was 48 hours and 5 days, respectively. In later passages the incubation period in young adult mice was shortened. Usually adult mice presented flaccid posterior paralysis followed by coma and death, although some acute deaths with no paralysis occurred. Brains from moribund mice contained 10^6 to 10^8 mouse LD_{50} doses. The virus was pathogenic for young hamsters and guinea pigs. It was reisolated and passaged in cell cultures of rhesus monkey kidney, hamster kidney, and HeLa cells. An agglutinin for sheep erythrocytes was demonstrated in infected mouse-brain tissue and in hamster-kidney culture fluid.

The agent was identified by neutralization tests in mice and tissue culture with a hyperimmune rabbit antiserum prepared against the American Type Culture prototype strain of encephalomyocarditis virus (*1*). The identification was confirmed at the Walter Reed Army Institute of Research (*2*). Viruses of the encephalomyocarditis group have never previously been isolated or studied in our Panama laboratories.

A pig, exhibiting fever, listlessness, and anorexia over a 48-hour period, developed a significant rise in neutralizing antibodies to the isolate. Neutralizing antibodies were found in the sera of rats (*Rattus rattus*) trapped on the farm in April 1959. Preliminary studies failed to demonstrate antibodies in the sera of ten men in contact with the swine at the time of the outbreak.

The disease was successfully reproduced by inoculation of two young pigs with the mouse-passaged virus. One pig was inoculated intercerebrally, the other, intraperitoneally. In both animals viremia was demonstrated on the 2nd day, and signs of disease appeared on the 3rd day. The pig inoculated intracerebrally developed progressive paralysis; it was bled and sacrificed on the 11th day. The neutralizing index of this serum was 60. Virus was not recovered from the organs tested. Severe myocarditis and moderate encephalitis were demonstrated histologically. The pig inoculated intraperitoneally became inappetent and listless and died on the 4th day after inoculation. The virus was recovered from the lung and pooled liver-spleen specimens. Histologically severe myocarditis was found. The brain was not examined. The virus strains from both animals were reidentified.

To our knowledge, this report describes for the first time the natural infection of swine with the encephalomyocarditis virus and the most extensive outbreak of encephalomyocarditis infection in man or animals in which the virus was recovered. The outstanding lesion in these naturally infected animals was severe myocarditis. The evidence presented suggests that myocardial failure was the primary cause of death. Contamination of food and water with the excreta of infected rodents and swine possibly contributed to the spread of the virus in this outbreak.

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Notes

1. The encephalomyocarditis immune serum was supplied by William Pond of the National Institute of Allergy and Infectious Diseases.
2. We thank Malcolm S. Artenstein and Nancy Rogers of the Walter Reed Army Institute of Research for reidentifying the viral isolate.
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18 September 1959

Transport of Strontium-90 in Runoff

Abstract. Only a small portion of the strontium-90 that fell on cultivated soils was removed in runoff. The concentration of strontium-90 was usually about 10 times higher in the soil carried by the runoff than in the soil from the plow layer of the plots. Thus, a considerable concentration of Sr^{90} could occur in areas where runoff sediments accumulate.

Strontium-90 is deposited on soil surfaces chiefly through rainfall (*1*). Thus it would seem to be especially likely to move in surface runoff. The extent of this movement was measured on plots which had previously been established for the study of soil losses by rainfall erosion.

Table 1. Strontium-90 in fallout and runoff from corn, oats, and clover plots at La Crosse, Wis., in 1957.

Period	Strontium-90 ($\mu\mu c/ft^2$)			
	Fallout	Runoff from plot		
		Corn	Oats	Clover
3/13–5/14	83	1.1	0.8	None
5/14–5/25	39	0.8	0.6	None
5/25–6/5	30	8.6	8.3	0.4
6/5 –6/15	12	0.2	0.2	0.1
6/15–7/3	20	0.1	0.3	0.2
7/3 –7/16	23	0.1	0.2	0.1
7/16–7/21	12	0.4	0.1	0.1
7/21–8/18	38	0.2	None	None
Total	257	11.5	10.5	0.9

Samples of runoff and rainfall were collected in 1957 at La Crosse, Wis., and Tifton, Ga. (*2*). The La Crosse plots are on Fayette silt loam with a 16-percent slope and are planted to corn, oats, and clover in rotation. The Tifton plots are on Tifton loamy sand with a 3-percent slope and are planted to corn, peanuts, and oats in rotation. One plot was sampled for each crop. The length of the plots was 72.5 ft at La Crosse and 83 ft at Tifton.

Rainfall and runoff samples were collected after each major runoff. The fallout of Sr^{90} was collected by taking rainfall samples in washtubs 3 ft in diameter, which were left in the open near the plot areas at all times. A few quarts of dilute $Sr(NO_3)_2$ solution were kept in the tub to aid in dust retention and to act as a carrier. At La Crosse, the runoff was stirred and an aliquot was taken for analysis. At Tifton, the sediment was dried and mixed, and a sample was taken for analysis. The supernatant was discarded at Tifton because it had a negligible calcium content (*3*)—a finding which indicated that the supernatant would also contain negligible amounts of Sr^{90} since strontium is adsorbed on soils more readily than calcium (*4*).

The Sr^{90} content of the samples was determined at Beltsville, Md. The runoff samples were dried, and 0.1 mole of $Sr(NO_3)_2$ was added as carrier. Strontium-90 was extracted by overnight digestion with hot 4*N* HCl, filtration, and leaching with 1*N* HCl. Interfering radioactive elements were removed by scavenging precipitations; $Y(OH)_3$ was formed in the first of these precipitations, $BaCrO_4$ in the next two and $Y(OH)_3$ in the last. Strontium-90 was determined by separation and by following the decay of its yttrium-90 daughter. Rainfall samples were dried and analyzed by the same procedure, except that no further $Sr(NO_3)_2$ was added and the samples were only slightly acidified with HCl.