

SOME DISEASES AND PARASITES AFFECTING
COTTONTAIL RABBITS IN VIRGINIA,

by

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Thesis submitted to the Graduate Faculty of the
Virginia Polytechnic Institute and State University
in partial fulfillment of the requirements for the degree of

MASTER OF SCIENCE

in

Fisheries and Wildlife

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December 1978

Blacksburg, Virginia 24061

ACKNOWLEDGEMENTS

I wish to express my sincere appreciation to my committee chairman,
and to for their support,
encouragement, guidance, and criticism throughout the study.

is gratefully acknowledged for his assistance and critical
review of the manuscript.

I wish to thank the Virginia Commission of Game and Inland Fisheries
for both financial (Project No. W-40-R-24) and field assistance,
especially , Game Biologist Supervisor and ,
Game Biologist. Thanks are due to the many Game Wardens who procured
raccoons.

The assistance in collecting specimens and providing accommodations
at Fort Pickett by and , Wildlife Management
Division, U. S. Army, Fort Pickett is gratefully acknowledged.

The assistance of , Deputy Director and his
staff at the Microbiology Section of the Consolidated Laboratories,
Richmond, Virginia is sincerely appreciated.

, University of Georgia, is sincerely thanked
for his assistance in the identification of Baylisascaris procyonis.

I am indebted to my fellow graduate student, ,
for her help in both the field and laboratory phases of this project.
The assistance of both and in the
laboratory phases of the project is gratefully acknowledged.

Finally, this would not have been possible without the moral,
financial, and physical support of my family, especially my loving and
understanding wife, .

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INTRODUCTION

The cottontail rabbit (Sylvilagus floridanus) is the most important small game species in North America. Its abundance and distribution make it favored by many groups of sportsmen, and it also holds a place in the heart of the nonconsumptive user of wildlife.

Cottontails in Piedmont, Virginia are not as plentiful as they once were. According to Reeves (1960) the number of cottontails in Virginia started to decline by 1933. The citizens became so concerned, that in 1941 it became unlawful for anyone in Virginia to engage in the previously common practice of buying and selling cottontail rabbits. The populations of cottontails continued to decline in the Commonwealth and the concern became so great that in 1964 the legislature passed a bill requesting the Commission of Game and Inland Fisheries to make a study of the situation. In recent studies, including the present one, there has been extreme difficulty in acquiring specimens for research.

Many factors are capable of limiting cottontail numbers and among these are inadequate food, inadequate cover, predation, parasitism, and disease (any deviation from the norm). The latter two factors can have an impact that is not quite as obvious or as easily remedied as the former factors.

Among the diseases that rabbits can contract, tularemia is considered to be the only one capable of decimating cottontail populations (Pelton 1968). The ability of tularemia to reduce

cottontail numbers drastically was observed by McGinnes (1964) at an enclosure near Roanoke, Virginia. At Fort Pickett, a semiactive military installation in southeastern Virginia, a drastic decline in the number of cottontails harvested occurred in 1960-61 (Woronecki 1961) with no apparent explanation. However, in the spring of 1962 Bellig (1962) found a dead cottontail on the post from which the tularemia organism was isolated; a possible explanation for the decline. Evidence in the form of a titer for tularemia antibodies in rabbit serum, was found again at Fort Pickett by Jacobson et al. (1978) in 1974 and was considered responsible for the low harvest of cottontails still persisting there.

In addition to bacterial diseases, cottontails are subject to infections by helminth parasites. Although a parasite infection can have adverse effects, ideally the parasite is not meant to destroy the host. If this happened, the parasite would soon exterminate its habitat and drive itself to extinction. When parasites become abundant in a host or when they invade an unnatural host they can cause severe damage which is sometimes fatal (Andrews 1969). Some parasites in their intermediate stages inflict damage to a host in the process of completion of the parasite's life cycle. A parasite of this sort caused an epizootic among cottontail rabbits captured at Center Woods, Virginia Polytechnic Institute and State University, Montgomery County, Virginia in 1974-75. Sixteen of 60 cottontails captured at Center Woods succumbed to cerebrospinal nematodiasis (Nettles et al. 1975). The causative agent was found to be the

larval stage of Baylisascaris procyonis, a nematode whose adult stage lives in the small intestine of the raccoon (Procyon lotor). This isolated epizootic exhibited the possibility that neurologic disease could also be a regulating factor in cottontails elsewhere in Virginia.

Wildlife are also subject to nutritional diseases including starvation. The relationship of partial starvation and parasitism are factors that have been associated and occasionally studied. However, the effects of this interaction are relatively unknown and could be of more importance in regulating wildlife abundance and distribution than previously regarded.

Recognizing that disease can have an impact on species abundance, the purpose of this study was to determine if disease could be involved in the continued low harvest of cottontails at Fort Pickett and elsewhere in the Piedmont of Virginia. The specific objectives for this study were:

1. To determine if mammals other than lagomorphs and birds serve as reservoirs for Francisella tularensis at Fort Pickett, Virginia.
2. To determine the possible arthropod vectors of Francisella tularensis at Fort Pickett.
3. To determine if the distribution of Baylisascaris procyonis in Virginia is such that the disease, cerebrospinal nematodiasis, could be considered a population regulatory factor of the cottontail rabbit in the Piedmont.
4. To determine the effects of a normal parasite load and restricted nutrition on the physiological parameters of the cottontail rabbit.

LITERATURE REVIEW

Tularemia

History

McCoy (1911) was the first to identify tularemia as a disease of animals. He discovered this new disease while working on plague among ground squirrels (Citellus beecheyi) in California.

Using guinea pigs as test animals for plague, he noted a difference in the lesions produced by the Yersinia pestis organism and the new bacteria. McCoy and Chapin (1912) named the bacteria causing this "plague-like disease of rodents" Bacterium tularense after Tulare County, California where it was first found. Not long after this, Wherry and Lamb (1914) reported the first documented human case of infection with Bacterium tularense; a meatcutter in Ohio who contracted the disease from a rabbit (probably Sylvilagus floridanus). Francis (1921) coined the name "tularemia" based upon the specific name of the organism and the septicemic nature of the disease. Ohara (1925) in Japan and Surorova et al. (1928) in Russia also reported this new bacterium which has since been found throughout the Northern Hemisphere with the exception of the British Isles. Bacterium tularense was later renamed Pasteurella tularensis and most recently Francisella tularensis (McCoy and Chapin 1912) in honor of Dr. Edward Francis, a pioneer in the study of the disease. Francisella tularensis is now classified in the same family as Brucella organisms and is closely related to the family containing Pasteurella spp. and Yersinia spp. which includes bubonic plague.

The lagomorphs and Francisella probably coevolved. Analysis of the adaptation to ectoparasites, geographic distribution, and its transmission in natural nidi indicates that Francisella tularensis originated at the end of the Miocene or the early Pliocene in the Northern Hemisphere (Reilly 1970), and at about this same time the split between Sylvilagus and Leporis in the family Leporidae occurred. The leporids probably originated in Asia but most of the early evolution was in North America during the Oligocene and Miocene. During the Pliocene, however, the advance subfamily Leporinae evolved in the Old World (Vaughan 1972).

One of the earliest references to a disease presumed to be tularemia is "Leemands Soet" (lemming fever) described by Worm in 1653 in Fenno-Scandia (Omeland et al. 1977). Many names have been applied to tularemia, most of which are descriptive of the human mode of infection. The most common are: rabbit fever, market man's disease, deer fly fever, Pahvant Valley fever, and in Japan Yato byo and Ohara's disease.

Types of Francisella tularensis

Francisella tularensis is a variable organism that produces a number of manifestations. There are 2 primary forms, distinguishable in virulence and mode of transmission; Type-A or tick borne is more virulent than Type-B which is water borne. One means of differentiation is that the more virulent strains ferment glycerol whereas the less virulent strains do not (Hornick and Eigelsbach 1969).

Type-A was referred to as Francisella tularensis tularensis and Type-

B as F. tularensis palearctica by Olsufiev et al. (1959). F. tularensis tularensis is found only in the Nearctic but F. tularensis palearctica is found not only in the Nearctic but throughout the remainder of the Northern Hemisphere. Since the subspecies names given by Olsufiev are not geographically accurate, the Type-A and Type-B designations of Jellison et al. (1961) are preferred. Olsufiev (1970) later amended his classification to make F. t. tularensis into F. t. nearctica and F. t. palearctica to F. t. holarctica and designated the 2 varieties of holarctica, japonica and mediasiatica. This new classification has not received wide acceptance.

Tularemia also has been classified by the clinical and epidemiological types observed. The clinical types compiled by Simpson (1929) and Francis (1947) as reported by Jellison (1974:18-19) are:

1. Ulceroglandular
2. Oculoglandular
3. Glandular in which no primary lesion is evident but regional or superficial lymph nodes are involved
4. Typhoidal where neither primary lesion or regional adenopathy is evident
5. Meningeal, a complication of the ulceroglandular type
6. Oropharyngeal, anginose or ingestion forms
7. Pulmonary type with lobar pneumonia, bronchopneumonia, pleuritis and pleurisy as prominent symptoms.

Pathology and Modes of Infection

Most infections with F. tularensis cause a bacteremia or lymphadenitis. The bacteremia causes necrotic foci in the spleen, liver, lungs, lymph nodes, and bone marrow. If unchecked the damage may be fatal (Reilly 1970). Prior to 1949 the fatality rate in the United States was 9.5 percent and, according to Brooks and Buchanan (1970), this dropped to 1.2 percent when streptomycin became available.

Several vaccines have become available for persons in high risk vocations or avocations, and a subclinical case will produce, in most cases, an immunity of unknown duration.

In cottontails the disease is quick acting. The diagnosis is based upon the isolation of the organism, observation of the enlarged liver and spleen with the characteristic lesions and the presence of a serum antibody titer. The latter method is used for diagnosis in man with a titer of 1:80 considered positive (McDowell et al. 1964). Death in cottontails normally occurs within a week after infection and often within 24 hours. The animals become lethargic and unable to eat or escape predation. According to Bell (cited in Jacobson 1976) cottontails are able to survive a fully virulent infection of F. tularensis, and Demaree (1970) was able to produce some protection in cottontails using an attenuated vaccine. McGinnes (1958), however, was not able to produce immunity in cottontails with a single injection of F. tularensis antigen. An injection of the attenuated antigen appeared to delay death by one day. Injections of streptomycin following the inoculation of a vaccine did not provide protection.

The primary modes of infection in man are by contact with infected rabbits, tick bites and deer fly bites, ingestion of infected water and, to some extent, inhalation of infected dust. Jellison and Parker (1945) estimated that 90 percent of all human cases were attributable to contact with rabbits, mainly Sylvilagus floridanus (dressing, cooking, or ingestion of inadequately cooked meat). This is true in the eastern and north central sections of the United States. However, in the South, infection due to tick bites is high (Calhoun et al. 1956; Cooney and

Burgdorfer 1974). In the West there is also a high incidence of disease due to bites from deer flies (Chrysops spp.) although infections from tick bites and lagomorphs are not unusual (Emmons et al. 1976; Hopla 1974; Jellison 1974). Sheep shearers and tenders have a high risk of infection because sheep are known carriers and several severe epizootics among sheep have occurred with concurrent infections in man (Jellison and Kohls 1955). Infection is common in beaver and muskrat trappers in the northern states and Canada. The largest epidemic among trappers occurred in Vermont in 1968 when 46 cases were confirmed (Young et al. 1969).

There have probably been many epizootics that have not been recorded. Brachman (1969) believed that "human disease occurs if 1 percent of the rodents in an area are infected." Bell (1965) and Omland et al. (1977) also believed that a high incidence in man is related to epizootics in animals.

Man has been infected also by skinning deer (Gilbert and Coleman 1932; Tartakow 1946; Emmons et al. 1976), opossums (Bernstein 1935; Hoff et al. 1975b) and also by being bitten by a cat, wild boar, coyote, dog, hog, and snapping turtle (Gelman 1961; McGinnes 1964). Gelman (1961) and McGinnes (1964) both reported that infection has occurred even from a cat scratch. However, there has not been a confirmed case of human-to-human transmission of the disease (Brachman 1969).

Experimental infection has also been produced by transmission through intact skin. Quan et al. (1955) compared the LD₅₀ doses for intracutaneous inoculation, oral (intubation and drinking water) and through skin in albino laboratory mice. In two separate intracutaneous

inoculation tests, the LD₅₀ was 0.96 and 0.30 organisms. The doses necessary for oral infection were 10⁶ and 10⁷ organisms for intubation and drinking water respectively. For contact through the skin the lowest amount necessary was 5 x 10⁶ organisms in 2-month-old mice. The LD₅₀'s for 1-week-old mice, 1-month-old mice, and 6-month-old mice were 2 x 10⁷, 2 x 10⁷, and 4 x 10⁷ respectively. Francis, as reported by Quan et al.(1955), also produced infections in guinea pigs through intact skin. Francis apparently placed suspensions of infected splenic tissue of recently dead guinea pigs onto unbroken skin of healthy guinea pigs. These guinea pigs were dead in 5 days and showed the characteristic lesions of the spleen and liver. F. tularensis was recovered from the animals also.

Trans-epidermal infections have not been observed or confirmed in the wild. The potential does exist and may be important in the transmission of the disease in water-borne epidemics and epizootics.

In Sweden, 95 percent of the cases were the result of insect bites, primarily mosquitoes (Aedes cinereus) (Dahlstrand et al. 1971). There has been an increase in recent years in the number of cases resulting from inhalation of infected dust. Dahlstrand et al. (1971) reported an incident in Sweden where an increase in the vole population occurred with a subsequent die-off. The voles had invaded the barns and contaminated the hay with infected feces. Several human cases were reported in workers in these barns. Bell and Stewart (1975) reported that urine from infected voles can be a source of the organism as well.

There is a seasonal pattern accompanying the incidence of tularemia in man. Where insects and arthropods are the primary vectors, the

disease occurs mainly in the summer months when these vectors are the most active (Bell 1965; Dahlstrand et al. 1971). Yeatter and Thompson (1952) demonstrated seasonal occurrence concurrent with the rabbit hunting season in Illinois. In a plot relating the number of cases of tularemia to the opening day and the mean date of the first 10 freezing nights, they found that there were fewer cases of tularemia when the freezing nights occurred soon after or before opening day of the season. The years of 1932 and 1933 had unusually warm autumns and there was a high population of rabbits. During these years Illinois reported the highest number of cases of tularemia in its history. Yeatter and Thompson (1952) recommended that the hunting season be delayed to occur after several freezing nights, thus lowering the risk of infection because vectors responsible would decline and any tularemic rabbits would be stressed and soon die. McGinnes (1964) observed a similar pattern in an enclosure in Virginia. He found that the incidence of tularemia abated in late November and did not occur again until the early part of March, when arthropod vectors resumed activity.

The incidence of tularemia in man peaked in 1939 in the United States (12 years after it became a reportable disease) when there was a total of 2,291 cases, 258 of which were fatal (Jellison 1974). This was an infection rate of 18.6 cases per 1,000,000 in population (Brooks and Buchanan 1970). The current incidence (1977) is 0.08 cases per 100,000.

Olsen (1975) gave two possible reasons for the decline in the number of reported cases:

1. Ecologically induced selection against the more virulent strains

2. Over the past 2 to 3 decades a general reduction in the amount of F. tularensis circulating in wild reservoirs.

The first explanation, i.e. virulence change, is similar to the virulence theory of Green (1943). He hypothesized that the virulence of the disease circulating in nature is dependent upon the number of times it is passed through hosts. In support of this theory, he found a difference in the virulence of strains isolated from cotton-tails and grouse, and that when serially passed through guinea pigs, the grouse strain became as virulent as the rabbit strain.

The second reason given by Olsen (1975) is only an assumption; there is at present no evidence to support it. Since the disease has been known for only about 60 years, there has not been enough time and research to determine if it does in fact cycle in nature.

In addition to the above two reasons, a third might be plausible. The use of antibiotics since 1949 for diseases similar to tularemia may have resulted in cures without proper diagnosis of the disease. Exposure to the bacillus may have led to mass immunization among people coming in contact with the disease.

Vectors

"It is frequently stated that there are more arthropod vectors for tularemia than for any other zoonotic disease" (Jellison 1974:79). Of the many groups of arthropods that can transmit tularemia, the most important in North America are ticks, deer flies, and fleas. Mites, lice, and mosquitoes have also been implicated as vectors.

Ticks of the family Ixodidae are the most important of all the vectors. The species most frequently transmitting tularemia are:

Dermacentor andersoni, D. variabilis, D. occidentalis, Haemaphysalis leporispalustris, Amblyomma americanum, and Ixodes spp. All but D. andersoni and D. occidentalis are common and have a wide distribution in the Southeast (Sonenshine and Stout 1971). These species are at least 2-host ticks, meaning that during its life cycle the tick changes hosts at least once, and is therefore a good candidate as a vector. The rabbit tick, Haemaphysalis leporispalustris, is a 3-host tick that does not bite man but is considered responsible for the maintenance of the disease among cottontails. It is found as a larva on quail and other ground inhabiting birds and as a nymph and an adult on rabbits.

Not only are ticks suited as vectors, but they can serve as reservoirs as well. Matheson (1940) and Brachman (1969) have shown that many species of ticks can transmit F. tularensis transstadially (from one stage to the next) and transovarially (from the female to her progeny). There has been, however, some difficulty in demonstrating transovarial passage in Dermacentor spp. (Bell 1945).

Fleas were the first to be suspected as possible vectors (McCoy 1911). This association was probably made because fleas were known to transmit plague at that time. There have been isolates of F. tularensis made from fleas, but this is relatively rare. Fleas are therefore of minor importance in transmission and maintenance of the disease (Jellison 1959). Francis and Lake (1921) reported the transmission of tularemia with the louse Haemodipsus ventricosus. The habits of lice (Order Anoplura) might be important in the transmission among host individuals of the same species, or maintenance in reservoir hosts. Lice are host specific thus making them poor candidates as vectors of

tularemia from reservoirs to susceptible animals.

In 1908, 6 cases of a febrile disease in man were associated with the bite of a "large horse fly" (Pearse 1911). Francis (1919) called the disease "deer fly fever", which was caused by the Bacterium tularense of McCoy and Chapin (1912). Francis believed that the primary vector was the deer fly, Chrysops discalis which readily bites man. Other tabanid species shown to transmit the disease mechanically are Chrysops noctifer, Tabanus septentrionalis, and Tabanus rupestris. Chrysops discalis is probably the only species important in the transmission to man, but the others could be important in transmission among wildlife species. There are only a few instances of the isolation of F. tularensis from naturally infected tabanids. Cox (1965) was able to isolate the bacterium from 2 pools of C. fulvaster and 1 of C. aestuans. Klock et al. (1973) isolated F. tularensis from 3 pools of C. discalis in Utah in an area of known human infection. Studies by Francis and Mayne (1921) showed that the longest interval between bites that produces infection was 4 days. However, Parker in 1933 observed viable F. tularensis bacteria in a C. noctifer one month after infection (Krinsky et al. 1975). The habit of feeding on several individuals and the ability to be infective for at least 4 days makes the tabanids good disseminators of the disease to man among wildlife and domestic species.

Mites and argasid ticks can also serve as vectors for tularemia, but their importance is inconsequential in the overall epidemiology of the disease (Jellison 1974). Mosquitoes have been suspected in the

United States, but their overall role is minimal in comparison to the role of ticks as vectors.

Reservoirs

Olsen (1975) stated that there were more than 100 species of wild mammals, 9 species of domestic mammals, 25 species of birds, and several species of fish and amphibians that can serve as reservoirs. The most complete list is found in Reilly (1970) and is reproduced and amended in the Appendix. In addition, F. tularensis has been isolated from water (Jellison 1974) and mud (Young et al. 1969; Bell and Stewart 1975), and as previously stated, ticks.

Epizootics

Several epizootics of tularemia have occurred most commonly among cottontail rabbits (Sylvilagus spp.) in the United States. The first epizootic reported in the literature was that of wild rabbits dying in large numbers in Kentucky across the Ohio River from Cincinnati, Ohio, in 1912 where the first infection in man was reported by Wherry and Lamb (1914). Hendrickson (1947) reported that epizootics among cottontails occurred "West to East" around 1928 and "East to West" in 1938. Major die-offs of rabbits in 1934 and again in 1938 in Wisconsin were observed by McCabe (1943). Baumgartner (1947) reported a similar die-off in Oklahoma in 1939. In these last 3 instances the exact cause of the die-off was unknown but was thought to be tularemia. Tularemia epizootics also occurred in Minnesota from 1932 to 1939 (Bell 1965), in 1937-1938 in New York (Allen 1954), in South Carolina in 1961 (McCahan et al. 1962) and in Indiana in 1968-1970 (Demaree 1970). The several epizootics that have occurred in Virginia will be discussed below.

Muskrats (Jellison et al. 1958; Young et al. 1969), beaver (Stenlund 1953; Lawrence et al. 1958), Microtus spp. (Jellison et al. 1958), sheep (Jellison and Kohls 1955), and ranch-raised mink and foxes fed infected rabbits (Jellison 1974; Henson et al. 1978) have all been involved in epizootics. In the case of the microtine epizootics, cannibalism is believed to be the transmission method responsible for the spread of the disease (Bell 1965). Omland et al. (1977) and Hornfeldt (1978) considered tularemia epizootics to be one of the major factors regulating microtine cycles in Fenno-Scandia.

Bell (1965) proposed a theory based on stress mechanisms to account for the epizootic spread of tularemia by cannibalism in excessively high populations of rodents. It is as follows: EXCESSIVE POPULATION -----> STRESS -----> ADRENAL (ALDOSTERONE) EXHAUSTION -----> SALT HUNGER -----> CANNIBALISM -----> EPIZOOTIC SPREAD OF TULAREMIA.

The work of Mohr (1961) and Green et al. (1943) has shown that vector populations (flea and tick) are proportional to their host density or spacing. These authors assumed that this fluctuation in vectors was important in the spread of arthropod-borne diseases in wild populations. The tularemia epizootics had a major impact on hunting not solely by reducing cottontail numbers. Both McCabe (1943) and Yeatter and Thompson (1952) reported that tularemia was of such significance in the Midwest that many rabbit hunters had abandoned the sport.

Serologic Surveys

Several serologic surveys of vertebrates for tularemia have been conducted. They were carried out usually after a reported or suspected epizootic and were done with public health as the main impetus. The

major surveys reporting the most extensive information on species and numbers infected are: McCahan et al. (1962) in South Carolina, McKeever et al. (1958) in Georgia and Florida, Burgdorfer et al. (1974) in Kentucky, Friend and Halterman (1967) in New York, Hoff et al. (1975a; 1975b) in Florida, Franklin et al. (1966) in Kansas, Calhoun et al. (1955) in Arkansas, Vest et al. (1965) and Woodbury and Parker (1953) in Utah, Philip et al. (1955) in Nevada, and Cook et al. (1965) in Texas.

Tularemia in Virginia

Tularemia has been in Virginia since at least 1925 when an outbreak among a family in Lee County was described by Dr. Francis to Dr. R. R. Parker in a letter dated August 7, 1925 (Jellison 1974). Reeves (1960) reported that an epizootic occurred in 1933 causing great concern among hunters. Another epizootic occurring in a private enclosure near Roanoke used for training beagles was reported by McGinnes (1964). The strain present in this enclosure was highly virulent and all attempts to restock the pen failed. Spencer (1961) reported that 25.7 percent of the cases of tularemia in Virginia between the years 1949 and 1958 occurred in a six-county area. This focus was in southcentral Virginia encompassing the counties of Campbell, Charlotte, Halifax, Mecklenburg, Pittsylvania, and Prince Edward.

In 1959-1960 a drastic reduction in the number of cottontails harvested at Fort Pickett was observed (Woronecki 1961). In March 1962 Bellig (1962) found a dead cottontail at Fort Pickett from which F. tularensis was isolated. This isolation provided the only obvious explanation for the decline. This area is close to the nidus reported by Spencer. This population has not recovered (Fig. 1) and prompted an

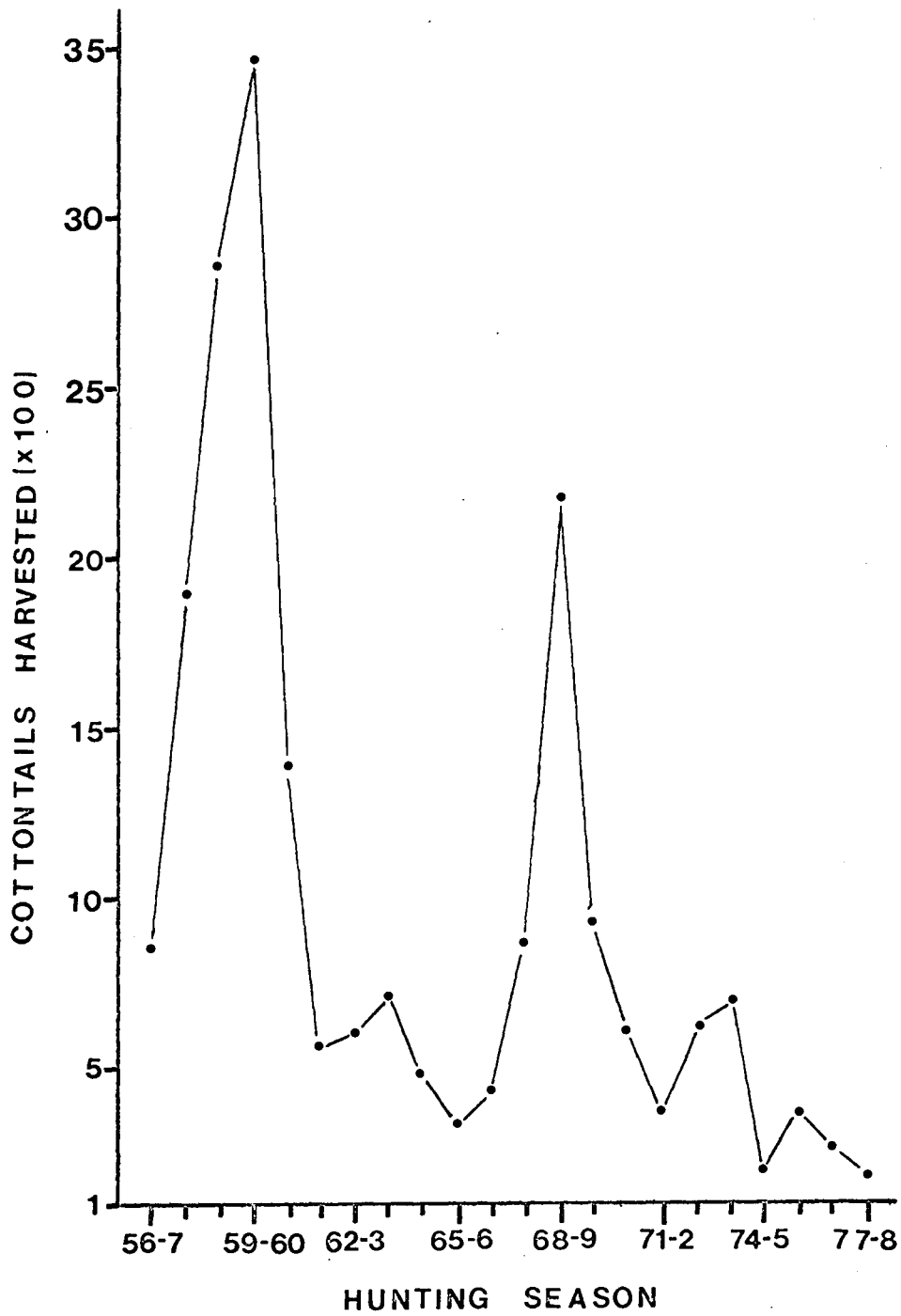


Fig. 1. Hunter harvest of cottontail rabbits at Fort Pickett, Virginia, 1956-1978.

extensive study by Jacobson et al. (1978). The serum from 4 of 17 cottontails collected at Fort Pickett in the fall of 1973 exhibited titers to tularemia. They felt that latent tularemia was the reason for the observed continued decline in hunter harvest at Fort Pickett.

Jacobson et al. (1978) also reported a titer from a cottontail collected at the Radford Army Ammunitions Plant in Pulaski County in September 1973. The most recent report of tularemia in wildlife in Virginia was from 2 Delmarva fox squirrels (Sciurus niger cinereus) found ill at the Chincoteague National Wildlife Refuge, Chincoteague, Virginia in the spring and summer of 1977 (J.C. Appel, pers. comm.). F. tularensis was isolated from the 2 squirrels by the Center for Disease Control, Atlanta, Georgia.

Baylisascaris procyonis

History

Baylisascaris procyonis (Stefanski and Zarnowski 1951; Sprent 1968) is an obligate parasite of the family Ascarididae that is found in the adult form in the small intestine of the raccoon (Procyon lotor). This species was once considered to be synonymous with Ascaris columnaris of North America and Ascaris procyonis of Europe. Taxonomic studies conducted by Hartwich (1962) and Sprent (1968) led to the description of a new genus, Baylisascaris. A. procyonis and A. columnaris, as found in raccoons, were grouped as the new species B. procyonis.

B. procyonis has also been reported from the kinkajou (Potos flavus), a close relative of the raccoon, from Colombia, South America (Overstreet 1970). Nettles (pers. comm.) has shown that the opossum (Didelphis marsupialis) can be experimentally infected as a definitive

host.

Life Cycle

The complete life cycle of B. procyonis is not known. Many of the ascarids have a direct life cycle, which means that an intermediate host is not necessary for the development of the larvae. For example, eggs of Ascaris lumbricoides are emitted in the feces of the definitive host; the eggs embryonate in the soil and are swallowed in food, water, or soil. The larvae hatch and penetrate the duodenal wall where they enter the blood and lymph channels. They then pass through the heart into the lungs and break out into the air sacs. After migration up the trachea they are swallowed and once in the small intestine, they mature into adults.

In species having an indirect life cycle the embryonated eggs are ingested by an intermediate host in which the parasite develops but does not reach sexual maturity. The larvae may encyst in the viscera or migrate into the central nervous system (CNS) or muscle tissue. When the intermediate host is ingested by the definitive host, the larvae develop into adults in the small intestine.

Tiner (1953) believed that Ascaris columnaris in the raccoon has an indirect life cycle with small rodents serving as the intermediate host. The larger small mammals such as woodchucks and rabbits may serve as intermediate hosts, but it is more likely that they are paratenic hosts.

The larvae in the body of the intermediate hosts penetrate the intestinal mucosa and migrate to the central nervous system. If

migration proceeds to the brain, normal neural functioning is impaired. Torticollis or wry neck, ataxia, and loss of motor control are signs of this CNS invasion. An animal suffering from neurologic disease becomes easy prey for the definitive host. The species which have been observed with neurologic disease are listed in Table 1.

Pathogenicity

The pathogenicity of B. procyonis in the intermediate host is great in comparison with other ascarids of carnivores. The "raccoon form" of Ascaris columnaris (i.e. B. procyonis) has often been found to be more pathogenic than the "skunk form" (Tiner 1951). One raccoon ascarid larva in the medulla or spinal cord of a mouse is sufficient to cause death. It takes, on the other hand, several skunk ascarid larvae (A. columnaris) to cause damage. Survival of mice infected with the "skunk form" and even destruction of the larvae is not unusual (Tiner 1951). Apparently the pathogenicity of the larvae is related to the size it reaches in the nervous tissue. B. procyonis larvae often attain a length greater than 1 mm which seems to be the critical length since all species with larvae less than 1 mm are less pathogenic than B. procyonis (Tiner 1953b).

Epizootics

B. procyonis has been responsible for several die-offs in rodents and lagomorphs. In Michigan, Dade et al. (1977) reported that 20 to 35 captive nutria (Myocaster coypus) succumbed to neurologic disease after local cottonwoods were given as a food source. The area from which the cottonwoods had been collected supported a heavy raccoon population. Another die-off in Michigan occurred among domestic rabbits (Oryctolagus

Table 1. Species known to have been infected naturally with Baylisascaris procyonis
or Ascaris columnaris.

Species	Common name	Source
Peromyscus leucopus	White-footed mouse	Tiner 1951
Citellus tridecemlineatus	Thirteen-lined ground squirrel	Fritz et al. 1968
Sciurus niger	Fox squirrel	Tiner 1951
Sciurus granatensis	Red squirrel	Schueler 1973
Myocaster coypus	Nutria	Dade et al. 1977
Marmota monax	Woodchuck	Richter and Kradel 1964
Marmota monax	Woodchuck	Swerczek and Helmboldt 1970
Marmota monax	Woodchuck	Jacobson et al. 1976
Marmota monax	Woodchuck	Fleming and Caslick 1978
Oryctolagus cuniculus	European rabbit	Dade et al. 1975
Sylvilagus floridanus	Eastern cottontail rabbit	Jacobson et al. 1976

cuniculus) (Dade et al. 1975). It was believed that raccoons had entered the barns where the rabbits were kept and had contaminated the pens.

Another epizootic occurred in Virginia in 1974-75. Several cotton-tail rabbits and woodchucks were observed with clinical signs of neurologic disease. This disease had not previously been noted in this area (Center Woods, Virginia Polytechnic Institute and State University, Montgomery County, Virginia) and occurred concurrent with an influx of raccoons into the woodlot (Nettles et al. 1975; Jacobson et al. 1976).

Tiner (1953a) suggested that as much as 10 percent of the small rodent mortality near Champaign, Illinois could be due to the raccoon ascarid. The effects of this ascarid on other rodent and small mammal populations is unknown and could be restricted to isolated areas of high raccoon populations. The potential public health hazard is unknown.

Distribution

The current distribution of B. procyonis is unknown. A search of the literature indicated that it is nearly ubiquitous in the United States (Tables 2,3,4,5). Its distribution in the southeast appears to be limited to the more mountainous regions (Nettles pers. comm.).

Parasitism and Nutrition

"So many and no more" was Allen's (1954) way of expressing the concept of limiting factors. In wildlife management the limiting factors are the nemesis of producing adequate populations for the consumptive and nonconsumptive wildlife users. One view of increasing wildlife populations is to remove from the environment those factors

Table 2. Species known to have been infected experimentally with Baylisascaris procyonis or Ascaris columnaris.

Species	Common name	Source
<i>Mus musculus</i>	House mouse	Tiner 1949
<i>Peromyscus leucopus</i>	White-footed mouse	Tiner 1949
<i>Sigmodon hispidus</i>	Cotton rat	Tiner 1949
<i>Phodopus</i> spp.	Hamster	Tiner 1949
<i>Cavia porcellus</i>	Guinea pig	Tiner 1949
<i>Marmota monax</i>	Woodchuck	Jacobson et al. 1976
<i>Sciurus carolinensis</i>	Gray squirrel	Tiner 1949
<i>Oryctolagus cuniculus</i>	European rabbit	Church et al. 1975
<i>Sylvilagus floridanus</i>	Eastern cottontail rabbit	Jacobson et al. 1976
<i>Didelphis marsupialis</i>	Opossum	Nettles (pers. comm.)

Table 3. Distribution of *Baylisascaris procyonis* in the United States based on the presence of adult worms in raccoons (*Procyon lotor*).

State	Reference
California	Overstreet 1970
Georgia	Babero and Shepperson 1958
Illinois	Tiner and Chin 1948
Illinois	Leigh 1940
Iowa	Morgan and Waller 1940
Iowa	Waller 1940
Minnesota	Olsen and Fenstermacher 1938
Ohio	Rausch 1946
Virginia	Jacobson et al. 1976
Washington	McNeil and Krogsdale 1953

Table 4. Distribution of Baylisascaris procyonis in the United States based on the diagnosis of larvae in accidental hosts.

State	Source
Connecticut	Church et al. 1975
Connecticut	Swerczek and Helmboldt 1970
Illinois	Ferris et al. 1960
Illinois	Fritz et al. 1968
Illinois	Tiner 1951
Maryland	Schueler 1973
Michigan	Dade et al. 1975; 1977
New York	Fleming and Caslick 1978
Pennsylvania	Richter and Kradel 1964
Virginia	Nettles et al. 1975
Virginia	Jacobson et al. 1976

Table 5. Areas in the United States where raccoons have been examined and Baylisascaris procyonis not found present.

Region	Source
Chesapeake Bay Area	Alexander et al. 1972
Ossabaw Island, Georgia	Jordan and Hayes 1959
East Texas	Chandler 1942
Wisconsin	Schiller and Morgan 1949
Alabama	Johnson 1970
South Carolina	Johnson 1970
North Carolina	Johnson 1970
Georgia	Johnson 1970
Florida	Johnson 1970
Virginia	Johnson 1970

which prevent full reproductive potential. Two such important factors are parasitism and lack of adequate nutrition.

Parasitism and nutrition can act separately or together. The effects of parasitism on cottontails have been reviewed by Jacobson (1976) with further references in Andrews (1969). The effects of partial starvation and other nutrition related conditions have been reviewed by Warren (1976) with additional results by that author. These authors concluded that both conditions have adverse effects on a cottontail's reproductive capacity and blood composition.

Newberne (1973) regarded parasitism and nutrition as interacting either synergistically or antagonistically. In a synergistic interaction an infection is likely to increase in severity if an animal has clinical or subclinical malnutrition. The animal probably does not have enough protein reserves to build up immune responses to the infection. The effects of the two interacting is greater or more severe than would be the sum of the effects of the two conditions separately.

In a few instances the conditions may be antagonistic. Malnutrition could, in fact, decrease the severity of the disease. This could occur in cases where a parasite has a specific nutrient demand that is not filled by the host's diet and, as a result, the parasite dies.

Many studies have been conducted on the effects of specific nutrient deficiencies in the host's diet on the parasites present. That subject is too extensive to be reviewed here. The reader is referred to Nelson et al. (1975; 1976) and Crompton and Neisham (1976) for further information.

Parasitism and Nutrition in Wild Species

Perhaps the first to observe the relationship between the two factors in wildlife was Clancey et al. (1940). Of 342 cottontails they examined, 50 percent of the ones showing signs of malnutrition were infected with 3 or more species of internal parasites. Erickson (1944) made similar observations on hares in Manitoba. He found that from 1931 to 1933, when the hare populations were increasing, parasites were not abundant but gradually increased, and in 1935-36 a peak of parasitism was reached and the hares began to die off. The highest percentages of concurrent infections of parasites were found from 1936 to 1940 when the hares were dying in the greatest numbers. Erickson concluded that the parasites killed the animals by both direct and indirect means. The direct means are presumably associated with reduction of the quality and quantity of food available.

This relationship between parasitism and environment is used as a monitor of herd evaluation in white-tailed deer. Eve and Kellogg (1977) have shown that as the density of animals increases, the number of parasites of all species in the abomasum also increases. The increase in density and the resulting increase in contact is the reason given for the associated increase in the number of abomasal parasites. The quality and quantity of food may also have an effect on the parasite abundance but unfortunately this has not been examined.

In an experiment using ground squirrels (species not reported) Noble (1961) evaluated the effect of various stressors on the presence of Trichomonas spp. The animals were all given the same dose and,

with the exception of the caged controls, exposed to one of the following stressors: crowding, confinement, light and heat, noise, annoyance, noxious stimulants, darkness, and hunger. In all cases except hunger there was an increase in the number of trichonomads present compared to the number found in the controls. Unfortunately Noble did not measure any physiological characteristics of the ground squirrels in this test.

Parasitism and Nutrition in Laboratory Animals

Sheppe and Adams (1957) showed that a normally nonpathogenic organism can become pathogenic when the host is stressed. Laboratory mice (Mus musculus) were subjected to one of any combination of the following treatments: infected or not with Trypanosoma duttoni, ad libitum or 50 percent ad libitum food consumption, and warm or cold environmental temperature. They found that the 50 percent ad libitum-parasitized animals died sooner than those not parasitized and that the parasitized mice on full ration averaged only half as much weight gain as did the nonparasitized mice.

Crompton et al. (1978) studied the effects of Nippostrongylus brasiliensis and protein malnutrition in rats. Three groups were used; 1) a group fed a 2 percent protein diet ad libitum, 2) rats infected with N. brasiliensis and fed the 2 percent protein diet, and 3) rats pair-fed the same diet and amount as the infected animals. Infection with N. brasiliensis produces anorexia, therefore the third group was restricted to the amount of feed the infected rats consumed. Both the infected and pair-fed rats had significantly higher erythrocyte counts and hemoglobin concentrations and lower leucocyte counts. Plasma

protein, plasma albumin, and plasma corticosterone concentrations were all higher in the pair-fed rats. The differences between the pair-fed and ad libitum animals were few. The pair-fed animals had lower body weights and leucocyte counts, and higher erythrocyte counts and hemoglobin concentrations. No differences were observed in the plasma protein levels between the ad libitum group and the pair-fed group, but there were differences between the infected and non-infected groups, indicating that parasitism has an effect on those blood parameters.

Parasitism and Nutrition in Domestic Animals

Bergstrom et al. (1977) found that a low-protein diet and infection with Trichostrongylus spp. significantly affected the wool fiber diameter of sheep. It did not, however, have any effect on weight gain or feed conversion of the animals.

It was previously noted that parasitic infections often cause decreased feed consumption by the host. Seebeck et al. (1971), Springell et al. (1971) and O'Kelly et al. (1971) designed an experiment to evaluate the effects of infestation by the tick Boophilus microplus and anorexia on cattle. Each tick-infested animal was paired with a tick-free animal that was restricted to the same amount of feed that the tick-infested animal had consumed. As a control, tick-free animals were fed ad libitum. These authors found that the anorectic effect accounted for about 65 percent of the depression in body weights due to tick infestation. The anorectic treatment had no significant effect on the blood composition, but the tick infestation did. There was a decrease in red cell volume and the amounts of circulating hemoglobin, albumins, and total cholesterol, and an increase in circulating globulin. Studies

by van Adrichem and Shaw (1977a, 1977b) support the findings of changes in blood composition in tick-infested cattle.

TECHNIQUES AND PROCEDURES

Tularemia Survey

Study Area

Fort Pickett is a semiactive military installation located in the Piedmont physiographic region of southeastern Virginia. Its 18,616 hectares are situated primarily in Nottoway County with portions in Brunswick and Dinwiddie Counties. It was a major staging and receiving area during World War II but is presently a training facility for personnel from other installations and for reserve units.

Fort Pickett has a rolling topography with an elevation ranging from 61 meters to 131 meters (Fortenberry 1959). It has a modified continental climate with mild winters and humid summers. The mean annual temperature is 14^o C and the mean annual precipitation is 107 cm. The vegetation is composed of mixed hardwoods and pine. Open fields are maintained on the post for maneuvers and wildlife management by controlled burning and by mechanical means (C.O. Martin pers. comm.). A study of the land use trends is found in Jacobson et al. (1978).

The installation was selected as a study area because of the decline in cottontail numbers and reported presence of tularemia (Jacobson et al. 1978; Bellig 1962). The post is open to public hunting. All game taken must be reported prior to leaving the post and records of hunter kills show multi-year trends of rabbit populations.

Collection Procedures

Small mammals and quail were trapped using wire cage traps and wooden box traps. Small rodents were captured using Sherman aluminum folding live traps. The small rodent traps were set in a grid pattern

10 meters apart. The larger traps were placed in areas of the highest probability of capture. Trapping was conducted between July and September 1977 and during February 1978. Cottontails were also collected between October 1976 and January 1977 with a shotgun.

Upon capture all small mammals and quail were sacrificed by shooting or chloroform, and a blood sample taken. Blood was collected from small rodents by either decapitation or by collecting it from the orbital capillary bed with a Pasteur pipette. Blood was collected from quail by decapitation. Cardiac puncture with a 12 cc or a 20 cc syringe and an 18 ga needle was the method used for other small mammals. The animal was immediately placed in a plastic bag containing chloroform soaked gauze to kill the ectoparasites.

Deer were immobilized with succinylcholine chloride. Blood was taken from the jugular vein using evacuated collection tubes and a 19 ga needle.

After collection all blood was transferred to test tubes. These tubes were placed on ice until they could be taken to the laboratory.

A sample of the ectoparasites present was taken from animals collected and preserved in 70 percent ethanol for later identification.

Serum Testing Procedures

All blood was centrifuged for 30 minutes at 1600 g's. The serum was removed and transferred to plastic Falcon culture tubes using Pasteur pipettes and then frozen. In addition to the serum collected by this investigator, the sera from 5 white-tailed deer collected at Fort Pickett in September 1977 by the Southeastern Cooperative Wildlife Disease Study Group were incorporated into the survey sample.

The frozen sera were taken later to the Commonwealth of Virginia's Consolidated Laboratories for testing for the presence of tularemia antibodies. Sera were tested by both the slide test as described by Difco Laboratories (1975) using a phenolized suspension of F. tularensis and by the tube test using a locally prepared antigen (Bennett, pers. comm.). A titer of 1:40 or greater for the tube test was considered significant, indicating infection.

Statistical Analysis

Where appropriate the binomial test described by Hollander and Wolfe (1973) was used.

Cottontail Population Age Structure

During the hunting season all hunters were asked to bring the rabbits collected to a central check station. Game Checking Station personnel removed the eyes and put them in vials containing 10 percent formalin and labeled the vial with the date and area of collection. The eye lenses from both hunter-killed rabbits and those collected by this investigator were processed for age determination as described by Edwards (1967).

Baylisascaris procyonis Survey

Collection Procedures

Raccoons were collected throughout Virginia between December 1976 and May 1978 by trapping or shooting. Collections were made by staff and students of the Department of Fisheries and Wildlife and by personnel from the Virginia Commission of Game and Inland Fisheries. Some animals were taken to the laboratory immediately after being captured and sacrificed. Others had to be frozen and stored until they could be trans-

ported. When possible sex and age data were collected.

Once in the laboratory, the small intestine was removed, opened with bandage scissors, and examined for the presence of parasites. All worms grossly visible were removed and placed in hot A-F-A (alcohol-formalin-acetic acid). The specimens were later cleared by evaporating the alcohol from an alcohol-glycerin solution. Specimens were then examined under a binocular dissecting microscope at which time they were sexed and counted. Identification of Baylisascaris procyonis was verified by Dr. V.F. Nettles, Southeastern Cooperative Wildlife Disease Study Group, University of Georgia, Athens, Georgia.

Statistical Analysis

Data were analyzed using the Wilcoxon Signed Rank, Sign Test, and Wilcoxon Rank Sum tests as described by Hollander and Wolfe (1973).

Parasite Nutrition Experiment

Procurement and Handling

Nineteen cottontail rabbits, 8 males and 11 females, were collected between January and March 1978 from the Turf Grass Center and Center Woods, Virginia Polytechnic Institute and State University. Upon capture each animal was sexed, weighed, and housed in an individual cage (55x25x33 cm steel cages; Hoeltge, Inc., Cincinnati, Ohio). The animals were given feed (pelleted deer ration containing 49.8 percent corn, 17 percent alfalfa, 10 percent soybean meal, 10 percent rice hulls, 10 percent molasses, 1.5 percent mineral salt, 1 percent DSVP, 0.7 percent PO_4 ; Big Spring Mills, Elliston, Virginia) and water ad libitum.

A 2x2 factorial design with two levels of nutrition (ad libitum

and 70 percent ad libitum) and two levels of parasitism (normal and reduced) was used. On April 10, 1978 animals of both sexes were randomly selected to be in one of the four treatment groups. Animals on reduced nutrition were given food at 70 percent of their previous consumption. Food consumption of all animals was monitored weekly and when necessary the restricted amounts were adjusted according to the mean fluctuation of the ad libitum animals.

Atgard[®] containing dichlorvos (2,2-dichlorovinyl dimethyl phosphate; Shell Chemical Company) as the active ingredient was used to purge helminth parasites, particularly nematodes. It was administered at the rate of 5 mg of active ingredient per pound of body weight. The drug, which comes in pelleted form, was given in the feed which was not replenished until the entire ration was consumed. The litter was closely examined to detect spilled or refused drug pellets. This treatment was repeated two weeks later.

Termination

The experiment was terminated after 7 weeks on May 30, 1978. Beginning at 1200 hours, randomly selected animals were sacrificed by cervical dislocation and a blood sample was taken immediately by cardiac puncture. Whole blood was centrifuged at 1100 g's for 30 minutes; the serum removed and frozen in plastic Falcon tubes. The animal was dissected removing viscera, head, hide, and feet. The stomach, small intestine, large intestine, and cecum were separated and preserved in 10 percent formalin.

Body and Organ Weights

Body weight was measured prior to dissection on a Mettler P11N

balance. The carcass was weighed and frozen. The liver was weighed fresh on an Ainsworth 200 balance. The kidneys, adrenals, eyes, and reproductive tracts were preserved in 10 percent formalin and later weighed on a Mettler H-45 balance. All samples of the same organ were weighed on the same date. One testis and one epididymis were placed in 10 ml of a solution of 0.05 percent Triton-x and 0.9 percent saline for later determination of number of spermatozoa as described by Warren (1976). Eye lens weights were used to determine age as described by Edwards (1967).

Nutritional Indices

At dissection, abdominal fat was scored as follows: 0 - no fat, 1 - fat present, 2 - moderately abundant, 3 - very abundant. Femur and tibia bone marrow fat was determined using ether extraction as described by Jacobson (1976).

Packed cell volume was determined by use of an Adams Readocrit centrifuge. Blood urea nitrogen levels were determined by the procedure described by Sigma Chemical Company (1974). The American Monitor Corporation (1974b) method was used to determine serum cholesterol levels. Serum corticoid levels were determined using a competitive protein binding method as modified by the laboratory of Dr. Frank Gwazdauskas, Department of Dairy Science, Virginia Polytechnic Institute and State University. Levels of serum albumin were determined using the method of the American Monitor Corporation (1974). Total serum protein levels and serum globulin were determined by the method of Bausch and Lomb (1965). Total serum protein was also determined using

the Goldberg Refractometer.

Parasitological Procedures

At dissection the cysticerci of Taenia pisiformis present in the body cavity were counted. The contents and scraped lining of the component parts of the gastrointestinal tract were washed through a 100 mesh screen (Preiser Scientific, Charleston, West Virginia). The material remaining in the screen was examined under a binocular dissecting scope and all parasites were collected and counted. Parasitological procedures were not employed to determine if more than one species of Trichostrongylus and Cittotaenia were present.

Statistical Analysis

The analysis of variance using a least squares regression procedure was used to obtain the mean squares for the main effects (drug and nutrition), the interaction (drug + nutrition), and the error. The Statistical Analysis System (SAS) of Barr and Goodnight (1972) was employed for data analysis.

RESULTS

Tularemia Survey

Serology

A sufficient amount of serum for antibody testing was collected from 90 of the 111 animals captured at Fort Pickett. Table 6 shows the number of each species captured and number of animals exhibiting antibody titers. Of the 21 samples unsuitable for testing, the majority came from white-footed mice (Peromyscus leucopus) or Microtus spp. Many of these animals were found dead in the trap. Blood collection from many of the cottontails collected by shooting was difficult due to shot damage.

In Table 7 the titers and sex are listed for each positive infection. A titer of 1:40 or greater was considered indicative of infection. Of the 7 samples with titers of 1:80 or greater, 4 were from raccoons (Procyon lotor), and 1 each from a skunk (Mephitis mephitis), Norway rat (Rattus norvegicus), and white-tailed deer (Odocoileus virginianus). Other species exhibiting titers of less than 1:40 were raccoon (1), opossum (Didelphis marsupialis) (3), chipmunk (Tamias striatus) (1), and bobwhite quail (Colinus virginianus) (1). Seven of the 12 species tested exhibited evidence of infection.

No significant difference was found in the incidence of disease between the areas of collection. Eight of 13 positive samples were from the Cantonment Area, which is off limits to hunting. The remaining 5 positive samples were from Training Area 1, which was open to public hunting. Forty-one of the samples tested came from Area 1 while 40 came from the Cantonment Area. The remaining 9 were collected

Table 6. Species of animals collected at Fort Pickett, 1976-1978, exhibiting positive evidence of infection with Francisella tularensis.

Species	Sex	Titer
Procyon lotor	M	1:160 ^a
Procyon lotor	M	1:320 ^a
Procyon lotor	M	1:160 ^a
Procyon lotor	M	1:320 ^a
Procyon lotor	F	1:20
Didelphis marsupialis	M	1:40
Didelphis marsupialis	M	1:20
Didelphis marsupialis	M	1:20
Mephitis mephitis	F	1:320 ^a
Rattus norvegicus	F ^b	1:80 ^a
Tamias striatus	? ^b	1:20
Odocoileus virginianus	F ^b	1:80 ^a
Colinus virginianus	? ^b	1:20

^a. Significant titer indicating infection.

^b. Sex undetermined.

Table 7. Numbers of each species tested for *Francisella tularensis* antibodies, number positive, percent infected, and percent infected with a titer $> 1:80$ from animals collected at Fort Pickett, Virginia 1976-1978.

Species	Number Captured	Number Tested	Number Positive	Percent Infected	Percent 1:80
<i>Sylvilagus floridanus</i>	36	33	0	0	0
<i>Peromyscus leucopus</i>	22	10	0	0	0
<i>Microtus</i> spp.	3	1	0	0	0
<i>Rattus norvegicus</i>	3	2	1	50	50
<i>Marmota monax</i>	1	1	0	0	0
<i>Tamias striatus</i>	2	2	1	50	0
<i>Odocoileus virginianus</i>	8	8	1	12.5	12.5
<i>Mephitis mephitis</i>	2	2	1	50	50
<i>Urocyon cinereoargenteus</i>	1	1	0	0	0
<i>Procyon lotor</i>	16	15	5	33.3	26.7
<i>Didelphis marsupialis</i>	13	13	3	23.1	0
<i>Blarina brevicauda</i>	1	0	0	0	0
<i>Colinus virginianus</i>	3	2	1	50	0
TOTAL	111	90	13	14.4	7.8

from Training Area 5, which is similar to Area 1 and was open to public hunting.

The binomial test (Hollander and Wolfe 1973) was used to determine if, among those infected, one sex was infected more frequently than the other. Sex could not be determined for 2 of the specimens, therefore the test was conducted on a sample size of 11, of which 4 were females and 7 were males. The test showed no significant difference between the sexes. Among those in which sex could be determined, 32 were females and 44 were males.

Ectoparasites

Ectoparasites collected from 9 wildlife species are listed in Table 8. No ectoparasites were found on Microtus spp., bobwhite quail, and chipmunks. Amblyomma americanum was the most common arthropod parasite, being found on all hosts except the Norway rat. Four of the host species were those that were positive for Francisella tularensis titer. Dermacentor variabilis was found frequently, but not on any of the cottontails collected. Haemaphysalis leporispalustris was found exclusively on the cottontail rabbit. Ixodes spp. were collected from both the cottontail and the white-footed mouse. The flea Cediopsylla simplex was found on the rabbits as well as the opossum. Nosopsyllus fasciatus is a common flea of rats, but was found in one case on a cottontail. This is the first report of this flea being found on a rabbit in Virginia.

Cottontail Population Age Structure

During the 1976-77 hunting season 54 cottontails were collected from which eye lenses were taken. Of these, 46 percent were adults.

Table 8. Ectoparasites and their hosts collected at Fort Pickett, Virginia, 1976-1978.

Host	Dermacentor variabilis	Amblyomma americanum	Haemaphysalis leporispalustris	Ixodes spp.	Cediopsylla simplex	Nosopsyllus fasciatus
Procyon lotor	X ^a	X				
Sylvilagus floridanus		X	X	X	X	X
Peromyscus leucopus		X		X		
Didelphis marsupialis	X	X			X	
Odocoileus virginianus		X				
Mephitis mephitis	X	X				
Marmota monax	X	X				
Rattus norvegicus	X					

^a X indicates that at least one stage of the parasite was present on at least one host specimen.

In 1977-78, 69 cottontails were collected and 58 percent were adults. Fig. 2 shows the number of animals for the different eye lens weight classes. There was a natural break in the groups at about 200-210 mg indicating the break between adults and juveniles for this area. The juveniles had eye lens weights of less than 200 mg. Using the tables of Edwards (1967), the birth dates for the animals were extrapolated using the eye lens weight and date of collection. This distribution is presented in Fig. 3.

Distribution of Baylisascaris procyonis

Between December 1976 and February 1978, 72 raccoons from 11 counties were examined for the presence of Baylisascaris procyonis (Table 9). The ascarid worm was found in the small intestine of 19 of these animals. The raccoons harboring the nematode were collected in Augusta, Carroll, and Montgomery Counties. These 3 counties lie west of the Blue Ridge, but the raccoons examined from these counties did not harbour B. procyonis (Fig. 4). B. procyonis was reported from Giles County by Jacobson et al. (1976).

In those infected raccoons, it was found that significantly more ($P < 0.0001$) of the worms were female than male. The mean number of female B. procyonis per infected host was 10.6 ± 20.2 and the mean number of males per infected host was 5.2 ± 8.5 (Table 10).

The degree of infection by host sex was also examined. No significant difference was found in the number of worms per infected host by sex in the 8 males and 11 females examined.

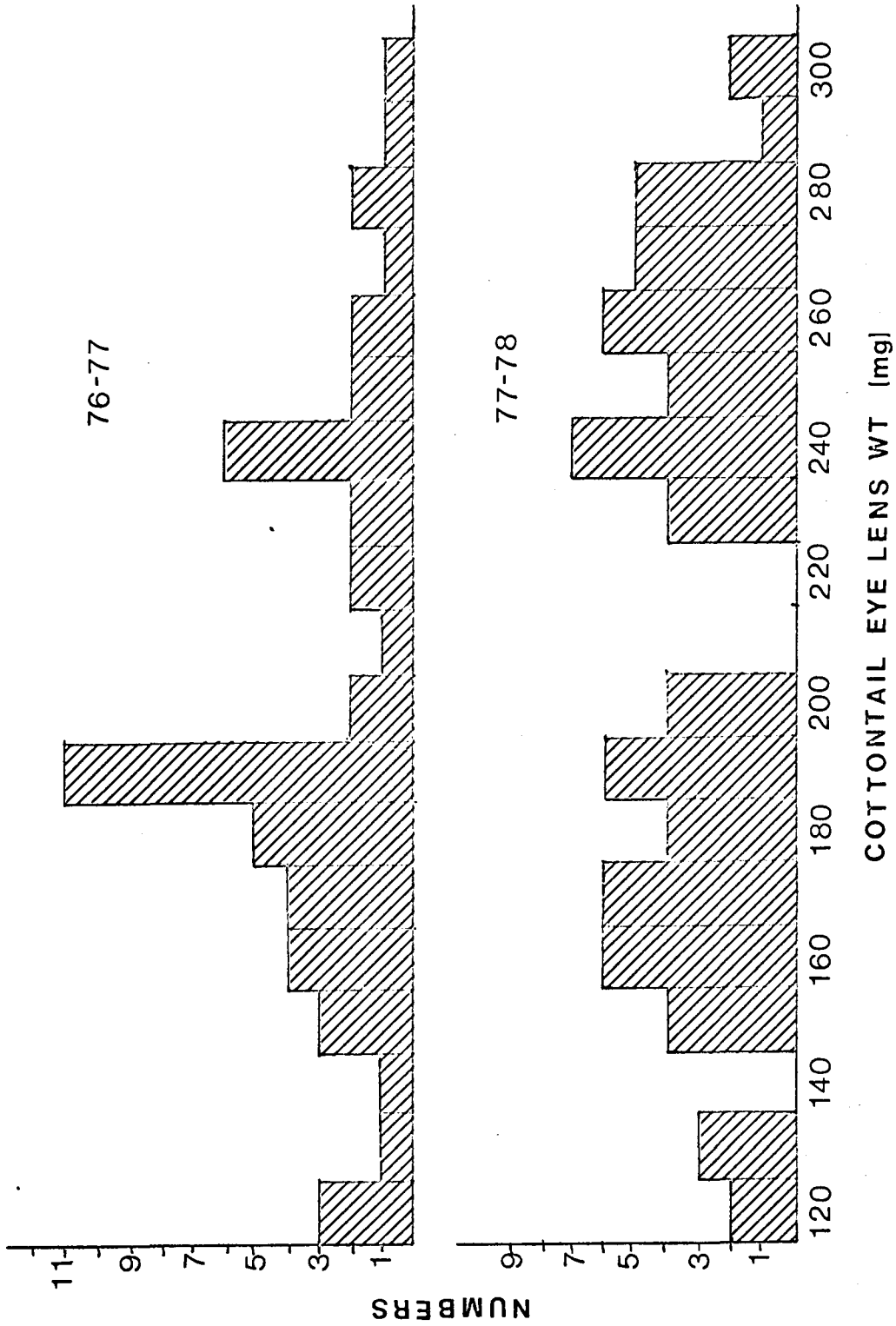


Fig. 2. Cottontail eye lens weight distribution for the cottontails collected at Fort Pickett, Virginia during the 1976-77 and 1977-78 hunting seasons.

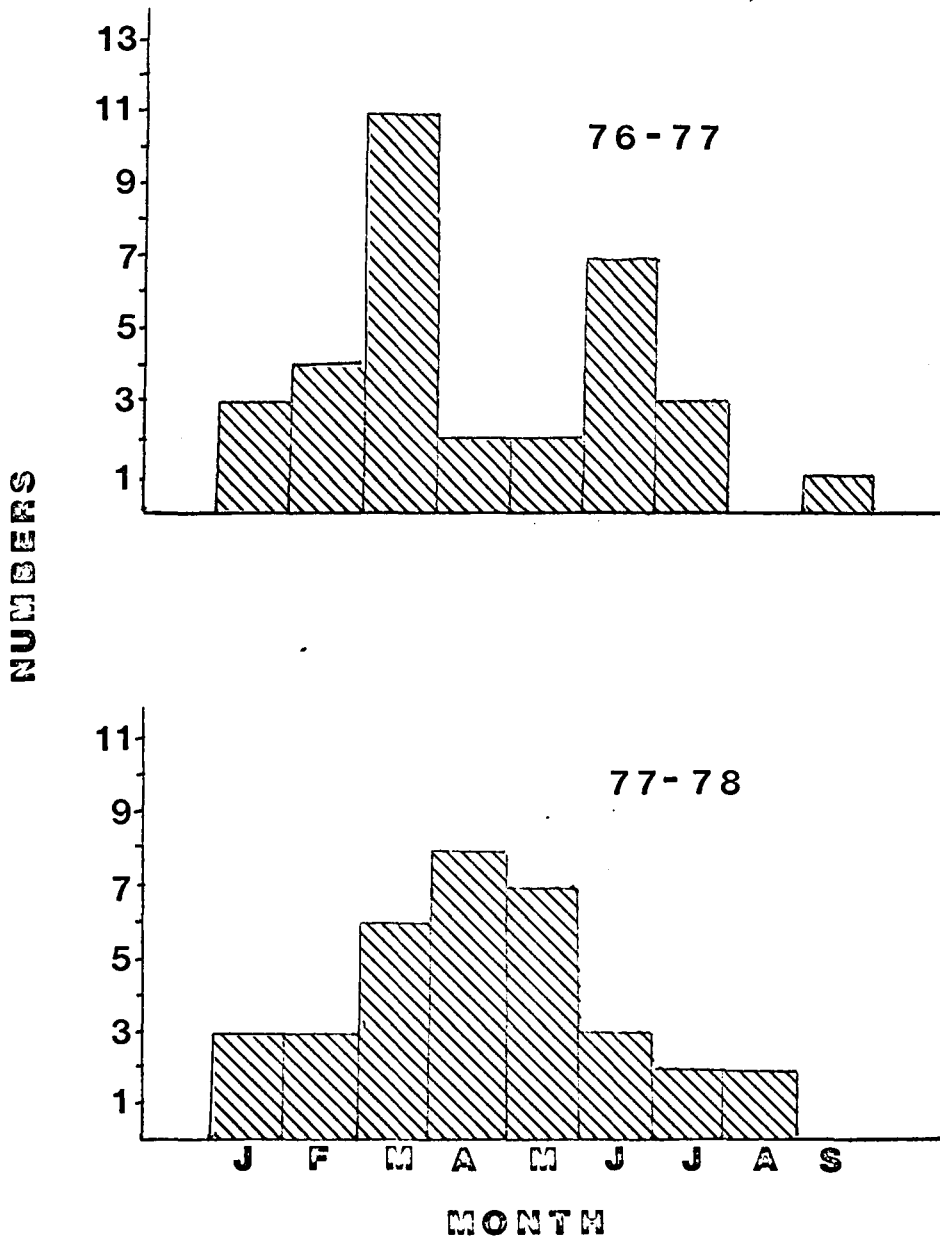


Fig. 3. Distribution of extrapolated birth dates of cottontails collected at Fort Pickett, Virginia during the 1976-77 and 1977-78 hunting seasons.

Table 9. Counties from which raccoons were examined, number of raccoons examined, and the number infected with Baylisascaris procyonis.

County	Number Examined	Number Infected
West of Blue Ridge		
Augusta	9	3
Botetourt	2	0
Carroll	1	1
Giles	1	0
Montgomery	20	16
Page	1	0
Piedmont		
Nottoway	20	0
Spotsylvania	1	0
Coastal Plain		
Lancaster	11	0
Northumberland	1	0
Prince George	5	0

Counties surveyed for *B. procyonis* but not found present.



Counties where *B. procyonis* was found.



▲▲ Blue Ridge Mountains

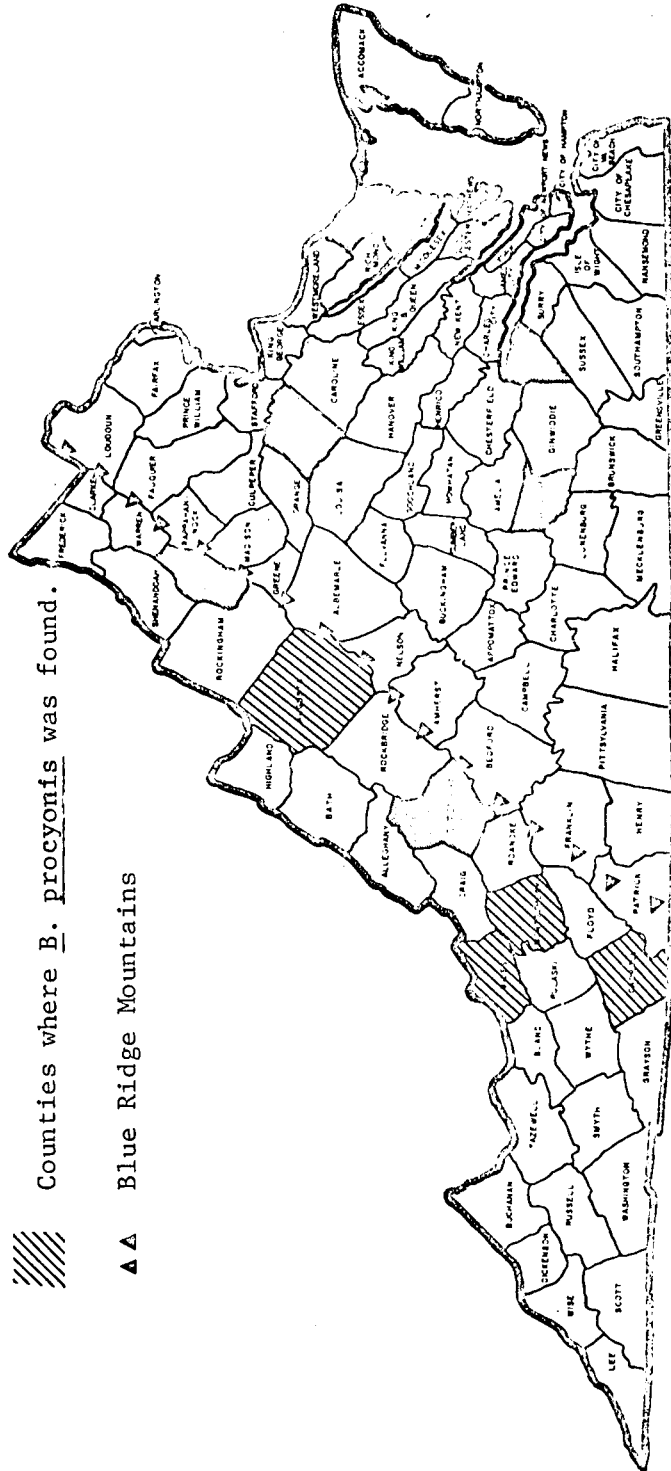


Fig. 4. Known distribution of *Baylisascaris procyonis* in Virginia.

Table 10. County, sex of raccoon, and number of each sex of Baylisascaris procyonis found in raccoons collected between 1976-1978.

County	Sex of Raccoon	Males	Females	Unknown	Total
Augusta	R	10	18	0	28
Augusta	F	1	3	0	4
Augusta	M	0	0	1	1
Carroll	F	4	5	0	9
Montgomery	M	9	16	1	26
Montgomery	F	1	0	0	1
Montgomery	M	38	91	0	129
Montgomery	F	4	4	0	8
Montgomery	M	7	12	1	20
Montgomery	F	3	9	0	12
Montgomery	F	1	1	0	2
Montgomery	M	4	4	0	8
Montgomery	F	1	4	0	5
Montgomery	F	1	11	0	12
Montgomery	M	1	10	0	11
Montgomery	M	0	4	0	4
Montgomery	M	4	3	0	7
Montgomery	F	7	5	1	13
Montgomery	F	2	1	0	3

Parasitism and Nutrition

Parasitology

The mean numbers of nematode parasites are presented in Table 11. Trichostrongylus spp. were significantly affected by drug treatment. The untreated animals had at least twice as many worms as the treated animals ($P < 0.05$; Table 12). There also tended to be more Trichostrongylus spp. and Obelicoidea cuniculi in the 70 percent ad libitum-fed animals as compared to their corresponding ad libitum-fed animals.

Nutritive restriction had no effect on the numbers of the parasite Dermatophyes veligera and Haemaphysalis tricolor present. There tended to be more numbers of Citrotaenia in the ad libitum regimens (Table 13; $P = 0.08$; Table 14).

Feed Consumption

The average daily feed consumption by the rabbits in each group prior to initiation of the treatments is shown in Table 15. The mean daily amount fed to the 70 percent ad libitum animals was 47.7 g and 47.4 g for the drug treated and untreated groups respectively. The mean feed consumption over the course of the experiment did not change appreciably except for one animal that consumed only 1 to 5 g per day over a period of 4 days. Its consumption returned to normal after this period. Two other animals that had been treated with the drug did not always consume all of the restricted ration (70 percent ad libitum) during the first 3 weeks of the experiment. The mean consumption for the ad libitum-fed groups at the end of the experiment was 61.4 g and 54.1 g for the treated and untreated groups respectively. This is a decrease of 4 g from the initial mean feed consumption for both groups.

Table 11. Regimen comparisons of the infections of Trichostrongylus spp., Obeliscoides cuniculi, and Dermatoxys veligera per host (mean $\pm$ SE).

Regimen	N	<u>Trichostrongylus</u>	<u>Obeliscoides</u>	<u>Dermatoxys</u>
Drug + Ad libitum	5	4.0 $\pm$ 3.8	7.0 $\pm$ 2.3	0.2 $\pm$ 0.2
Drug + 70 % ad lib	6	15.8 $\pm$ 8.4	39.0 $\pm$ 22.4	0.2 $\pm$ 0.2
No drug + Ad lib	3	31.7 $\pm$ 16.9	23.7 $\pm$ 15.6	1.0 $\pm$ 1.0
No drug + 70 % ad lib	5	33.2 $\pm$ 10.7	47.8 $\pm$ 27.2	0.4 $\pm$ 0.2

Table 12. Mean square values for regimen comparisons of Trichostrongylus spp., Obeliscoides cuniculi, and Dermatoxys velligera.

Source of Variation	df	<u>Trichostrongylus</u>	<u>Obeliscoides</u>	<u>Dermatoxys</u>
Drug	1	2253.33*	720.61	1.19
Nutrition	1	198.52	3501.06	0.45
Drug + Nutrition	1	117.88	68.76	0.36
Error	15	429.62	2090.63	0.59

* $P < 0.05$

Table 13. Regimen comparisons of mean (+SE) infections per host of Cittotaenia spp., Taenia pisiformis cysticerci, and Hasstilesia tricolor.

Regimen	N	<u>Cittotaenia</u>	<u>Taenia</u>	<u>Hasstilesia</u>
Drug + Ad libitum	5	0.8 + 0.4	18.4 + 6.2	1193.4 + 278.4
Drug + 70 % Ad libitum	6	0.2 + 0.2	69.0 + 35.7	1057.2 + 339.1
No drug + Ad libitum	3	0.3 + 0.3	5.7 + 3.5	1919.0 + 418.1
No drug + 70 % Ad libitum	5	0.0	45.6 + 18.4	1748.6 + 553.1

Table 14. Mean square values for regimen comparisons of Cittotaenia spp., Taenia pisiformis, and Hasstilesia tricolor.

Source of Variation	df	<u>Cittotaenia</u>	<u>Taenia</u>	<u>Hasstilesia</u>
Drug	1	0.45	1450.69	2231092.74
Nutrition	1	1.04 [*]	9106.98 ^{**}	104471.11
Drug X Nutrition	1	0.10	126.42	1297.07
Error	15	0.29	3054.07	811246.88

^{*}P=0.0764

^{**}P=0.1047

Table 15. Mean ($\pm$ SE) feed consumption per animal per day by groups prior to initiation of regimens.

Regimen	N	Feed Consumption (g/day)
Drug +Ad libitum	5	65.2 $\pm$ 5.9
Drug + 70 % Ad libitum	6	69.2 $\pm$ 2.0
No drug + Ad libitum	4	58.5 $\pm$ 7.0
No drug + 70% Ad libitum	6	67.6 $\pm$ 2.8

Body and Organ Weights

The data on initial body weight, final body weight, carcass weight, and body weight change over the course of the experiment are presented in Table 16. As indicated at the start of the experiment, there was no significant difference in the mean body weight among the groups (Table 16). At the termination there was a significant difference ($P < 0.05$; Table 17) among the body weights by nutritive restriction. The animals fed the ad libitum diet were 100 to 300 g heavier than the animals fed on the 70 percent ad libitum diet. This latter group lost weight while the ad libitum animals gained weight. This difference was significant at the $P = 0.07$ (Table 17) level. The carcass weights were also lower for the feed-restricted animals at the $P = 0.07$ (Table 17) significance level.

The means for the organ weights are listed in Table 18. The feed-restricted animals had mean liver weights of 21.25 g and 23.85 g and the ad libitum animals averaged 29.75 g and 33.18 g. This difference was significant at $P < 0.01$ (Table 19). The interaction term was significant ($P < 0.05$; Table 19) for the paired kidney weights. The means were the lowest for the group that was treated with the drug and fed 70 percent ad libitum and the group fed ad libitum, but not given the drug. This same trend was also noted for the eye lens weights. The drug treatment significantly affected the paired adrenal weights ($P < 0.05$; Table 19). The adrenals of the animals given the drug averaged 324 mg and 318 mg while the other groups had means of 241 mg and 275 mg.

The mean values and the mean square values for the male reproductive

Table 16. Regimen comparisons of mean ($\pm$ SE) initial body weights, final body weights, body weight change, and carcass weights.

Regimen	N	Initial Body Weight (g)	Final Body Weight (g)	Body Weight Change (g)	Carcass Weight (g)
Drug + Ad libitum	5	1209 $\pm$ 91	1226 $\pm$ 118	4 $\pm$ 113	712 $\pm$ 53
Drug + 70 % Ad libitum	6	1087 $\pm$ 22	959 $\pm$ 48	-151 $\pm$ 43	575 $\pm$ 37
No drug + Ad libitum	3	1059 $\pm$ 136	1184 $\pm$ 133	82 $\pm$ 85	710 $\pm$ 80
No drug + 70 % Ad libitum	5	1075 $\pm$ 89	1056 $\pm$ 64	-59 $\pm$ 45	641 $\pm$ 53

Table 17. Mean square values for the regimen comparisons of initial body weights, final body weights, body weight change, and carcass weights.

Source of Variation	df	Initial Body Weight	Final Body Weight	Body Weight Change	Carcass Weight
Drug	1	29134.81	3357.85	32417.84	4492.05
Nutrition	1	12210.37	174322.14*	97410.32	47051.98
Drug X Nutrition	1	21386.51	21603.90	223.94	5167.54
Error	15	29961.90	35464.99	26459.44	190946.97

* $P < 0.05$

Table 18. Regimen comparisons of the mean values ($\pm$ SE) for fresh liver(g), paired kidney (g), paired adrenal (mg), and mean eye lens weights (mg).

Regimen	N	Liver (g)	Paired Kidney (g)	Paired Adrenals (mg)	Eye Lens (mg)
Drug + Ad libitum	5	29.6 $\pm$ 4.3 ^a	6.6 $\pm$ 0.5	324 $\pm$ 27	244 $\pm$ 14
Drug + 70 % Ad libitum	6	21.3 $\pm$ 1.9 ^a	5.5 $\pm$ 0.2	318 $\pm$ 14	214 $\pm$ 7
No drug + Ad libitum	3	33.2 $\pm$ 5.7	5.4 $\pm$ 0.9	241 $\pm$ 24	221 $\pm$ 7
No drug + 70 % Ad libitum	5	23.9 $\pm$ 0.9	6.5 $\pm$ 0.4	275 $\pm$ 25	251 $\pm$ 17

^aOne less observation than indicated.

Table 19. Mean square values for regimen comparisons of liver weight, paired kidney weight, paired adrenal weight, and mean eye lens weight.

Source of Variation	df	Liver	Paired Kidney	Paired Adrenal	Eye Lens
Drug	1	39.21	0.01	16959.64 [*]	217.78
Nutrition	1	316.69 ^{**}	0.00	802.82	0.00
Drug X Nutrition	1	1.06	5.41 [*]	1637.45	4053.51 [*]
Error	15	38.77 ^a	0.84	2537.10 ^b	742.77

^{*}P<0.05

^{*}P<0.01

^aBased on 2 fewer degrees of freedom than indicated.

^bBased on 1 less degree of freedom than indicated.

organ weights and spermatozoa counts are in Tables 20 and 21. No significant differences among the groups were observed.

The mean values of paired ovarian and uteri weights are in Table 22. Neither drug treatment nor nutritive restriction nor the interaction had a significant effect on the female reproductive organs (Table 23). The mean paired weights tended to be lower for the animals not given the drug and the uteri weights were higher in the animals fed ad libitum.

Nutritional Indices

Table 24 presents the mean fat index values, percent femur marrow fat, and percent tibia marrow fat. Nutritive restriction had a significant ($P < 0.01$; Table 25) effect on the fat index. The restricted groups had means of 0.7 and 1.4 while the ad libitum groups had means of 2.6 and 2.3. Although the differences are not statistically significant, (Table 25) the femur marrow fat and tibia marrow fat percentages were greater in the animals fed ad libitum.

No statistically significant differences were found among the groups for packed cell volume, blood urea nitrogen, serum corticoids, and serum cholesterol levels (Tables 26 and 27). As seen in Table 26, the feed-restricted groups tended to have higher mean BUN, serum corticoids, and serum cholesterol levels and lower PCV's.

The mean values for the serum protein parameters are listed in Table 28. No significant differences were observed among groups in total serum protein, serum albumin, and serum globulin (Table 29).

Correlation Analysis

The results of a simple correlation analysis between parasites and physiological parameters are presented in Table 30. There was a strong

Table 20. Regimen comparisons of mean ($\pm$ SE) male reproductive organ weights and spermatozoa counts.

Regimen	N	Paired Testes (g)	Seminal Vesicles (mg)	Prostate (mg)	Spermatozoa (#/mg testes)
Drug + Ad libitum	3	12.1 $\pm$ 3.2	547 $\pm$ 100	308 $\pm$ 78	3.2 $\pm$ 1.0
Drug + 70 % Ad libitum	2	11.1 $\pm$ 1.9	397 $\pm$ 28	302 $\pm$ 63	5.1 $\pm$ 1.2
No drug + Ad libitum	1	1.9	-	-	1.1
No drug + 70 % Ad libitum	2	10.0 $\pm$ 2.0	652 $\pm$ 362	159 $\pm$ 57	1.7 $\pm$ 0.5

Table 21. Mean square values for the regimen comparisons of paired testes weights, seminal vesicle weights, prostate gland weights, and spermatozoa counts.

Source of Variation	df	Paired Testes	Seminal Vesicle	Prostate	Spermatozoa Counts
Drug	1	53.52	65101.52	20334.76	12.62
Nutrition	1	21.70	27084.07	47.13	2.67
Drug X Nutrition	1	36.00	0.00	0.00	0.65
Error	4	19.01	80723.62	12778.09	2.44

Table 22. Regimen comparisons of the mean ($\pm$ SE) paired ovary and uterus weights.

Regimen	N	Paired Ovaries (mg)	Uteri (g)
Drug + Ad libitum	2	106 $\pm$ 1	4.53 $\pm$ 1.72
Drug + 70 % Ad libitum	4	139 $\pm$ 32	2.59 $\pm$ 0.30
No drug + Ad libitum	2	95 $\pm$ 37	1.96 $\pm$ 0.47
No drug + 70 % Ad libitum	3	65 $\pm$ 20	2.71 $\pm$ 0.58

Table 23. Mean square values for the regimen comparisons of paired ovary and uterus weights.

Source of Variation	df	Paired Ovaries	Uteri
Drug	1	0.0045	3.797
Nutrition	1	0.0000	0.895
Drug X Nutrition	1	0.0025	4.563
Error	7	0.0025	1.356

Table 24. Regimen comparisons of the mean ($\pm$ SE) fat index, percent femur bone marrow fat, and percent tibia marrow fat.

Regimen	N	Fat Index	Femur Marrow Fat (%)	Tibia Marrow Fat (%)
Drug + Ad libitum	5	2.6 $\pm$ 0.4	70.6 $\pm$ 13.9	83.8 $\pm$ 6.6
Drug + 70 % Ad libitum	6	0.7 $\pm$ 0.3	47.7 $\pm$ 14.0	57.9 $\pm$ 17.8
No drug + Ad libitum	3	2.3 $\pm$ 0.7	75.0 $\pm$ 2.3	88.8 $\pm$ 1.1
No drug + 70 % Ad libitum	5	1.4 $\pm$ 0.6	55.2 $\pm$ 14.9	71.6 $\pm$ 12.0

Table 25. Mean square values for regimen comparisons of the fat index, femur marrow fat, and tibia marrow fat.

Source of Variation	df	Fat Index	Femur Marrow Fat	Tibia Marrow Fat
Drug	1	0.24	156.35	391.93
Nutrition	1	9.13 [*]	2036.74	2068.36
Drug X Nutrition	1	1.11	9.87	85.21
Error	18	1.09	943.63	886.46

^{*}P<0.01

Table 26. Regimen comparisons of the mean ($\pm$ SE) values for packed cell volume, blood urea nitrogen, serum corticoids, and serum cholesterol.

Regimen	N	Packed Cell Volume (%)	Blood Urea Nitrogen (mg/100ml)	Serum Corticoids (ng/ml)	Serum Cholesterol (mg/100ml)
Drug + Ad libitum	5	49.6 $\pm$ 2.1	18.4 $\pm$ 3.1	5.5 $\pm$ 3.0	94.8 $\pm$ 12.0
Drug + 70 % Ad libitum	6	43.7 $\pm$ 2.6	25.7 $\pm$ 3.4	18.9 $\pm$ 10.2 ^a	99.5 $\pm$ 15.3
No drug + Ad libitum	3	47.3 $\pm$ 3.3	16.3 $\pm$ 0.9	4.5 $\pm$ 2.7	67.7 $\pm$ 20.8
No drug + 70 % Ad libitum	5	44.8 $\pm$ 5.6	29.6 $\pm$ 10.8	6.7 $\pm$ 1.2	110.2 $\pm$ 16.5

^aOne less observation than indicated.

Table 27. Mean square values for the regimen comparisons of packed cell volume, blood urea nitrogen, serum corticoids, and serum cholesterol.

Source of Variation	df	Packed Cell Volume	Blood Urea Nitrogen	Serum Corticoids	Serum Cholesterol
Drug	1	1.43	2.50	185.11	1.43
Nutrition	1	79.65	466.94	260.34	79.65
Drug X Nutrition	1	12.84	40.45	135.65	12.84
Error	15	65.20	189.73	167.08 ^a	978.00

^aOne less degree of freedom than indicated.

Table 28. Regimen comparisons of the means ($\pm$ SE) of the total serum protein (Refractometer), total serum protein (determined by assay), serum albumin, and serum globulin levels.

Regimen	N	Total Serum Protein (Refractometer) (g/100ml)	Total Serum Protein (Assay) (g/100ml)	Serum Albumin (g/100ml)	Serum Globulin (g/100ml)
Drug + Ad libitum	5	6.6 $\pm$ 0.3	7.4 $\pm$ 1.0	3.3 $\pm$ 0.3	4.1 $\pm$ 0.8
Drug + 70 % Ad libitum	6	6.5 $\pm$ 0.4	6.7 $\pm$ 0.3	3.5 $\pm$ 0.2	3.0 $\pm$ 0.4
No drug + Ad libitum	3	7.1 $\pm$ 0.2	7.7 $\pm$ 0.7	3.3 $\pm$ 0.3	4.3 $\pm$ 0.4
No drug + 70 % Ad libitum	5	6.9 $\pm$ 0.6	8.4 $\pm$ 1.2	4.0 $\pm$ 0.6	4.4 $\pm$ 0.6

Table 29. Mean square values for regimen comparisons of total serum protein (Refractometer), total serum protein (Assay), serum albumin, and serum globulin.

Source of Variation	df	Total Serum Protein (Refractometer)	Total Serum Protein (Assay)	Serum Albumin	Serum Globulin
Drug	1	1.05	4.44	0.34	2.66
Nutrition	1	0.10	0.00	0.80	1.08
Drug X Nutrition	1	0.01	2.39	0.29	1.75
Error	15	1.03	3.49	0.80	1.76

negative correlation between the nematodes Obeliscoides cuniculi and Trichostrongylus and the fat index and bone marrow fat. Positive correlations were observed between Cittotaenia and body and organ weights, fat index, and femur marrow fat. Other significant correlations were between Hasstilesia tricolor and total serum protein as measured by both methods.

Table 30. Results of a simple correlation analysis between parasite incidence and physiological parameters.

Physiological Parameter	Parasites					
	<u>Obeliscoides</u> <u>cuniculi</u>	<u>Trichostrongylus</u> <u>spp.</u>	<u>Dermatoxys</u> <u>veligera</u>	<u>Cittotaenia</u> <u>spp.</u>	<u>Taenia</u> <u>pisiformis</u>	<u>Hasstilesia</u> <u>tricolor</u>
Final Body Weight	-0.37	-0.30	-0.02	0.63**	-0.20	-0.39
Carcass Weight	-0.42	-0.27	-0.10	0.55**	-0.17	-0.40
Liver Weight ^a	-0.38	-0.29	0.14	0.67**	-0.21	-0.22
Kidney Weight	0.07	-0.25	-0.25	0.56*	-0.09	-0.28
Adrenal Weight ^b	0.39	-0.32	-0.02	0.01	-0.03	-0.16
Eye lens Weight	-0.18	0.02	-0.35	0.34	-0.21	-0.30
Fat Index	-0.52*	-0.54	0.38	0.47*	0.01	-0.01
Femur Fat	-0.58*	-0.45*	0.25	0.46*	0.26	-0.04
Tibia Fat	-0.62**	-0.27	0.26	0.32	0.26	-0.03
BUN	0.02	-0.09	0.04	-0.30	0.21	0.41
PCV	-0.58**	-0.29	0.19	-0.09	0.17	0.16
Corticoids ^b	-0.01	0.13	-0.09	-0.20	0.06	-0.18
Cholesterol	-0.48	-0.23	0.20	-0.36	0.23	-0.13
Albumin	-0.17	-0.12	0.43	-0.38	0.25	0.48
Globulin	-0.24	-0.07	0.16	0.24	0.12	0.42
Total Protein (R)	-0.01	0.09	0.05	0.05	0.10	0.02*
Total Protein (A)	-0.27	-0.15	0.35	-0.04	0.20	0.49*

a 2 fewer observations than indicated.

* P < 0.05

b 1 less observation than indicated.

** P < 0.01

DISCUSSION

Tularemia Survey

Thirteen samples exhibited antibody titers for tularemia and 7 had titers of 1:80 or greater which were considered conclusive of infection. A titer of less than 1:40 is not often considered evidence of infection since cross reactions may occur. However, most serologic surveys of wildlife for tularemia regard a titer of 1:20 as evidence of infection. In this survey such low titers have been regarded as demonstrating probable infection.

All of the species found infected have been reported infected previously. In most of the past surveys in the United States the raccoon, opossum, and skunk, when present in the sample, were among the most commonly infected species. In this study 33.3 percent of the 15 raccoons, 50 percent of the 2 skunks, and 15.4 percent of the 13 opossums were positive for titer. Marchette et al. (1961) made the assumption that wild carnivores which possess tularemia antibodies could be assumed to have been recently exposed to tularemia. Calhoun et al. (1956) supported a similar idea from studies that showed high titers to persist for as long as 10 months in dogs and cats. These studies indicate that carnivores can serve as reservoirs and are probably refractive to the disease.

The populations of raccoons and opossums were high at Fort Pickett. During the course of the study there was no difficulty in capturing these species. Because hunting at night was not allowed at Fort Pickett, the numbers of raccoons and opossums taken by hunters were small. Only

5 and 8 raccoons were harvested during the 1976-77 and 1977-78 hunting seasons respectively.

Marchette et al. (1961) determined that F. tularensis produced fatal disease in most rodents. This was supported by evidence of die-offs of muskrats (Jellison et al. 1958; Young et al. 1969), beaver (Stenlend 1953; Lawrence et al. 1956), voles both in the United States (Jellison et al. 1958; Kartman et al. 1959) and in Europe (Dahlstrand et al. 1971). Marchette et al. (1961), however, reported that the Norway rat was an exception and was able to withstand infection. This resistance would account for the presence of a significant titer (1:80) in a Norway rat and a paucity of other rodent species captured. In view of the trapping effort, only a small number of voles and white-footed mice were captured. None of the specimens that were captured exhibited antibody response. These small rodents could be regulated by tularemia as was believed to have happened in Sweden (Hornfeldt 1978). The chipmunk that exhibited the low titer might not be refractive to the disease, but only recently infected as the titer does not peak for several days. The only other report in the literature of infection in chipmunks was by Breen (1933; cited in Reilly 1970) in Minnesota. Other Sciuromorpha have been found infected and very susceptible to tularemia. In 1977, 2 Delmarva fox squirrels (Sciuris cinereoargenteus) were found at the Chincoteague National Wildlife Refuge from which F. tularensis was isolated and believed to have been the cause of death (J.C. Appel, pers. comm.).

No other members of the Sciuromorpha were examined from Fort

Pickett. An attempt was made to capture squirrels, but this investigator was unable to do so. During the period of the study very few squirrels were sighted. The population apparently had not declined because there was a 139 percent increase in the number of squirrels harvested during the 1977-78 hunting season (the season following the study) over the number harvested the previous season (J.B. Redd, pers. comm.).

Infections have been reported previously in deer by several investigators (Cook et al. 1965; Burgdorfer et al. 1974; Hoff et al. 1975b). Human infection has even resulted from contact with infected deer (Emmons et al. 1976). Deer are the primary wildlife species for which Fort Pickett is managed, and at present the base supports a very large population. The abundance of deer and their high degree of mobility make them excellent disseminators of disease. Only 1 of 8 (12.5 percent) was found with a significant titer.

Quail are abundant, but the harvest has decreased in recent years presumably due to drought. Green and Wade (1929) reported that quail were susceptible to F. tularensis, and this was the known cause of death of 2 out of a covey of 6. The remaining 4 birds were never sighted again. This indicated that tularemia could be a major cause of mortality among quail and that the low titer reported in this study suggests that quail at Fort Pickett are infected with F. tularensis and, if not fatally infected they could be a carrier.

Rabbits

None of the 33 cottontails exhibited antibody titers. The strain

of F. tularensis present might have been virulent enough to cause death in all rabbits infected. A strain of low virulence might have permitted some animals to develop immunity to the bacillus, and thus exhibit antibody response. The paucity of rabbits at Fort Pickett would tend to corroborate this idea, otherwise an increase in cottontail numbers might be expected. The harvest of cottontails decreased by 22 percent in 1977-78 over the 1976-77 harvest.

In the fall of 1973, Jacobson et al. (1978) found 4 of 17 cottontails to have antibodies against tularemia. The titers reported were 1:5, 1:5, 1:10, and 1:20+. These titers are low, and would not have been considered significant in most circumstances. These titers could be indicative of recent exposure of the animals to the disease, in which case significant immune response had not developed before these animals were sacrificed. Also the virulence may have been low during that year, but this is doubtful because the rabbit population has continued to decline (the 1977-78 cottontail harvest was approximately 6 percent of the 1959-60 harvest (Fig. 1)).

Ectoparasites

All of the animals found to be infected with F. tularensis have arthropod parasites in common with the cottontail rabbit. As seen in Table 8, Amblyomma americanum was the most prevalent arthropod. In this study the cottontail was found to share this ectoparasite with the raccoon, white-footed mouse, opossum, white-tailed deer, striped skunk, and woodchuck. All but the woodchuck and white-footed mouse were found infected with F. tularensis at Fort Pickett. Cooney and Burgdorfer

(1974) also reported D. variabilis on cottontails in Kentucky.

The rabbit tick Haemaphysalis leporispalustris is probably the most important vector for tularemia among cottontails and quail. This tick is host specific for rabbits and birds and could be responsible for transmission from rabbit to rabbit and bird to rabbit. H. leporispalustris was found frequently on cottontails at Fort Pickett. It has been reported on quail elsewhere in the Virginia Piedmont by Sonenshine and Stout (1970).

Summary

The high incidence of titers and prevalence of potential vectors indicate that tularemia could be a major limiting factor for cottontails at Fort Pickett and perhaps throughout the Piedmont. McKeever et al. (1958), Burgdorfer et al. (1974), Hoff et al. (1975b), and Omland et al. (1977) also concluded that high incidence of titers in wild animals in an area indicated that tularemia was a limiting factor for cottontails or voles.

Most authors agree that further serologic studies should be conducted on indicator species. Since rodents and lagomorphs are normally lethally susceptible to the disease, they do not contribute much to the information regarding incidence in the wild. The present study indicated that attention should be focused on carnivores, opossums, deer, Norway rats, and possibly quail. Other birds could also be important in maintenance and dissemination of F. tularensis. Cabelli et al. (1964) has shown that both mourning doves and quail are refractive to tularemia and could act as reservoirs.

A great number of dogs are used in hunting at Fort Pickett. These dogs could not only maintain the disease in the area but also introduce it into other places. It is also possible that hunting dogs from other areas are reintroducing F. tularensis.

With such a moderately high incidence of infection in wild species, human infections could be expected. According to the Virginia State Department of Health, Bureau of Epidemiology, there have been no reported cases of tularemia in the counties surrounding Fort Pickett for the years 1976-1978. Only 14 cases of tularemia have been reported in Virginia (primarily northern Virginia) from 1976 to October 1978. Eight of these were in 1976, 3 in 1977, and 3 in 1978.

Although there were very few reported cases, human infections could have occurred. The varied clinical manifestations of the disease could have resulted in misdiagnosis and the use of antibiotics could have masked further manifestations.

Cottontail Population Age Structure

The results indicated a high proportion of adults in the population. According to Hill (1972) a normal fall population is 20 to 30 percent adults. This reduction in juveniles indicates a high juvenile mortality. In 1976-77, a bimodal distribution of birth dates was seen (Fig. 3). These 2 peaks in litters (March and June) did not appear in 1977-78 when the peak was in April.

These data suggest that juvenile recruitment is continually declining, exhibiting a total population decline. The persistence of tularemia in the environment could be instrumental in preventing recruitment.

If the juveniles were exposed to tularemia they would readily succumb and could contribute to the maintenance of the disease by being ingested by raccoons or other carnivores and scavengers. Also, if a female rabbit contracts the disease and dies while nursing, her litter would be lost through starvation. This mortality would be a function of vector activity which would start in March and continue through the summer. Environmental conditions would be important in regulating vector abundance and activity.

Distribution of Baylisascaris procyonis

Although this survey did not encompass all counties of Virginia, each physiographic region was represented. Since B. procyonis was found only in the mountainous section, cerebrospinal nematodiasis might not be important as a cottontail regulatory factor in the Piedmont. These findings concur with those made by Nettles (pers. comm.) in Georgia.

The spread of this ascarid is probably related to the translocation of raccoons. If B. procyonis is present in a raccoon population, the population of rabbits and rodents present would be affected. However, Dade et al. (1975) indicated that immunity was possible.

The reason more ($P < 0.001$) female worms (mean 10.6 ± 8.5) were found could be due to size dimorphism. The females are larger and more easily noticed and are therefore more likely to be collected. It was attempted, however, to collect all worms visible. Jacobson et al. (1976) found 49 female and 44 male B. procyonis in one raccoon.

Neither sex of raccoons was infected more often than the other. No such difference would be expected because both sexes have the same

foraging habits and would have had equal probability of infestation.

All but 2 animals examined were of sufficient age to be susceptible to infestation. These 2 animals were captured together in the same trap in Center Woods. They were estimated to be 6 weeks old, and were captured at the site where a lactating female had been captured the previous day. All other animals taken from Center Woods had been heavily infested with B. procyonis.

Parasitism and Nutrition

Effects of Nutritive Restriction

Parasite loads

Only in the case of the 3 groups of nematode species was there a difference in parasite loads due to nutrition. The treated and diet restricted animals and the control animals tended to harbor more Trichostrongylus, Dermatoxys veligera, and Obeliscoides cuniculi than did the ad libitum animals. No trends were observed in the other endoparasites observed.

Body and organ weights

Nutritive restriction had a significant effect ($P < 0.05$ Table 13) on final body weight. Carcass weights tended to be lower for the feed-restricted animals, but not significantly different. This indicated a reduction in fat content and probably no muscle tissue break down. Since mature animals were used, no changes due to growth would be expected.

Nutritive restriction had a significant effect ($P < 0.01$) on the fresh liver weights. The restricted animals had lower liver weights;

a difference not observed by Warren and Kirkpatrick (1978). This difference was probably due to the depletion of liver glycogen.

Nutritive restriction did not have an effect on the paired adrenal, paired kidney, eye lens, and male and female reproductive organ weights. Kirkpatrick and Kibbe (1971) and Warren (1976) found that nutritive restriction reduced paired ovarian weights. However, in another experiment by Warren (1976) no differences were found. The extremely small sample sizes of this study did not permit adequate evaluation of the treatment.

No differences in male reproductive characteristics due to nutritive restriction were observed. Kibbe (1969) reported one experiment where 20 percent restriction caused a decrease in the weight of male reproductive organs. The length of the experiment and the paucity of male rabbits in this study may have influenced the expression of the effects of nutritive restriction on these reproductive parameters.

Nutritional indices

Nutritive restriction significantly reduced the amount of abdominal fat present in the feed-restricted animals. This loss of abdominal fat emphasized the severity of the restriction. This lack of fat would also account for much of the difference in final body weights, and the lack of difference in the carcass weights among the regimens. The 70 percent ad libitum animals probably utilized their fat stores while the ad libitum animals increased their fat reserves.

The nutritive restriction did not have a significant effect on the femur and tibia marrow fat, but it tended to be lower in the feed-

restricted animals. This parameter is sensitive to nutritional status in ungulate species (Cheatham 1949; Riney 1955; Greer 1968; and Neiland 1970). The values reported here correspond to those reported by Jacobson et al. (1978) and were slightly higher than those of Warren and Kirkpatrick (1978). As also reported by the other investigators, the tibia marrow fat percentage was higher than the percent fat in the femur marrow.

Nutritive restriction did not have a significant effect on any of the blood parameters measured (Tables 23 and 25). Blood urea nitrogen (BUN) has been considered an indicator of stress. Although the BUN levels tended to be higher in feed-restricted animals because these animals were catabolizing tissue, the difference was not consistent enough to warrant its use as a nutritional index. BUN is too sensitive to immediate stress, such as trapping and handling (Jacobson et al. 1978a) to adequately reflect nutritional condition.

Serum cholesterol has been used as a nutritional index in cervids (LeResche et al. 1974). In cottontails it is related to stress as shown by Jacobson et al. (1978). In this study the restricted animals tended to have higher levels of serum cholesterol indicating a stressful condition. Serum corticoids, which are also indicators of stress, were only slightly greater in the restricted animals. The values reported in this study were considerably less than those reported by Jacobson et al. (1978) and Warren (1976).

Serum protein levels were not significantly different among the groups. These characteristics were apparently unaffected by the condi-

tions imposed on the animals.

Effects of Drug Treatment

Parasite loads

Drug treatment effectively ($P < 0.05$) reduced the number of Trichostrongylus present (Table 27). The numbers of Dermatoxys veligera present were also affected, but this parasite's infection was so low that no significant differences were discernable.

As expected, treatment had little effect on the numbers of trematodes and cestodes present.

Body and organ weights

Animals treated with the anthelmintic had significantly ($P < 0.05$; Table 16) heavier paired adrenal weights than the untreated animals. This trend was not observed by Jacobson et al. (1974) and is not what would be expected under the stress hypothesis of Selye (1973). It was expected that the parasitized animals would be stressed thereby eliciting an adrenal cortical response causing an increase in the size of the adrenal gland. A possible explanation for the observed difference is that the drug influenced the size of the adrenal glands by eliciting increased functioning.

None of the other body and organ parameters were significantly affected solely by drug treatment. These results agree with those reported by Jacobson and Kirkpatrick (1974) and Yuill (1964).

Nutritional indices

Parasite infection had no significant effect on any of the nutritional indices measured. Jacobson and Kirkpatrick (1974) found the drug

treatment significantly ($P < 0.05$) affected total serum protein, serum globulin, and differential cell counts. In this study the increased serum protein responses to infection did not occur as expected. Serum globulin normally increases with infection (Guyton 1971; Aljeboori and Ivey 1970) but van Adricken and Shaw (1977) reported infected calves to have lower serum protein levels. Apparently in this study the difference in nematode numbers between treated and untreated groups was not sufficient to elicit a significant difference in the blood parameters measured.

Effects of Parasite-Nutrition Interaction

Paired kidney weights and paired mean eye lens weights were the only parameters significantly ($P < 0.05$; Table 16) affected by the drug treatment+feed-restriction interaction. This difference, as seen in Table 15, was probably a function of the small sample size.

The lack of significant effects from parasitism on the physiological parameters measured was probably due to the relatively small numbers of parasites harbored by the untreated animals. The animals used in this study were captured in late winter and were in healthy condition with relatively low parasite loads.

SUMMARY AND CONCLUSIONS

Tularemia

1. The records of cottontail hunter harvest at Fort Pickett indicated a sharp decline in the early 1960's. Evidence for tularemia was found in 1962 and again in 1973. This study was undertaken to determine if mammals other than lagomorphs, and ground nesting birds serve as reservoirs for Francisella tularensis.
2. Ninety serum samples from 11 species of mammals and 1 avian species were tested for tularemia antibodies.
3. Evidence for infection was found in 5 raccoons, 3 opossums, 1 striped skunk, 1 Norway rat, 1 chipmunk, 1 white-tailed deer, and 1 bobwhite quail.
4. Ectoparasites were also collected. Dermaacentor variabilis, Amblyomma americanum, and Haemaphysalis leporispalustris were the major known tularemia vectors collected. The cottontail was found to share one or more vector species with those hosts found with tularemia antibodies except for the Norway rat and bobwhite quail.
5. The use of eyelens weights of hunter harvested cottontails to determine the age of the collected animals indicated that in 1976-77, 46 percent were adults and in 1977-78, 58 percent were adults.
6. The results of this survey indicated that tularemia could be a limiting factor among cottontails at Fort Pickett. The high incidence of infection and the different species infected could account for the persistence of the disease. The vectors shared by the reservoirs and cottontails allowed the rabbits to be easily exposed to the pathogen. The age structure of the population was biased towards adults,

indicating a decline which could be the result of tularemia infections.

Baylisascaris procyonis

7. An epizootic caused by Baylisascaris procyonis found among cottontails collected at Center Woods, Virginia Polytechnic Institute and State University, produced a diseases that could be a regulatory factor elsewhere in Virginia.
8. A survey was undertaken to determine the distribution of the adult stage of B. procyonis. Between December 1976 and February 1978, 72 raccoons from 11 counties were examined. The ascarid was found in 19 raccoons from the counties of Augusta, Carroll, and Montgomery; all of which lie west of the Blue Ridge.
9. This study indicated that B. procyonis might be a population regulatory factor in cottontail populations west of the Blue Ridge.

Parasitism and Nutrition

10. A study was undertaken to assess the effects of parasitism and nutritive restriction. In a 2 x 2 factorial design, 19 cottontails were fed ad libitum or 70 percent ad libitum and a group was treated with the anthelmintic Atgard[®] and another group was not treated.
11. Trichostrongylus were the only parasites affected by the drug treatment. The untreated rabbits had at least twice as many nematodes as the treated group.
12. Final body weights, carcass weights, and liver weights were significantly lower in the feed-restricted animals.
13. The paired kidney weights and eye lens weights were the lowest in the control group and in the group treated with the drug and fed the 70 percent ad libitum diet.

14. Paired adrenal weights were higher in the drug treated animals.
15. Neither drug treatment nor nutritive restriction had a significant effect on male reproductive organ weights, spermatozoa counts, paired ovarian weights, and uteri weights.
16. Femur and tibia marrow fat and abdominal fat indices were lower in the animals fed the 70 percent ad libitum diet.
17. Serum globulin, serum albumin, serum corticoids, serum cholesterol, total serum protein, blood urea nitrogen, and packed cell volumes were not significantly affected by drug treatment. The feed-restricted animals tended to have higher blood urea nitrogen, serum corticoids, and serum cholesterol levels and lower packed cell volumes.
18. In a correlation analysis, it was found that Obeliscoides cuniculi and Trichostrongylus were negatively correlated with fat index and bone marrow fat. Cittotaenia presence was positively correlated with the body and organ weights, fat index, and femur marrow fat. Presence of Hasstilesia tricolor abundance was correlated with total serum protein.
19. It was concluded that the lack of significant effects from drug treatment indicated that the parasite loads harbored were light and did not significantly affect the host. Nutritive restriction did have significant effects on several body characteristics. The small sample size and low parasite loads contributed to the lack of observed significant interaction between parasitism and nutritive restriction.

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APPENDIX

Appendix Table I. Species reported to have been naturally or experimentally infected with Francisella tularensis.

Species	Common Name	Source
Class Mammalia		
Marsupialia		
Didelphidae		
<u>Didelphis marsupialis</u>	Opossum	Calhoun et al. 1956 McKeever et al. 1958 Burgdorfer et al. 1974 Hoff et al. 1975a
Insectivora		
Soricidae		
<u>Sorex vagrans monticola</u>	Wandering Shrew	Kohls and Steinhaus 1943
Edentata		
Dasypodidae		
<u>Dasypus novemcinctus</u>	Armadillo	Hoff et al. 1975a
Lagomorpha		
<u>Lepus</u> spp.	Jack Rabbit	Francis 1921
<u>Lepus americanus</u>	Snowshoe Rabbit	Green and Schilling 1934 Green 1943

Appendix Table I. Species reported to have been naturally or experimentally infected with Francisella tularensis. (continued).

Species	Common Name	Source
<u>L. a. columbiensis</u>	Varying Hare	Parker et al. 1931 in <u>Burroughs et al. 1945</u>
<u>L. californicus</u>	Black-tailed Jack Rabbit	Burroughs et al. 1945 Marchette et al. 1961
<u>L. c. texanus</u>	Black-tailed Jack Rabbit	Marchette et al. 1961
<u>L. townsendii campanius</u>	White-tailed Jack Rabbit	Bow and Brown 1943
<u>Sylvilagus audubonii</u>	Desert Cottontail	Marchette et al. 1961
<u>S. floridanus</u>	Eastern Cottontail Rabbit	Wherry and Lamb 1914 Green 1943 McKeever et al. 1958 Bellig 1962 McCahan et al. 1962 McGinnes 1964 Franklin et al. 1966 Burgdorfer et al. 1974 Jacobson et al. 1978
<u>S. palustris</u>	Marsh Rabbit	Hoff et al. 1975b
Rodentia		
Sciuridae		
<u>Tamias striatus</u>	Eastern Chipmunk	Green 1933 in <u>Reilly 1970</u>

Appendix Table I. Species reported to have been naturally or experimentally infected with Francisella tularensis (continued).

Species	Common Name	Source
<u>Eutamias</u> spp.	Chipmunk	Parker 1945 in Reilly 1970 Nakamura 1950a
<u>E. minimus</u>	Least Chipmunk	Marchette et al. 1961
<u>E. m. pictus</u>	Least Chipmunk	Thorpe et al. 1965 Vest et al. 1965
<u>E. dorsalis</u>	Cliff Chipmunk	Green 1933 in Reilly 1970
<u>Marmota monax</u>	Woodchuck	Burgdorfer et al. 1974
<u>M. flaviventris</u>	Yellow-bellied Marmot	Lillie and Francis 1936
<u>Citellus armatus</u>	Uinta Ground Squirrel	Nakamura 1950a
<u>C. beecheyi</u>	California Ground Squirrel	McCoy 1911 McCoy and Chapin 1912 Simons et al. 1953
<u>C. b. douglasii</u>		Simons et al. 1953
<u>C. b. fisheri</u>		Simons et al. 1953
<u>C. columbianus columbianus</u>	Columbian Ground Squirrel	Burroughs et al. 1945
<u>C. leucurus</u>		Stagg et al. 1956 Vest and Marchette 1958 Marchette et al. 1961 Vest et al. 1965 Thorpe et al. 1965

Appendix Table I. Species reported to have been naturally or experimentally infected with Francisella tularensis (continued).

Species	Common Name	Source
<u>C. richardsonii</u>	Richardson's Ground Squirrel	Brown and Roy 1943 in Reilly 1970
<u>C. r. elegans</u>	Wyoming Ground Squirrel	Burroughs et al. 1945
<u>C. townsendii</u>	Townsend's Ground Squirrel	Vest and Marchette 1958
<u>C. t. mollis</u>	Piute Ground Squirrel	Francis 1919 Francis 1921 Simons et al. 1953
<u>Cynomys leucurus</u>	White-tailed Prairie Dog	Davis 1935
<u>Sciuris carolensis</u>	Eastern Gray Squirrel	Green 1933 in Reilly 1970 Burgdorfer et al. 1974
<u>Sciuris niger</u>	Fox Squirrel	McKeever et al. 1958
<u>S. n. cinereoargenteus</u>	Delmarva Fox Squirrel	Appel (pers. comm.)
<u>Tamiasciuris hudsonicus</u>	Red Squirrel	Francis 1934 in Reilly 1970
<u>T. h. ventorum</u>	Wind River Pine Squirrel	Nakamura 1950a
Heteromyidae		
<u>Perognathus parvus</u>	Great Basin Pocket Mouse	Thorpe et al. 1965 Vest and Marchette 1958
<u>P. formosus</u>	Long-tailed Pocket Mouse	Vest et al. 1965
<u>P. f. inoculatus</u>	Long-tailed Pocket Mouse	Vest and Marchette 1958 Marchette et al. 1961

Appendix Table I. Species reported to have been naturally or experimentally infected with Francisella tularensis (continued).

Species	Common Name	Source
<u>Dipodomys ordii</u>	Ord's Kangaroo Rat	Thorpe et al. 1965 Vest et al. 1965
<u>D. o. pallidus</u>		Vest and Marchette 1958
<u>D. o. utahensis</u>		Marchette et al. 1961
<u>D. microps</u>	Chisel-toothed Kangaroo Rat	Vest et al. 1965
<u>D. m. bonnevilli</u>	Chisel-toothed Kangaroo Rat	Vest and Marchette 1958 Thorpe et al. 1965
Castoridae		
<u>Castor canadensis</u>	Beaver	Scott 1940 Hammersland and Joneschild 1940 Stenlund 1953 Banfield 1954 Lawrence et al. 1956 Owen et al. 1961 Bell et al. 1962 Thorpe et al. 1965 Bell and Stewart 1975
Cricetidae		
<u>Reithrodontomys megalotis</u>	Western Harvest Mouse	Thorpe et al. 1965 Vest et al. 1965
<u>R. m. megalotis</u>	Western Harvest mouse	Marchette et al. 1961

Appendix Table I. Species reported to have been naturally or experimentally infected with Francisella tularensis (continued).

Species	Common Name	Source
<u>Peromyscus</u> spp.		Simons et al. 1953 Stagg et al. 1956
<u>P. crinitus</u>	Canyon Mouse	Vest and Marchette 1958
<u>P. c. pergracilis</u>	Canyon Mouse	Marchette et al. 1961
<u>P. gossypinus</u>	Cotton Mouse	Hoff et al. 1975b
<u>P. maniculatus</u>	Deer Mouse	Vest and Marchette 1958 Vest et al. 1965
<u>P. m. osgoodi</u>	Osgood White-footed Mouse	Ozburn 1944
<u>P. m. artemisiae</u>	White-footed Mouse	Nakamura 1950a
<u>P. m. ribidus</u>	Redwood's White-footed Mouse	Burroughs et al. 1945
<u>P. m. sonoriensis</u>	Sonoran White-footed Mouse	Stagg et al. 1956 Marchette et al. 1961
<u>P. polionotus</u>	Oldfield Mouse	Hoff et al. 1975b
<u>P. truei</u>	Pinyon Mouse	Vest and Marchette 1958 Thorpe et al. 1965 Vest et al. 1965
<u>Onychomys leucogaster</u> <u>utahensis</u>	Northern Grasshopper Mouse	Marchette et al. 1961
<u>Sigmodon hispidus</u>	Cotton Rat	Hoff et al. 1975b
<u>Neotoma albigula</u>	White-throated Wood Rat	Ecke and Holdenreid 1952

Appendix Table I. Species reported to have been naturally or experimentally infected with Francisella tularensis-(continued).

Species	Common Name	Source
<u>Neotoma fuscipes</u>	Dusky-footed Wood Rat	Burroughs et al. 1945
<u>N. lepida lepida</u>	Desert Wood Rat	Stagg et al. 1956 Vest and Marchette 1958 Marchette et al. 1961
<u>Microtus californicus</u>	California vole	Burroughs et al. 1945
<u>M. c. aesturatus</u>	Tule Meadow Vole	Perry 1928
<u>M. montanus</u>	Montana Vole	Jellison et al. 1958 Owens et al. 1961
<u>M. m. nexus</u>	Montane Meadow Vole	Marchette et al. 1961
<u>M. ochragaster</u>	Prairie Vole	Burgdorfer et al. 1974
<u>M. pennsylvanicus</u>	Meadow Vole	Bell and Stewart 1975
<u>M. p. modestus</u>	Sawatch Meadow Vole	Kohls and Steinhaus 1943
<u>M. p. drummondi</u>	Drummond's Meadow Vole	Ozburn 1944
<u>Ondatra zibethica</u>	Muskrat	Jellison et al. 1942 Banfield 1954 Young et al. 1969
Muridae		
<u>Rattus norvegicus</u>	Norway Rat	McCoy and Chapin 1912 Dieter and Rhodes 1926 Burroughs et al. 1945 Marchette et al. 1961

Appendix Table I. Species reported to have been naturally or experimentally infected with Francisella tularensis (continued).

Species	Common Name	Source
Zapoidae		
<u>Zapus princeps</u>	Western Jumping Mouse	Nakamura 1950b
Carnivora		
Canidae		
<u>Canis familiaris</u>	Dog	Ey and Daniels 1941 Johnson 1944 Calhoun 1954 Calhoun et al. 1956 Burgdorfer et al. 1974
<u>Canis latrans</u>	Coyote	Guilford 1947 Stagg et al. 1956 Thorpe et al. 1965 Vest et al. 1965
<u>C. l. lestes</u>	Coyote	Marchette et al. 1961
<u>Vulpes fulva</u>	Red Fox	Lillie and Francis 1936 McKeever et al. 1958
<u>V. macrotis</u>	Kit Fox	Thorpe et al. 1965
<u>V. m. nevadensis</u>	Kit Fox	Marchette et al. 1961
<u>Urocyon cinereoargenteus</u>	Gray Fox	Schlotthauer et al. 1935 McKeever et al. 1958 Burgdorfer et al. 1974

Appendix Table I. Species reported to have been naturally or experimentally infected with Francisella tularensis (continued).

Species	Common Name	Source
Procyonidae		
<u>Procyon lotor</u>	Raccoon	Calhoun et al. 1956 McKeever et al. 1958 Burgdorfer et al. 1974 Hoff et al. 1975b
Mustelidae		
<u>Mustela vison</u>	Mink	Nakamura 1950a Henson et al. 1978
<u>Taxidea taxus taxus</u>	Badger	Nakamura 1950a Marchette et al. 1961
<u>Spilogale putorius</u>	Spotted Skunk	McKeever et al. 1958
<u>Mephitis mephitis</u>	Stripped Skunk	Francis 1937 McKeever et al. 1958 Burgdorfer et al. 1974
Felidae		
<u>Felis domesticus</u>	Cat	Green and Wade 1928 McKeever et al. 1958
<u>Lynx rufus</u>	Bobcat	McKeever et al. 1958 Thorpe et al. 1965
Artiodactyla		
Cervidae		

Appendix Table I. Species reported to have been naturally or experimentally infected with Francisella tularensis (continued).

Species	Common Name	Source
<u>Dama dama</u>	Fallow Deer	Burgdorfer et al. 1974
<u>Odocoileus hemionus</u>	Mule Deer	Thorpe et al. 1965 Vest et al. 1965 Emmons et al. 1976
<u>O. virginianus</u>	White-tailed Deer	Cook et al. 1965 Thorpe et al. 1965 Burgdorfer et al. 1974 Hoff et al. 1975b
Bovidae		
<u>Bos taurus</u>	Domestic Cattle	Burgdorfer et al. 1945 Calhoun et al. 1956
<u>Ovis aries</u>	Domestic Sheep	Parker and Butler 1929 Jellison and Kohls 1955
Perissodactyla		
Equidae		
<u>Equus caballus</u>	Domestic Horse	Claus et al. 1959 in Reilly 1970
Hystriocomorpha		
Erethizontidae		
<u>Erethizon dorsatum</u>	Porcupine	Nakamura 1950b

Appendix Table I. Species reported to have been naturally or experimentally infected with Francisella tularensis (continued),

Species	Common Name	Source
Class Aves		
Anseriformes		
Anatidae		
<u>Anas carolinensis</u>	Green-winged Teal	Hopla 1974
<u>Anas platyrhynchos</u>	Mallard	Hopla 1974
Galliformes		
Tetraonidae		
<u>Dendragapus obscurus</u>	Blue Grouse	Parker 1929
<u>Bonasa umbellus</u>	Ruffed Grouse	Green and Shillinger 1932 Green 1943
<u>Pedioecetes phasianellus</u>	Sharp-tailed Grouse	Green and Shillinger 1932 Green 1943
<u>Centrocercus urophasianus</u>	Sage Hen	Parker et al. 1932
Phasianidae		
<u>Colinus virginianus</u>	Bobwhite Quail	Green and Wade 1928

Appendix Table I. Species reported to have been naturally or experimentally infected with Francisella tularensis (continued).

Species	Common Name	Source
Charadriiformes		
Laridae		
<u>Larus pipixcan</u>	Franklin Gull	Ozburn 1944 Stahl et al. 1969
<u>Anous stolidus</u>	Common Noddy	Cabelli et al. 1964
<u>A. tenuirostris</u>	White-capped Noddy	Cabelli et al. 1964
<u>Gyris alba</u>	White Tern	Cabelli et al. 1964
<u>Sterna fuscata</u>	Sooty Tern	Cabelli et al. 1964
Columbiformes		
Columbidae		
<u>Zenaida macroura</u>	Mourning Dove	Cabelli et al. 1964
Strigiformes		
Strigidae		
<u>Bubo virginianus</u>	Horned Owl	¹⁹³⁸ Green and Evans in Burroughs et al. 1945

Appendix Table I. Species reported to have been naturally or experimentally infected with Francisella tularensis (continued).

Species	Common Name	Source
Class Insecta		
Diptera		
Tabanidae		
<u>Chrysops discalis</u>	Deer Fly	Francis and Mayne 1921
<u>C. fulvaster</u>		Cox 1965
<u>C. aestuans</u>		Cox 1965
<u>C. noctifer</u>		Parker 1933
<u>Tabanus rupestris</u>		Parker 1933
<u>T. septentrionalis</u>		Parker 1933
Muscidae		
<u>Musca domestica</u>	House Fly	Wayson 1914
<u>Stomoxys calcitrans</u>	Stable Fly	Wayson 1914
Class Arachnida		
Acarina		
Ixodidae		
<u>Amblyomma americanum</u>	Lonestar Tick	Calhoun and Alford 1955
<u>Dermacentor albipictus</u>	Winter Tick	Bell 1965
<u>D. andersoni</u>	Rocky Mountain Wood Tick	Parker et al. 1924

Appendix Table I. Species reported to have been naturally or experimentally infected with Francisella tularensis (continued).

Species	Common Name	Source
<u>D. occidentalis</u>	Wood Tick	Parker et al. 1929
<u>D. parumpertus</u>		Philip et al. 1955
<u>D. variabilis</u>	American Dog Tick	Green 1931 Davis et al. 1934 Green 1943 Burgdorfer et al. 1974
<u>Haemaphysalis leporispalustris</u>	Rabbit Tick	Parker et al. 1931 Green 1943
<u>H. chordeilis</u>		Parker et al. 1932
<u>Ixodes dentatus</u>		Parker et al. 1952
<u>I. pacificus</u>		Davis and Kohls 1937
<u>I. scapularis</u>	Black-legged Tick	Hopla 1960

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SOME DISEASES AND PARASITES AFFECTING
COTTONTAIL RABBITS IN VIRGINIA

by

Edwin John Jones

(ABSTRACT)

A serologic survey at Fort Pickett, Virginia was undertaken to determine if tularemia could be a factor in the continued low hunter harvest of cottontail rabbits. Between December 1976 and February 1978, ninety serum samples were collected from 11 species of mammals and 1 avian species, and tested for antibodies against Francisella tularensis. Evidence of infection was found in 5 raccoons, 3 opossums, 1 striped skunk, 1 Norway rat, 1 chipmunk, 1 white-tailed deer, and 1 bobwhite quail. This indicated that tularemia was present at Fort Pickett in a number of species and could be responsible for the low numbers of cottontails present.

As the result of an epizootic of cerebrospinal nematodiasis among rabbits caused by Baylisascaris procyonis, a survey of the presence of B. procyonis in its definitive host was undertaken. Between December 1976 and February 1978, 72 raccoons from 11 counties were examined. B. procyonis was found in raccoons from Augusta, Carroll, and Montgomery Counties. It was not found in any raccoons collected from the 6 counties east of the Blue Ridge. This indicates that B. procyonis may only be a cottontail regulatory factor west of the Blue Ridge Mountains.

A final phase of the study was to determine the effects of parasitism and nutritive restriction. In a 2x2 factorial design experiment 19 cottontails were placed on an ad libitum or 70 percent ad libitum diet and treated with Atgard^R, an anthelmintic, or untreated control. It was found that Trichostrongylus spp. were the only parasites significantly affected by drug treatment. The animals on the 70 percent ad libitum diet had lower final body weights, carcass weights, liver weights, tibia and femur marrow fat levels, and lower abdominal fat indices. It was concluded that the parasite loads were too light to significantly affect the host.