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# Salmonellosis in White-tailed Deer Fawns*

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**Summary**

Experimental infection of white-tailed deer fawns with *Salmonella meleagridis* was accomplished. The fawns suffered clinical illness, similar to spontaneous cases observed in the field. This disease may be an important factor in fawn survival in wild herds based on the frequency with which Salmonellae could be isolated in wild fawns.

The clinical disease was acute, characterized by rapid depression and dehydration. Death ensued in three of eight experimental cases. The survivors suffered clinical illness.

## Introduction

Salmonellosis in ruminants is caused by a group of organisms which have little host specificity and which have an impressive list of antigenic combinations by which they are classified.[8] The disease is seen most commonly in the young, but if environmental conditions are unfavorable, infection of adult ruminants may occur.[7] Bacterial contamination occurs at birth or during the first few hours of the life of calves.[1] The fetus is capable of reacting effectively to some antigens while the response to others is inadequate. This is particularly true of Salmonella antigens, to which the fetal lamb does not respond.[9,10] Postnatally, the neonatal ruminant rapidly and non-selectively absorbs antibody from the colostral milk, and thus may have demonstrable antibody by two days of age.[8]

Data obtained in studies on the Welder Wildlife Refuge indicated that 72% of white-tailed deer fawns died before they reached one year of age.[4] Data from pilot studies in other East Texas areas have suggested that fawn losses are usually high during the first month of life. Debbie[6] and Cox[5] reported the occurrence of *Salmonella typhimurium* infections in captive white-tailed fawns and that diarrhea was a paramount problem during the first few weeks of postnatal life. No source of infection was mentioned, but it appeared that such infections

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played an important role in fawn survival in the field; and that the stress of changed diet and captivity might allow exacerbation of latent infections. Bruner and Moran (1949) reported the isolation of *S. derby* from a deer, but no mention was made of clinical disease. This investigation was initiated to explore the potential of Salmonella infections as a cause of deaths of fawns.

Two definitions are here given to clarify the statements given in the article. Any animal from which positive cultures may be obtained are considered "infected". Those animals which have obvious signs of illness are considered "clinically affected".

## Materials and Methods

*Field Studies:* Field studies were conducted in Brazos and Grimes Counties from 1966 to 1969 during the last week of May and the month of June. This period coincided with the peak fawning season. Fawns were located at night by use of a spotlight and captured with dip nets. The fawns were identified with a numbered tag in each ear, measured and weighed; and a rectal culture obtained. The fawns were then released at the site of capture. Cultures were taken by everting the anus and inserting a sterile cotton swab which was dampened with sterile tryptose broth taking care not to contact the external skin. The swab was rotated several times, withdrawn and inserted in a tube of sterile tryptose broth. The tube was capped and returned to the laboratory for processing. Attempts were made to recapture fawns already sampled when they were encountered at later dates. When captured, they were again cultured and released.

The cultures were processed in the laboratory through differential media as outlined in Figure 1. Positive cultures were sent to the State-Federal Animal Disease Laboratory, Phoenix, Arizona for serological identification.

The annual loss of deer in the Brazos-Grimes County areas was determined by counts of surviving fawns made by the Hahn method and on dead deer counts determined by walking over a given area.

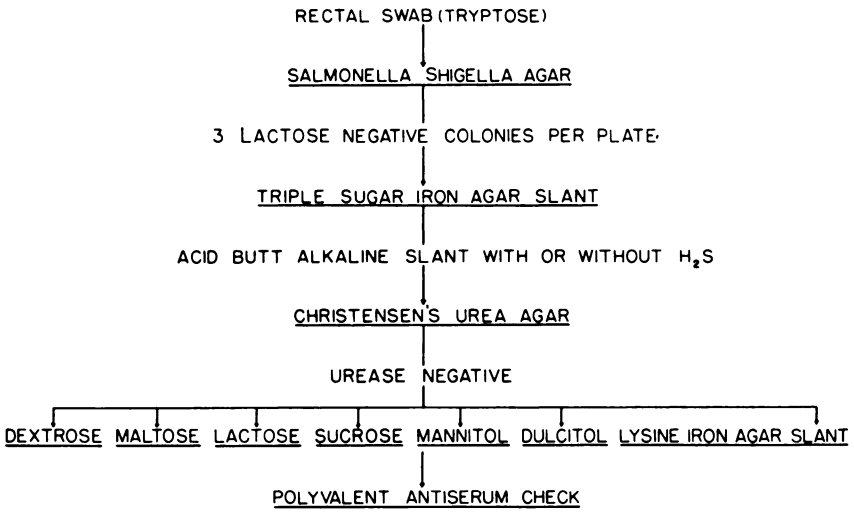


FIGURE 1. *Procedure for cultural identification of* Salmonella species.

*Experimental Studies:* All experimental fawns were born in captivity. This group had the same paternal parent, but the maternal ancestry varied. All experimental fawns and does were cultured prior to experimental use and were negative for *Salmonellae*.

Two groups of four fawns were inoculated orally with 10 ml. of a 12-hour culture as soon after birth as possible with *S. meleagridis* isolated from wild fawns. One group was left with the does. The second group was colostrum deprived. The colostrum deprived animals were maintained on raw, post colostral goats' milk. A third group of five fawns was maintained with the doe as control animals. Those fawns infected at birth were cultured by rectal sample before experimental infection to determine if prenatal infection may have taken place.

Blood samples were taken daily from all experimental fawns. Total leucocyte counts, packed cell volumes, and differential leucocyte counts were determined. Serum was separated and frozen for serological tests. Serological testing was performed using commercially available Salmonella somatic O antigen. This procedure employed the macroscopic tube test and was adapted to use a microdilution system to allow for a greater number of samples to be processed.

Necropsies were performed on seven spontaneous field cases of fawn enteritis, and all experimentally infected animals. The experimental fawns that survived were killed at the end of three weeks post-inoculation. Cultures were taken from the mesenteric lymph nodes and intestinal contents of all experimental fawns at necropsy and from field necropsies when possible. Tissue samples from all organs were fixed in 10% buffered formalin, embedded in paraffin, and sectioned at four microns for histological study. The routine stains used were hematoxylin and eosin. The periodic acid-Schiff stain was used in some cases to delineate basement membranes and capillary walls.

## Results

*Field Studies:* During the fawn season of 1969, thirty-three rectal cultures were obtained from wild fawns in an isolated study area in Grimes and Brazos Counties. Six positive cultures were obtained representing an incidence of 18.1%. Cultural studies in this area yielded 11.6% incidence in 1968, 21.4% incidence in 1967, and 43% incidence in 1966, a year of unusually high rainfall during June. *Salmonella* species isolated in 1969 from wild deer were *Salmonella derby, S. muenchen, S. anatum, S. newport, S. oranienburg, S. mississippi* and *S. meleagridis*. Deer mortality in the general area was 17.9% in 1967, 31.3% in 1968, and 17.10% in 1969; in the study area, losses were 31.6% in 1968 and 8.8% in 1969. Counts were not available in the study area during the 1966-67 season.

Fawns experimentally infected with Salmonella did not gain weight as rapidly as did the controls. Only two of eight clinically affected fawns made near-normal weight gains. Fawns which died in both clinically affected groups began to lose weight at birth and lost weight until death ensued (Table 1). Mortality was greater and weight gain less in colostrum deprived infected fawns than those which remained with the doe. Three clinically affected fawns died the second, third and fifth days post-inoculation.

Rectal temperature was not a good index of infection, as those fawns which were fatally infected had reduced rectal temperatures within 12 hours and the fawns that survived did not have elevated temperatures when compared to the uninfected controls. Normal temperature ranged from 101 to 102 degrees farenheit. Exercise or difficulty in restraint can result in one or two degrees rise in rectal temperature.

The clinical behavior of the infected fawns was different from the controls which remained alert and active during the trials. The clinically affected fawns spent considerable time lying down. Depending on the clinical course of infection, the signs abated or became more

severe. The fatal cases weakened rapidly, and the fawns became recumbent and comatose (Figure 2). The comatose state preceded death by 6 to 12 hours. Surviving fawns appeared more alert and the hair coat was again groomed although some individuals appeared to relapse after a few days and become depressed.

TABLE 1. *Weight gain (pounds) comparison of experimental fawns.*

| GROUP | AT BIRTH | AT 3 WKS | GAIN |
|---|---|---|---|
| 1. CONTROLS | 4.8 | 12.4 | 7.6 |
| | 5.6 | 12.4 | 6.8 |
| | 4.8 | 11.5 | 6.7 |
| | 4.8 | 12.8 | 8.0 |
| | 4.6 | 11.5 | 6.9 |
| 2. INOCULATED | 5.8 | 10.0 | 4.2 |
| | 4.4 | 11.0 | 6.6 |
| | 4.2 | 11.2 | 7.0 |
| | 4.6 | 3.9* | —.5 |
| 3. INOCULATED COLOSTRUM DEPRIVED | 5.2 | 4.7* | —.5 |
| | 5.1 | 4.5* | —.6 |
| | 5.0 | 8.9 | 3.9 |
| | 6.0 | 9.1 | 3.1 |

* Death ensued before 3 wks.

FIGURE 2. *Characteristic appearance of a fawn suffering from salmonellosis. Note the unkempt hair and listless carriage.*

An elevation in total leucocyte count was not seen in the fatal cases. The most reliable index of active infection by *Salmonella* was a rise in the monocytes in the differential count. Monocyte values for the control fawns seldom rose above 4%, while those of infected fawns rose in all examples to levels of 7-25% of the differential. In most of the animals, this monocytosis ocurred three to five days following infection, and was apparent in the fatal cases as well as in the survivors. One case which survived did not show an increase in monocytes until nineteen days following infection.

Packed cell volumes in the control animals were within 20-30% at birth and slowly rose to between 30-40% at three weeks of age. Packed cell volumes in clinically affected fawns with colstrum remained in the 20-30% range, and those of colostrum deprived (survivors) dropped to the 10-20% range during the three-week trial.

*Gross lesions:* Clinically affected animals were grouped into two classes: the acute fatal infection and the survivors. No difference in necropsy findings was seen between the two experimentally infected groups. In the acute fatal infections the carcasses were emaciated and dehydrated; the hair was dry and stuck together in peaks; and the skin had little resilience. Yellowish fecal material matted the perianal hair. The eyes were sunken and had a dull appearance. The small intestines were distended about four times their normal size with gas and contained a pale yellow liquid ingesta. The caecum was severely distended with gas. The mesenteric lymph nodes were enlarged and clear fluid exuded from their cut surface. The bladder was distended with clear urine in two of the three fatal cases. The other visceral and thoracic organs were normal.

The survivors were killed three weeks post-inoculation. The external appearance of this group was normal. The intestinal tract also appeared normal; however, the ingesta of the small intestine were pale yellow but had the sour odor of normal ingesta. No excess gas was noted. The size of the mesenteric lymph nodes in this group varied from normal to three times the normal size. The mesenteric lymphatic chain was whitish and granular in appearance and when cut, exuded a considerable quantity of clear fluid. Cultures of these nodes were positive for *Salmonella* in all animals except one. No other significant lesions were seen.

The necropsy observations of the field cases were not as uniform as those in the experimental animals due to varying periods of time between death and necropsy. None of the carcasses were found at the time of death, and autolytic changes were superimposed on the changes described for acute infections. Greenish staining of viscera by bile was pronounced. The livers were pale and soft, and the intestines were greatly distended. The mesenteric lymph nodes were enlarged.

*Histopathological Studies:* The mucosal epithelium of the villi of the small intestine in the acute fatal cases was eroded leaving a bare capillary bed exposed to the lumen. The lamina propria was infiltrated with reticuloendothelial cells, lymphocytes, and a few eosinophils. Neutrophils occurred in small foci but were not a major part of the cellular reaction. This reaction in the mucosal layer was most pronounced in the ileum. There was variation in the degree of involvement in the different segments of intestine. Depletion of the lymphoid follicles of the Peyer's patches was evident in all fatal cases.

The mesenteric lymph nodes were depleted of lymphocytes, and the sinusoidal spaces were filled with reticuloendothelial cells in all examined lymph nodes of the fatal cases, regardless of location. The germinal centers of the spleen were reduced in size, and there was proliferation of the reticuloendothelial cells in the red pulp.

The only other lesion in the fatal cases was a secondary bacterial pneumonia which was present in one deer. Focal bacterial colonies were accompanied by neutrophils and reticuloendothelial cells in the pulmonary parenchyma. No changes were seen in the central nervous system.

Histopathological changes in the survivors were seen in the same organs as in the organs of fatal cases. The changes

were, in general, less severe. The intestine was the major organ affected; the lamina propria was infiltrated with reticuloendothelial cells and large numbers of lymphoyctes. The germinal centers were pale and full of actively dividing lymphoblasts. Small foci of neutrophils were seen in the Peyer's patches, the mesenteric lymph nodes and the spleen. The size of splenic germinal centers varied from small to normal. Hematopoiesis was a common finding in the spleens of surviving fawns. Filling of the lymph node sinusoids by reticuloendothelial cells was evident but not as prominent as in the fatal cases. Pulmonary involvement was not seen in any of the surviving animals and no changes in the central nervous system could be demonstrated.

*Serological Studies:* This testing was done using a macroscopic tube test and a microdilution system. Agglutination was demonstrable and general increase in the rate of agglutination could be demonstrated as the clinical disease progressed; however, control animals' sera yielded similar results.

## Discussion

Neonatal enteritis causes a significant mortality in domestic ruminants and observations in these hosts would be comparable with the data in young deer. Infection by opportunistic bacteria of the newborn fawn would be highly variable, depending on the normal bacterial flora of the doe and the environment in which the fawn was born. Once contaminated, the ability of the fawn to respond to infection with adequate antibody production would determine if survival was possible. It seemed that the neonatal fawn was highly susceptible to infection and subsequent clinical illness with *Salmonellae* until colostral antibody was received and present in the circulation. This period of time during which antibody levels were low would be an optimum time for infection through fecal contamination by the doe or environmental contamination.

*Salmonella* infection in fawns appeared capable of producing significant mortality. As in any wild population, this problem was not the entire cause of mortality but was one of the rather constant factors which produced annual deer loss. Although there were several different species of *Salmonella* cultured in the field, it was felt that there was little difference in the clinical course of the disease, regardless of *Salmonella* species once infection had been achieved. During the four-year study, only one animal that had a positive rectal culture was ever seen again, and that animal was killed as a yearling deer. It thus appears that most culturally positive fawns probably do not survive, or at least have a lesser chance of surviving. An additional point considered in the field studies was that *Salmonella* carriers would probably not be readily identified by rectal swab techniques. It was felt that the incidence of this disease in fawns may have been considerably higher.

Experimental evidence of the pathogenicity of this group of organisms for fawns was demonstrated when experimental fawns were infected and all suffered clinical illness. Even the fawns which survived challenge had histological evidence of active disease and may have succumbed to predation or concomitant parasitism had they not been in a relatively stable environment. Mortality was higher in the experimental fawns which were deprived of colostrum. This treatment was performed to simulate interruption of the doe in the field, abandonment or capture and artificial rearing. The pathological findings on carcasses found in the field were not considered adequate for a definitive diagnosis. Confirmatory cultures of mesenteric lymph nodes was considered mandatory for a conclusive diagnosis, coupled with suggestive histopathological changes.

The major pathological changes seen in the small intestine were denudation of the mucosa leaving the capillaries exposed to the lumen. This resulted in a large surface area to which the bare capillary walls were exposed, promoting rapid dehydration and electrolyte imbalance which contributed to death. In those animals which survived, replacement of

the injured mucosa matched or surpassed the rate of cellular death, and the pronounced histiocytic response coincided with remission of clinical signs. The host reaction appeared to be unable to completely remove the infection since cultures of the mesenteric lymph nodes were positive in all survivors except one. The one surviving fawn for which no cultural confirmation was available had histopathological evidence suggestive of the disease.

Once injury of the intestinal mucosa permitted systemic invasion by the infective organism, the salmonellae appeared to have a great tendency to localize in the lymph nodes, particularly in the mesentery, which were constantly infected organs in all experimental infections. This constant finding was also present in the spontaneous cases encountered during the study and in previous experiences with fawn loss in captivity.

Serological studies were not productive with the methods used in this study. It was felt that this was due to cross reaction to other normal bacterial flora in the gastrointestinal tract, and that commercially available antigen was not adequate for definitive work. Since the serological reactions were similar in infected fawns and the controls which remained culturally negative throughout the study, it is suggested that antibody production against Somatic O Salmonella antigen is low in fawns for the first day after birth. After that time, antibody activity increases and remains at a relatively constant level. It was felt that inconclusive serologic findings were probably due to the quality of the commercial antigen, as other variables were carefully regulated.

Weight gains in the surviving colostrum deprived fawns were less than those of the other groups. Due to a limited number of newborn fawns from culturally negative does available, control colostrum deprived fawns were not studied. Reduced weight gains in this group may be due in part to colostrum deprivation, although confirmatory cultures were made on all fawns in the group.

The most probable route of infection taken by this organism is orally at, or shortly after, birth. The birth process is characterized by copious fluid escaping from the amniotic sac which drenches the hindquarters of the doe and the newborn fawn. This fluid creates a good transport medium for fecal or environmental contaminants. The fawn is soon licked dry; but in the period which follows during which the fawn stands and attempts to nurse, most of the ventrum of the doe is repeatedly nuzzled. Attempts at nursing are not confined to the mammae but are also applied to any warm moist area including the perineum. This provides ample opportunity for the fawn to acquire an abundant bacterial flora, both from the doe and the environment. As this problem appears to be largely in the moist river bottom environments of the State of Texas, one can assume that a relatively few carrier animals that shed the organism in their feces could maintain efficient environmental contamination.

## Management Implications

One cause of poor fawn survival has been described. The presence of this disease in fawns in the moister areas of the State indicates that the deer herds of these enzootic areas have less reproductive success than herds in the drier areas of the State. This disease, and in all probability others also, would result in slower recovery of deer numbers after heavy harvest than is seen in other areas. A larger base herd is therefore required to maintain deer numbers at a high level.

Reproductive success is the key to propagation of any species; be it for recreation, protein production, or survival of an endangered species. High reproductive rates in game species are required for optimal utilization of this natural resource for hunting. Conversely, species with low reproductive rates are, in general, those which become threatened with extinction from hunting pressure or habitat destruction.

As the demand for recreation expands in proportion to increases in human population, intensified management of

game species for sport will inevitably evolve. As intensified management schemes are developed, greater concern must be attached to nutritional and disease problems of game species. "Natural balance" will become, if it has not already done so, a reference point; for so widespread has the influence of man become that few environments remain free of alterations by the human species.

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