

Lethal Factors in Experimental Infections of *Trypanosoma equiperdum* in Rats.*

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A biochemical and pathological study of experimental *Trypanosoma equiperdum* infections in laboratory-bred rats has disclosed that the animals died with a significantly reduced blood pH, lowered alkali reserve, and greatly diminished blood oxygen content, i. e., they finally succumbed to a condition of non-volatile, uncompensated acidosis. These findings are in harmony with the previously reported excessive lactic acid content and reduced oxygen content of the blood of trypanosome-infected rats (Kligler and Geiger;¹ Kligler, Geiger and Comaroff²). A progressive hypoglycemia accompanied these terminal manifestations as has been demonstrated by a number of investigators.

Tissue changes found at death were pronounced vasodilatation and congestion throughout the body, extreme enlargement of the spleen, varying degrees of pulmonary edema and occasionally emphysema, distension of the right heart and engorgement of the coronary vessels. The most significant development was the formation of intravascular blood clots which were most prominent in the left side of the heart, but which were also found in the right heart and the vessels of the lungs. These clots were not post-mortem clots nor were they typical laminated thrombi. They were composed of red cells in a mesh of fibrin enclosing an agglomeration of trypanosomes.

It is believed by the authors that the obstruction to the circulation occasioned by these blood clots results in pulmonary edema which leads progressively to the manifestations of dyspnea, suffocation, and finally asphyxia. These changes account for the terminal syndrome, viz., convulsions, almost total lack of oxygen and excessive amounts of lactic acid in the blood, lowered alkali reserve and subsequently reduced pH of the blood. The reduction of blood

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¹ Kligler, I. J., and Geiger, A., *Proc. Soc. Exp. Biol. and Med.*, 1928, xxvi, 229.

² Kligler, I. J., Geiger, A., and Comaroff, R., *Ann. Trop. Med. and Parasit.*, 1929, xxiii, 325.

sugar is tentatively explained as follows: the utilization of glucose by the rapidly multiplying trypanosomes exercises a progressively increasing demand upon the glycogenolytic function of the liver. The release of glycogen by the liver is probably facilitated under the existing condition of reduced alkali reserve. This may account for the occasional temporary hyperglycemia noted and for the increase in muscle glycogen (Bruynoghe, Dubois, and Bouckaert³). It seems probable that the central necrosis of the liver, caused by the anoxemia, interferes with the glycogenic function of that organ, so that it ultimately fails to supply sufficient glycogen to maintain the normal level of glycemia.

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The Passage of the Spermatozoa and Ova Through the Oviducts of the Rabbit.*

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In earlier papers^{1, 2, 3} I pointed out that the oviducts of the turtle and of the pigeon possess narrow pro-ovarian tracts of cilia by which spermatozoa may be carried from the lower part of these ducts to the neighborhood of the ovary. It was a matter of interest to ascertain whether such tracts obtain in the mammals. For this purpose I have studied the oviducts of the rabbit.

In the female rabbit the vagina is a single median tube about 5 cm. long. This is followed by two independent uteri each about 7 cm. long, opening separately into the vagina. From each uterus a narrow Fallopian tube approximately 7 cm. long is continued forward to its termination in an infundibulum close to the ovary of its own side. After coition spermatozoa reach the anterior end of the uterus in about 2 hours and are found at the infundibulum in 2 hours more. Ovulation takes place about 10 hours after coition. Spermatozoa will live in the female ducts of the rabbit for about 30 hours, the eggs

³ Bruynoghe, M., Dubois and Bouckaert, *Bull. L'Acad. Roy. de Med. de Bel.*, 1927, v, 7, 142.

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¹ Parker, G. H., *PROC. SOC. EXP. BIOL. AND MED.*, 1928, xxvi, 52.

² Parker, G. H., *Am. J. Physiol.*, 1926, lxxxvii, 93.

³ Parker, G. H., *PROC. SOC. EXP. BIOL. AND MED.*, 1930, xxvii, 704.