SERUM POTASSIUM LEVELS AND ADRENAL HYPERTROPHY IN RATS, (Rattus norwegicus), INFECTED BY Trypanosoma lewisi. *

Author: WOLDOW, NORMAN

Source: Bulletin of the Wildlife Disease Association, 5(3) : 297-301

Published By: Wildlife Disease Association

URL: https://doi.org/10.7589/0090-3558-5.3.297

SERUM POTASSIUM LEVELS AND ADRENAL HYPERTROPHY IN RATS, (Rattus norwegicus), INFECTED BY Trypanosoma lewisi.*

NORMAN WOLDOW

Department of Biology
Rice University
Houston, Texas

Among the central issues in ecology are the mechanisms by which population size is regulated. Many factors must be involved in this process and many of these have come under investigation.

One of the most prominent of the recently proposed mechanisms has been the relationship between various stress producing factors, and the secretions of the adrenal cortex. Stress such as infection, cold, hunger, and psychological unpleasantness often has been shown to induce adrenal hypertrophy and increases cortical secretion. This in turn hinders the organism's resistance to further stress and reduces the reproductive rate as well. Both of these reductions are thought to play important roles in the regulation of population size, especially in small mammals.[1,2,3]

Considering their ubiquitous distribution among animals, it is surprising that parasites have received little attention for their possible roles as stressors in the "General adaptation sydrome." Blatantly pathogenic forms may not have been interesting from this standpoint, since their effect on population number may be most easily observed as outright killing of individuals. Less virulent parasites have a peculiar way of appearing to do such little harm to their hosts as to be thought of as non-pathogens.

For whatever reasons, parasitism has not been associated with the adreno-pituitary secretions in the regulation of host population. The purpose of this research was to determine in the laboratory, whether or not a mild, non-pathogenic, endoparasite could induce a stress, such as has been implicated in population control.

Trypanosoma lewisi represents a very common sub-group of the trypanosomes which cause non-pathogenic infections of rodents throughout the world. *T. lewisi* has for its mammalian host the domestic Brown Rat, *Rattus norwegicus,* and is therefore readily available for experimental work. Over the years, *T. lewisi* has become one of the best known parasites and the classic example of a non-pathogen.

---

*This research was part of a thesis submitted to the Biology Department, University of Miami, Miami, Florida.

Trypanosoma as a whole is a widely distributed and abundant parasitic genus, inhabiting all vertebrate classes as well as many invertebrate forms. The genus contains some most virulent pathogens, as well as many mild non-pathogens like *T. lewisi*.[5]

The basic procedure was to inject the experimental animals intraperitoneally with a small sample of infective blood from a carrier rat. Control animals were injected in the same way, using normal rat blood. After two or three weeks, the animals were sacrificed, and careful weighings of the adrenal glands was made. Notation was made of the animals' body weight both before and after the experiment. Also, histological preparations of the adrenals were made in order to determine the relative size of the cortex and medulla.

The results of this experiment for adult males can be seen in Figure 1. Control animals in this run had a mean adrenal weight of 25.5 mg. with a low of 14.5 and a high of 37.3 mg. Infected animals had a mean adrenal weight of 30.5 mg. with a low of 18.5 and a high of 48.6 mg. Although there is a considerable area of overlap, comparison of the two distributions by means of Students' T test shows them to be distinct at the 95% level of significance.

Another way to consider adrenal weight is to divide the weight of the gland by the total body weight. This gives a ratio, of milligrams of adrenal gland per gram of body weight, which is very useful to distinguish hypertrophy from simple growth of the whole body, in which the adrenal glands would also increase. The control animals had a mean value of 0.0883 mg/g while the experimental mean was 0.1191. Histological examination of the enlarged and control glands showed that the cortex was effected (it did not appear that any of the specific zones of the cortex were defferentially enlarged, but cell counts were not made).

Figure 2 shows the result of an experiment using weanling male rats, weighing approximately 50 to 75 grams. The difference between experimental and control animals is more dramatic than in adults. Here the controls had a mean of 16.2 mg, while the experimentals had a mean of 20.7 g, about 25% higher. The level of significance for this difference, computed by Students' T test is greater than 97.5%.

Again, the relative weight of the adrenals was considered. Controls had a 0.247 mg/g mean, and the experimentals had an elevated mean of 0.337 mg/g.

During infection by *T. lewisi*, several important physiological derangements accompany the adrenal hypertrophy, and may be involved in both the causation of the hypertrophy and in the general weakening of the animal. Perhaps the most dramatic is the hosts' substantial antibody response to *T. lewisi* antigens. This has been well documented in the literature of parasitology and need not be discussed here.[5] Also well known is the link between antibody formation and adrenal hypertrophy.[1,2,3]

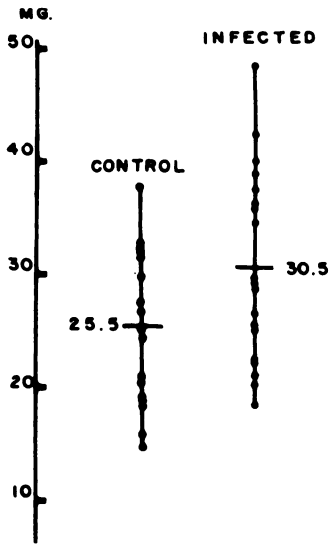

FIGURE 1

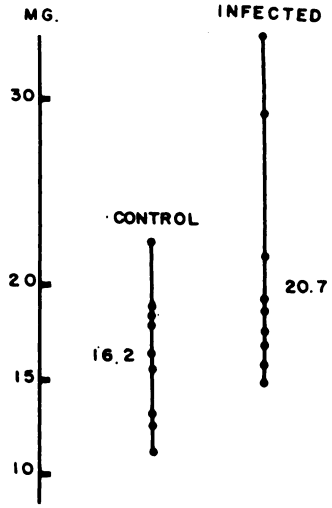

FIGURE 2

FIGURE 1. *Adrenal weight of adult male rats. Control, and after two weeks' infection with* T. lewisi.

FIGURE 2. *Adrenal weight of weanling male rats. Control, and after two weeks' infection with* T. lewisi.

The area of electrolyte balance in trypanosome infection has received some attention in the era prior to the advent of reliable flame photometers. Ikejiani[4] compared the effect of various pathogenic and non-pathogenic Trypanosomes on the quantity of potassium in the blood using a wet chemical colorometric assay.

As in the case of many pathogens, the virulent trypanosomes exerted a cytolytic effect which released large quantities of intra-cellular potassium into the blood. Non-pathogenic forms, including *T. lewisi* were not found to have a cytolytic effect, and host blood potassium was found to remain normal.

Working in conjunction with Neil Chernoff,[6] an effort was made to up date the data on potassium levels in *T. lewisi* infection using modern flame photometric equipment (Beckman IL 200). The results were altogether unexpected.

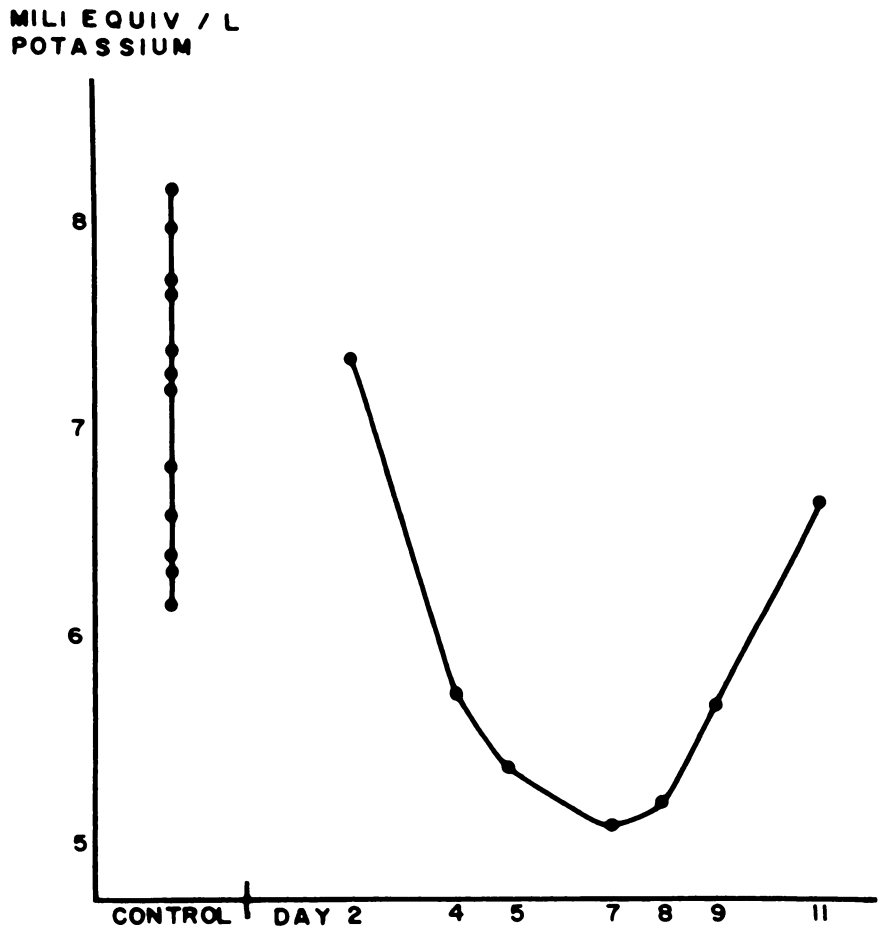

FIGURE 3. *Serum Potassium in rats on days 2 through 11 of infection with* T. lewisi, *measured as milliequivalents of Potassium per liter. Line at the left shows the range of control values. Each point on the curve represents the average of five rats.*

Figure 3 shows the very substantial *decrease* in serum potassium observed during the first week of the infection, with a gradual return to normal levels. This is a temporal relationship between the potassium reduction and the period of most rapid growth of the parasites.

The causes of this reduction and its physiological implications are not known at this time. However, it does seem that reductions of potassium levels such as seen on days 4-7 of infection would be debilitating in some

way, perhaps interfering with neural function by altering potentials across the nerve membrane. The condition is very temporary, and may be of little direct consequence in matters of population control. However, stress to the osmoregulatory system could contribute to the overall picture of debilitation.

These data establish a relationship between trypanosome infection and an important symptomatic factor in the general adaptation syndrome. There is further seen to be a difference in magnitude between adult and weanling rats.

Although these findings do not necessarily mean that parasites such as *T. lewisi* do exert a regulative effect on population in the field, they do suggest that parasitic infections should not be ignored in field studies concentrating on stress and population size.

Literature Cited

1. CHRISTIAN, J. J. 1959 (Jan.). Control of Population Growth in Rodents by Interplay Between Population Density and Endocrine Physiology. Wildlife Disease, 2wd-59.
2. CHRISTIAN, J. J., and D. E. DAVIS. 1964. Endocrines, Behavior, and Population. Science, 146: 1550-1560.
3. CHRISTIAN, J. J., J. A. LLOYD, and D. E. DAVIS. 1965. The Role of Endocrines in the Self Regulation of Mammalian Populations. Rec. Prog. Hormone Res., 21: 501-578., New York, Academic Press.
4. IKEJIANI, O. 1946. Studies in Trypanosomiasis II. The Serum Potassium Levels of Rats During Infection with *Trypanosoma lewisi, Trypanosoma brucei,* and *Trypanosoma equiperdum.* J. Parasit., 32: 374-379.
5. NOBLE, E. R., and G. A. NOBLE. 1961. *Parasitology: The Biology of Animal Parasites.* Philadelphia, Lea & Febiger.
6. WOLDOW, N. L., and N. CHERNOF, in preparation.