THE EFFECT OF NUTRITION ON THE SUSCEPTIBILITY OF SPLENECTOMIZED CALVES TO ANAPLASMOSIS

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INTRODUCTION

Bovine anaplasmosis is an important cause of cattle losses in the United States, particularly in the southern half of the country. Additional cases of the disease are constantly being reported from the northern states. Thus, what has been considered primarily a problem of the southern region is becoming a problem of nationwide importance. While anaplasmosis will probably not reach the magnitude in the North that it holds in the South, due to the difference in insect vector population, there will undoubtedly be an increasing incidence of this disease in the North as the number of carrier cattle in the northern states increase.

The livestock producer is interested, mainly, in reducing losses from anaplasmosis by any methods available. Methods to accomplish this are limited, for either the producer or veterinarian, at the present time. Serologic diagnosis of carrier cattle, and their subsequent removal from the herd, is the only perfected method of reducing losses from anaplasmosis, and this still awaits general acceptance and implementation by livestock officials before it will become effective. Treatment during the acute course of the disease is ineffective. Although the broad spectrum antibiotics are capable of inhibiting the development of the causative agent, cattle sick under practical field conditions are not diagnosed before the appearance of acute anemia, on which the antibiotics have no beneficial effect. Therefore, it would aid in the reduction of losses from anaplasmosis if additional methods could be found to adversely affect the development of the causative organism and to reduce the degree of anemia.

This disease in cattle in the United States is reputed to be caused by an infectious organism, Anaplasma marginale. The classification of this organism has not been definitely established. It shows some characteristics of an intracellular nature similar to virus and rickettsia and, yet, it is generally classified as belonging among the protozoa. The fact that a large body of literature reports the effects of nutrition on the resistance-susceptibility of animals to infectious disease suggests a new approach to reducing losses in cattle infected with anaplasmosis.

The literature indicates that the viral organisms produce less intense infections in hosts fed deficient rations. The developmental course of rickettsia infection is altered and the susceptibility of the host is frequently increased by deficient diets. Anaplasma marginale, while much is yet to be learned concerning its nature, is known to be intracellular for at least a part of its existence. Deficiency nutrition of the host, likewise, affects the development and apparent virulence of protozoan parasites. A. marginale has generally been considered to be a protozoan. Therefore, from previous work, it would be expected that deficiency nutrition of the host would produce some change in the relationship of A. marginale to its definitive host, the bovine.

The experiment reported here was designed as a preliminary investigation of the relationship between cattle nutrition and anaplasmosis.

LITERATURE REVIEW

The period during and immediately following World War II afforded the opportunity for observation of disease in populations under the stress of starvation and the social and economic disorganization concommitant with war. Markowski (1945) has stated that when typhus broke out in the prisoner of war camp in which he was interned at Hammerstein, the mortality rate among the Russian prisoners was only 30 percent of those infected while among the German guards infected, 100 percent died. The prisoners lived under the worst possible conditions of sanitation, housing and nutrition. Similarly Keys et al. (1950) presents reports from the Warsaw Ghetto during the worst period there of overcrowding, starvation and almost complete lack of sanitation that epidemics were very rare and ran a benign course. Typhus was endemic in the Ghetto for the last two years, but deaths from typhus declined during the worst period of starvation.

Keys (1949) makes the additional statements concerning the Warsaw Ghetto that measles and meningitis in the starving children were reduced in incidence and those that were affected had only mild cases. The better fed the infected children were, the more severe were the courses of the two diseases. Rheumatic fever among the children of the Ghetto almost disappeared during the period of starvation.

In summarizing his observations, made under starvation conditions of the war, Keys et al. (1950) thinks that, in general, the infections that primarily involve the mucocutaneous barrier were increased, due largely to the breakdown of sanitation and hygiene rather than to any

direct influence of nutrition. On the other hand, infections requiring blood transport and internal lodgement in the body did not seem to be increased in either incidence or severity by a general state of severe undernutrition.

The above observations emphasize the change in thinking on the relation of nutrition to infectious disease that has taken place during the past two decades. These observations and the experimental work to follow indicate that the host-parasite relationship is an extremely delicate adjustment between a specific host and a specific infectious agent in which the nutrition of the host may either increase or decrease the susceptibility of that host to a specific parasite.

Virus:

Much of the work on virus infection has been with poliomyelitis virus in mice due to the widespread interest and funds available for poliomyelitis research. Generally speaking, most of the work has shown that nutritional deficiencies reduced the susceptibility of the host to infection by the virus as measured by death or paralysis. In fact, Foster et al. (1944) has shown that restriction of the adequate control diet alone without a deficiency of any one element will produce less paralysis and fewer deaths in mice infected with the Lansing strain of poliomyelitis virus than in infected mice full fed the control diet.

When various elements of the diet are considered, it has been found that deficiencies of some compounds reduce susceptibility to certain virus while others have no effect. Sprunt (1948) found that a low protein diet reduced the mortality in mice infected with swine influenza over the controls fed a complete diet. If methionine was included in the low protein diet, there was an increase in the mortality of the infected mice.

Somewhat different effects of low protein diet were noted by Jones et al. (1946) on mice infected with poliomyelitis. In this experiment, it was found that the low protein diet increased the length of the incubation period but that there was no change in the ultimate mortality of the mice on the low protein diet over the control mice on the normal diet.

Davies et al. (1952) determined the effect on susceptibility of deficiency of certain of the amino acids in mice infected with the Lansing strain of poliomyelitis virus. They found that lysine deficiency produced the least effect - only prolonging the incubation time and a slight decrease in the number of paralyzed animals. Valine and phenylalamine deficiencies caused the next greatest decrease in paralysis while deficiencies of tryptophan and isoleucine were most effective in lowering the number of paralyzed animals and increasing the incubation period. These workers also measured the quantity of virus in the central nervous system and found that the virus multiplied more rapidly and reached higher levels in the non-deficient mice than in the tryptophan deficient mice. Also, it was found that the central nervous system of the tryptophan deficient mice supported virus growth longer than that of the control mice.

Deficiencies of various vitamins in the ration and their effect on susceptibility to virus infection have been given much attention. Thiamine deficient diets result in a lower incidence of infection in mice to Theiler's virus than in control mice given optimum levels of thiamine (Rasmussen et al., 1944). When these workers then gave thiamine to the deficient survivors those mice then became paralyzed after a prolonged incubation period. These workers also found that reduced caloric values in the presence of adequate vitamins produced similar but less marked results. Foster et al. (1944) showed results conforming with this work.

Jones, Foster and Henle (1948) went a step further to show that a thiamine inhibitor, oxythiamine, gave significant degree of protection when fed to mice infected with the Lansing virus but that the protection was less than when a thiamine deficient diet was fed.

Pyridoxine deficiency in virus infection in mice show results similar to those found in thiamine deficiency. Leftwich and Mirick (1949) inoculated mice with the pneumonia virus of mice and at the same time placed the mice on a pyridoxine free synthetic diet. The pyridoxine free mice were less susceptible to the infection than the control mice. They conclude that pyridoxine is apparently necessary for optimal virus multiplication. In contrast to some of the previously reported work, these authors could not find any change in susceptibility to this virus when protein in the diet was restricted. A different approach to the problem was made by the same workers Mirick and Leftwich (1949) by feeding the pyridoxine deficient diet for eight days before exposing the mice to the virus. In this case, the pyridoxine deficient mice were more susceptible to the virus than were the controls.

The effect of minerals in the diet on the susceptibility of mice to Theiler's virus (GD VII) was investigated by Lickstein et al. (1946). Deficiencies of calcium, magnesium and chlorine had no effect on susceptibility while sodium had a slight effect. However, a progressively increased susceptibility was found as the amount of potassium and phosphorus was increased in the diet.

Rickettsia:

While the rickettsia are intracellular organisms, or closely associated with living cells, the little experimental work available on their resistance-susceptibility relationship to the host, in relation to nutrition, indicates that some deficiency states cause an increased susceptibility

of the host to rickettsial infection as contrasted to the decreased susceptibility of the host to viral infection under similar conditions. Increased susceptibility to infection of rats with typhus, as measured by mortality, has been shown by Fitzpatrick (1948) for deficiencies of protein, pantothenic acid, riboflavin and thiamine. There was no change in susceptibility when the infected rats were given diets deficient in pyridoxine, choline, niacin or para-aminobenzoic acid. Pinkerton and Bessy (1939) had previously shown similar results with riboflavin.

Pinkerton (as cited by Clark et al. 1949) has also indicated that the symptoms of murine typhus infection in guinea pigs is changed by starvation from scrotal inflammation with febrile reaction to a non-inflammatory, non-febrile reaction with increased numbers of rickettsia in a gelatinous exudate in the peritoneal cavity and scrotum.

Bacteria:

In general, the work with bacterial infections under experimental conditions has indicated that deficiency conditions in the diet of the host animals has resulted in increasing the susceptibility of the host to bacterial invasion.

Dubos (1955), using mice as the host and the tubercule bacillus as the infectious agent, has presented extensive data on the resistance-susceptible state as related to nutrition. He used three diets: 1) Purina lab chow, 2) synthetic diet 191 containing 18 percent casein and 5 percent fat, 3) dried skim milk (36.8 percent protein and 0.8 percent fat) and wheat flour. Mice fasted 30 hours per week but otherwise fed ad lib. were more susceptible to tuberculosis than the mice on any of the diets fed continuously. In contrast, susceptibility was not affected by limitation of food intake to a low constant daily level.

Susceptibility of the mice was independent of the protein content of the diet but low protein with high carbohydrate produced high susceptibility. However, if a part of the carbohydrate was replaced with peanut oil susceptibility was similar to that in the controls. Susceptibility was increased by the addition of sodium citrate to any of the diets and, also, if dinitrophenol or thyroxine were given in amounts to limit the weight gain. It is interesting that fatal infections of the attenuated BCG organism could be produced in the mice by any of these methods of increasing susceptibility. Some of Dubos'conclusions are that these findings are consistent with, but do not prove, the hypothesis that increased susceptibility can be brought about by metabolic disturbances that cause a depletion of glycogen reserves, reduction of glycolytic activity of the inflammatory cells or by an increase in the concentration of certain polycarboxylic acids and ketones. Higgins and Feldman (1943) did not find any influence on susceptibility of rats to avian tuberculosis of deficiencies of thiamine and riboflavin. Excess amounts of vitamin C, given by Heise and Stunken (1939) both before and after inoculation of guinea pigs with tubercle bacilli, did not influence susceptibility.

Schneider (1946, 1949) has isolated from wheat germ by extracting with acetone what he termed the susceptibility factor in the acetone soluble fraction and the resistance factor in the residue from the acetone extraction. Seventy-five percent of the mice infected with Salmonella enteriditis survived on a natural diet containing whole wheat and whole dried milk. Sixty-five percent of such mice survived on a basal synthetic diet containing 20 percent wheat germ and also on the basal diet containing the resistance factor. None of the mice survived on the basal diet plus the acetone soluble susceptibility factor.

Mice infected with Type one pneumococci on diets deficient in thiamine or riboflavin were more susceptible than those on a complete diet (Wooley and Sebrell, 1942) while, in mice and rats with the same infection on a pantothenic acid deficient diet, there was no change in susceptibility. However, Robinson and Siegel (1944) report that deficiencies in thiamine and possibly pyridoxine reduced resistance while there was no change in riboflavin or pantothenic acid deficiencies in mice exposed to pneumococci.

An increase in susceptibility due to vitamin C deficiency is reported by Kelly (1944) in guinea pigs infected with Spirillum sputigenum. Normally, this organism is non-pathogenic for the guinea pig but, under this condition, produced local inflammation and abscesses.

Protozoa:

Due to the interest in malaria these protozoan parasites in poultry as well as in mammals, have been the subject of many investigations into the host-parasite relationship as modified by nutrition. Among the protozoan parasites, some nutritional deficiencies will cause a reduction in the number of parasites in the host while deficiencies of other dietary factors increase the number of parasites and the severity of the reaction in the host.

Reduction of the total food intake of chicks infected with Plasmodium lophurae by Seeler and Ott (1944a) resulted in higher parasite counts and increased mortality over the ad lib. fed infected control birds. Studies by the same workers (1945) show a similar increase in susceptibility of chicks to P. lophurae when fed a protein deficient diet.

Deficiencies of certain vitamins in malarial infections result in fewer parasites in the blood but an increase in deaths. This is indicated

by Seeler and Ott (1944) in P. lophurae infected chicks fed riboflavin deficient diets. Also, administration of riboflavin to the deficient chicks during infection resulted in an increase in total parasites. A similar reduction in the total number of parasites in chickens infected with P. gallinaceum is reported by Brackett et al. (1946) when he gave the birds a diet deficient in pantothenic acid. The results were similar when analogues of pantothenic acid were administered to the chickens fed a diet adequate in pantothenic acid.

Trager (1943) found that calcium pantothenate favors survival of P. lophurae in chicken erythrocytes in vitro.

Biotin deficiency also increases the number of P. lophurae in chickens (Seeler and Ott, 1944b) and Trypanosoma lewisi in rats (Caldwell and György, 1947). The latter writers also report that injected hyperimmune serum did not protect the biotin deficient animals from infection with T. lewisi. They attribute this to abnormality in the production of complement due to the deficiency.

In a study using monkeys infected with Plasmodium knowlesi, McKee and Geiman (1946) report that plasma and whole blood ascorbic acid levels were significantly lower in the parasitized than in the control monkeys. In these spontaneous deficient animals, and in some monkeys in which they induced ascorbic acid deficiency, there was a longer period of development of the parasite. Administration of ascorbic acid to the deficient animals resulted in a normal course of parasitism.

Antibody Formation:

Since the formation of specific antibodies is part of the resistance response of a host to infection with a specific parasite, the measurement of the amount of antibody found should be an indirect measure of the

amount of resistance response of the host to infection. This measure of resistance has been widely used in the study of the relation of nutrition to resistance.

Cannon and co-workers have presented extensive data to support their view that the proteins are important in the formation of antibodies. (Cannon, 1944, 1945). This group, in a study by Wissler et al. (1946), reported that when rats were fed a protein depletion diet for 183 days, then given specific crystalline amino acids for 7 days and inoculated on the seventh day with sheep erythrocytes, there was a marked increase of antibody formation over the low-protein controls. This was true for the following amino acids: arginine, histidine, lysine, tryptophan, phenylalanine, methionine, threonine, leucine, isoleucine, valine, tyrosine, cystine, glutamic acid, aspartic acid, glycine and alanine. Other workers (Berry et al., 1945) fed rats a diet similar to that consumed in an area of endemic malnutrition as a basal ration and then supplemented this diet with casein, minerals and B vitamins. Antibody response was much higher in the supplemented group than in the basal ration group.

In contrast to the work of Cannon's group, Zilva (1919) and Metcoff (1949) could find no relation between protein nutrition and immune response. These workers studied not only the antibody response but that of complement and amboceptor. It may well be that the differences in the reports of these workers is not in fact but in technique. It is logical to assume that there would be correlation between diet protein and antibody production.

Certain of the vitamins have been investigated in their relation to antibody response. Pyridoxine deficient diets have produced a lowered antibody response (Stoerk et al., 1947; Agnew and Cook, 1949). Pteroylglutamic acid deficiency also inhibits the response of antibodies in the

host (Little et al. 1950; Ludovici and Axelrod, 1951). Ludovici and Axelrod (ibid.) also investigated the effect of niacin-tryptophan, vitamin B_{12}, vitamin A and vitamin D deficiencies on antibody response and have grouped the deficiencies by their effect on antibody response as follows: Severe impairment - pantothenic acid, pyridoxine, pteroylglutamic acid; moderate impairment - riboflavin, thiamine, biotin, vitamin A, niacin-tryptophan; no impairment - vitamin B_{12} and vitamin D. They also found that methionine was capable of a sparing action in the requirement for pantothenic acid in antibody formation but not for growth. Wertman et al. (1951, 1952) also shows a reduced antibody response, using the rickettsia of murine typhus as antigen, in pantothenic acid, thiamine, riboflavin and folacin deficiencies.

It is the opinion of Schneider (1951) that the confusion and inconsistent findings of the workers in the field of nutrition, as related to resistance-susceptibility of the host to infectious disease, has arisen from the failure to recognize that resistance and susceptibility are two separate biological attributes of the host and not different ends of the same attribute with interchange of the terms "more susceptible" or "less resistant." Since resistance and susceptibility are distinct attributes, they are capable of separate manipulation and are inherited as separate traits (Webster, 1937). Schneider (1949) shows that the effect of nutrition on resistance-susceptibility cannot be demonstrated except on a host heterogenetic to resistance and susceptibility and a pathogen heterogenetic for virulence and avirulence.

EXPERIMENTAL OBJECTIVE

To determine the effect of nutrition on resistance-susceptibility of splenectomized calves to infection with anaplasmosis as measured by:

a. Mortality

b. Degree of infection

c. Blood pathology

d. Serology

e. Histopathology

PROCEDURE

Twenty calves of mixed breeding, but predominately of dairy stock, ranging in age from 4 to 14 weeks, were splenectomized and placed in a randomized block design of four experimental groups and five outcome groups based on age. This design was adopted because the wide difference in age would influence the calves' ability to eat and utilize the various rations.

The basal ration used in this experiment is given in Table I. The objective was to feed a full amount of the basal ration for growth and maintenance to the calves in Group II. This full ration was to be supplemented with access to nurse cows for the calves in Group I. The basal ration, calculated to provide only maintenance, was fed to the calves in groups III and IV. The amount of concentrate and hay necessary for each calf in each group was calculated from Morrison's feeding standards--based on the initial and again on the mid-experiment weights of the calves. (See appendix, Table VII.)

Table I. Approximate analysis of basal ration.

Item	Protein %	Fat %	Fiber %	N F E %
Concentrate*	12	2	7	59
Alfalfa hay	14.7	2	29	36.4

* The concentrate was composed of oats, barley, corn, wheat middlings, linseed oil meal, alfalfa meal, molasses, vitamin D, limestone, dicalcium phosphate, salt, and trace minerals.

The calves had been splenectomized a minimum of three weeks before they were divided into groups and placed on their respective rations.

The measured feeding was begun 18 days before the calves were inoculated with blood from an acute case of anaplasmosis.

The inoculation procedure was designed to provide each calf with as nearly as possible the same number of infectious organisms. The procedure adopted was as follows: A calf acutely infected with anaplasmosis was exsanguinated at the maximum number of infected erythrocytes; and 100 ml. of this blood was injected intravenously into each experimental calf in Groups I, II and III. The Group IV calves were not infected with anaplasmosis but were held as uninfected controls for Group III, the infected group fed the low TDN ration.

Blood samples were taken at approximately weekly intervals for a month before inoculation and at daily intervals after the incubation period and through convalescence until relapse was indicated. Then samples were taken at thrice weekly intervals until the experiment was terminated. They were analyzed for hematocrit, hemoglobin, erythrocyte count, percent of infected erythrocytes, number of reticulocytes, serum protein, and titre of the complement-fixing antibody.

Hematocrit measurements were made by the method of Wintrobe (1946). Hemoglobin determinations were made on a Haden-Hausser hemoglobinometer. The blood used for determination of percent infected erythrocytes and the number of reticulocytes was first vitally stained with a 1 percent aqueous solution of brilliant cresyl blue, then smeared on slides and stained with Wright's stain buffered at pH 6.8. Serum protein was determined by the specific gravity method. (Phillips, et al., 1945). Titration of the complement-fixing antibodies was by the methods used in this laboratory (Price, Brock and Miller, 1954).

In addition to the blood samples, tissue sections were obtained

at autopsy on animals that died. These were stained with hematoxylin and eosin. The hemosiderin was brought out in additional sections with ferrocyanide by the method described by Gomori (1952).

RESULTS

Mortality:

The best measure of resistance-susceptibility in a host to infection, as judged by reproducible and unequivocal results, is the mortality among that host when exposed to a specific infection (Clark et al., 1949). On the basis of past experience, it would have been necessary to use 75 calves in this experiment to obtain statistically valid observations on mortality in the calves infected with anaplasmosis as influenced by nutrition. Therefore, the results shown in Table II can only indicate a trend.

Table II. Mortality of calves infected with anaplasmosis.

Block	Group I	Group II	Group III	Group IV
1	462 (died)	499	464 (died)	460
2	458	463 (died)	498	459
3	452	500 (died)	457 (died)	461
4	453	451 (died)	497 (died)	456
5	454 (died)	449	450 (died)	455 (died)

Infected Erythrocytes:

The degree of infection, as measured by both the maximum percent of infected erythrocytes and the maximum number of infected erythrocytes per cubic millimeter of blood is given in Table III. Numbers of infected erythrocytes were obtained by multiplying the total erythrocyte count by the percent of infected erythrocytes. The uninfected calves, comprising Group IV, are not included in Table III since they had no infected erythrocytes.

There was no significant difference among the experimental groups as measured by the degree of infection developed in the inoculated calves by either criteria.

Table III. Highest percent and number of infected erythrocytes

Group I			Group II			Group III		
An.No.	%Inf. RBC	No.Inf. RBCx10^6	An.No.	%Inf. RBC	No.Inf. RBCx10^6	An.No.	%Inf. RBC	No.Inf. RBCx10^6
462	54.7	2.77	499	34.8	2.40	464	32.3	1.72
458	23.9	1.23	463	37.2	2.70	498	26.4	1.59
452	26.0	1.21	500	56.5	2.20	457	30.2	1.05
453	37.5	1.89	451	57.6	2.84	497	55.1	3.02
454	72.1	3.81	449	41.4	1.97	450	82.1	2.53

Anemia:

To determine the amount of anemia developed by the anaplasmosis infected calves during the course of the disease, measurements were made daily of the number of erythrocytes per cubic millimeter of blood, the amount of hemoglobin per 100 ml. of blood, and the hematocrit in percent. In Table IV are presented the results of the minimum reading for each of the calves.

There was no significant difference in the low erythrocyte counts among the groups of infected calves. Due to the fact that the Haden-Hausser hemoglobinometer will not read values below 3.5 gm. per 100 ml. of blood, and all the infected calves dropped below this value, the hemoglobin values cannot be analyzed. However, the difference in hematocrit values among the infected groups is significant with most of the difference being attributable to the difference between Group I, the highest TDN group, and Groups II and III.

From the measurements in Table IV, values for mean corpuscular

Table IV. Lowest RBC count, hemoglobin and hematocrit values.

Group	Calf	RBCx10^6	Hemoglobin gm./100 ml.	Hematocrit %
I	462	2.58	3.5	10
	458	1.82	3.5	9
	452	1.40	3.5	11
	453	1.58	3.5	9
	454	1.54	3.5	8
		$\bar{x}$1.78		$\bar{x}$9.4
II	499	2.02	3.5	9
	463	1.73	3.5	6
	500	1.46	3.5	7
	451	1.64	3.5	7
	449	1.17	3.5	10
		$\bar{x}$1.60		$\bar{x}$7.8
III	464	1.59	3.5	8
	498	1.50	3.5	7
	457	1.82	3.5	7
	497	1.43	3.5	5
	450	1.93	3.5	8
		$\bar{x}$1.65		$\bar{x}$7.0
IV	460	8.90	10.5	33
	459	9.90	12.0	38
	461	10.60	11.0	32
	456	7.15	8.5	23
	455	6.03	7.0	23
		$\bar{x}$8.32	$\bar{x}$9.8	$\bar{x}$29.8

volume, mean corpuscular hemoglobin and mean corpuscular hemoglobin concentration were calculated. (See appendix, Table VIII). All of these values remained within normal ranges until the animals started to recover from the disease. Those calves which died did not show any variation from normal values before they died.

Similar results are seen in the appearance of reticulocytes among the erythrocytes. No reticulocytes were found on the smears until after the calves that eventually lived had reached the crisis. The calves that died of the infection did not develop reticulocytes.

(See appendix, Table VIII.)

Antibody Response:

The maximum complement-fixing antibody titre developed by the infected calves in the experimental groups cannot logically be compared since many of the calves died before the development of maximum titre. Throughout this study the lowest hematocrit value has proved to be the most accurate guide to the crisis in this disease, at which time the animal either dies or starts recovery. Therefore, a proper point in the course of the disease at which to compare antibody titrations would seem to be at the lowest hematocrit value for each animal. Such antibody titre values are shown in Table V. There is no significant difference in the titrations among the three infected nutritional groups.

Table v. Complement-fixing antibody titres at the lowest hematocrit values

Group					
I		II		III	
Animal	Titre	Animal	Titre	Animal	Titre
462	1-640	499	1-1280	464	1-20480
458	1-1280	463	1-2560	498	1-2560
452	1-640	500	1-2560	457	1-640
453	1-5120	451	1-2560	497	1-2560
454	1-320	449	1-1280	450	1-80

Infected Erythrocyte/Hematocrit Ratio:

In examining the data for a criterion that would most accurately indicate the severity of anaplasmosis in a given calf it was found that, by combining the hematocrit value and the number of infected erythrocytes on a given day into a ratio--infected erythrocytes divided by hematocrit, a value was obtained with a higher correlation to the eventual outcome of the disease in a given calf than any other value so far discovered.

The higher the ratio, the less chance a calf has to survive the disease. In Table VI is presented the highest ratio obtained for each infected calf. These values are not significantly different among experimental groups.

Table VI. Ratio of infected erythrocytes to hematocrit values.

Group					
I		II		III	
Animal	Ratio	Animal	Ratio	Animal	Ratio
462*	14.1	499	10.4	404*	9.0
458	7.0	463*	12.3	498	8.8
452	5.7	500*	15.2	457*	8.3
453	11.2	451*	16.7	497*	13.7
454*	19.0	449	11.6	450*	19.7

*Eventually died.

Serum Protein:

Serum protein was measured, not to obtain direct information on the objective of this study, but to study the effect of the three experimental rations on protein in the serum. However, it was found that there was an average depression of about 1 gm. per 100 ml. of serum at the peak of the acute attack of anaplasmosis with rapid recovery to normal of 6 to 7 gm. per 100 ml. of serum. (See appendix, Table VIII.) No significant difference in low serum protein values was found among the experimental groups of calves.

No effect of the low TDN rations on serum protein was seen until the last week of the experiment. Except for the two or three most critical days during the acute attack of anaplasmosis, the serum protein values were normal in all four experimental groups. However, after 50 to 57 days on the low TDN ration, the calves in Groups III and IV showed

an average decline of 1.5 gm. of protein per 100 ml. of serum from the average preinfection value of 6.88 gm. per 100 ml. of serum.

Histopathology:

The tissue sections prepared at autopsy of the calves that died from the infection show degeneration of the parenchymal cells of the liver around the central veins, becoming less severe toward the periphery of the liver lobules. The kidneys have a distension of the vessels with blood just under the capsule with marked cloudy swelling of the cells of the proximal convoluted tubules in the cortex, and hyaline casts in the collecting tubules of the medullary substance. There is no correlation between the amount of tissue damage and the nutritional groups in which the calves were fed.

There were large quantities of hemosiderin in the Kupffer cells of the liver, primarily near the central vein. Macrophage cells located in the cortical substance of the kidney, immediately beneath the capsule and also between the tubules, contained hemosiderin. Likewise, many of the phagocytes in the blood contained hemosiderin. No relation between the amount of hemosiderin present in the tissue and the nutrition of the calf from which it came could be determined.

DISCUSSION AND CONCLUSIONS

Of the data obtained in this study the only significant difference among the high, medium and low TDN level groups was found in the hematocrit values. From Table VIII in the appendix it can be seen that a rise in the hematocrit value was the first indication that the animal would recover. The hematocrit value is also found as one factor in the ratio producing the best estimate of the severity of the disease. Thus, it appears that the hematocrit, a combined measure of erythrocyte number and size, is of primary importance in determining the severity and prognosis of anaplasmosis infection in these calves.

Other evidence concerning resistance-susceptibility to anaplasmosis obtained in this study is inconclusive. Certainly the trend in mortality indicates that there may be some effect of nutrition on survival in anaplasmosis infection since twice as many calves died in the low TDN group as in the full ration group. However, one of the low nutrition infected group may have died because of the low nutrition rather than the infection since the youngest of the uninfected group died.

The percent and number of infected erythrocytes are very nearly the same for all infected groups. Measures of anemia, other than hematocrit values, were not significantly different among the groups. Antibody formation, which has been greatly influenced by nutrition in studies of other diseases, was not significantly changed in this experiment.

It is possible that the calves were not held on the low nutrition ration for a long enough period to develop the severe stress that may be required to produce significant variation in the physiologic

functions measured in this experiment, particularly when as few as 15 animals were used. This is indicated by the fact that serum protein levels were normal until the last week of the experiment, at which time there was a marked drop in these values in the low nutrition calves. That the rations were having the effect for which they were designed is shown by the lack of weight gain from December to January in the low nutrition groups. (See appendix, Table VII.)

The massive inoculation dose of 100 ml. of blood highly infected with anaplasmosis which was used, in part, to minimize the error in sampling the infective organisms for inoculation into the experimental calves, is probably another factor in the failure of this experiment to produce more conclusive evidence on the effect of nutrition in anaplasmosis infection. The calves were overwhelmed by the infection, as evidenced by the extremely short incubation period of a week for practically all of the calves and the short development period of the disease of one week in most cases. This is an atypical course of the disease in which the incubation period is normally three weeks with a development period of 10 to 12 days. The overwhelming infection and resultant abbreviated course of the disease probably tended to reduce the variation in many of the functions measured in this experiment.

Variation due to individuals in this study was reduced far below that found in previous experiments with splenectomized calves infected with anaplasmosis and fed the same ration. The incubation periods of the calves in this study varied by only two days; the deaths of calves are closely grouped into a four day period; and the maximum reaction to the infection is grouped around the fourteenth day following inoculation. Since variation in these major landmarks of the course of anaplasmosis

is sharply restricted in these calves over the variation normally found in anaplasmosis infected calves fed the same, any significant difference found in the calves on this experiment should receive consideration weighed by the factors discussed above--lack of variation in the measurements and individuals.

When the results reported here are viewed in this manner, the significant difference found in the hematocrit values among the nutritional groups acquires additional importance. Therefore, while these data do not conclusively prove, they are consistent with, the hypothesis that susceptibility to anaplasmosis in splenectomized calves is increased by low levels of nutrition.

The results obtained by this work are sufficiently indicative of a relation between anaplasmosis infection and nutrition that additional experiments, modified by the experience of this preliminary study, should be conducted. Changes in the design of such experiments, as indicated by analysis of the present experiment, would be: 1) limit the age of the calves to 8 to 15 weeks of age and use a completely randomized design; 2) a much smaller inoculating dose of anaplasmosis infected blood; 3) a 60 day depletion period on the various rations before inoculation.

SUMMARY

A preliminary study was made on the relation of nutrition to susceptibility of splenectomized calves to infection with anaplasmosis.

Twenty splenectomized calves, aged 4 to 14 weeks, of mixed breeding, were divided into four groups of five calves each. Group I was then fed a basal ration designed to maintain normal growth in addition to access to nurse cows. Group II was given the same ration as Group I without the addition of milk. Group III was fed the basal ration calculated to maintain weight. Group IV received the same ration as Group III. Groups I, II and III were infected with anaplasmosis while Group IV was held as uninfected inanition controls. The calves were weighed at the beginning, the middle and the end of the experiment. Measurements of hematocrit, hemoglobin, erythrocyte count, percent of infected erythrocytes, number of reticulocytes, serum protein and antibody titre were made before and during infection. Tissue sections were made from the liver and kidney of the calves which died during the experiment.

Hematocrit values were the only measurements with a significant difference among infected nutritional groups. The mortality among the infected calves in the experiment indicates a trend from fewest deaths in the high nutrition group to most deaths in the low nutrition group. Hematocrit values were found to be more important than any other measure taken in this experiment for prognosis and, when combined into a ratio with the number of infected erythrocytes, produced the most accurate evaluation of the severity of the disease.

The results obtained from this experiment are consistent with, but

do not prove, the hypothesis that low levels of nutrition increase the susceptibility of splenectomized calves to infection with anaplasmosis.

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A P P E N D I X

APPENDIX

Table VII. Feed given each calf daily.

Group	Calf	Weight			Concentrate lbs		Hay lbs		Milk
		Dec. 23	Jan. 21	Feb. 19	Dec. 23	Jan. 21	Dec. 23	Jan. 21	
I	462	240	275		5.4	5.5	2.2	2.3	ad lib.
	458	125	149	152	2.7	3.6	1.2	1.6	ad lib.
	452	120	157	168	2.7	3.6	1.2	1.6	ad lib.
	453	125	158	165	2.7	3.6	1.2	1.6	ad lib.
	454	75	107		1.6	2.2	0.8	1.0	ad lib.
II	499	215	226	217	4.8	5.3	2.2	2.2	
	463	160	182		3.6	4.1	1.6	1.8	
	500	100	120		2.2	2.7	1.0	1.2	
	451	100	108		2.2	2.2	1.0	1.0	
	449	70	69	72	1.5	1.5	0.7	0.7	
III	464	260	244		1.9	1.8	0.8	0.7	
	498	160	149	142	1.3	1.2	0.5	0.5	
	457	130	137		1.0	1.0	0.4	0.4	
	497	80	78		0.6	0.6	0.3	0.3	
	450	65	67		0.5	0.5	0.2	0.2	
IV	460	180	177	195	1.4	1.4	0.6	0.6	
	459	170	151	165	1.3	1.2	0.6	0.5	
	461	120	122	125	0.9	0.9	0.4	0.3	
	456	110	116	122	0.8	0.9	0.4	0.4	
	455	75			0.6		0.3		

Table VIII. Original values from whole blood samples

Date	Ht. %	Hb. gm.	RBCx10^6	MCV	MCH	MCHC	% Inf. RBC	Reticu-Locytes,%	Protein gm.	Titre
				Animal 462, Group I, Block 1						
12/6/54	34	11.0	7.05	47.9	15.5	32.4	-	0.0	6.45	-
12/23	29	10.0	8.30	34.9	12.0	34.5	-	0.0	6.28	-
12/30	32	9.5	8.10	39.5	11.7	29.7	-	0.0	6.50	-
1/6/55	29	10.5	8.04	36.3	13.1	36.2	-	0.0	6.38	-
1/10	31	10.0	7.64	40.8	13.2	32.2	-	0.0	6.28	-
1/13	30	9.5	8.48	35.3	11.2	31.7	-	0.0		-
1/17	31	10.0	8.02	38.8	12.5	32.3	1.0	0.0	6.97	-
1/19	34	10.0	8.42	40.5	11.9	29.4	3.9	0.0	7.05	-
1/20	32	10.0	8.36	38.1	11.9	31.3	10.5	0.0		<1-10
1/21	29	9.0	7.56	38.2	11.8	31.0	12.2	0.0	7.00	1-40
1/22	28	9.0	7.72	36.4	11.7	32.1	20.4	0.0	7.05	1-320
1/23	20	6.0	5.32	37.7	11.3	30.0	52.0	0.0	7.15	1-320
1/24	10	<3.5	2.58	38.5			54.7	0.0	6.55	1-640
Died, p.m. 1/24/55										
				Animal 458, Group I, Block 2						
12/6/54	36	10.0	8.75	40.9	11.4	27.8	-	0.0	7.15	-
12/23		9.5	9.54		10.0		-	0.0	7.05	-
12/30	35	9.5	9.34	37.6	10.2	27.1	-	0.0	7.10	-
1/6/55	34	11.0	11.14	30.6	9.9	32.4	-	0.0	6.91	-
1/10	33	10.0	10.30	32.0	9.7	30.3	-	0.0	7.14	-
1/13	38	11.0	11.06	34.2	9.9	28.9	-	0.0	7.50	-
1/17	33	10.0	9.40	35.1	10.6	30.3	1.0	0.0	6.98	-
1/19	33	9.5	9.80	33.7	9.7	28.8	3.4	0.0	7.14	-
1/20	31	9.0	9.76	31.6	9.2	29.0	4.1	0.0	7.14	-
1/21	27	8.5	9.12	29.8	9.3	31.5	7.2	0.0	6.74	<1-10
1/22	25	8.0	7.84	32.1	10.3	32.0	12.3	0.0	6.55	1-80

Table VIII (continued)

Date	Ht. %	Hb. gm.	RBCx10^6	MCV	MCH	MCHC	% Inf. RBC	Reticulocytes, %	Protein	Titre
1/23	17	6.0	5.14	34.3	11.8	34.3	23.9	0.0	6.40	1-160
1/24	13	3.5	3.66	35.1	9.7	26.9	16.5	0.0	6.05	1-1280
1/25	9	<3.5	2.50	36.0			15.6	1.0	6.10	1-1280
1/26	11	<3.5	1.82	61.1			7.7	1.3	6.28	1-2560
1/27	13	3.5	1.85	68.4	18.4	26.9	7.6	4.8	6.68	1-5120
1/28	20	4.0	2.35	83.3	16.7	20.0	4.3	2.3	6.74	1-5120
1/29	17	4.5	2.30	76.1	19.6	25.7	2.4	1.4	6.68	1-1280
1/30	19	5.0	2.60	75.0	19.2	25.6	1.3	1.2	6.74	1-1280
1/31	19	5.0	2.90	65.5	17.2	26.3	1.0	0.0	6.68	1-640
2/1	22	6.0	3.40	64.7	17.6	27.3	-	0.0	6.51	1-1280
2/2	23	6.5	3.53	65.7	18.6	28.3	-	0.0	6.68	1-5120
2/3	23	6.5	3.90	59.0	16.7	28.3	-	0.0	6.20	1-5120
2/4	24	6.5	4.32	55.8	15.1	27.1	-	0.0	6.20	1-5120
2/7	25	8.0	4.44	58.0	18.2	31.4	-	0.0	6.41	1-1280
2/9	23	8.0	4.83	47.9	16.7	34.8	3.0	0.0	6.10	1-1280
2/11	20	7.0	5.22	38.5	13.5	35.0	8.8	0.0	6.05	1-320
2/14	8	4.0	3.37	25.0	11.8	47.1	9.4	0.0	5.98	1-320
2/16	13	3.5	2.57	51.9	14.4	28.1	11.2	4.1	6.68	1-1280
2/18	16	3.5	2.34	68.4	14.9	21.9	7.6	5.2	6.48	1-2560
				Animal 452, Group I, Block						
12/6/54	31	9.0	7.50	41.3	12.0	29.0	-	0.0	6.28	-
12/23	29	8.5	6.85	42.0	12.3	29.3	-	0.0	5.50	-
12/30	30	7.5	8.24	36.6	9.1	25.0	-	0.0	6.52	-
1/6/55	31	9.5	8.80	35.2	10.8	30.6	-	0.0	6.68	-
1/10	31	9.5	9.00	34.4	10.6	30.6	-	0.0	6.49	-
1/13	35	9.5	9.28	37.6	10.2	27.1	-	0.0	6.68	-
1/17	29	8.0	8.14	35.8	9.9	27.6	1.0	0.0	6.78	-

Table VIII (continued)

Date	Ht. %	Hb. gm.	RBCx10^6	MCV	MCH	MCHC	% Inf. RBC	Reticulocytes, %	Protein	Titre
1/19	31	9.5	8.62	36.0	11.0	30.6	3.5	0.0	7.04	-
1/20	27	9.0	7.02	38.6	12.9	33.3	9.5	0.0	6.36	-
1/21	26	8.0	6.74	38.8	11.9	30.8	12.4	0.0	6.52	<1-10
1/22	25	7.5	6.25	39.7	11.9	30.0	19.3	0.0	6.68	<1-10
1/23	19	5.0	4.50	42.2	11.1	26.3	24.0	0.1	5.42	<1-10
1/24	17	4.0	3.71	45.9	10.8	23.5	25.6	1.0	6.68	1-160
1/25	11	<3.5	1.92	60.5			26.0	3.4	5.90	1-640
1/26	12	3.5	1.40	85.7	25.0	29.2	10.2	6.8	5.90	1-1280
1/27	15	4.0	1.88	78.9	20.0	26.7	8.8	7.1	5.75	1-10240
1/28	18	4.5	1.85	94.7	23.7	25.0	6.2	9.4	6.55	1-2560
1/29	21	5.5	2.23	95.4	25.0	26.2	3.6	12.1	6.78	1-2560
1/30	22	5.5	2.50	93.7	22.9	24.4	1.9	4.0	6.55	1-1280
1/31	23	5.5	2.52	92.0	22.0	23.9	<1.0	2.2	6.28	1-1280
2/1	27	7.0	2.90	94.8	24.1	25.5	<1.0	1.3	6.68	1-320
2/2	28	7.0	3.26	84.8	21.2	25.0	-	0.1	6.49	1-320
2/3	29	7.5	4.00	72.5	18.8	25.9	-	0.0	6.48	1-320
2/4	30	8.0	4.05	75.0	20.0	26.7	-	0.0	6.38	1-320
2/7	33	8.5	4.47	73.3	21.1	25.8	<1.0	0.0	6.28	1-320
2/9	31	9.0	5.50	56.4	16.4	29.0	5.0	0.0	6.48	1-320
2/11	27	8.5	5.27	51.9	16.0	30.9	13.2	0.0	6.20	1-320
2/14	10	4.0	2.70	37.0	14.8	40.0	14.0	2.1	6.51	1-640
2/16	17	4.5	2.03	87.5	23.8	27.4	9.5	8.6	6.68	1-640
2/18	18	5.0	3.00	90.0	25.0	27.8	8.4	0.1	6.60	1-1280
				Animal 453, Group I, Block 4						
12/ 6/54	34	11.0	9.70	35.1	11.3	32.4	-	0.0	6.38	-
12/23	31	9.0	8.44	26.9	10.7	29.0	-	0.0	6.10	-
12/30	34	9.5	9.22	36.9	10.3	28.0	-	0.0	6.10	-

Table VIII (continued)

Date	Ht. %	Hb. Gm.	RBCx10^6	MCV	MCH	MCHC	% Inf. RBC	Reticulocytes, %	Protein	Titre
1/6/55	37	12.0	9.95	37.0	12.0	32.4	-	0.0	6.68	-
1/10	36	11.5	10.30	35.0	11.2	32.0	-	0.0	6.68	-
1/13	37	11.0	10.40	35.6	10.6	29.7	-	0.0	6.85	-
1/17	36	12.0	10.64	34.0	11.3	33.3	-	0.0	7.05	-
1/19	37	11.5	9.74	38.1	11.9	31.1	<1.0	0.0	7.13	-
1/20	37	11.0	10.00	37.0	11.0	29.7	1.0	0.0	6.52	-
1/21	37	11.0	9.68	38.7	11.3	29.3	3.4	0.0	6.60	-
1/22	37	11.0	9.08	40.7	12.1	29.7	2.4	0.0	5.90	<1-10
1/23	35	10.0	9.80	35.7	10.2	28.6	6.6	0.0	5.90	<1-10
1/24	35	9.5	9.75	35.7	9.7	27.1	8.1	0.0	5.90	1-80
1/25	29	9.0	7.98	36.3	11.3	31.0	15.2	0.0	5.43	1-320
1/26	22	8.5	7.49	29.3	11.3	38.6	25.2	0.0	5.66	1-2560
1/27	22	7.5	6.10	36.1	12.3	34.1	29.2	0.0	5.39	1-2560
1/28	18	5.5	5.40	34.3	10.2	29.7	32.0	0.0	5.82	1-2560
1/29	11	3.5	3.44	33.8	10.3	30.4	37.5	0.0	5.90	1-5120
1/30	9	<3.5	1.70	52.9			24.6	0.1	6.10	1-1280
1/31	11	<3.5	1.80	61.1			17.6	5.4	6.55	1-5120
2/1	13	<3.5	1.58	81.3			9.0	9.1	6.51	1-10240
2/2	17	4.0	2.10	83.3	19.0	22.9	3.4	12.7	6.38	1-10240
2/3	19	4.5	2.26	82.6	19.6	23.7	1.0	10.3	6.21	1-10240
2/4	23	5.5	2.65	85.2	20.4	23.9	<1.0	7.0	6.52	1-5120
2/5	24	6.0	3.03	81.7	20.0	24.5	<1.0	5.2	6.52	1-5120
2/7	28	7.5	3.94	73.1	19.2	26.3	-	0.0	6.74	1-1280
2/9	30	8.5	4.34	69.8	19.8	28.3	-	0.0	6.38	1-1280
2/11	28	8.5	4.61	62.0	18.5	29.8	3.2	0.0	6.29	1-1280
2/14	30	8.5	5.00	60.0	17.0	28.3	4.1	0.0	6.29	1-1280
2/16	25	8.0	4.56	55.4	17.4	31.4	13.0	0.0	6.10	1-1280
2/18	19	7.0	3.72	51.1	18.8	35.9	21.2	0.0	5.90	1-1280

Table VIII (continued)

Date	Ht. %	Hb. Gm.	RBCx10^6	MCV	MCH	MCHC	% Inf. RBC	Reticulocytes, %	Protein	Titre
				Animal 454, Group I, Block 5						
12/6/54	36	10.5	7.80	46.2	13.5	29.2	-	0.0	7.58	-
12/23	34	9.5	8.50	40.0	11.2	27.9	-	0.0	6.68	-
12/30	36	10.0	8.10	44.4	12.3	27.8	-	0.0	6.40	-
1/6/55	31	10.0	8.70	35.6	11.5	32.3	-	0.0	5.35	-
1/10		11.0	8.56		12.8		-	0.0	5.98	-
1/13	38	10.0	8.22	46.3	12.2	26.3	-	0.0	6.38	-
1/17	34	11.0	9.18	37.0	12.0	32.4	-	0.0	6.81	-
1/19	35	10.0	8.26	42.2	12.0	28.6	1.2	0.0	6.68	-
1/20	34	10.0	8.50	40.0	11.8	29.4	3.2	0.0	6.40	-
1/21	31	9.0	7.50	41.3	12.0	29.0	13.3	0.0	5.82	-
1/22	32	9.0	7.30	43.8	12.3	28.1	26.0	0.0	6.40	-
1/23	25	7.5	6.32	40.5	12.0	29.4	39.0	0.0	6.68	<1-10
1/24	20	5.5	5.50	36.4	10.5	29.0	69.2	0.0	6.21	<1-10
1/25	10	<3.5	2.64	40.4			72.1	0.0	6.78	1-80
1/26	8	<3.5	1.54	53.3			45.5	0.0	6.82	1-320
Died p.m. 1/26/55										
				Animal 499, Group II, Block 1						
12/6/54	47	13.0	12.50	37.6	10.4	27.7	-	0.0	7.04	-
12/23	41	12.0	12.30	33.3	9.8	29.3	-	0.0	7.04	-
12/30	42	12.0	11.88	35.3	10.1	28.6	-	0.0	7.45	-
1/6/55	36	13.0	10.80	33.3	12.0	36.1	-	0.0	7.15	-
1/10	41	12.0	11.08	36.9	11.2	29.3	-	0.0	7.45	-
1/13	37	12.0	10.44	35.6	11.5	32.4	-	0.0	7.80	-
1/17	37	11.0	10.00	37.0	11.0	29.7	<1.0	0.0	7.14	-
1/19	37	11.0	10.88	33.9	10.1	29.7	3.4	0.0	6.85	-

Table VIII (continued)

Date	Ht. %	Hb. gm.	RBCx10^6	MCV	MCH	MCHC	% Inf. RBC	Reticulocytes, %	Protein	Titre
1/20	34	11.5	9.80	34.7	11.7	33.8	5.1	0.0	7.04	-
1/21	31	10.0	9.10	34.1	11.0	32.3	8.9	0.0	6.48	<1-10
1/22	31	10.0	9.34	33.3	10.8	41.3	12.8	0.0	5.45	<1-10
1/23	23	8.5	6.62	34.8	12.9	37.0	23.2	0.0	6.28	1-40
1/24	23	7.0	6.90	33.3	10.1	30.4	34.8	0.0	6.75	1-160
1/25	19	6.0	6.25	30.2	9.5	31.6	26.2	0.0	5.98	1-160
1/26	21	4.0	4.15	50.0	9.5	19.0	20.8	0.0	5.39	1-160
1/27	11	3.5	3.49	31.4	10.0	31.8	18.6	0.0	5.59	1-160
1/28	9	3.5	2.51	36.0	12.0	33.3	15.4	0.0	5.98	1-1280
1/29	10	3.5	2.17	45.5	11.4	25.0	12.4	<0.1	5.90	1-320
1/30	13	3.5	2.13	61.9	16.7	26.9	5.6	3.1	6.85	1-1280
1/31	16	4.0	2.60	61.5	15.4	25.0	2.6	3.8	6.97	1 640
2/1	16	3.5	2.32	71.7	15.2	21.2	<1.0	4.6	6.81	1-320
2/2	16	4.0	2.40	66.6	16.7	25.0	<1.0	<0.1	6.78	1-320
2/3	17	4.5	2.02	85.0	22.5	26.5	-	0.0	6.38	1-1280
2/4	19	4.5	2.50	76.0	18.0	23.7	-	0.0	6.21	1-320
2/5	19	5.0	3.00	63.3	16.7	26.3	-	0.0	6.21	1-1280
1/7	20	5.0	2.60	76.9	19.2	27.5	-	0.0	6.18	1-160
2/9	20	5.5	3.15	62.5	17.2	27.5	-	0.0	6.21	1-640
2/11	20	6.0	3.33	60.6	18.2	30.0	1.2	0.0	6.18	1-160
2/14	23	6.0	3.54	65.7	17.1	26.1	5.4	0.0	6.10	1-160
2/16	20	6.0	4.10	50.0	14.6	29.3	18.4	0.0	6.10	1-160
2/18	16	4.5	2.70	59.3	16.7	28.1	27.0	0.0	6.18	1-320
				Animal 463, Group II, Block 2						
12/6/54	43	11.5	11.50	37.4	10.0	26.7	-	0.0	6.64	-
12/23	43	14.5	13.00	33.1	11.2	33.7	-	0.0	6.81	-
12/30	42	12.0	11.80	35.6	10.2	28.6	-	0.0	6.85	-

Table VIII (continued)

Date	Ht. %	Hb. gm.	RBCx10^6	MCV	MCH	MCHC	% Inf. RBC	Reticulocytes, %	Protein	Titre
1/6/55	39	12.0	11.68	33.3	10.3	30.8	-	0.0	7.04	-
1/10	42	11.0	10.90	38.5	10.1	26.2	-	0.0	7.13	-
1/13	37	11.0	10.30	35.9	10.7	29.7	-	0.0	7.15	-
1/17	40	12.0	10.50	38.1	11.4	30.0	1.0	0.0	7.20	-
1/19	38	12.0	10.50	36.2	11.4	31.6	3.5	0.0	7.04	-
1/20	38	11.0	10.43	36.5	10.6	28.9	2.7	0.0	7.52	<1-10
1/21	35	10.0	9.74	36.1	10.3	28.6	6.4	0.0	7.04	1-160
1/22	35	11.0	8.95	38.9	12.2	31.4	15.1	0.0	7.04	1-320
1/23	27	8.5	7.37	36.5	11.5	31.5	26.7	0.0	6.89	1-320
1/24	22	7.0	6.90	31.9	10.1	31.8	30.1	0.0	6.68	1-640
1/25	15	5.0	4.36	34.1	11.4	33.3	36.6	0.0	6.41	1-640
1/26	9	<3.5	2.69	33.3			37.2	0.0	5.98	1-1280
1/27	6	<3.5	1.73	35.3			25.2	0.0	6.68	1-2560
Died, p.m. 1/27/55										
				Animal 500, Group II, Block 3						
12/6/54	48	13.0	12.30	39.0	10.6	27.1	-	0.0	7.13	-
12/23	48	14.0	12.60	38.1	11.1	29.2	-	0.0	6.92	-
12/30	47	14.0	11.10	42.3	12.6	29.8	-	0.0	6.92	-
1/6/55	43	13.0	11.12	38.7	11.7	30.2	-	0.0	7.04	-
1/10	42	12.5	10.20	41.2	12.3	29.8	-	0.0	6.74	-
1/13	39	12.0	9.50	41.1	12.6	30.8	<1.0	0.0	7.15	-
1/17	38	11.5	9.40	40.4	12.2	30.3	1.0	0.0	6.25	-
1/19	36	10.0	8.54	42.4	11.8	27.8	7.4	0.0	5.98	-
1/20	35	10.5	8.00	43.8	13.1	30.0	9.5	0.0	6.68	-
1/21	29	9.5	7.72	37.7	12.3	32.8	22.6	0.0	6.10	<1-10
1/22	29	9.0	7.86	36.7	11.4	31.0	26.0	0.0	5.74	1-80
1/23	18	6.0	4.15	42.9	14.3	33.3	53.1	0.0	5.78	1-160

Table VIII (continued)

Date	Ht. %	Hb. gm.	RBCx10^6	MCV	MCH	MCHC	% Inf. RBC	Reticulocytes,%	Protein	Titre
1/24	12	3.5	3.23	37.5	10.9	29.2	56.5	0.0	5.35	1-160
1/25	7	<3.5	1.46	46.7			44.4	0.0	5.82	1-2560
Died p.m. 1/25/55										
				Animal 451, Group II, Block 4						
12/6/54	41	12.0	9.90	41.4	12.1	29.3	-	0.0	6.68	-
12/23	48	14.0	11.40	42.1	12.3	29.2	-	0.0		-
12/30	44	14.0	11.00	40.0	12.7	31.8	-	0.0	6.49	-
1/6/55	31	13.0	10.20	30.4	12.7	41.9	-	0.0	6.74	-
1/10	39	12.0	10.60	36.8	11.3	30.8	-	0.0	6.74	-
1/13	39	11.5	10.58	36.8	10.8	29.5	-	0.0	6.78	-
1/17	37	12.0	9.00	41.1	13.3	32.4	1.0	0.0	6.74	-
1/19	36	10.5	9.64	37.5	10.9	29.2	8.6	0.0	6.49	<1-10
1/20	33	10.5	9.68	34.0	10.8	33.9	11.2	0.0	6.78	<1-10
1/21	33	10.0	8.68	37.9	11.5	30.3	13.4	0.0	6.45	<1-10
1/22	30	9.0	8.38	35.7	10.7	30.0	15.2	0.0	6.38	<1-10
1/23	25	7.5	5.91	42.4	12.7	30.0	33.3	0.0	6.49	1-160
1/24	17	5.5	4.93	34.7	11.2	32.4	57.6	0.0	5.70	1-320
1/25	11	3.5	2.98	36.7	11.7	31.8	60.2	0.0	5.98	1-640
1/26	7	<3.5	1.64	43.8			43.1	0.0	5.98	1-2560
				Animal 449, Group II, Block 5						
12/6/54	30	8.5	7.32	41.1	11.6	28.3	-	0.0	7.29	-
12/23	37	10.0	9.32	39.8	10.8	27.0	-	0.0	6.81	-
12/30	37	10.0	10.14	36.6	9.9	27.0	-	0.0	6.38	-
1/6/55	31	10.0	8.50	36.5	11.8	32.3	-	0.0	5.90	-

Table VIII (continued)

Date	Ht. %	Hb. gm.	RBCx10^6	MCV	MCH	MCHC	% Inf. RBC	Reticu-locytes,%	Protein	Titre
1/10	27	9.5	7.80	34.6	12.2	35.2	-	0.0	5.70	-
1/13	31	10.0	7.77	39.7	12.8	32.3	-	0.0	7.04	-
1/17	40	12.0				30.0	-	0.0	6.28	-
1/19	29	8.5	8.40	33.3	10.1	29.3	1.3	0.0	6.49	-
1/20	28	8.5	8.80	31.8	9.7	30.4	3.9	0.0	7.04	-
1/21	29	9.0	7.66	37.7	11.7	31.0	4.2	0.0	6.74	-
1/22	28	8.5	7.72	37.0	11.0	29.8	4.7	0.0	6.68	-
1/23	28	8.0	6.90	40.6	11.6	28.6	5.1	0.0	6.28	-
1/24	25	6.5	5.80	43.1	11.2	26.0	12.2	0.0	6.28	-
1/25	22	7.5	6.00	37.5	12.5	33.3	16.6	0.0	5.98	<1-10
1/26	18	5.5	4.75	39.1	11.5	30.5	28.7	0.0	5.50	1-80
1/27	17	5.0	4.77	35.4	10.4	29.4	41.4	0.0	5.50	1-160
1/28	13	4.0	3.65	36.5	10.8	29.6	36.7	0.0	6.10	1-640
1/29	10	<3.5	2.22	47.7			30.2	<0.1	5.66	1-1280
1/30	10	<3.5	1.35	71.4			28.6	3.7	6.49	1-1280
1/31	11	<3.5	1.17	91.7			11.3	5.2	6.60	1-2560
2/1	14	<3.5	1.30	111.5			8.2	8.6	6.68	1-1280
2/2	16	3.5	1.65	94.1	20.6	21.9	5.4	4.0	6.28	1-7280
2/3	16	4.0	1.75	91.7	22.2	24.2	2.0	3.4	5.82	1-5120
2/4	16	4.0	1.76	88.9	22.2	25.0	1.0	2.5	6.10	1-1280
2/5	17	4.5	2.02	85.0	21.3	25.3	-	0.0	6.10	1-1280
2/7	18	4.5	2.19	81.8	19.3	23.9	-	0.0	6.10	1-5120
2/9	19	4.5	2.50	76.0	17.0	22.6	2.1	0.0	5.70	1-2560
2/11	19	4.5	3.08	61.3	15.0	23.7	2.8	0.0	4.91	1-1280
2/14	18	5.5	2.90	62.1	18.1	29.4	3.7	0.0	5.12	1-1280
2/16	18	5.5	3.29	56.1	16.7	29.7	6.6	0.0	5.00	1-1280
2/18	17	5.0	3.10	54.8	16.1	29.4	7.9	0.0	5.12	1-1280

Table VIII (continued)

Date	Ht. %	Hb. gm.	RBCx10^6	MCV	MCH	MCHC	% Inf. RBC	Reticulocytes, %	Protein	Titre
				Animal 464, Group III, Block 1						
12/6/54	30	9.5	7.70	38.9	12.3	31.7	-	0.0	7.04	-
12/23	29	10.0	8.00	36.3	12.5	34.5	-	0.0	7.04	-
12/30	31	9.5	9.20	33.7	10.3	30.6	-	0.0	6.68	-
1/6/55	33	10.0	8.26	39.8	12.0	30.3	-	0.0	7.24	-
1/10	32	10.0	9.00	35.6	11.1	31.3	-	0.0	7.38	-
1/13	34	10.0	7.94	43.0	12.7	29.4	-	0.0	7.49	-
1/17	34	10.0	9.16	37.0	10.9	29.4	1.0	0.0	7.61	-
1/19	32	9.5	7.38	43.2	12.8	29.7	3.0	0.0	7.65	-
1/20	33	9.5	8.00	41.3	11.9	28.8	5.5	0.0	7.61	<1-10
1/21	30	9.0	7.28	41.1	12.3	30.0	7.6	0.0	6.81	1-80
1/22	27	8.0	7.00	38.6	11.4	29.6	10.7	0.0	7.20	1-640
1/23	24	7.0	6.00	40.0	11.7	29.2	18.8	0.0	7.15	1-640
1/24	19	6.5	5.33	35.8	12.3	34.2	32.3	0.0	6.78	1-2560
1/25	13	4.0	3.00	45.0	13.3	29.6	25.2	0.0	6.68	1-5120
1/26	8	<3.5	2.05	38.1			28.2	0.0	6.68	1-10240
1/27	6	<3.5	1.59	40.7			26.6	0.0	7.20	1-20480
Died p.m. 1/27/55										
				Animal 498, Group III, Block 2						
12/6/54	45	13.0	12.20	36.9	10.7	28.9	-	0.0	6.78	-
12/23	38	11.0	10.80	35.2	10.2	28.9	-	0.0	6.60	-
12/30	38	11.0	10.50	36.2	10.5	28.9	-	0.0	6.68	-
1/6/55	38	13.0	10.10	37.6	12.9	34.2	-	0.0	7.04	-
1/10	37	10.0	11.40	32.5	8.8	27.0	-	0.0	6.92	-
1/13	38	11.5	10.46	36.2	11.0	30.3	-	0.0	6.97	-
1/17	36	11.0	10.08	35.6	10.9	30.6	-	0.0	7.09	-

Table VIII (continued)

Date	Ht. %	Hb. gm.	RBCx10^6	MCV	MCH	MCHC	% Inf. RBC	Reticulocytes, %	Protein	Titre
1/19	37	11.0	9.70	38.1	11.3	29.7	1.8	0.0	7.20	-
1/20	36	12.0	8.90	40.4	13.5	33.3	1.9	0.0	7.15	-
1/21	36	11.0	9.80	36.7	11.2	30.6	4.3	0.0	7.04	1-10
1/22	35	11.0	10.20	34.3	10.8	31.4	6.8	0.0	6.68	1-10
1/23	33	10.0	8.43	39.3	11.9	30.3	8.5	0.0	6.85	1-80
1/24	30	9.5	8.45	35.3	11.2	31.7	15.7	0.0	6.49	1-160
1/25	27	9.0	8.20	32.9	11.0	33.3	19.4	0.0	6.18	1-160
1/26	13	6.5	5.72	22.8	11.4	44.0	26.4	0.0	6.01	1-640
1/27	17	5.0	4.79	35.4	10.4	29.4	23.8	0.0	5.50	1-640
1/28	12	3.5	3.63	34.7	9.7	28.0	19.6	0.0	5.58	1-2560
1/29	7	3.5	1.98	35.0			16.0	0.1	5.31	1-1280
1/30	7	3.5	1.56	43.8			11.1	0.1	6.83	1-1280
1/31	8	3.5	1.50	53.3			7.2	6.9	6.85	1-640
2/1	13	3.5	2.10	61.9			4.2	14.0	6.89	1-2560
2/2	14	3.5	1.95	70.0	18.4	25.0	2.2	10.2	7.13	1-5120
2/3	15	4.0	2.00	75.0	20.0	26.6	1.0	6.1	6.68	1-10240
2/4	17	4.0	2.17	79.5	18.2	22.8	1.0	0.1	6.49	1-1280
2/5	17	4.5	2.54	70.0	16.0	25.7	-	0.0	5.90	1-1280
2/7	20	5.5	2.80	73.2	19.6	26.8	-	0.0	6.28	1-20480
2/9	22	6.0	3.25	66.7	18.2	27.3	-	0.0	6.10	1-5120
2/11	19	6.0	3.19	59.4	18.8	31.6	4.4	0.0	5.90	1-1280
2/14	16	4.5	3.30	50.0	12.9	26.1	9.6	0.0	6.28	1-1280
2/16	13	4.0	2.55	51.9	15.4	29.6	14.4	0.0	5.12	1-2560
2/18	9	3.5	2.10	45.2	16.7	36.8	15.0	0.0	4.91	1-2560
				Animal 457, Group III, Block 3						
12/6/54	39	11.0	9.90	38.4	11.1	28.2	-	0.0	7.13	-
12/23	41	11.5	9.90	41.4	11.6	28.0	-	0.0	6.74	-
12/30	40	10.0	9.80	40.8	10.2	25.0	-	0.0	6.74	-

Table VIII (continued)

Date	Ht. %	Hb. Gm.	RBCx10^6	MCV	MCH	MCHC	% Inf. RBC	Reticulocytes, %	Protein	Titre
1/6/55	35	10.0	11.20	31.3	8.9	28.6	-	0.0	6.49	-
1/10	35	10.0	10.56	33.0	9.4	28.6	-	0.0	6.49	-
1/13	37	10.0	10.20	36.3	9.8	27.0	-	0.0	7.04	-
1/17	36	10.0	10.14	35.6	9.9	27.8	1.0	0.0	7.04	-
1/19	31	9.0	9.48	32.6	9.5	29.0	2.0	0.0	6.60	-
1/20	32	9.0	9.92	32.8	9.1	27.7	2.1	0.0	6.74	1-10
1/21	32	9.5	8.32	38.6	11.4	29.7	5.7	0.0	6.81	1-10
1/22	27	8.0	8.28	32.5	9.6	29.6	12.2	0.0		1-10
1/23	22	7.0	5.31	41.5	13.2	31.8	15.3	0.0	6.28	1-160
1/24	18	5.0	5.00	36.0	10.5	29.4	21.0	0.0	5.90	1-320
1/25	12	4.0	3.30	36.4	12.1	33.3	30.2	0.0	5.50	1-640
1/26	7	3.5	2.02	35.0			24.4	0.0	5.31	1-640
1/27	7	3.5	1.82	38.9			23.7	0.0	6.10	1-1280
Died p.m. 1/27/55										
				Animal 497, Group III, Block 4						
12/6/54	42	11.0	11.10	37.8	9.9	26.2	-	0.0	7.65	-
12/23	46	13.0	11.10	41.4	11.7	28.3	-	0.0	7.52	-
12/30	40	12.0	10.88	36.7	11.0	30.0	-	0.0	7.24	-
1/6/55	38	11.0	10.28	36.9	10.7	28.9	-	0.0	7.20	-
1/10	40	11.0	10.90	36.7	10.1	27.5	-	0.0	7.04	-
1/13	36	11.0	9.60	37.5	11.5	30.6	-	0.0	7.13	-
1/17	34	10.5	10.40	32.7	10.1	30.9	1.0	0.0	7.13	-
1/19	33	10.0	8.62	38.4	11.6	30.3	3.4	0.0	7.15	-
1/20	33	10.0	7.96	41.3	12.5	30.3	6.7	0.0	7.24	-
1/21	32	9.5	8.00	40.0	11.9	29.7	8.2	0.0	6.68	-
1/22	31	9.5	8.14	38.3	11.7	30.6	11.3	0.0	6.68	1-10
1/23	26	8.0	6.62	39.4	12.1	30.8	32.1	0.0	6.78	1-10
1/24	22	6.5	6.38	34.4	10.2	29.5	47.4	0.0	6.68	1-160

Table VIII (continued)

Date	Ht. %	Hb. gm.	RBCx10^6	MCV	MCH	MCHC	% Inf. RBC	Reticulocytes, %	Protein	Titre
1/25	15	5.0	3.28	46.9	15.2	32.3	55.1	0.0	6.28	1-320
1/26	9	3.5	2.40	37.5			50.6	0.0	6.49	1-640
1/27	5	3.5	1.43	38.5			46.6	0.0	6.78	1-2560
Died p.m.	1/27/55									
				Animal 450, Group III, Block 5						
12/6/54	42	11.0	8.45	49.4	12.9	26.2	-	0.0	7.38	-
12/23	47	12.0	11.00	42.7	10.9	25.5	-	0.0	7.15	-
12/30		11.0	9.46		11.6		-	0.0	6.68	-
1/6/55	41	12.0	10.16	40.2	11.8	29.3	-	0.0	7.13	-
1/10	40	10.0	9.04	44.4	11.1	25.0	-	0.0	6.38	-
1/13	42	12.0	10.50	40.0	11.4	28.6	-	0.0	7.41	-
1/17	40	12.0	10.30	38.8	11.7	30.0	1.0	0.0	7.20	-
1/19	36	10.0	9.76	36.7	10.2	27.8	7.0	0.0	6.97	-
1/20	40	11.0	10.10	39.6	10.9	27.5	12.5	0.0	8.62	-
1/21	37	11.0	8.90	41.6	12.4	29.7	25.0	0.0	7.13	-
1/22	31	9.0	8.80	35.2	10.2	29.0	30.1	0.0	6.68	1-10
1/23	19	5.0	5.12	38.2	9.8	25.6	40.5	0.0	6.68	
1/24	14	4.5	3.95	35.0	11.3	32.1	64.0	0.0	5.90	1-10
1/25	8	3.5	1.92	42.1			82.1	0.0	6.55	1-80
Died a.m.	1/25/55									

Table VIII (continued)

Animal	Date	Ht. %	Hb. gm.	RBCx10^6	MCV	MCH	MCHC	Reticulocytes,%	Protein gm.
Mean values for animals in Group IV									
460	12/23/54 to 2/11/55	37	11.3	9.29	40.9	12.2	29.8	0.0	7.03
	2/14/55 to 2/18/55	35	11.2	9.39	37.4	11.9	31.7	0.0	6.07
459	12/23/54 to 2/11/55	43	12.8	10.38	42.2	12.3	29.6	0.0	7.02
	2/14/55 to 2/18/55	40	12.8	9.78	40.6	13.1	32.3	0.0	6.07
461	12/23/54 to 2/11/55	44	12.9	10.95	39.8	11.8	29.5	0.0	7.04
	2/14/55 to 2/18/55	35	11.2	10.36	33.8	10.8	31.9	0.0	5.80
456	12/23/54 to 2/11/55	32	9.9	9.04	35.1	10.9	31.3	0.0	6.61
	2/14/55 to 2/18/55	25	6.8	7.13	35.7	9.6	27.2	0.0	5.35
455	Died 1/19/55								

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