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A Highly Fatal Disease of Guinea Pigs.

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Our guinea pig colony is carefully watched for infectious diseases and every attempt is made to eliminate any that appear. For several years deaths have been few and the common guinea-pig pathogens have not been observed. This year, in a group of apparently normal guinea pigs removed for observation, a few young animals were found to have elevated temperatures. Inoculations of organ-suspensions were made into other animals and an agent was secured that is highly fatal for guinea pigs.

When injected subcutaneously, intraperitoneally, intracerebrally, or intranasally, guinea pigs show an elevated temperature in from 2 to 5 days, which persists for approximately 5 days and then drops to normal or below. Leucocyte counts show from 3000 to 5000 cells, while the red corpuscles are normal in number and volume. The infection can be transmitted by contact, but so far attempts to infect by feeding have been unsuccessful. Guinea pigs die in about 2 weeks after inoculation, and to date not a single animal has survived.

Autopsy shows a loss of subcutaneous fat, with dilated blood vessels in the subcutis. Animals weighing 250 g will lose from 50 to 75 g, while those weighing 500 g will lose from 125 to 175 g. The kneefold lymph-nodes are enlarged and reddened. The stomach is collapsed and the abdominal cavity appears too large for its contents. The abdominal and thoracic viscera show no characteristic gross pathology. Pneumonia is absent except in animals inoculated intranasally and those infected by contact.

When guinea pigs carrying hemolytic streptococcus or *B. bronchisepticus* are inoculated extensive pneumonia is usually found at autopsy. The combined infection of hemolytic streptococcus and the agent can be transmitted by contact and in exposed animals the pulmonary lesions are the predominating feature. Exposure-experiments with *B. bronchisepticus* and the agent have not been done.

Young white mice are not favorable animals for use, since after intracerebral, intranasal, or intraperitoneal injection only a portion die. Intracerebral injection into rabbits produces a fever of short duration. Cutaneous inoculation of rabbits causes no local lesion. Intratesticular injection has resulted in an orchitis in one animal;

subinoculations from this animal have produced orchitis in all injected rabbits, and the testicular suspension injected into guinea pigs has caused the disease described above. Intraperitoneal injection into 2 cats has been negative.

The agent causing this infection is in suspensions of various organs, in tracheal washings, and in the blood serum. Titrations of sera from infected guinea pigs have shown that from 1×10^{-3} to 1×10^{-5} cc will cause disease. Heating of infective serum for 20 minutes at 50°C has not destroyed its activity. Serum heated at 55°C for the same time gave a prolonged incubation period, while serum heated for 20 minutes at 60°C was inactive.

Cultures of infective organ suspensions or serum show no growth on ordinary media. The agent readily passes through tested Berkefeld N filters, and Berkefeld W filtrates cause disease but death occurs 2 to 3 days later than in the controls. The agent has been propagated on the chorioallantoic membrane of embryonated chicken eggs, and suspensions of membranes from the 18th serial transfer have produced the characteristic disease in guinea pigs.

The source of this infection has not been determined. It is probably not of human origin, for the sera of the 2 caretakers and of 4 of us who have been working with the disease all fail to neutralize the agent.

This agent appears to be a filtrable virus capable of producing disease by itself as well as in association with the common guinea-pig pathogens.

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Metabolism of the Alcoholate of the Trimer of Hydroxypyruvic Aldehyde.

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Hydroxypyruvic aldehyde has been prepared previously by Evans and Waring[1] and by Hynd.[2] Hynd also studied its toxicity and action in insulin shock. Elsewhere the authors[3] have described a

1 Evans, W. L., and Waring, C. E., *J. A. C. S.*, 1926, **48**, 2678.

2 Hynd, A., *Biochem. J.*, 1931, **25**, 11.

3 Evans, W. E., Jr., Carr, C. J., and Krantz, J. C., Jr., *J. A. C. S.*, 1938, **60**, 1628.