

mammary duct systems of spayed female mice. Differences in numbers of duct junctions per unit area must be interpreted cautiously because, in the mammae of intact or oestrogen-treated spayed mice the density of duct junctions is not related in a simple manner to the area of the duct system (Flux)(9). However the number of duct junctions per unit area in the mammae of the mice treated with vinyltestosterone was much greater than in those of comparable sizes in either intact or oestrone treated spayed mice in other experiments (Flux)(9) and it appeared certain that vinyltestosterone stimulated the growth of side branches in the duct systems. As far as effects on the weight of the uteri, and thymus and adrenal glands of spayed mice were concerned the results of this experiment were in accordance with those of Kochakian(1) and Lewis *et al.*(3).

Summary. 17-vinyltestosterone promoted growth in the extent and in the degree of branching of the mammae of spayed female mice but did not cause growth of alveoli on the duct systems. It also caused increases in the weight of the uteri of spayed mice and

depressed the weights of the thymus and adrenal glands.

I am indebted to Dr. S. J. Folley for his interest in this experiment which was carried out in his laboratory and to Ciba Ltd., Horsham, England, through the courtesy of Dr. C. W. S. Taylor, for vinyltestosterone given to Dr. Folley. During the course of the work I received financial support from Dairy Research Institute, New Zealand, and from Department of Scientific and Industrial Research (N.Z.) as a National Research Fellow.

1. Kochakian, C. D., *Recent Progress in Hormone Research*, ed. G. Pincus, 1947, v1, 177.
2. Leatham, J. H., *Trans. New York Acad. Sci.*, Series II, 1950, v12, 234.
3. Lewis, R. A., de Majo, S., and Rosenberg, E., *Endocrinology*, 1949, v45, 564.
4. Emmens, C., and Parkes, A. S., *Nature*, Lond., 1939, v143, 1064.
5. Chamorro, A., *C. R. Soc. Biol.*, Paris, 1943, v137, 87.
6. ———, *ibid.*, 1944, v138, 71.
7. ———, *ibid.*, 1945, v139, 958.
8. Folley, S. J., in *Marshall's Physiology of Reproduction*, 1952, v2, ed. A. S. Parkes, Longmans.
9. Flux, D. S., Thesis, University of Reading, 1953.

Received November 18, 1953. P.S.E.B.M., 1954, v85.

Effect of Aureomycin in Preventing Liver Abscess in Cattle.* (20772)

J. MATSUSHIMA, T. W. DOWE, AND C. H. ADAMS. (Introduced by E. L. R. Stokstad.)

From the Department of Animal Husbandry, University of Nebraska, Lincoln.

Liver abscesses result in the condemnation of about 5% of the livers from cattle slaughtered in Federally inspected meat packing plants in the United States. This loss amounts to more than \$2,000,000 annually. According to the National Livestock Loss Prevention Board(12), the regional incidence of abscesses, telangiectasis and "sawdust"[†] is highest in the western states. Jensen, *et al.*(8)

found that liver abscesses develop in significant numbers, both in the feed lot and on the range. Abscesses appear to develop 80 to 120 days after the cattle are placed in the feed lot. These investigators also observed that telangiectasis develops early and abscesses form later in the fattening period. The studies of Smith(14) indicate that there is a definite relationship between ulcers of the rumen and abscesses of the liver in beef cattle. A recent study by Jensen(9) shows a direct relationship between liver abscesses and stomach injuries. Newsom(13) and Yamamoto(15) in separate investigations found that the predominating organism in abscessed livers was *Actinomyces necrophorus*. Merchant(11)

* Published with the approval of the Director as Paper No. 640, Journal Series, Nebraska Agric. Exp. Station.

[†] A term used to designate a condition of liver characterized by many areas of focal necrosis spread throughout the liver or localized giving the appearance of sawdust on the liver.

classifies the same organism as *Spherophorus necrophorus*. The organism appears to be a normal inhabitant of animal tissues. Animals are not immunized against liver abscesses when cultured filtrates of *Spherophorus necrophorus* are given intraperitoneally(9). Since the organism is frequently a secondary invader in other diseases in which primary necrosis or destruction of tissue exists(11), this may account for its failure to be cultured under *in vivo* conditions in cattle. However, acute hepatic necrosis has been produced experimentally in rats kept on a diet low in casein (2,3) and also on rations containing yeast as the sole source of protein(7). When aureomycin was added to a necrogenic yeast diet(4) it was found to have a significantly beneficial effect by preventing experimental hepatic necrosis in rats. The assumption is made that aureomycin might act through the suppression of the intestinal flora. Abell and Bweridge(1) demonstrated that sulfaguanidine also delayed the development of liver necrosis in rats. Dietary imbalances, such as excessive fat(5,10), carbohydrate(6), or proteins (3) have not been consistent in the development of necrosis in the liver of rats.

The primary objective of the experiment in which the following results are reported was to study the effect of aureomycin in controlling calf scours. The observations of the condition of the livers were made at the time of slaughter following feeding experiments.

Methods. The Milking Shorthorn calves used in these experiments were born during the 1950-51 or the 1951-52 seasons. The newborn calves were given special aureomycin tablets or aureomycin capsules† orally each day for 12 weeks. The tablets and capsules were composed primarily of sugar base and contained 15 mg aureomycin. The calves were paired as evenly as possible with respect to birth weight, age, and age of dam. One calf of each pair was given an aureomycin tablet immediately or shortly after receiving colostrum. Some of the paired calves were fed milk from nipple pails while others were put

on nurse cows. The antibiotic tablets or capsules were given to the test calves in both groups (bucket-fed and calves on nurse cows) at the rate of 15 mg per head daily. The animals used in the 1951-52 fattening test were given one aureomycin tablet or capsule daily during the first 12 weeks after birth while the animals in the 1952-53 feeding test were given two aureomycin tablets every other day.

Results. No case of scours was observed in the test groups which received aureomycin. The calves in the control groups which received no aureomycin scoured intermittently. Severe cases of scours were observed during the first 6 weeks following birth. Approximately 20 percent larger gains were made by the calves which received a daily intake of 15 mg of aureomycin. All of the calves were weaned at approximately 196 days of age. The animals fattened during 1951 and 1952 were put in a dry-lot and shortly after weaning they were fed a ration consisting of 60% corn, 20% oats, 13% dried beet pulp and 7% soybean oil meal. Alfalfa hay was also fed. The animals used in the 1952-53 fattening experiment were turned out to pasture during the summer of 1952 after weaning. Then they were fed a ration consisting of the same grain mixture as above with corn silage and alfalfa hay for 128 days. After this period the silage was eliminated from the ration.

The results of the feeding trials are presented in Table I.

The animals were to be slaughtered when they reached about 1000 pounds but this plan could not be adhered to strictly. Thus, it will be noted that certain animals were fed considerably longer than others.

The liver from each of the steers was examined at the time of slaughter. In several previous feeding experiments with dual-purpose steers where no antibiotics were fed, approximately 75% of the livers were found to be abscessed and those which were not abscessed showed scar tissues. When healthy, normal livers without scar tissues were observed among the group of animals slaughtered in 1952, the histories of these animals were examined. It was found that the animals which received aureomycin during the first

† The aureomycin-hydrochloride for the capsules and the aureomycin tablets were supplied through the courtesy of Lederle Laboratories Division, American Cyanamid Co., Pearl River, N. Y.

LIVER ABSCESS PREVENTION BY AUREOMYCIN

TABLE I. Feed Lot and Slaughter Data.

	Pair	Steer No.	Birth wt, lb	Initial wt, lb	Days on feed	Avg daily gain, lb	Slaughter wt, lb*	Condition of liver
1951-1952 feeding period								
Control	1	314	74	440	314	2.02	1042	Abscess
	2	334	93	610	202	1.63	930	"
	3	337	83	350	314	2.07	999	Sawdust†
	4	313	78	460	314	1.93	1024	Abscess
	5	339	72	335	341	1.83	920	"
		Avg	80	439	297	1.90	983	
Treated	1	325	87	550	202	2.03	905	Normal
	2	308	84	450	282	2.09	940	"
	3	338	82	505	202	2.10	900	"
	4	309	77	415	289	2.02	1000	"
	5	327	63	320	341	1.91	950	Small scar‡
		Avg	79	448	263	2.03	939	
1952-1953 feeding period								
Control	1	56	72	675	284	1.13	981	Abscess
	2	65	72	660	197	1.70	920	"
	3	61	69	620	197	2.11	985	Normal
	4	67	83	565	284	1.81	1020	Abscess
	5	73	60	460	284	2.08	973	Scar‡
		Avg	71	596	249	1.77	976	
Treated	1	58	74	655	197	2.08	970	Normal
	2	64	73	580	284	1.83	1033	"
	3	62	86	550	197	2.36	940	"
	4	70	74	525	284	1.88	997	"
	5	74	65	510	197	2.28	890	"
		Avg	74	564	232	2.09	966	

* 24-hr shrinkage.

† A term used to designate liver that contains many areas of focal necrosis spread throughout the liver giving the appearance of sawdust on the liver.

‡ Passed.

twelve weeks after birth had the normal livers, as shown in Table I. Similar observations were made in the second group of steers slaughtered in 1953.

Summary. Under the conditions of these experiments, it appears that aureomycin prevented scouring during the early preweaning period and thereby did not predispose the treated animals to necrophorus infection.

1. Abell, M. R., and Bweridge, J. M. R., *Can. J. Res.*, 1949, v27, 316.
2. György, P., and Goldblatt, H., *J. Exp. Med.*, 1942, v75, 355.
3. ———, *ibid.*, 1949, v89, 245.
4. György, P., Stokes, Joseph Jr., Smith, W. H., and Goldblatt, H., *Am. J. Med. Sci.*, 1950, v220, 671.
5. György, P., *Med. Clin. N. American*, 1949, v33, 1657.
6. Himsworth, H. P., and Glynn, L. E., *Clin. Sci.*

1944, v5, 93.

7. Himsworth, H. P., and Lindan, O., *Nature*, 1949, v163, 30.8. Jensen, Rue, Frey, P. R., Cross, Floyd, and Connell, W. E., *J. Am. Vet. Med. Assn.*, 1947, v110, 256.

9. Jensen, Rue, Ph.D. Thesis. Univ. of Minnesota, June, 1953.

10. McLean, J. R., and Bweridge, J. M. R., *J. Nutrition*, 1952, v47, 41.11. Merchant, I. A., *Veterinary Bacteriology and Virology*, 4th ed., Iowa State College Press, 1950, p474.

12. National Livestock Loss Prevention Board, Meat Wasted in Marketing Livestock, 1944 Report.

13. Newsom, I. E., *J. Infect. Dis.*, 1938, v63, 232.14. Smith, H. A., *Am. J. Vet. Res.*, 1944, v5, 234.15. Yamamoto, S., *J. Jap. Soc. Vet. Sci.*, 1938, v17, 40.

Received November 18, 1953. P.S.E.B.M., 1954, v85.